Evidence-based Physical Therapy for the Pelvic Floor

Bridging science and clinical practice
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It is both a pleasure and compliment to be asked to write a foreword for the second edition of this excellent book, *Evidence-based Physical Therapy for the Pelvic Floor: Bridging science and clinical practice*, edited by Kari Bø, Bary Berghmans, Siv Mørkved and Marijke Van Kampen.

The emphasis is on evidence-based practice with recommendations for all healthcare professionals who manage patients with pelvic floor dysfunction.

In particular, this latest edition builds on the success of the first with new chapters on evidence-based physical therapy for female and male LUTS, sexual dysfunction, anal incontinence and pelvic pain.

Pelvic floor muscle training is recommended as first-line therapy for pelvic floor disorders; and education and training in how best to provide this is essential. This book provides the evidence and recommendations.

The chapters are well written by international experts and readers will be impressed by the quality of the information provided.

Addressing the often challenging clinical problems related to specific patient groups such as children and the elderly is welcome and, in particular, the section on the management of pelvic floor dysfunction in elite athletes where such problems can affect performance.

Prevention is arguably more important than cure and understanding how pregnancy and childbirth affect the pelvic floor and how dysfunction can be prevented is well described.

This book will be a valuable reference not just for physical therapists but for students, nurse specialists, urologists, urogynaecologists, colorectal surgeons and all who manage patients with these distressing problems. I can thoroughly recommend it.

*Professor Robert Freeman, MD, FRCOG*
It is a great pleasure to write a foreword for the second edition of this important reference on evidence-based physical therapy for the pelvic floor. This book has been written by a number of the key experts working in this field and provides a comprehensive and structured overview of the subject. The basic principles are reviewed, in particular the important issue of evaluating the evidence by randomised trials and systematic reviews of these data, describing the functional anatomy of the female pelvic floor, the neuroanatomy and neurophysiology of the pelvic floor and how this interacts with the associated structures in the urinary and colorectal systems.

Accurate assessment of the pelvic floor muscle function is essential as is defining the anatomical defects, and this is covered in detail. The next important aspect, having defined structure and function, is to consider the disorders associated with dysfunction of the pelvic floor in both the male and the female, and how this relates to the underlying and associated symptom complex that we see affecting urinary, colorectal and sexual function.

Whilst pelvic floor dysfunction is of particular importance in the female, it can also be relevant in many male patients with associated pathology, either following trauma or after surgery. Pelvic floor dysfunction can also occur in other groups such as the paediatric population and with increasing age. It is a particular problem in the elderly as well as in the patient with neurological disorders, and also in patients such as the elite athlete where the pelvic floor is particularly stressed in these very fit people.

This excellent overview of the subject concludes with the importance of developing clinically meaningful practice guidelines.

I can thoroughly recommend this superb book which is particularly relevant, not only to those with an interest in sourcing information in this area, but as a reference guide for experts.

Professor Christopher Chapple, BSc, MD, FRCS (Urol), FEBU
Foreword

The new edition of *Evidence Based Physical Therapy for the Pelvic Floor: Bridging Science and Clinical Practice* by my physical therapy colleagues Kari Bø, Bary Berghmans, Marijke Van Kampen, and Siv Mørkved continues to provide physical therapists and other health professionals involved in the management of pelvic floor disorders with a wealth of knowledge and background information. The extensive worldwide use of the first 2007 edition gives strong credence for the need for the revised edition of this book. Pelvic floor disorders are a global health problem affecting women and men and have been estimated to have a prevalence of almost 50% in women around the world (Milsom et al., 2013). Urinary incontinence affects millions of men and women worldwide with substantial economic burdens to societies, thus all the more supporting the extensive need for this book’s timely information (Milsom et al., 2014).

When the first edition of this book was published, it was just four years after World Confederation for Physical Therapy (WCPT) had adopted its first declaration of principle on “Evidence-based Practice”. Since then, we have revised the position statement twice, but still we believe that physical therapists have a responsibility to use evidence to inform practice and ensure that the management of patients/clients, carers and communities is based on the best available evidence. Physical therapists have a responsibility not to use techniques and technologies that have been shown to be ineffective or unsafe. Thus, evidence should be integrated with clinical experience, taking into consideration beliefs, values, and the cultural context of the local environment, as well as patient/client preferences.

This edition has further refined and expanded the examination tests and measures used for assessing the pelvic floor and the evidence-based interventions for managing pelvic floor disorders in a wide variety of patients and clients. This book’s underlying philosophy is that we must always return to the evidence that makes us reflective practitioners regarding the selection of the appropriate tests and measures needed to be better able to devise and select intervention strategies concurrent with examination findings. It shows us the importance of maintaining currency with the evidence supporting practice and shows the importance of the randomized trials and systematic reviews in informing practice.

The content areas span the spectrum of information needed to bridge the science with clinical practice and spans the age spectrum from children to child-bearing years to aging adults. This edition includes the: functional anatomy; neuroanatomy and neurophysiology; measurement of pelvic floor muscle function, strength, and pelvic organ prolapse; the science of pelvic floor exercise prescription; separate chapters on pelvic floor dysfunction and evidence-based physical therapy for female pelvic floor dysfunction, male pelvic floor dysfunction, and pelvic floor dysfunctions affecting both women and men; and information needed for the physical therapy management of individuals with neurological disorders and elite athletes. Thus, the effectiveness of physical therapy is extensively covered throughout all clinical situations.

The authors have brought together all of the leading figures in pelvic floor dysfunction and management. The authors share their experiences from the perspectives of physical therapy, biomechanics, epidemiology, kinesiology, medicine, nursing, sports science, and surgery. The interdisciplinary model of practice is apparent for effective service delivery for these patients and clients.
There is absolutely no doubt that this latest edition of *Evidence Based Physical Therapy for the Pelvic Floor: Bridging Science and Clinical Practice* will continue to be the book of choice for students, clinicians, and faculty involved in these crucial aspects of the examination and intervention for evidence-based management of the pelvic floor. This book is indeed the “gold standard” for practice in this area.

Marilyn Moffat, PT, DPT, PhD, DSc (hon), GCS, CSCS, CEEAA, FAPTA
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**REFERENCES**


It is with great pleasure and excitement that we present this new edition of our textbook! We hope it will attract all physical therapists interested in the broad area of function and dysfunction of the pelvic floor. The editors of this book have more than 25 years experience in clinical practice and research in the prevention and treatment of symptoms of pelvic floor dysfunction. Between us our experience covers most areas of physical therapy for the pelvic floor, from children, women and men, to special groups such as pregnant and post-partum women, athletes, the elderly and patients with special health problems. In addition, we also have extensive background in other areas of physical therapy such as sports physiotherapy, neurology, rehabilitation, musculoskeletal, ergonomics, exercise science, health promotion, clinical epidemiology, biomechanics, motor control and learning and implementation of guidelines.

Prevention and treatment of pelvic floor dysfunction is truly a multidisciplinary field in which every profession should play its own evidence-based role for the highest benefit of the patients. With this in mind, we are very proud that so many leading international clinicians, researchers and opinion leaders from different professions have participated in the realisation of this book. Our sincere and warmest thanks to all of you for your unique contribution and the time and effort you have put in to making this book a truly evidence-based and up-to-date textbook.

We sincerely hope to have created a special and important book for the physical therapy profession for pelvic floor dysfunction. As we anticipated it appears to be useful for physical therapy schools and can be found in scientific libraries worldwide. Moreover, our hope that this book would become the base for postgraduate studies in pelvic floor physical therapy, became a reality. We hope that again the multidisciplinary nature of the authorship of this book will be reflected in the readership, serving nurses, gynaecologists, urologists, general practitioners and other health professionals working in conservative treatment and pelvic floor muscle training, as well as those in the physical therapy field.

As in the medical profession, clinical practice of physical therapy in pelvic floor has built up from a base of clinical experience, through small experimental studies to clinical trials. Today clinicians more and more can build on protocols from high-quality randomized clinical trials (RCTs) showing sufficient effect size (the difference between the change in the intervention group and the change in the control group). A quick search on PEDro (the Physiotherapy Evidence Database, Sydney, Australia, www.pedro.org.au) shows that physical therapy is changing rapidly from being a non-scientific field to a profession with a strong scientific platform. In November 2013 there were more than 21,000 RCTs, 4,369 systematic reviews and 473 evidence-based clinical practice guidelines in different areas of physical therapy listed in the database. While this book recognises that much more research is needed into the prevention and treatment of many conditions in the pelvic floor area, there are already more than 65 RCTs evaluating the effect of pelvic floor muscle training for stress and mixed incontinence. Hence, in good clinical practice the physical therapist should adapt individual patient training programmes according to the protocols from these studies rather than using theories or models which are not backed by clinical data. In addition, good clinical practice should always be individualized and should be based on a combination of clinical experience, knowledge from high-quality RCTs and patient preferences. Next to this, good clinical practice should always be based on respect, empathy and strong ethical grounding.
In 2001, Lewis Wall, Professor of Urogynecology, wrote an editorial in the International Urogynecology Journal describing 7 stages in the life of medical innovations:

1. Promising report, clinical observation, case report, short clinical series
2. Professional and organizational adoption of the innovation
3. The public accepts the innovation – state or third party pays for it
4. Standard procedure – into textbooks (still no critical evaluation)
5. RCT
6. Professional denunciation
7. Erosion of professional support, discredit

He stated that by the time stage 7 is concluded, or even before the RCT has started, the procedure may already have given way to a new procedure or method which has grown in its wake. This cycle continues with these new methods and procedures being prescribed to patients without patients being informed about the effect, risk factors or complications. It is also noteworthy that, in most cases, patients are unaware of the fact that there is no scientific base for the proposed treatment. While Wall’s description of the lifecycle applies specifically to medical innovations, we are subject to the same scrutiny and criticism in physical therapy. (Wall, 2001)*

Although physical therapy modalities, in comparison with surgery, rarely produce serious side effects or complications, we suggest that Wall’s 7 stages may also be very useful to show how different theories, and not science, impact on physical therapy practice. We are keenly aware and concerned that in the long run such unscientific evolution of practice will damage patients, the physical therapy profession itself and parties responsible for compensation. In particular, the use of such untested models and theories as a background for implementing new interventions when there evidence is in fact available for alternative and proven treatment strategies, must be considered bad clinical practice, and may even be considered unethical. Hence, it is our hope that this book will be a big step towards evidence-based practice in all symptom areas of pelvic floor dysfunction.

This does not mean that we should not treat conditions for which there are no or only few/weak controlled studies to support clinical practice. However, we sincerely believe that all physical therapists should be aware of the different level and value of statements, theories, clinical experience, knowledge from research designs other than RCTs and knowledge from high-quality research. It is a duty to openly explain to patients and other parties that the proposed treatment is not based on high-quality studies, but only on the best available knowledge at that time. The profession should never confuse statements, clinical experience and theories, with evidence from high-quality RCTs, and optimally, we should not use new modalities in regular clinical practice until they have proved to be effective in RCTs. In this book we have tried our best to differentiate between the different levels of knowledge and evidence and to be very clear about the limitations of the research underlying the recommendations for practice. In line with this, we have left out those areas that were not convincing because of lack of evidence. These areas include:

- The role or effect of PFMT on core stability to prevent/treat low back and pelvic girdle pain
- The effect of “functional training”
- The role of motor control training as the sole treatment of pelvic floor dysfunction
- The definition, assessment and treatment for “hypertone pelvic floor”
- The effect of body posture on the pelvic floor

These areas have recently been covered by Bø and Herbert (2013) in a systematic review. The conclusion was there is not yet evidence to support alternative exercises in pelvic floor muscle training for female stress urinary incontinence. Our aim is continue updating the evidence in all areas of research in pelvic floor physical therapy. Therefore, in this edition we have included more areas because of the continuing growth of knowledge based on high-quality research, and we hope that the next edition will contain even more.

The evidence presented in this book is based on reviews from the Cochrane Library, the five International Consensus Meetings on Incontinence, other systematic reviews and updated searches on newer RCTs. However, the conclusions of these high-quality systematic reviews can differ because they are a product of how the authors have posed their research questions, what type of studies they have included, what choice of outcome measures they have made, and how they have classified the studies. Therefore, not all conclusions in this book are in line with other conclusions. The goal of the editors of this book is to evaluate only clinically relevant research
questions. Moreover, our selection procedure and strategy for the in- and exclusion of studies should be transparent and easy to understand for the readers of the book.

Active exercise is the core of physical therapy interventions. Passive treatments may be used to stimulate non-functioning muscles, to inhibit an overactive detrusor muscle and to manage pain so that active exercise becomes possible. The following is a quote from Hippocrates (c. 450 BC) which elegantly lends itself to the philosophy of physical therapy:

“All parts of the body which have a function, if used in moderation and exercised in labours in which each is accustomed, become thereby healthy, well-developed and age more slowly, but if unused and left idle they become liable to disease, defective in growth, and age quickly”.

It is the role of the physical therapist to motivate patients and to facilitate exercise and adapted physical activity throughout the lifespan.

We hope that new students in this exciting and interesting field will find enough guidance in this book to begin to prevent, assess and treat pelvic floor dysfunction effectively in their clients/patients, but they must also learn to be critical of new theories and modalities that have not yet been tested sufficiently. For experienced physical therapists we hope that providing contemporary scientific evidence to support or contradict clinical practice will affect changes in practice and will push for more high-quality clinical research projects. Hopefully, you will enjoy reading the book just as much as we have enjoyed working with it. Through working on the book we have certainly become aware of many unanswered questions, and have identified many new research areas that need to be addressed in this challenging area. We encourage the readers interested in research to continue with formal education in research methodology (MSc and PhD programmes) and join us in trying to make high-quality clinical research in the future. We appreciate any constructive feedback for chapters to be changed or included for the next edition.

Professor Kari Bø
Dr Bary Berghmans
Dr Siv Mørkved
Professor Marijke Van Kampen

Overview of physical therapy for pelvic floor dysfunction

Kari Bø

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PELVIC FLOOR DYSFUNCTION

The framework of this book is based on the approach to disorders of the pelvic floor in women described by Wall and DeLancey (1991). Wall and DeLancey (1991) stated that ‘pelvic floor dysfunction, particularly as manifested by genital prolapse and urinary or faecal incontinence, remains one of the largest unaddressed issues in women’s health care today’ (p. 486). In their opinion, lack of success in treating patients with pelvic floor dysfunction is due to a professional ‘compartmentalization’ of the pelvic floor.

Each of the three outlets in the pelvis has had its own medical specialty, with the urethra and bladder belonging to the urologist, the vagina and female genital organs belonging to the gynaecologist, and the colon and rectum belonging to the gastroenterologist and the colorectal surgeon (Fig. 1.1).

Wall and DeLancey (1991) argue that instead of concentrating on the three ‘holes’ in the pelvis, one should look at the ‘whole pelvis’, with the pelvic floor muscles (PFM), ligaments and fasciae as the common supportive system for all the pelvic viscera.

The interaction between the PFM and the supportive ligaments was later elaborated by DeLancey (1993) and Norton (1993) as the ‘boat in dry dock theory’. The ship is analogous to the pelvic organs, the ropes to the ligaments and fasciae and the water to the supportive layer of the PFM (Fig. 1.2).

DeLancey (1993) argues that as long as the PFM or levator ani muscles function normally, the pelvic floor is supportive and the ligaments and fascia are under normal tension. When the PFM relax or are damaged, the pelvic organs must be held in place by the ligaments and fasciae alone. If the PFM cannot actively support the organs, over time the connective tissue will become stretched and damaged.

Bump and Norton (1998) also used this theoretical framework in their overview of the epidemiology and natural history of pelvic floor dysfunction. They suggested that pelvic floor dysfunction may lead to the following conditions:

- urinary incontinence (stress, urge and mixed incontinence);
- faecal incontinence;
- pelvic organ prolapse;
- sensory and emptying abnormalities of the lower urinary tract;
- defecatory dysfunction;
- sexual dysfunction;
- chronic pain syndromes.

Bump and Norton (1998) also described three stages in the development of pelvic floor dysfunction:

1. A perfect pelvic floor that is anatomically, neurologically and functionally normal.
2. A less than perfect, but well-compensated pelvic floor in an asymptomatic patient.
3. A functionally decompensated pelvic floor in the patient with end-stage disease, with urinary incontinence, anal incontinence, or pelvic organ prolapse.

A model describing aetiological factors possibly leading to or causing pelvic floor dysfunction in women has been developed, classifying the factors into:

- predisposing factors (e.g. gender, genetic, neurological, anatomical, collagen, muscular, cultural and environmental);
inciting factors (e.g. childbirth, nerve damage, muscle damage, radiation, tissue disruption, radical surgery);
• promoting factors (e.g. constipation, occupation, recreation, obesity, surgery, lung disease, smoking, menstrual cycle, infection, medication, menopause);
• decompensating factors (e.g. ageing, dementia, debility, disease, environment, medications).

In 2008 DeLancey et al further developed this model to what they named the Lifespan Model. They described a graphical tool to integrate pelvic floor function related to pelvic floor disorders in three major phases: (1) development of functional reserve during an individual’s growth; (2) variations in the amount of injury and potential recovery that occur during and after vaginal birth; and (3) deterioration occurring with advancing age. The authors suggest that the Lifespan Model should be used to focus on more refined preventive strategies of pelvic floor dysfunction risk in an individual woman as opposed to more general recommendations for all women (DeLancey et al., 2008).

Wall and DeLancey (1991) argued that progress in the treatment of pelvic floor dysfunction in women would occur more rapidly if a unified, cross-disciplinary approach to disorders of the pelvic support was developed. Wall and DeLancey (1991) mentioned only the different medical professions as part of a multidisciplinary team. In this book we will argue that physical therapists (PTs), having assessment and treatment of the musculoskeletal system in general as their specialty, should be core professionals in a multidisciplinary approach to pelvic floor dysfunction.

PHYSICAL THERAPY FOR THE PELVIC FLOOR

The nature of physical therapy

In May 1999, at the 14th General Meeting of the World Confederation for Physical Therapy (WCPT), a position statement describing the nature and process of physical therapy/physiotherapy was approved by all member nations (WCPT, 1999). This description will be used as a foundation and framework to give an overview of physical therapy/physiotherapy in the area of pelvic floor dysfunction. The term ‘physical therapy’ will be used throughout this book, in accordance with the guidelines of the WCPT Europe.

According to the WCPT, physical therapy is ‘providing services to people and populations to develop, maintain and restore maximum movements and functional ability throughout the lifespan’. The main area of practice for PTs is musculoskeletal pain and dysfunction. However, many PTs also specialize in other areas, such as the cardiorespiratory field, neurology and coronary disease. In all areas, PTs aim to improve functional capacity and improve patients’ ability to maintain or increase physical activity level.

![Figure 1.1 Gynaecologists, urologists and colorectal surgeons concentrate on their areas of interest and tend to ignore the pelvic floor common to them all.](image1)

![Figure 1.2 The ‘boat in dry dock’ analogy. Reproduced with permission from Norton, 1993.](image2)
The PFM are not responsible for gross motor movements alone, but work in synergy with other trunk muscles. Therefore, pelvic floor dysfunction may lead to symptoms during movement and perceived restriction in the ability to stay physically active (Bø et al., 1989; Nygaard et al., 1990). Several studies have shown that, for example, urinary incontinence may lead to a change in movement patterns during physical activities (Bø et al., 1989; Nygaard et al., 1990), withdrawal from regular fitness activities and troublesome difficulties when being active (Brown and Miller, 2001; Nygaard et al., 1990).

Lifelong participation in regular moderate physical activity is important in the prevention of several diseases, and is an independent factor in the prevention of osteoporosis, obesity, diabetes mellitus, high blood pressure, coronary heart disease, breast and colon cancer, depression and anxiety (Bouchard et al., 1993).

In addition, limitations in the ability to move or conduct activities of daily living either due to age or injuries, may also lead to other problems, such as secondary incontinence. Physical therapy for pelvic floor dysfunction may therefore also include physical activities for increasing general function and fitness level.

Physical therapy includes the provision of services in circumstances where movement and function are threatened by the process of ageing or that of injury or disease.

Hippocrates (5th to 4th centuries BC) claimed that ‘all parts of the body which have a function, if used in moderation and exercised in labours in which each is accustomed, become thereby healthy, well-developed and age more slowly, but if unused and left idle they become liable to disease, defective in growth, and age quickly’.

The PFM are subject to continuous strain throughout the lifespan. In particular, the pelvic floor of women is subject to tremendous strain during pregnancy and childbirth (Morkved, 2003; DeLancey et al., 2008). In addition, hormonal changes may influence the pelvic floor and pelvic organs and a decline in muscle strength may occur due to ageing. Hence, the PFM may need regular training to stay healthy throughout life.

Physical therapy is concerned with identifying and maximizing movement potential, within the spheres of health promotion, prevention, treatment, and rehabilitation.

Physical therapists may promote PFM training (PFMT) by writing about the issue in newspapers and women's magazines, informing all their regular patients about PFMT, including PFMT in regular exercise classes and in particular in antenatal and postnatal training, as well as before and after pelvic surgery in men and women. Physical therapists who treat pelvic floor dysfunction should be fully trained in this specialty or should refer to colleagues who have the thorough knowledge to treat patients according to the principles of evidence-based physical therapy.

Physical therapy is an essential part of the health services delivery system

P'Ts practice independently of other health care providers and also within multidisciplinary rehabilitation/habilitation programmes for the restoration of optimal function and quality of life in individuals with loss and disorders of movement.

In most countries physical therapy work is by referral from medical practitioners. However, during recent decades this has changed in some countries such as Australia and New Zealand. In 2006 Dutch PTs have also become primary contact practitioners. Both systems require good collaboration between the medical and physical therapy professions.

The referral system implies that the medical practitioner is aware of what the PT can offer, and also has PTs available to send referrals to. One of the weaknesses of this system is that medical practitioners who are not motivated or who have insufficient knowledge about the evidence for different physical therapy interventions will not send suitable patients to physical therapy. The patients will more likely be offered traditional medical treatment options such as medication or surgery. These treatments may have adverse effects and are more expensive than exercise therapy (Black and Downs, 1996; Smith et al., 2002). In addition, the referral system is expensive because it involves an extra consultation.

The argument against PTs as primary contact practitioners has been that PTs do not have enough education to make differential diagnoses, and may therefore not detect more serious diseases such as cancer or neurological disease underlying the symptoms.

The editors of this book do not take a stand for either system of physical therapy service. We believe that prevention and treatment of pelvic floor dysfunction needs a multidisciplinary approach and would encourage collaboration between physicians and PTs at all levels of assessment and treatment:

Physical therapy involves … using knowledge and skills unique to physical therapists and, is the service ONLY [author's emphasis] provided by, or under the direction and supervision of a physical therapist.
The educational standard of PTs differs between countries throughout the world. In the United States, physical therapy is at master’s degree level (although this is based on an undergraduate degree other than physical therapy), whereas in most countries in Europe, Asia and Africa it is a 3-year bachelor degree and in Australia and New Zealand it is a 4-year bachelor degree. In most countries PTs can now continue with a master’s degree and PhD.

Physical therapy schools are within the university in many countries, but in other countries physical therapy is taught in polytechnic schools or colleges below university level.

There can be different educational requirements for entry into undergraduate programmes within one country and from country to country. In most countries, however, physical therapy is a professional education and the entry level for physical therapy undergraduate studies is very high, in some countries being at the same level as medicine. In the area of pelvic floor dysfunction, traditionally the level of scientific background has been very high with several professors of physical therapy and many practitioners and researchers with master’s and PhDs.

The emphasis on pelvic floor dysfunction in undergraduate physical therapy curricula varies between countries at both undergraduate and postgraduate physical therapy level. The broad knowledge of anatomy and physiology, medical science, clinical assessment and treatment modalities learnt by all PTs can be applied to the pelvic floor. Several countries also have postgraduate education programmes for PTs specializing either in women’s health or pelvic or pelvic floor physical therapy with education level and content varying between countries.

The physical therapy process includes assessment, diagnosis, planning, intervention, and evaluation.

Assessment

Assessment includes both the examination of individuals or groups with actual or potential impairments, functional limitations, disabilities, or other conditions of health by history taking, screening and the use of specific tests and measures, and evaluation of the results of examination through analysis and synthesis within a process of clinical reasoning.

In patients with pelvic floor dysfunction, after thorough history taking, the PT will assess the function of the pelvic floor by visual observation, vaginal palpation and/or measurement of muscle activity (measurement of vaginal or urethral squeeze pressure, electromyography [EMG] and ultrasound) (Bo and Sherburn, 2005).

Diagnosis

In carrying out the diagnostic process, physical therapists may need to obtain additional information from other professionals.

Most PTs in private practice obtain referrals of patients from general practitioners. These medical practitioners themselves seldom have access to urodynamics, EMG, magnetic resonance imaging (MRI) or ultrasound.

According to the Report from the Standardization Subcommittee of the International Continence Society (Abrams et al., 2002), a diagnosis of stress or urge incontinence or pelvic pain syndrome cannot be based on history taking alone. Therefore, interdisciplinary collaborations with other professionals are highly recommended. In real life most PTs in private practice treat patients who have not undergone a thorough diagnostic investigation.

DeLancey (1996) has suggested that the cure and improvement rates of PFMT would be higher for stress urinary incontinence (SUI) if more detailed knowledge about the pathophysiology of each patient was available.

Planning

A plan of intervention includes measurable outcome goals negotiated in collaboration with the patient/client, family or care giver. Alternatively it may lead to referral to another agency in cases which are inappropriate for physical therapy.

It is extremely important that the patient decides the final goal of the treatment. For example, not all women need to be totally dry during jumping because they may never perform this activity.

One goal for an elderly woman might be to be able to lift her grandchild without leaking or feeling heaviness or bugling from a pelvic organ prolapse. If she is able to contract the PFM with a certain degree of strength this may be quite easy to accomplish with proper instruction of precontraction of the PFM before and during lifting.

Another woman may have the goal of being totally dry or having good organ support while playing tennis (Bo, 2004a). To achieve this she may need much more intensive PFMT training because she needs to build up muscle volume and stiffness of the pelvic floor and gain an automatic PFMT action during an increase in abdominal pressure or a high ground reaction force (Bo, 2004b).
Overview of physical therapy for pelvic floor dysfunction

Because most PTs treat patients with pelvic floor dysfunction without a full diagnosis it is of utmost importance that they communicate with other medical professions if they discover discrepancies between expected outcomes, or suspect other underlying conditions to be the cause of the patient’s complaints. For example, urgency and urgency incontinence may be the first signs of multiple sclerosis.

**Intervention**

*In general physical therapy intervention is implemented and modified in order to reach agreed goals and may include: manual handling; movement enhancing; physical, electro-therapeutic and mechanical agents; functional training (muscle strength and endurance, coordination, motor control, body-awareness, flexibility, relaxation, cardiorespiratory fitness); provision of aids and appliances; patient/client related instruction and counseling; documentation and coordination, and communication.*

WCPT

In treating pelvic floor dysfunction the mainstay of physical therapy is education about the dysfunction, information regarding lifestyle interventions, manual techniques and PFMT.

PFMT can be taught with or without the use of biofeedback or other adjunctive therapies, such as electrical stimulation or mechanical agents. It includes teaching of the correct contraction, muscle and body awareness, coordination and motor control, muscle strength and endurance, and relaxation.

The PT will choose different treatment programmes for different conditions and different patients. In some cases the PT will also provide preventive devices to the patients, and teach them how to use them. Interventions may also be aimed at preventing impairments, functional limitations, disability and injury, and include the promotion and maintenance of health, quality of life, and fitness in all ages and populations. To prevent urinary incontinence, teaching pelvic floor exercises in pregnancy and after childbirth is essential.

The choice of interventions should always be based on the highest level of evidence available. Ideally, the PT will choose the protocol from a randomized controlled trial (RCT) where the intervention has been shown to be effective and adjust this to the patient’s needs and practical requirements (Bo and Herbert, 2009).

In the area of SUI there is sufficient knowledge from RCTs to choose an effective training protocol. However, in other conditions that may be caused by pelvic floor dysfunction such knowledge is not yet available. The PT then has to develop a programme on the basis of clinical experience (his or her own, or that of other experts), small experimental studies or theories. It is essential that such experience or theories are quickly developed into research hypotheses and tested in RCTs by trained researchers to see if there is a clinically worthwhile effect (Bo and Herbert, 2009).

Collaboration between experienced clinicians and researchers is extremely important in planning clinical research. Experienced clinicians should not jump at new theories and ideas or change their practice based on theories and small experimental studies alone. Ideally, the only information that should lead to a drastic change of clinical practice is results (positive or negative) from RCTs (Bo and Herbert, 2009).

When undertaking research and deciding on a physical therapy intervention, the PT must be aware that the ‘quality of the intervention’, particularly the intensity of the physical therapy intervention, will affect the outcome. Ineffective (low-dose) or even harmful treatments can be used in a RCT that has high-quality methodology. These research challenges are the same when conducting RCTs that include both surgery and PFMT, and the methodological quality of studies of both surgery and PFMT has been variable (Hay-Smith et al., 2011; Dmochowski et al., 2013; Moore et al., 2013).

When participating in research led by other professionals it is important that the physical therapy intervention meets quality standards. No drug company would dream of conducting a study with a non-optimal dosage of the drug. In published RCTs, there are several PFMT programmes with low dosage showing little or no effect (Hay-Smith et al., 2011).

**Evaluation**

*Evaluation necessitates re-examination for the purpose of evaluating outcomes.*

WCPT

Using the same outcome measures before and after treatment is mandatory for the purpose of evaluating outcomes in clinical practice.

In treating symptoms of pelvic floor dysfunction the PT uses different forms of PFMT (independent variable in experimental research) to change the condition (named dependent variable in experimental research, e.g. stage of pelvic organ prolapse, pelvic pain or SUI).

It is mandatory that PTs use the concept of the International Classification of Impairment, Disability and Handicap (ICIDH) (1997), later changed to International Classification of Function (ICF) (2002), to evaluate efficacy of the intervention. The ICF is a World Health Organization (WHO) approved system designed to classify health and health-related states. According to this system (see Chapter 5.1), different health components are related to specific diseases and conditions:

- body functions: physiological and psychological functions of body systems (e.g. delayed motor latency of the nerves to the PFM);
• body structures: anatomical parts (e.g. rupture or atrophy of the PFM);
• impairments: problems in body function or structure such as significant deviation or loss (e.g. weak or non-coordinated PFM);
• activity: execution of a task or action by an individual (e.g. to stay continent during increase in abdominal pressure);
• participation: involvement in a life situation (being able to participate in social situations such as playing tennis or aerobic dancing without fear or embarrassment of leaking);
• environment (e.g. easy access to the bathroom).

Physical therapy aims to improve factors involving all these components. Therefore we need to select different outcome measures for different components. For example, PFMT may improve timing of the co-contraction during cough (ICF: body functions; neurophysiology). This may be measured using MRI or ultrasound.

One of the aims for PFMT in treating pelvic organ prolapse (POP) is to alter the length/stiffness of the PFM so they sit at a higher anatomical location inside the pelvis (ICF: body structure, anatomy). This may be measured using MRI or ultrasound.

Impairment of the PFM can result from inability to produce optimal strength (force). Muscle strength can be measured by manometers or dynamometers during attempts of maximal contraction.

Ambulatory urodynamics of urethral pressure during physical activities may be developed as a future measure of automatic co-contraction during activity.

Urinary leakage could be classified as disability in the ICIDH and as activity in the new ICF system. The actual leakage can be measured by number of leakage episodes (self report) or pad tests.

Physical therapy also aims at, for example, reducing urinary leakage to a point where this is no longer restricting the patient from participation in social activities (ICF: participation). This can be measured by quality of life questionnaires. PTs can also work politically to improve the environment, such as advocating for easy access to toilets in public buildings.

Ideally, PTs should assess the effect of the physical therapy intervention in all these components using outcome measures with high responsiveness (measurement tools that can detect small differences), reliability (intra- and inter-tester reproducibility), and validity (to what degree the measurement tool measures what it is meant to measure). The WCPT states that PTs should ‘use terminology that is widely understood and adequately defined’ and ‘recognize internationally accepted models and definitions’.

In the area of pelvic floor dysfunction we are fortunate to have international committees working on standardization and terminology. The International Continence Society (ICS) constantly revises its standardization of terminology (Abrams et al., 2002), and the Clinical Assessment Group within the same society has also delivered a standardization document (www.icsoffice.org). Recently, joint working group documents from the International Urogynecological Association (IUGA) and the ICS have been published, and work by several joint terminology groups is currently underway (Haylen et al., 2010).

Physical therapists must refer to definitions and terminology from the WHO, the WCPT, and for definitions and standards developed in exercise science and motor learning and control to be able to communicate effectively with other professions.

## Linking research and practice

*Emphasise the need for practice to be evidence-based whenever possible … [and] appreciate the interdependence of practice, research and education within the profession.*

WCPT

Sackett et al. (2000) has defined evidence-based medicine as ‘the conscientious, explicit and judicious use of current best evidence in making decisions about care of individual patients’. Neither the best available external clinical evidence (RCTs) nor clinical expertise alone is good enough for decision making in clinical practice. Without clinical experience, ‘evidence’ can ignore the individual’s needs and circumstances, and without evidence, ‘experience’ can become old-fashioned/out of date.

Evidence-based physical therapy practice has a theoretical body of knowledge, uses the best available scientific evidence in clinical decision making and uses standardized outcome measures to evaluate the care provided (Herbert et al., 2005).

Herbert et al. (2005) have stated that research conducted as part of routine clinical practice can be prone to bias because there is often a lack of comparison of outcomes with outcomes of randomized controls. In such studies it may be difficult to distinguish between effects of intervention and natural recovery or statistical regression. In addition, self-reported outcomes may be biased because patients may feel obliged to the therapist. There may be no record or follow-up of drop-outs, outcome measures may be distorted by assessors’ expectations of intervention, adherence to the training protocol is seldom reported and long-term results are often not available. The best evidence of effects of intervention comes from randomized trials with adequate follow-up and blinding of assessors and, where possible, blinding of patients too.

Our understanding of the mechanisms of therapies is often incomplete, and it is unknown whether the effects of some physical therapy interventions are large enough to be worthwhile (effect size).

Only high-quality clinical research (RCTs) potentially provides unbiased estimates of the effect size (Herbert, 2000a,b). This provides several challenges in clinical practice.
To increase their level of knowledge in clinical practice, PTs need to:

- stay updated in pathophysiology;
- use interventions for which we have evidence-based knowledge of dose–response issues;
- if possible: use interventions/protocols based on results/protocols from high-quality RCTs with positive results (clinically relevant effect-size);
- use pre- and post-treatment tests that are responsive, reliable, and valid;
- measure adherence and adverse effects!

**ROLE OF THE PHYSICAL THERAPIST IN PELVIC FLOOR DYSFUNCTION**

- Work in a team with other professionals in medicine (e.g. general practitioner, urologist, gynaecologist, radiologist).
- Evaluate the degree of pelvic floor dysfunction symptoms and complaints and overall condition by covering all components of the ICF.
- Fully evaluate PFM performance, including ability to contract, resting condition and strength.
- Set individual treatment goals and plan treatment programmes in collaboration with the patient.
- Treat the condition individually and/or conduct PFM exercise classes.
- Teach preventive PFM exercise individually or in classes during pregnancy and postnatally.
- Clinicians without a research background can participate in high-standard research as deliverers of high-quality physical therapy and conduct evaluation of the intervention. They should, however, refuse to be involved in studies with low-quality methodology and/or low-quality intervention (e.g. inadequate dosage).
- Research PTs should:
  - conduct basic research on tissue adaptation to different treatment modalities;
  - participate in the development of responsive, reliable and valid tools to assess PFM function and strength and outcome measures;
  - conduct high-quality methodological and interventional RCTs to evaluate effect of different physical therapy interventions.

**REFERENCES**


Evidence-Based Physical Therapy for the Pelvic Floor


Critical appraisal of randomized trials and systematic reviews of the effects of physical therapy interventions for the pelvic floor

Rob Herbert

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In the preceding chapter, Kari Bø described her vision of physical therapy for the pelvic floor. A core part of that vision is that practice should be guided by evidence in the form of high-quality clinical research. This chapter develops that theme by considering one specific sort of evidence: evidence about the effects of interventions. The chapter begins by identifying the sorts of evidence that tell us about the effects of intervention. It then explores how readers of the research literature can differentiate between high- and low-quality evidence. The chapter concludes by briefly considering how high-quality evidence of the effects of intervention can be used to assist clinical decisions.

RANDOMIZED TRIALS AND SYSTEMATIC REVIEWS

Randomized trials

Randomized trials (also called randomized controlled trials or randomized clinical trials [RCTs]) are used to estimate the effects of interventions. They involve sampling people (trial ‘subjects’ or ‘participants’) from clinical populations who either have a health disorder (in studies of treatment) or are at risk of a health disorder (in studies of prevention). The key feature of randomized trials is that each participant in the trial is randomly allocated to receive the intervention of interest or not. The group of participants that does not receive the intervention of interest is often called the ‘control group’. The effect of the intervention is determined by comparing the outcomes of participants in the intervention and control groups.

There are a number of variations of this broad approach (Herbert et al., 2005). In the simplest version, each participant is randomized either to a group that receives the intervention or a group that does not receive the intervention. Alternatively, participants in both groups could receive standard care but participants in one group could receive, in addition, the intervention of interest. Or one group could receive an intervention and the other group could receive a different intervention. All of these variations can be called randomized trials.

Two features differentiate randomized trials from other types of studies of the effects of intervention: in randomized trials there is comparison between outcomes of groups that do and do not receive a particular intervention, and participants are allocated to conditions using a random procedure. These features make it possible to separate out the effects of intervention from other factors that influence clinical outcomes, such as the natural history of a condition, or statistical phenomena such as statistical regression. The logic is as follows: randomization generates groups that are likely to have similar characteristics, especially in large trials. So when we give the intervention of interest to one group and not the other, differences in the outcomes of the two groups cannot be attributable to differences in the groups’ characteristics but must instead be attributable to the intervention. A complication is that, because
randomization produces groups with similar but not identical characteristics, differences in outcomes could be due to small differences in the groups’ characteristics at baseline. Statistical methods can be used to assess whether this is plausible or not. So it is possible to use the difference between the outcomes of the two groups in a randomized trial to provide an estimate of the effect of intervention.

Importantly, randomization is the only completely satisfactory way to generate two groups that we can know are comparable (have similar characteristics). No other method can assure a ‘fair comparison’ between intervention and control groups. (Some empirical evidence suggests well-conducted non-randomized trials often produce similar results to randomized trials [Benson and Hartz, 2000; Concato et al., 2000; but see Kunz and Oxman, 1998], but there is no reason why we should expect that to be so.) For this reason randomized trials can claim to be the only method that can be expected to generate unbiased estimates of the effects of interventions.

**Systematic reviews**

Many physical therapy practices, including several interventions for the pelvic floor, have been subjected to multiple randomized trials. Where more than one trial has examined the effects of the same intervention we can potentially learn more from a careful examination of the totality of evidence provided by all relevant randomized trials than from any individual trial. Potentially we can get more information about the effects of an intervention from literature reviews rather than from individual studies.

Until a couple of decades ago, reviews of the literature were conducted in an unsystematic way. Authors of reviews would find what they considered to be relevant trials, read them carefully, and write about the findings of those trials. The authors of the best reviews were able to differentiate between high- and low-quality trials to bring together a balanced synthesis that fairly reflected what existing trials said about the effects of the intervention.

Nonetheless, traditional (narrative) reviews have always had one important shortcoming: their methods are inscrutable. It is hard for readers of narrative reviews to know if the review was carried out optimally. Readers cannot determine, without specific knowledge of the literature under review, whether the reviewer identified all of the relevant trials or properly weighted the findings of high-quality and low-quality studies. Also, readers usually cannot know how the reviewer went about drawing together the findings of the relevant trials to synthesize the review’s conclusions. There must always be some concern that the evidence provided in narrative reviews is biased by selective reporting of studies, unbalanced assessment of trial quality, or partial interpretations of what the best trials mean.

The method of systematic reviews was developed in the late 1970s to overcome some of the shortcomings of narrative reviews (Hunt, 1997). The most important characteristic of systematic reviews is that they explicitly describe the methods used to conduct the review; typically systematic reviews have a Methods section that describes how the search was conducted, how trials were selected, how data were extracted and how the data were used to synthesize the findings of the review. Thus, in systematic reviews, the methods are transparent. This means the reader can make judgements about how well the review was conducted. Most systematic reviews attempt to minimize bias by attempting to find all relevant trials, or at least a representative subset of the relevant trials. Also, predetermined criteria are used to assess the quality of trials, and to draw together the findings of individual trials to generate an overall conclusion. (See Box 2.1.)

**What can’t randomized trials and systematic reviews tell us?**

Theoretically, randomized trials could provide us with estimates of the effects of every physical therapy intervention and every component of every physical therapy intervention. In practice, we are a long way from that position, and it is likely we will never get there.

Randomized trials are cumbersome instruments. They are able to provide unbiased estimates of the effects of interventions, but they do so at a cost. Many trials enroll hundreds or even thousands of participants and follow them for months or years. The magnitude of this undertaking means that it is not possible to conduct trials to examine the effects of every permutation of every component of every intervention for every patient group.

In practice the best that randomized trials can provide us with is indicative estimates of effects of typical interventions administered in a small subset of reasonable ways to typical populations, even though we know that when the intervention is applied in clinical settings its effects will vary depending on precisely how the intervention is administered and precisely who the intervention is administered to.

Randomized trials can suggest treatment approaches, but the fine detail of how interventions are implemented will always have to be informed by clinical experience, by our understandings of how the intervention works, and by common sense.

Randomized trials and systematic reviews of randomized trials are suited to answering questions about the effects of interventions, but are not able to answer other

**Box 2.1**

The best information about the effects of physical therapy interventions for the pelvic floor is provided by randomized trials or, where there has been more than one randomized trial, by systematic reviews of randomized trials.
sorts of questions. For example, different sorts of designs are required to answer questions about the prognosis of a particular condition or about the interpretation of a diagnostic test (Herbert et al., 2005).

A major limitation of randomized trials is that the methods developed for analyzing randomized trials can only be applied to quantitative measures of outcomes. But it is not possible to quantify the full complexity of people’s thoughts and feelings with quantitative measures (Herbert and Higgs, 2004). If we want to understand how people experience an intervention we need to consult studies that employ qualitative methods, such as focus groups or in-depth interviews, rather than randomized trials. In general, qualitative methods cannot tell us about the effects of intervention but, because they can tell us about people’s experiences of intervention, they can inform decisions about whether or not to intervene in a particular way.

**How can the evidence be located, and how much evidence is there?**

Several databases can be used to locate randomized trials and systematic reviews of the effects of intervention. PubMed indexes the general health literature and can be accessed free of charge at www.pubmed.gov.

CENTRAL, part of the Cochrane Library (www.thecochranelibrary.com/), specifically indexes randomized trials and is free in many countries. (To see a list of countries from which CENTRAL can be accessed free of charge, go to the Cochrane Library web page, click on the ACCESS tab and follow the link to Access Options)

The only database that specifically indexes randomized trials and systematic reviews of physical therapy interventions is PEDro. It is freely available at www.pedro.org.au. In July 2014 a quick search of the PEDro database for records indexed as relevant to the ‘perineum or genitourinary system’ yielded 870 randomized trials and 167 systematic reviews.

**Dimensions of quality of randomized trials and systematic reviews**

Randomized trials and systematic reviews vary greatly in quality. There are high-quality studies that have been carefully designed, meticulously conducted and rigorously analysed, and there are low-quality studies that have not! Physical therapists must be able to differentiate between high- and low-quality studies if they are to be able to discern the real effects of intervention.

A key characteristic of high-quality randomized trials and systematic reviews is that they are relatively unbiased. That is, they do not systematically underestimate or overestimate effects of intervention. And of course high-quality trials and reviews must also be relevant to clinical practice. That is, they must tell us about the effects of interventions when administered well to appropriate patients, and about the effects of the intervention on outcomes that are important. Finally, high-quality trials and reviews provide us with precise estimates of the size of treatment effects. The precision of the estimates is primarily a function of the sample size (the number of subjects in a trial or the number of subjects in all studies in the review). Thus the highest-quality trials and reviews, those that best support clinical decision making, are large, unbiased and relevant.

The following sections consider how readers of trials and reviews can assess these aspects of quality.

**SEPARATING THE WHEAT FROM THE CHAFF: DETECTING BIAS IN TRIALS AND REVIEWS**

**Detecting bias in randomized trials**

When we read reports of randomized trials we would like to know if the trials are biased or not. Another way of saying this is that we need to assess the validity (or ‘internal validity’) of the trials.

One way to assess internal validity is to see how well the trial has been designed. Over the past 50 years methodologists have refined the methods used to conduct randomized trials to the extent that there is now consensus, at least with regards to the main features of trial design, about what constitutes best practice in the design of clinical trials (Pocock, 1984; Moher et al., 2001). This suggests we could assess internal validity of individual trials by examining how well their methods correspond to what is thought to be best practice in trial design.

Alternatively, we could base judgments about the validity of trials on empirical evidence of bias. Several studies have shown that, all else being equal, certain design features are associated with smaller estimates of the effects of intervention (e.g. Chalmers et al., 1983; Colditz et al., 1989; Moher et al., 1998; Schulz et al., 1995). This has been interpreted as indicating that these design features are markers of bias.

Potentially we could use either of these approaches: we could base decisions about the validity of trials either on expert opinion or empirical evidence. There is much debate about which is the best way to assess validity. But fortunately both approaches suggest that trial validity should be assessed by looking for the presence of similar features of trial design (Box 2.2).

**Random allocation**

Most methodologists believe that true random allocation reduces the possibilities for bias, and some empirical evidence supports this position (Kunz and Oxman,
Box 2.2 Key features conferring validity to clinical trials

- True (concealed) random allocation of participants to groups
- Blinding of participants and assessors
- Adequate follow-up

To ensure that allocation is truly randomized it is important that a truly randomized process is used. Contemporary clinical trialists nearly always use a computer to generate the random allocation sequence but in older trials random number tables were often used. Perhaps a more critical issue is that it is necessary that the person who recruits patients into the trial is unaware, at the time he or she makes decisions about whether or not to admit a patient into the trial, which group the patient would subsequently be allocated to. Similarly, it is important that patients do not know, prior to choosing to participate in the trial, which group they would be allocated to if they were to participate in the trial. This is referred to concealment of the allocation schedule.

Failure to conceal allocation potentially distorts randomization because experimenters might be reluctant to let patients with the most serious symptoms into the trial if they know the patient is to be allocated to the control group, and patients may be less likely to agree to participate in the trial if they know they will subsequently be allocated to the control group. This would generate groups that are not comparable at baseline with regard to disease severity, so it introduces potential for serious bias. For this reason concealment is thought to protect against bias in randomized trials. Indeed, empirical evidence suggests failure to conceal allocation may be one of the most important indicators of bias (Chalmers et al., 1983; Schulz et al., 1995).

Of the trials of physical therapy for the pelvic floor listed on the PEDro database, only 30% explicitly conceal the allocation schedule.

Blinding

A second key design feature is blinding. The process of blinding implies that the allocation of each trial participant (whether the participant is in the intervention group or the control group) is hidden from people associated with the trial (such as trial participants or physical therapists administering the intervention or the people assessing trial outcomes).

Blinding of the participants in a trial is achieved by giving a sham intervention to subjects in the control group. Sham interventions are interventions that resemble the intervention of interest but which are thought to have no specific therapeutic effect. An example of an attempt to use a sham condition in a trial of an intervention for the pelvic floor is the trial by Sand et al. (1995) which compared the effects of active transvaginal electrical stimulation with sham stimulation.

By providing a sham intervention all trial participants can appear to receive intervention, but only the intervention group receives active intervention. Consequently trial participants can be ‘kept in the dark’ about whether they are receiving the intervention or control condition.

The usual justification for blinding trial participants is that this makes it possible to determine if an intervention has more of an effect than just a placebo effect. In so far as placebo effects occur, they are expected to occur to an equal degree in intervention and sham-intervention groups so it is thought that in sham-controlled trials the estimated effect of intervention – the difference between group outcomes – is not influenced by placebo effects.

An additional and perhaps more important justification is that, in trials with self-reported outcomes, blinding of participants removes the possibility of bias created by patients misreporting their outcomes. In unblinded trials, patients in the intervention group could exaggerate improvements in their outcomes and patients in the control group could understate improvements in their outcomes, perhaps because they think this is what assessors want to hear. When participants are blinded (when they do not know if they received the intervention or control conditions) there should be no difference in reporting tendencies of the two groups, so it is thought estimates of the effect of intervention (the difference between groups) cannot be biased by differential reporting.

In most trials of physical therapy interventions for the pelvic floor it is difficult to administer a sham intervention that is both credible and inactive. For example, it is difficult to conceive of a sham intervention for training pelvic floor muscles. In that case the best alternative may be to deliver an inactive intervention to the control group, even if the inactive intervention does not exactly resemble the active intervention. An example is the trial by Dumoulin et al. (2004) that compared pelvic floor rehabilitation (electrical stimulation of pelvic floor muscles plus pelvic floor muscle exercises) with biofeedback. These authors gave the control group relaxation massage to the back and extremities in the belief that this would control, to some degree, the effects of placebo and misreporting of outcomes. Such trials provide some control, but perhaps not complete control, of the confounding effects of placebo and misreporting of outcomes.

The difficulties of providing an adequate sham intervention preclude participant blinding in most trials of physical therapy interventions for the pelvic floor. Only 11% of these trials truly blind participants.
It is also desirable that the person assessing trial outcomes is blinded. Blinding of assessors ensures that assessments are not biased by the assessor’s expectations of the effects of intervention. When objective outcome measures are used, blinding of assessors is easily achieved by using assessors who are not otherwise involved in the study and are not told about which patients are in the intervention and control groups. However, blinding of assessors is more difficult when trial outcomes are self-reported (as, for example, in studies that ask women whether they ‘leak’). In that case the assessor is really the participant, and the assessor is only blind if the participant is blind.

Follow-up

A third feature of trial design that is likely to determine a trial’s validity is the completeness of follow-up.

In most trials participants are randomized to groups, but for various reasons outcome measures are not subsequently obtained from all participants. Such ‘loss to follow-up’ occurs, for example, when subjects become too ill to be measured, or they die, go on holiday, or have major surgery, or because the researchers lose contact with the participant. Loss to follow-up potentially ‘unrandomizes’ allocation, and can produce systematic differences in the characteristics of the two groups, so it potentially biases estimates of the effects of intervention.

How much loss to follow-up is acceptable in a randomized trial? When is loss to follow-up so extreme that it potentially causes serious bias? There is no simple and universally applicable answer to these questions. However, methodologists have applied threshold losses to follow-up of between about 10 and 20%. Losses to follow-up of less than 10% of randomized subjects are usually considered unlikely to produce serious bias, and losses to follow-up of greater than 20% are thought be a potential source of serious bias.

Fortunately most trials of physical therapy interventions for the pelvic floor have adequate follow-up: 61% of the relevant trials have loss to follow-up of less than 15%.

A related but more technical issue concerns problems with deviations from the trial protocol. Protocol deviations occur when, for example, people do not receive the intervention as allocated (e.g. if participants in an exercise group do not do their exercises), or if outcome measures are not measured at the allocated times. This presents a dilemma for the person analyzing the data: should data from these subjects be excluded? Should data from subjects who did not receive the intervention be analysed as if those subjects had been allocated to the control group? The answer to both questions is no!

Most methodologists believe that the best way to deal with protocol violations is to analyse the data as if the protocol violation did not occur. In this approach, called ‘analysis by intention to treat’ (Hollis and Campbell, 1999), all subjects’ data are analysed, regardless of whether they received the intervention as allocated or not, and their data are analysed in the group to which they were allocated. Analysis by intention to treat is thought to be the least biased way to analyse trial data in the presence of protocol violations. Of the relevant trials on PEDro, 24% explicitly analyse by intention to treat.

Detecting bias in systematic reviews

The search strategy

Systematic reviewers attempt to provide an unbiased summary of the findings of relevant trials. Ideally systematic reviews would summarize the findings of all relevant trials that had ever been conducted. That would achieve two ends: it would ensure that the reviewer had taken full advantage of all of the information available from all extant trials, and it would mean that the summary of the findings of the trials was not biased by selective retrieval of only those trials with atypical estimates of the effects of the intervention.

Unfortunately, it is usually not possible to find complete reports of all relevant trials: reports of some trials are published in obscure journals, others are published in obscure languages, many are published only in abstract format, and some are not published at all. Consequently even the most diligent reviewers will fail to find some trial reports.

Given that it is usually not possible to find reports of all relevant trials, the next best thing is for reviewers to obtain reports of nearly all trials. We can use reviews that summarize nearly all relevant trial reports to tell nearly all of what is known about the effectiveness of the intervention.

Incomplete retrieval of trial reports raises another problem. If reviewers do not identify all trial reports then there is the possibility that they have retrieved a particular subset of trials with exceptionally optimistic or pessimistic estimates of the effect of the intervention. We would like to be reassured when reading a systematic review that the reviewer has located a representative subset of all trials. That is, we would like to know that the reviewer has not selectively reported on trials that provide overly optimistic or pessimistic estimates of the effects of intervention. Even if we cannot expect reviewers to find reports of all trials we can require that they find an unbiased subset of nearly all trials.

To this end, most reviewers conduct quite thorough literature searches. For a Cochrane systematic review of pelvic floor muscle training (PFMT), for urinary incontinence in women, Hay-Smith et al. (2000) searched the Cochrane Incontinence Group trials register, Medline, Embase, the database of the Dutch National Institute of Allied Health Professions, CENTRAL, Physical Therapy Index and the reference lists of relevant articles. They also
searched the proceedings of the International Continence Society page by page. Some reviewers include trials published only as abstract form, whereas others include only full papers on the grounds that most abstracts have not been peer reviewed and often contain too little information to be useful.

Occasionally systematic reviewers conduct limited searches, for example by searching only Medline. This is potentially problematic: even though Medline is the largest database of medical literature such searches are likely to miss much of the relevant literature. It has been estimated that Medline only indexes between 17 and 82% of all relevant trials (Dickersin et al., 1994).

When reading a systematic review it is important to check that the literature search in the review is reasonably recent. If a report of a systematic review is more than a few years old it is likely several trials will have been conducted since the search was conducted, and the review may provide an out-of-date summary of the literature.

Assessment of trial quality

Systematic reviewers may find a number of trials that investigate the effects of a particular intervention, and often the quality of the trials is varied. Obviously it is not appropriate to weight the findings of all trials without regard to trial quality. Particular attention should be paid to the highest-quality trials because these trials are likely to be least biased; the poorest-quality trials should be ignored. Systematic reviews should assess the quality of the trials in the review, and quality assessments should be taken into account when drawing conclusions from the review.

A range of methods have been used to assess the quality of trials in systematic reviews. The most common approach is to use a quality scale to assess quality, and then to ignore the findings of trials with low-quality scores. Commonly used scales include the Maastricht scale (Verhagen et al., 1998), the PEDro scale (Maher et al., 2003) and the Cochrane risk of bias tool; a copy of the PEDro scale is shown in Box 2.3. These scales assess quality based on the presence or absence of design features thought to influence validity, including true concealed randomization, blinding of participants and assessors, adequate follow-up and intention to treat analysis.

This approach sounds sensible, but there are some reasons to think that it may discriminate inappropriately between trials. The available evidence suggests there is only moderate agreement between the ratings of different quality scales (Colle et al., 2002). Nonetheless, it is not known how better to assess trial quality, so these rudimentary procedures must suffice for now. For the time being we should expect systematic reviews to take into account the quality of trials, but we cannot be too discerning about how quality is assessed (Box 2.4).

### Box 2.3 The PEDro scale

1. Eligibility criteria were specified.  
2. Subjects were randomly allocated to groups (in a crossover study, subjects were randomly allocated an order in which treatments were received).  
3. Allocation was concealed.  
4. The groups were similar at baseline regarding the most important prognostic indicators.  
5. There was blinding of all subjects.  
6. There was blinding of all therapists who administered the therapy.  
7. There was blinding of all assessors who measured at least one key outcome.  
8. Measures of at least one key outcome were obtained from more than 85% of the subjects initially allocated to groups.  
9. All subjects for whom outcome measures were available received the treatment or control condition as allocated or, where this was not the case, data for at least one key outcome was analysed by ‘intention to treat’.  
10. The results of between-group statistical comparisons are reported for at least one key outcome.  
11. The study provides both point measures and measures of variability for at least one key outcome.

Total score is determined by counting the number of criteria that are satisfied, except that ‘eligibility criteria specified’ score is not used to generate the total score. Total scores are out of 10. More details on this scale are available from [http://www.pedro.org.au/english/faq/#question_five](http://www.pedro.org.au/english/faq/#question_five).

### Box 2.4 Key features conferring validity to systematic reviews

- An adequate search strategy (that finds an unbiased subset of nearly all relevant trials).
- The review considers trial quality when drawing conclusions about the effects of intervention.

### Assessing Relevance of Trials and Systematic Reviews

Not all valid trials are useful trials. Some provide valid tests of poorly administered interventions, others provide valid tests of the effects of intervention on inappropriate samples of patients, and yet others provide valid tests of the effect of intervention on meaningless outcomes. The following sections consider how the quality of the intervention, the selection of patients and outcomes can influence the relevance of randomized trials and systematic reviews.
Quality of intervention

Randomized trials are most easily applied to pharmacological interventions. In one sense pharmacological interventions are relatively simple: they involve the delivery of a drug to a patient. Because pharmacological interventions are quite simple they tend to be administered in quite similar ways in all trials. (One possible exception is the dose of the drug, but toxicity studies, pharmacokinetic studies and dose-finding studies often constrain the range of doses before definitive trials are carried out, so even this parameter is often fairly consistent across studies.) In contrast, many physical therapy interventions are complex. In trials of physical therapy interventions the intervention is often tailored to the individual patient based on specific examination findings, and sometimes the intervention consists of multiple components, perhaps administered in a range of settings by a range of health professionals. Consequently a single intervention (such as PFMT) may be administered in quite different ways across trials.

Wherever there is the possibility of administering the intervention in a range of ways we need to consider whether, in a particular trial, the intervention was administered well (Herbert and Bø, 2005). It is reasonable to be suspicious of the findings of trials where the intervention was administered in a way that would appear to be suboptimal.

Criticisms have been leveled against trials because the interventions were administered by unskilled therapists (Brock et al., 2002) or because the intervention was administered in a way that was contrary to the way in which the intervention is generally administered (Clare et al., 2004), or because the intervention was not sufficiently intense to be effective (Ada, 2002; Herbert and Bø, 2005). Such criticisms are sometimes reasonable and sometimes not.

Of course it is impossible to know with any certainty how an intervention should be administered before first knowing how effective the intervention is. Trials must necessarily be conducted before good information is available about how to administer the intervention. Consequently a degree of latitude ought to be offered to clinical trialists: we should be prepared to trust the findings of trials that test interventions that are applied in ways other than the ways we might choose to apply the intervention, as long as the application of the intervention in the trial was not obviously suboptimal.

Patients

Trials of a particular intervention may be carried out on quite different patient groups. Readers need to be satisfied that the trial was applied to an appropriate group of patients. It could be reasonable to ignore the findings of a trial if the intervention was administered to a group of patients for whom the intervention was generally considered inappropriate. An example might be the application of pelvic floor exercises to reverse prolapse in women who already have complete prolapse of the internal organs. Most therapists would agree that once prolapse is complete conservative intervention is no longer appropriate and surgical intervention is necessary.

The same caveat applies here: it is impossible to know with certainty, at the time a trial is conducted, who an intervention will be most effective for. Again we must be prepared to give trialists some latitude: we should be prepared to trust the findings of trials that test interventions on patients other than the patients we might choose to apply the intervention to as long as the patient group was not obviously inappropriate.

Outcomes

The last important dimension of the relevance of a clinical trial concerns the outcomes that are measured. Ultimately, if an intervention for the pelvic floor is to be useful it must improve quality of life. Arguably there is little value in an intervention that increases the strength of pelvic floor muscles if it does not also increase quality of life.

Studies of variables such as muscle strength can help us understand the mechanisms by which interventions work, but they cannot tell us if the intervention is worth doing. The trials that best help us to decide whether or not to apply an intervention are those that determine the effect of intervention on quality of life.

Many trials do not measure quality of life directly, but instead they measure variables that are thought to be closely related to quality of life. For example, Bø et al. (2000) determined the effect of PFMT for women with SUI on the risk of incontinence-related problems with social life, sex life and physical activity. It would appear reasonable to expect that problems with social life, sex life and physical activity directly influence quality of life, so this trial provides useful information with which to make decisions about PFMT for women with SUI.

In general, trials can help us make decisions about intervention in so far as they measure outcomes that are related to quality of life.

The most useful piece of information a clinical trial can give us is an estimate of the size of the effects of the intervention. We can use estimates of the effect of intervention to help us decide if an intervention does enough good to make it worth its expense, risks and inconvenience (Herbert, 2000a,b).
Obtaining estimates of the effects of intervention from randomized trials and systematic reviews

Most people experience an improvement in their condition over the course of any intervention. But the magnitude of the improvement only partly reflects the effects of intervention. People get better, often partly because of intervention, but usually also because the natural course of the condition is one of gradual improvement or because apparently random fluctuations in the severity of the condition tend to occur in the direction of an improvement in the condition. (The latter is called statistical regression; for an explanation see Herbert et al., 2005.) In addition, part of the recovery may be due to placebo effects or to patients politely overstating the magnitude of the improvements in their condition.

As several factors contribute to the improvements that people experience over time, the improvement in the condition of treated patients cannot provide a measure of the effect of intervention. A far better way to estimate the effects of intervention is to look at the magnitude of the difference in outcomes of the intervention and control groups. This is most straightforward when outcomes are measured on a continuous scale. Examples of continuous outcome measurements are pad test weights, measures of global perceived effect of intervention, or duration of labour. These variables are continuous because it is possible to measure the amount of the variable on each subject.

An estimate of the mean effects of intervention on continuous variables is obtained simply by taking the difference between the mean outcomes of the intervention and control groups. For example, a study by Bø et al. (1999) compared pelvic floor exercises with a no-exercise control condition for women with SUI. The primary outcome was urine leakage measured using a stress pad test. Over the 6-month intervention period women in the control group experienced a mean reduction in leakage of 13 g whereas women in the PFMT group experienced a mean reduction of 30 g. Thus the mean effect of exercise, compared to controls, was to reduce leaking by about 17 g (or about 50% of the initial leakage).

Other outcomes are dichotomous. Dichotomous outcomes cannot be quantified on a scale; they are events that either happen or not. An example comes from the trial by Chiarelli and Cockburn (2002) of a programme of interventions designed to prevent postpartum incontinence. Three months post partum, women were classified as being continent or incontinent. This outcome (incontinent/continent) is dichotomous, because it can have only one of two values.

When outcomes are measured on a dichotomous scale we would not normally talk about the mean outcome. Instead we talk about the risk (or probability) of the outcome; our interest is in how much intervention changes the risk of the outcome.

Chiarelli and colleagues found that 125 of the 328 women in the control group were still incontinent at 3 months, and 108 of 348 women in the intervention group were still incontinent at 3 months. Thus the risk of being incontinent at 3 months was 125/328 (38.1%) for women in the control group, but this risk was reduced to 108/348 (31%) in the intervention group. So the effect of the 3-month intervention was to reduce the risk of incontinence at 3 months post partum by 7.1% (i.e. 38.1–31.0%). This figure, the difference in risks, is sometimes called the absolute risk reduction. An absolute risk reduction of 7.1% is equivalent to preventing incontinence in one in every 14 women treated with the intervention.

Using estimates of the effects of intervention

Estimates of the effects of intervention can be used to inform the single most important clinical decision: whether or not to apply a particular intervention for a particular patient.

Decisions about whether to apply an intervention need to weigh the potential benefits of intervention against all negative consequences of intervention. So, for example, when deciding whether or not to undertake a programme of PFMT, a woman with SUI has to decide if the effects of intervention (including an expected reduction in leakage of about one-half) warrants the inconvenience of daily exercise. And when deciding whether to embark on a programme to prevent postpartum incontinence a woman needs to decide whether she is prepared to undertake the programme for a 1 in 14 chance of being continent when she otherwise would not be.

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Functional anatomy of the female pelvic floor

James A Ashton-Miller, John O L DeLancey

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INTRODUCTION

The anatomic structures that prevent incontinence during elevations in abdominal pressure are primarily sphincteric, augmented secondarily by musculofascial supportive systems. In the urethra, for example, the action of the vesical neck and urethral sphincteric mechanisms at rest constrict the urethral lumen and keep urethral closure pressure higher than bladder pressure. The striated urogenital sphincter, the smooth muscle sphincter in the vesical neck and the circular and longitudinal smooth muscle of the urethra all contribute to this closure pressure. In addition, the mucosal and vascular tissues that surround the lumen provide a hermetic seal via coaptation, aided by the connective tissues in the urethral wall. Decreases in the number of striated muscle sphincter fibres occur with age and parity, but changes in the other tissues are not well understood.

A supportive hammock under the urethra and vesical neck provides a firm backstop against which the urethra is compressed during increases in abdominal pressure to maintain urethral closure pressures above the rapidly increasing bladder pressure. This supporting layer consists of the anterior vaginal wall and the connective tissue that attaches it to the pelvic bones through the pubovaginal portion of the levator ani muscle and the uterosacral and cardinal ligaments comprising the tendinous arch of the pelvic fascia.

At rest the levator ani acts to maintain the urogenital hiatus closed in the face of hydrostatic pressure due to gravity and slight abdominal pressurization. During the dynamic activities of daily living the levator ani muscles are additionally recruited to maintain hiatal closure in the face of inertial loads related to having to decelerate caudal movements of the viscera as well as the additional load related to increases in abdominal pressure resulting from activation of the diaphragm and abdominal wall musculature.

Urinary incontinence is a common condition in women, with prevalence ranging from 8.5% to 38% depending on age, parity and definition (Thomas et al., 1980; Herzog et al., 1990). Most women with incontinence have stress urinary incontinence (SUI), not infrequently with urge incontinence (Diokno et al., 1987). Both types of incontinence are primarily due to an inadequate urethral sphincter which develops too little urethral closure pressure to prevent urine leakage (DeLancey et al., 2008, 2010). Usually this is treated using conservative therapy or, if that fails, then surgery. Despite the common occurrence of SUI, there have been few advances in our understanding of its cause in the past 40 years. Most of the many surgical procedures for alleviating SUI involve the principle of improving bladder neck support (Colombo et al., 1994; Bergman & Elia, 1995). Treatment selection based on specific anatomic abnormalities has awaited identification, in each case, of the muscular, neural and/or connective tissues involved.
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Understanding how the pelvic floor structure/function relationships provide bladder neck support can help guide treatment selection and effect. For example if, while giving vaginal birth, a woman sustains a partial tear of a portion of her pelvic muscles that influence her continence, then pelvic muscle exercises may be effective.

On the other hand, if portions of those muscles are irretrievably lost, for example due to complete and permanent denervation, then no amount of exercising will restore them; pelvic muscle exercises may well lead to agonist muscle hypertrophy, but whether or not this will restore continence will depend upon whether the agonist muscles can compensate for the lost muscle function.

This chapter reviews the functional anatomy of the pelvic floor structures and the effects of age on urethral support and the urethral sphincter, and attempts to clarify what is known about the different structures that influence stress continence. This mechanistic approach should help guide research into pathophysiology, treatment selection and prevention of SUI. In addition, we also review the structures that resist genital prolapse because vaginal delivery confers a 4- to 11-fold increase in risk of developing pelvic organ prolapse (Mant et al., 1997).

**HOW IS URINARY CONTINENCE MAINTAINED?**

Urethral closure pressure must be greater than bladder pressure, both at rest and during increases in abdominal pressure, to retain urine in the bladder and prevent leakage. The resting tone of the urethral muscles maintains a favorable pressure relative to the bladder when urethral pressure exceeds bladder pressure. The primary factor that determines continence is the maximum urethral closure pressure developed by the urethral sphincter (DeLancey et al., 2008, 2010).

During activities such as coughing, when bladder pressure increases several times higher than urethral pressure, a dynamic process increases urethral closure pressure to enhance urethral closure and maintain continence (Enhörning 1961). Both the magnitude of the resting closure pressure in the urethra and the increase in abdominal pressure generated during a cough determine the pressure at which leakage of urine occurs (Kim et al., 1997).

Although analysis of the degree of resting closure pressure and pressure transmission provides useful theoretical insights, it does not show how specific injuries to individual component structures affect the passive or active aspects of urethral closure. A detailed examination of the sphincteric closure and the urethral support subsystems (Fig. 3.1) is required to understand these relationships.

The dominant element in the urethral sphincter is the striated urogenital sphincter muscle, which contains a striated muscle in a circular configuration in the middle of the urethra and strap-like muscles distally. In its sphincteric portion, the urogenital sphincter muscle surrounds two orthogonally-arranged smooth muscle layers and a vascular plexus that helps to maintain closure of the urethral lumen.

**THE URINARY SPHINCTERIC CLOSURE SYSTEM**

Sphincteric closure of the urethra is normally provided by the urethral striated muscles, the urethral smooth muscle and the vascular elements within the submucosa (Figs 3.2 and 3.3) (Strohbehn et al., 1996; Strohbehn & DeLancey, 1997). Each is believed to contribute equally to resting urethral closure pressure (Rud et al., 1980).

Anatomically, the urethra can be divided longitudinally into percentiles, with the internal urethral meatus representing point 0 and the external meatus representing the 100th percentile (Table 3.1). The urethra passes through the wall of the bladder at the level of the vesical neck where the detrusor muscle fibres extend below the internal urethra meatus to as far as the 15th percentile.

The striated urethral sphincter muscle begins at the termination of the detrusor fibres and extends to the 64th percentile. It is circular in configuration and completely surrounds the smooth muscle of the urethral wall.
Figure 3.2 Midsagittal section showing the anatomy of the urethra. © DeLancey 1997.

Figure 3.3 Transverse histologic section of the mid-urethra of a 21-year-old woman. (A) Structures are visualized using a sigmactin smooth muscle stain, which shows the pubovesical muscle (PVM), the circumferential smooth muscle (CMU) layer, and the longitudinal smooth muscle (LMU) layer. (B) The contralateral side is stained with Mason's trichrome to show the arcus tendineus fascia pelvis (ATFP), the striated urogenital sphincter (SUG), the levator ani (LA), the anterior vaginal wall (AV), and the submucosa of the urethra (SM).

From Strohbehn et al., 1996, with permission of Lippincott Williams & Wilkins, Baltimore, MD.
Starting at the 54th percentile, the striated muscles of the urogenital diaphragm, the compressor urethrae and the urethrovaginal sphincter can be seen. They are continuous with the striated urethral sphincter and extend to the 76th percentile. Their fibre direction is no longer circular. The fibres of the compressor urethrae pass over the urethra to insert into the urogenital diaphragm near the pubic ramus. The urethrovaginal sphincter surrounds both the urethra and the vagina (Fig. 3.4). The distal terminus of the urethra runs adjacent to, but does not connect with, the bulbocavernosus muscles (DeLancey 1986).

Functionally, the urethral muscles maintain continence in various ways. The U-shaped loop of the detrusor smooth muscle surrounds the proximal urethra, favoring its closure by constricting the lumen.

The striated urethral sphincter is composed mainly of type 1 (slow-twitch) fibres, which are well suited to maintaining constant tone as well as allowing voluntary increases in tone to provide additional continence protection (Gosling et al., 1981). Distally, the recruitment of the striated muscle of the urethrovaginal sphincter and the compressor urethrae compress the lumen.

The smooth muscle of the urethra may also play a role in determining stress continence. The lumen is surrounded by a prominent vascular plexus that is believed to contribute to continence by forming a watertight seal via coaptation of the mucosal surfaces. Surrounding this plexus is the inner longitudinal smooth muscle layer. This in turn is surrounded by a circular layer, which itself lies inside the outer layer of striated muscle.

The smooth muscle layers are present throughout the upper four-fifths of the urethra. The circular configuration of the smooth muscle and outer striated muscle layers suggests that the contraction of these layers has a role in constricting the lumen. The mechanical role of the inner longitudinal smooth muscle layer is presently unresolved. Contraction of this longitudinal layer may help to open the lumen to initiate micturition rather than to constrict it.

### Table 3.1 Urethral topography and urethral and paraurethral structures

<table>
<thead>
<tr>
<th>Percentile of urethral length</th>
<th>Location: Region of the urethra</th>
<th>Structures</th>
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<tr>
<td>0–20</td>
<td>Intramural</td>
<td>Internal urethral meatus, Detrusor loop</td>
</tr>
<tr>
<td>20–60</td>
<td>Mid-urethra</td>
<td>Striated urethral sphincter muscle, Smooth muscle</td>
</tr>
<tr>
<td>60–80</td>
<td>Urogenital diaphragm</td>
<td>Compressor urethrae muscle, Urethrovaginal sphincter, Smooth muscle</td>
</tr>
<tr>
<td>80–100</td>
<td>Distal urethra</td>
<td>Bulbocavernosus muscle</td>
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Figure 3.4 Lateral view of urethral and pelvic floor muscular anatomy. BC, bulbocavernosus; CU, compressor urethrae; D, detrusor; LA, levator ani; US, urethral sphincter; UVS, urethrovaginal sphincter. Puborectalis muscle is removed for clarity. © DeLancey 2004.

**CLINICAL CORRELATES OF URETHRAL ANATOMY AND EFFECTS OF AGING**

There are several important clinical correlates of urethral muscular anatomy. Perhaps the most important is that SUI is caused by problems with the urethral sphincter mechanism as well as with urethral support. Although this is a relatively new concept, the supporting scientific evidence is strong.

The usual argument for urethral support playing an important role in SUI is that urethral support operations cure SUI without changing urethral function. Unfortunately, this logic is just as flawed as suggesting that obesity is caused by an enlarged stomach because gastric stapling surgery, which makes the stomach smaller, is effective in alleviating obesity. The fact that urethral support operations cure SUI does not implicate urethral hypermobility as the cause of SUI.

Most studies have shown not only that there is substantial variation in resting urethral closure pressures in normal women compared with those with SUI, but also that the severity of SUI correlates quite well with resting urethral closure pressure.
Loss of urethral closure pressure probably results from age-related deterioration of the urethral musculature as well as from neurologic injury (Hilton & Stanton, 1983; Snooks et al., 1986; Smith et al., 1989a, 1989b). For example, the total number of striated muscle fibres within the ventral wall of the urethra has been found to decrease seven-fold as women progress from 15 to 80 years of age, with an average loss of 2% per year (Fig. 3.5) (Perucchini et al., 2002a).

Because the mean fibre diameter does not change significantly with age, the cross-sectional area of striated muscle in the ventral wall decreases significantly with age; however, nulliparous women seemed relatively protected (Perucchini et al., 2002b). This 65% age-related loss in the number of striated muscle fibres found in vitro is consistent with the 54% age-related loss in closure pressure found in vivo by Rud et al., 1980, suggesting that it may be a contributing factor. However, prospective studies are needed to directly correlate the loss in the number of striated muscle fibres with a loss in closure pressure in vivo.

It is noteworthy that in our in vitro study thinning of the striated muscle layers was particularly evident in the proximal vesical neck and along the dorsal wall of the urethra in older women (Perucchini et al., 2002b). The concomitant seven-fold age-related loss of nerve fibres in these same striated urogenital sphincters (Fig. 3.6) directly correlated with the loss in striated muscle fibres (Fig. 3.5) in the same tissues (Pandit et al., 2000); and the correlation supports the hypothesis of a neurogenic source for SUI and helps to explain why faulty innervation could affect continence.

We believe that the ability of pelvic floor exercise to compensate for this age-related loss in sphincter striated muscle may be limited under certain situations. Healthy striated muscle can increase its strength by about 30% after an intensive 8–12 weeks of progressive resistance training intervention (e.g. Skelton et al., 1995). For example, suppose an older woman had a maximum resting urethral closure pressure of 100 cmH₂O when she was young but it is now 30 cmH₂O due to loss of striated sphincter muscle fibres. If she successfully increases her urethral striated muscle strength by 30% through an exercise intervention and there is a one-to-one correspondence between urethral muscle strength and resting closure pressure, she will only be able to increase her resting closure pressure 20 cmH₂O.
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by 30%, from 30 cmH\textsubscript{2}O to 39 cmH\textsubscript{2}O, an increment less than one-tenth of the 100 cmH\textsubscript{2}O increase in intravesical pressure that occurs during a hard cough. It remains to be determined whether pelvic floor muscle exercise is as effective in alleviating SUI in women with low resting urethral pressures as it can be in women with higher resting pressures, especially for women participating in activities with large transient increases in abdominal pressure.

**URETHRAL (AND ANTERIOR VAGINAL WALL) SUPPORT SYSTEM**

Support of the urethra and vesical neck is determined by the endopelvic fascia of the anterior vaginal wall through their fascial connections to the arcus tendineus fascia pelvis and connection to the medial portion of the levator ani muscle.

It is our working hypothesis that both urethral constriction and urethral support contribute to continence. Active constriction of the urethral sphincter maintains urine in the bladder at rest. During increases in abdominal pressure, the vesical neck and urethra are compressed to a closed position when the raised abdominal pressure surrounding much of the urethra exceeds the fluid pressure within the urethral lumen (see Fig. 3.1). The stiffness of the supportive layer under the vesical neck provides a backstop against which abdominal pressure compresses the urethra. This anatomic division mirrors the two aspects of pelvic floor function relevant to SUI: urethral closure pressure at rest and the increase in urethral closure caused by the effect of abdominal pressure.

Support of the urethra and distal vaginal wall are inextricably linked. For much of its length the urethra is fused with the vaginal wall, and the structures that determine urethral position and distal anterior vaginal wall position are the same.

The anterior vaginal wall and urethral support system consists of all structures extrinsic to the urethra that provide a supportive layer on which the proximal urethra and mid-urethra rest (DeLancey 1994). The major components of this supportive structure are the vaginal wall, the endopelvic fascia, the arcus tendineus fasciae pelvis and the levator ani muscles (see Fig. 3.1).

The endopelvic fascia is a dense, fibrous connective tissue layer that surrounds the vagina and attaches it to each arcus tendineus fascia pelvis laterally. Each arcus tendineus fascia pelvis in turn is attached to the pubic bone ventrally and to the ischial spine dorsally.

The arcus tendineus fasciae pelvis are tensile structures located bilaterally on either side of the urethra and vagina. They act like the catenary-shaped cables of a suspension bridge and provide the support needed to suspend the urethra on the anterior vaginal wall. Although it is well defined as a fibrous band near its origin at the pubic bone, the arcus tendineus fascia pelvis becomes a broad aponeurotic structure as it passes dorsally to insert into the ischial spine. It therefore appears as a sheet of fascia as it fuses with the endopelvic fascia, where it merges with the levator ani muscles (see Fig. 3.1).

**Levator ani muscles**

The levator ani muscles also play a critical role in supporting the pelvic organs (Halban & Tandler, 1907; Berglas & Rubin, 1953; Porges et al., 1960). Not only has evidence of this been seen in magnetic resonance scans (Kirschner-Hermanns et al., 1993; Tunn et al., 1998) but histological evidence of muscle damage has been found (Koelbl et al., 1998) and linked to operative failure (Hanzal et al., 1993).

There are three basic regions of the levator ani (Kearney et al., 2004) (Figs 3.8 and 3.9):

- the first region is the iliococcygeal portion, which forms a relatively flat, horizontal shelf spanning the potential gap from one pelvic sidewall to the other;
- the second portion is the pubovisceral muscle, which arises from the pubic bone on either side and attaches to the walls of the pelvic organs and perineal body;

![Figure 3.8 Schematic view of the levator ani muscles from below after the vulvar structures and perineal membrane have been removed showing the arcus tendineus levator ani (ATLA); external anal sphincter (EAS); puboanal muscle (PAM); perineal body (PB) uniting the two ends of the puboperineal muscle (PPM); iliococcygeal muscle (ICM); puborectal muscle (PRM). Note that the urethra and vagina have been transected just above the hymenal ring.](image-url)
Evidence-Based Physical Therapy for the Pelvic Floor

Injuries to the levator ani are associated with genital prolapse (Jing et al., 2012). Biomechanical computer simulations suggest this injury most likely occurs when stretch and tension in the muscle nearest the pubic bone peak near the end of the second stage of labour (Jing et al., 2012). Injuries to the levator ani are associated with genital prolapse (DeLancey et al., 2007) and in the next section we shall discuss why.

**Figure 3.9** The levator ani muscle seen from above looking over the sacral promontory (SAC) showing the pubovaginal muscle (PVM). The urethra, vagina and rectum have been transected just above the pelvic floor. PAM, puboanal muscle; ATLA, arcus tendineus levator ani; ICM, iliococcygeal muscle. (The internal obturator muscles have been removed to clarify levator muscle origins.)


- the third region, the puborectal muscle, forms a sling around and behind the rectum just cephalad to the external anal sphincter.

The connective tissue covering on both superior and inferior surfaces are called the superior and inferior fasciae of the levator ani. When these muscles and their associated fasciae are considered together, the combined structures make up the pelvic diaphragm.

The opening within the levator ani muscle through which the urethra and vagina pass (and through which prolapse occurs), is called the urogenital hiatus of the levator ani. The rectum also passes through this opening, but because the levator ani attaches directly to the anus it is not included in the name of the hiatus. The hiatus, therefore, is supported ventrally (anteriorly) by the pubic bones and the levator ani muscles, and dorsally (posteriorly) by the perineal body and external anal sphincter.

The normal baseline activity of the levator ani muscle keeps the urogenital hiatus closed by compressing the vagina, urethra and rectum against the pubic bone, the pelvic floor and organs in a cephalic direction (Taverner 1959). This constant activity of the levator ani muscle is analogous to that in the postural muscles of the spine. This continuous contraction is also similar to the continuous activity of the external anal sphincter muscle, and closes the lumen of the vagina in a manner similar to that by which the anal sphincter closes the anus. This constant action eliminates any opening within the pelvic floor through which prolapse could occur.

A maximal voluntary contraction of the levator ani muscles causes the pubovisceral muscles and the puborectalis muscles to further compress the mid-urethra, distal vagina and rectum against the pubic bone distally and against abdominal hydrostatic pressure more proximally. It is this compressive force and pressure that one feels if one palpates a pelvic floor muscle contraction intravaginally. Contraction of the bulbocavernosus and the ventral fibres of the iliococcygeus will only marginally augment this compression force developed by the pubovisceral and puborectalis muscles because the former develops little force and the latter is located too far dorsally to have much effect intravaginally.

Finally, maximal contraction of the mid and dorsal iliococcygeus muscles elevates the central region of the posterior pelvic floor, but likely contributes little to a vaginal measurement of levator strength or pressure because these muscles do not act circumvaginally.

When injury to the levator ani occurs it is usually caused by vaginal birth (DeLancey et al., 2003). Biomechanical computer simulations suggest this injury most likely occurs when stretch and tension in the muscle nearest the pubic bone peak near the end of the second stage of labour (Jing et al., 2012). Injuries to the levator ani are associated with genital prolapse (DeLancey et al., 2007) and in the next section we shall discuss why.

### Interactions between the pelvic floor muscles and the endopelvic fasciae

The levator ani muscles play an important role in protecting the pelvic connective tissues from excess load. Any connective tissue within the body may be stretched by subjecting it to a tensile force. Skin expanders used in plastic surgery stretch the dense and resistant dermis to extraordinary degrees, and flexibility exercises practised by dancers and athletes elongate leg ligaments. Both these observations underscore the adaptive nature of connective tissue when subjected to repeated tension over time.

If the ligaments and fasciae within the pelvis were subjected to continuous stress imposed on the pelvic floor by the great force of abdominal pressure, they would stretch. This stretching does not occur because the constant tonic activity of the pelvic floor muscles (Parks et al., 1962) closes the urogenital hiatus and carries the weight of the abdominal and pelvic organs, preventing constant strain on the ligaments and fasciae within the pelvis.

The interaction between the pelvic floor muscles and the supportive ligaments is critical to pelvic organ support. As long as the levator ani muscles function to properly maintain closure of the genital hiatus, the ligaments and fascial structures supporting the pelvic organs are under minimal tension. The fasciae simply act to stabilize the organs in their position above the levator ani muscles.
When the pelvic floor muscles relax or are damaged, the pelvic floor opens thereby placing the distal vagina between a zone of high abdominal pressure and the lower atmospheric pressure outside the body. The resulting pressure differential, which acts across the distal vaginal wall much like the wind on a sail, causes it to cup thereby increasing tension in the vaginal wall. This tension pulls the cervix caudally placing the uterine suspensory ligaments under tension and allowing the distal anterior vaginal wall to further cup (Chen et al., 2009). Although the ligaments can sustain these loads for short periods of time, if the pelvic floor muscles do not close the pelvic floor then the connective tissue will eventually fail, resulting in pelvic organ prolapse.

The support of the uterus has been likened to a ship in its berth floating on the water attached by ropes on either side to a dock (Paramore, 1918). The ship is analogous to the uterus, the ropes to the ligaments and the water to the supportive layer formed by the pelvic floor muscles. The ropes function to hold the ship (uterus) in the centre of its berth as it rests on the water (pelvic floor muscles). If, however, the water level falls far enough that the ropes are required to hold the ship without the supporting water, the ropes would break.

The analogous situation in the pelvic floor involves the pelvic floor muscles supporting the uterus and vagina, which are stabilized in position by the ligaments and fasciae. Once the pelvic floor musculature becomes damaged and no longer holds the organs in place, the supportive connective tissue is placed under stretch until it fails.

While the attachment of the levator ani muscles into the perineal body is important, it is uni- or bilateral damage to the pubic origin of this ventral part of the levator ani muscle during delivery that is one of the irreparable injuries to the pelvic floor. Recent magnetic resonance imaging (MRI) has vividly depicted these defects and it has been shown that up to 20% of primiparous women have a visible defect in the levator ani muscle on MRI (DeLancey et al., 2003), with a concomitant loss in levator muscle strength (DeLancey et al., 2007).

It is likely that this muscular damage is an important factor associated with recurrence of pelvic organ prolapse after initial surgical repair. Moreover, these defects were found to occur more frequently in those individuals complaining of SUI (DeLancey et al., 2003). An individual with muscles that do not function properly has a problem that is not surgically correctable.

**PELVIC FLOOR FUNCTION RELEVANT TO STRESS URINARY INCONTINENCE**

Functionally, the urethral sphincter is primarily responsible for maintaining urinary continence, aided secondarily by interactions between the levator ani muscle and the endopelvic fascia which help maintain continence and provide pelvic support. Impairments usually become evident when the system is stressed.

One such stressor is a hard cough that, driven by a powerful contraction of the diaphragm and abdominal muscles, can cause a transient increase of 150 cmH₂O, or more, in abdominal pressure. This transient pressure increase causes the proximal urethra to undergo a downward (caudodorsal) displacement of about 10 mm in the midsagittal plane that can be viewed on ultrasonography (Howard et al., 2000a). This displacement is evidence that the inferior abdominal contents are forced to move caudally during a cough.

Because the abdominal contents are essentially incompressible, the pelvic floor and/or the abdominal wall must stretch slightly under the transient increase in abdominal hydrostatic pressure, depending on the level of neural recruitment. The ventrocaudal motion of the bladder neck that is visible on ultrasonography indicates that it and the surrounding passive tissues have acquired momentum in that direction. The pelvic floor then needs to decelerate the momentum acquired by this mass of abdominal tissue.

The resulting inertial force causes a caudal-to-cranial pressure gradient in the abdominal contents, with the greatest pressure arising nearest the pelvic floor. While the downward momentum of the abdominal contents is being slowed by the resistance to stretch of the pelvic floor, the increased pressure compresses the proximal intra-abdominal portion of the urethra against the underlying supportive layer of the endopelvic fasciae, the vagina, and the levator ani muscles.

We can estimate the approximate resistance of the urethral support layer to this displacement. The ratio of the displacement of a structure in a given direction to a given applied pressure increase is known as the compliance of the structure.

If we divide 12.5 mm of downward displacement of the bladder neck (measured on ultrasonography) during a cough by the transient 150 cmH₂O increase in abdominal pressure that causes it, the resulting ratio (12.5 mm divided by 150 cmH₂O) yields an average compliance of 0.083 mm/cmH₂O in healthy nullipara (Howard et al., 2000a). In other words, the cough displaces the healthy intact pelvic floor 1 mm for every 12 cmH₂O increase in abdominal pressure. (Actually, soft tissue mechanics teaches us to expect ever smaller displacements as the abdominal pressure increments towards the maximum value.)

The increase in abdominal pressure acts transversely across the urethra, altering the stresses in the walls of the urethra so that the anterior wall is deformed toward the posterior wall, and the lateral walls are deformed towards one another, thereby helping to close the urethral lumen and prevent leakage due to the concomitant increase in intravesical pressure.

If pelvic floor exercises lead to pelvic floor muscle hypertrophy, then the resistance of the striated components of
the urethral support layer can be expected to also increase. This is because the longitudinal stiffness and damping of an active muscle are linearly proportional to the tension developed in the muscle (e.g., Blandpied & Smidt, 1993); for the same muscle tone, the hypertrophied muscle contains more cross-bridges in the strongly-bound state (across the cross-sectional area of the muscle) and these provide greater resistance to stretch of the active muscle.

If there are breaks in the continuity of the endopelvic fascia (Richardson et al., 1981) or if the levator ani muscle is damaged, the supportive layer under the urethra will be more compliant and will require a smaller pressure increment to displace a given distance.

Howard et al. (2000a) showed that compliance increased by nearly 50% in healthy primipara to 0.167 mm/cmH₂O and increased even further in stress-incontinent primipara by an additional 40% to 0.263 mm/cmH₂O. Thus, the supportive layer is considerably more compliant in these incontinent patients than in healthy women; it provides reduced resistance to deformation during transient increases in abdominal pressure so that closure of the urethral lumen cannot be ensured and SUI becomes possible.

An analogy that we have used previously is attempting to halt the flow of water through a garden hose by stepping on it (DeLancey, 1990). If the hose was lying on a noncompliant trampoline, stepping on it would change the stress in the wall of the hose pipe, leading to a deformation and flattening of the hose cross-sectional area, closure of the lumen and cessation of water flow, with little indentation or deflection of the trampoline. If, instead, the hose was resting on a very compliant trampoline, stepping on the hose would tend to accelerate the hose and underlying trampoline downward because the resistance to motion (or reaction force) is at first negligible, so little flattening of the hose occurs as the trampoline begins to stretch. While the hose and trampoline move downward together, water would flow unabated in the hose. As the resistance of the trampoline to downward movement increasingly decelerates the downward movement of the foot and hose, flow will begin to cease. Thus, an increase in compliance of the supporting tissues essentially delays the effect of abdominal pressure on the transverse closure of the urethral lumen, allowing leakage of urine during the delay.

Additionally, the constant tone maintained by the pelvic muscles relieves the tension placed on the endopelvic fascia. If the nerves to the levator ani muscle are damaged (such as during childbirth) (Allen et al., 1990), the denervated muscles would atrophy and leave the responsibility of pelvic organ support to the endopelvic fascia alone. Over time, these ligaments gradually stretch under the constant load and this viscoelastic behaviour leads to the development of prolapse.

There are several direct clinical applications for this information. The first concerns the types of damage that can occur to the urethral support system. An example is the paravaginal defect, which causes separation in the endopelvic fascia connecting the vagina to the pelvic sidewall and thereby increases the compliance of the fascial layer supporting the urethra. When this occurs, increases in abdominal pressure can no longer effectively compress the urethra against the supporting endopelvic fascia to close it during increases in abdominal pressure. When present, this paravaginal defect can be repaired surgically and normal anatomy can thus be restored.

Normal function of the urethral support system requires contraction of the levator ani muscle, which supports the urethra through the endopelvic fascia. During a cough, the levator ani muscle contracts simultaneously with the diaphragm and abdominal wall muscles to build abdominal pressure. This levator ani contraction helps to tense the suburethral fascial layer, as evidenced by decreased vesical neck motion on ultrasonographic evaluation (Miller et al., 2001), thereby enhancing urethral compression. It also protects the connective tissue from undue stresses. Using an instrumented speculum (Ashton-Miller et al., 2002), the strength of the levator ani muscle has been quantified under isometric conditions (Sampselle et al., 1998), the maximum levator force available to close the distal vagina has been shown to differ in the supine and standing postures (Morgan et al., 2005), and racial differences have been found in the levator muscle contractile properties (Howard et al., 2000b).

Striated muscle takes 35% longer to develop the same force in the elderly as in young adults, and its maximum force is also diminished by about 35% (Thelen et al., 1996a). These changes are due not to alterations in neural recruitment patterns, but rather to age-related changes in striated muscle contractility (Thelen et al., 1996b) due to the age-related loss of fast-twitch fibres (Claffin et al., 2011). Happily, and unlike that in the adjacent obturator internus muscle, the decrease in levator ani cross-sectional area or volume is not significant with older age (Morris et al., 2012), presumably due to the levator being comprised of slow-twitch muscle fibres. If the striated muscle of the levator ani becomes damaged or if its innervation is impaired, the muscle contraction will take even longer to develop the same force. This decrease in levator ani strength, in turn, is associated with decreased stiffness, because striated muscle strength and stiffness are directly and linearly correlated (Sinkjaer et al., 1988).

Alternatively, if the connection between the muscle and the fascia is broken (Klutke et al., 1990), then the normal mechanical function of the levator ani during a cough is lost. This phenomenon has important implications for clinical management. Recent evidence from MRI scans, reviewed in a blinded manner shows the levator ani can be damaged unilaterally or bilaterally in certain patients (DeLancey et al., 2003). This damage, which most often occurs in the pubo- visceral muscle near its pubic enthesis (Kim et al., 2011), has been shown to be associated with vaginal birth (Miller et al., 2010). Injury to the levator ani may also be related to urethral sphincter dysfunction (Miller et al., 2004).
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**URETROVESICAL PRESSURE DYNAMICS**

The anatomical separation of sphincteric elements and supportive structures is mirrored in the functional separation of urethral closure pressure and pressure transmission. The relationship between resting urethral pressure, pressure transmission and the pressure needed to cause leakage of urine are central to understanding urinary continence. These relationships have been described in what we have called the ‘pressuregram’ (Kim et al., 1997). The constrictive effect of the urethral sphincter deforms the wall of the urethra so as to maintain urethral pressure above bladder pressure, and this pressure differential keeps urine in the bladder at rest. For example, if bladder pressure is 10 cmH₂O while urethral pressure is 60 cmH₂O, a closure pressure of 50 cmH₂O prevents urine from moving from the bladder through the urethra (Table 3.2, Example 1).

Bladder pressure often increases by 200 cmH₂O or more during a cough, and leakage of urine would occur unless urethral pressure also increases. The efficiency of this pressure transmission is expressed as a percentage. A pressure transmission of 100% means, for example, that during a 200 cmH₂O increase in bladder pressure (from 10 cmH₂O to 210 cmH₂O), the urethral pressure would also increase by 200 cmH₂O (from 60 to 260 cmH₂O) (see Table 3.2, Example 1).

The pressure transmission is less than 100% for incontinent women. For example, abdominal pressure may increase by 200 cmH₂O while urethral pressure may only increase by 140 cmH₂O, for a pressure transmission of 70% (see Table 3.2, Example 2).

If a woman starts with a urethral pressure of 30 cmH₂O, resting bladder pressure of 10 cmH₂O and her pressure transmission is 70%, then with a cough pressure of 100 cmH₂O her bladder pressure would increase to 110 cmH₂O while urethral pressure would increase to just 100 cmH₂O and leakage of urine would occur (see Table 3.2, Example 3).

In Table 3.2, Example 4 shows the same elements, but with a higher urethral closure pressure; and similarly, Example 5 shows what happens with a weaker cough.

According to this conceptual framework, resting pressure and pressure transmission are the two key continence variables. What factors determine these two phenomena? How are they altered to cause incontinence? Although the pressuregram concept is useful for understanding the role of resting pressure and pressure transmission, it has not been possible to reliably make these measurements because of the rapid movement of the urethra relative to the urodynamic transducer during a cough.

**CLINICAL IMPLICATIONS OF LEVATOR FUNCTIONAL ANATOMY**

Pelvic muscle exercise has been shown to be effective in alleviating SUI in many, but not all, women (Bø & Talseth, 1996). Having a patient cough with a full bladder and measuring the amount of urine leakage is quite simple (Miller et al., 1998a). If the muscle is normally innervated and is sufficiently attached to the endopelvic fascia, and if by contracting her pelvic muscles before and during a cough a woman is able to decrease that leakage (Fig. 3.10) (Miller et al., 1998b), then simply learning when and how to use her pelvic muscles may be an effective therapy. If this is the case, then the challenge is for the subject to remember to use this skill during activities that transiently increase abdominal pressure.

If the pelvic floor muscle is denervated as a result of substantial nerve injury, then it may not be possible to rehabilitate the muscle sufficiently to make pelvic muscle exercise an effective strategy. In order to use the remaining innervated muscle, women need to be told when to contract the muscles to prevent leakage, and they need to learn to strengthen pelvic muscles.

A stronger muscle that is not activated during the time of a cough cannot prevent SUI. Therefore, teaching proper timing of pelvic floor muscles would seem logical as part of a behavioural intervention involving exercise. The efficacy of this intervention is currently being tested in a number of ongoing randomized controlled trials. In addition, if the muscle is completely detached from the fascial tissues, then despite its ability to contract, the contraction may no longer be effective in elevating the urethra or maintaining its position under stress.

**ANATOMY OF THE POSTERIOR VAGINAL WALL SUPPORT AS IT APPLIES TO RECTOCELE**

The posterior vaginal wall is supported by connections between the vagina, the bony pelvis and the levator ani muscles (Smith et al., 1989b). The lower one-third of the vagina is fused with the perineal body (Fig. 3.11), which is the attachment between the perineal membranes on either side. This connection prevents downward descent of the rectum in this region.

If the fibres that connect one side with the other rupture then the bowel may protrude downward resulting in a posterior vaginal wall prolapse (Fig. 3.12).
### Table 3.2  Effects of changes in cough pressure and pressure transmission ratio on urethral closure pressure and the potential leakage of urine

<table>
<thead>
<tr>
<th>Example</th>
<th>$P_{ves_R}$</th>
<th>$P_{ura_R}$</th>
<th>$UCP_R (Pura - Pves)$</th>
<th>Cough</th>
<th>PTR (%)</th>
<th>$\Delta P_{ura_c}$</th>
<th>$P_{ves_c}$</th>
<th>$P_{ura_c}$</th>
<th>$UCP_c$</th>
<th>Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>10</td>
<td>60</td>
<td>+50</td>
<td>200</td>
<td>100</td>
<td>200</td>
<td>210</td>
<td>260</td>
<td>+50</td>
<td>C</td>
</tr>
<tr>
<td>2</td>
<td>10</td>
<td>60</td>
<td>+50</td>
<td>200</td>
<td>70</td>
<td>140</td>
<td>210</td>
<td>200</td>
<td>−10</td>
<td>I</td>
</tr>
<tr>
<td>3</td>
<td>10</td>
<td>30</td>
<td>+20</td>
<td>100</td>
<td>70</td>
<td>70</td>
<td>110</td>
<td>100</td>
<td>−10</td>
<td>I</td>
</tr>
<tr>
<td>4</td>
<td>10</td>
<td>60</td>
<td>+50</td>
<td>100</td>
<td>70</td>
<td>70</td>
<td>110</td>
<td>130</td>
<td>+20</td>
<td>C</td>
</tr>
<tr>
<td>5</td>
<td>10</td>
<td>30</td>
<td>+20</td>
<td>50</td>
<td>70</td>
<td>35</td>
<td>60</td>
<td>55</td>
<td>−5</td>
<td>I</td>
</tr>
</tbody>
</table>

Parameters that have been varied are italicized to show how changes in specific parameters can change continence status. All pressures are expressed as cmH₂O.

C, continent; $\Delta P_{ura}$, change in urethral pressure; I, incontinent; PTR, pressure transmission ratio; $P_{ura_c}$, urethral pressure during cough; $P_{ura_R}$, urethral pressure at rest; $P_{ves_c}$, vesical pressure during cough; $P_{ves_R}$, vesical pressure at rest; $UCP_c$, urethral closure pressure during cough; $UCP_R$, urethral closure pressure at rest.
Figure 3.10 The effect of learning the ‘Knack’ (precontracting the pelvic muscles before a cough) on reducing the total amount of urine leaked during three separate medium-intensity coughs (left panel) and during three separate deep coughs (right panel) measured 1 week after the women had learned the skill. Each line joins the wet area on one trifold paper towel for each of the 27 women observed coughing without the Knack (denoted by ‘Without’) with that observed on a second paper towel when the same women used the Knack (denoted ‘With’) (Miller et al., 1998b). With regard to the units on the ordinate, a calibration test showed that every cm² of wetted area was caused by 0.039 ml urine leakage.

From Miller et al., 1998b, with permission of Blackwell Science, Malden, MA.

Figure 3.11 (A) The perineal membrane spans the arch between the ischiopubic rami with each side attached to the other through their connection in the perineal body. (B) Note that separation of the fibres in this area leaves the rectum unsupported and results in a low posterior prolapse.

© DeLancey 1999.

Figure 3.12 Posterior prolapse due to separation of the perineal body. Note the end of the hymenal ring, which lies laterally on the side of the vagina, is no longer united with its companion on the other side.

The midposterior vaginal wall is connected to the inside of the levator ani muscles by sheets of endopelvic fascia (Fig. 3.13). These connections prevent ventral movement of the vagina during increases in abdominal pressure. The medial most aspect of these paired sheets is referred to as the rectal pillars.

In the upper one-third of the vagina, the vaginal wall is connected laterally by the paracolpium. In this region there is a single attachment to the vagina, and a separate system for the anterior and posterior vaginal walls does not exist. Therefore when abdominal pressure forces the vaginal wall downward towards the introitus, attachments between the posterior vagina and the levator muscles prevent this downward movement.

The uppermost area of the posterior vagina is suspended, and descent of this area is usually associated with the clinical problem of uterine and/or apical prolapse. The lateral connections of the midvagina hold this portion of the vagina in place and prevent a midvaginal posterior prolapse (Fig. 3.14). The multiple connections of the perineal body to the levator muscles and the pelvic sidewall (Figs 3.15 and 3.16) prevent a low posterior prolapse from descending downward through the opening of the vagina (the urogenital hiatus and the levator ani muscles). Defects in the support at the level of the perineal body most frequently occur during vaginal delivery and are the most common type of posterior vaginal wall support problem.
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Figure 3.15 Levator ani muscles seen from below the edge of the perineal membrane (urogenital diaphragm) can be seen on the left of the specimen. © DeLancey 1999.

Figure 3.16 Position of the perineal membrane and its associated components of the striated urogenital sphincter, the compressor urethrae and the urethrovaginal sphincter. © DeLancey 1999.

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INTRODUCTION

Pelvic floor muscles (PFM) support pelvic organs, they are actively involved in their function, and probably the main culprits in some dysfunctions. A good example is stress urinary incontinence (SUI), which may develop due to weakness and/or activation and coordination disturbances of PFM. All activity of PFM is mediated (controlled) by the nervous system.

INNERVATION OF PELVIC FLOOR MUSCLES

Somatic motor pathways

The motor neurons that innervate the striated muscle of the external urethral and anal sphincters originate from a localized column of cells in the sacral spinal cord called Onuf’s nucleus (Mannen et al., 1982), expanding in humans from the second to third sacral segment (S2–S3) and occasionally into S1 (Schroder, 1985). Within Onuf’s nucleus there is some spatial separation between motor neurons concerned with the control of the urethral and anal sphincters. Spinal motor neurons for the levator ani group of muscles seem to originate from S3 to S5 segments and show some overlap (Barber et al., 2002).

Sphincter motor neurons are uniform in size and smaller than the other alpha motor neurons. They also differ with respect to their high concentrations of amino acid, neuropeptide, noradrenaline (norepinephrine), serotonin and dopamine-containing terminals, which represent the substrate for the distinctive neuropharmacological responses of these neurons, and differ from those of limb muscles, the bladder and the PFM.

The somatic motor fibres leave the spinal cord in the anterior roots and fuse with the posterior roots to constitute the spinal nerve. After passing through the intravertebral foramen the spinal nerve divides into a posterior and an anterior ramus (Bannister, 1995). Somatic fibres from the anterior rami (also called the sacral plexus) form the pudendal nerve.

Traditionally the pudendal nerve is described as being derived from the S2–S4 anterior rami, but there may be some contribution from S1, and possibly little or no contribution from S4 (Marani et al., 1993).

The pudendal nerve continues through the greater sciatic foramen and enters in a lateral direction through the lesser sciatic foramen into the ischiorectal fossa (Alcock’s canal). In the posterior part of Alcock’s canal the pudendal nerve gives off the inferior rectal nerve; then it branches into the perineal nerve, and the dorsal nerve of the penis/clitoris.

Although still controversial, it is generally accepted that the pudendal nerve supplies not only the anal but also the urinary sphincter. On the other hand it is mostly agreed that the main innervation for the PFM is through direct branches from the sacral plexus (‘from above’) rather than
predominantly by branches of the pudendal nerve (‘from below’) (Fig. 4.1).

Significant variability of normal human neuroanatomy is probably the source of remaining controversies originating from anatomical studies of peripheral innervation of the pelvis, which have so far been performed in only a small number of cases.

Higher nervous system regions control spinal cord motor nuclei by descending pathways; these inputs to PFM motor neurons are manifold, and mostly ‘indirect’ (through several interneurons). More direct connections to Onuf’s nucleus are from some nuclei in the brainstem (raphe, ambiguous) and from paraventricular hypothalamus.

Functional brain imaging is a powerful new tool to demonstrate functional anatomy of the human brain, and has already increased our knowledge in the realm of neural control of the lower urinary tract (LUT). Functional brain imaging techniques are based in particular on registering – directly or indirectly – the blood flow in the living human brain. Those brain areas that during a particular manoeuvre (e.g. pelvic floor contraction) are controlling that particular activity, are more metabolically active than other ‘nonactive’ brain areas. The increase in metabolism is accompanied by an increase in blood flow through the particular area, and this can be recorded.

The established way of recording the ‘amount’ of blood flow in parenchymatous organs is by nuclear medicine techniques, by making the blood flow ‘visible’ by a radioisotope injected into the blood. Positron emission tomography (PET) relies on this principle and is able to render enough anatomical detail to be useful also for functional anatomical studies.

Using a different recording principle (but based on similar physiological facts), functional magnetic resonance tomography (fMR) is even better for providing detailed functional anatomical data. (These techniques can also demonstrate brain areas with ‘less activity’, as in the ‘resting state’, thus indicating inhibition of certain brain areas during execution of some manoeuvres.)

PET studies have revealed activation of the (right) ventral pontine tegmentum (in the brainstem) during holding of urine in human subjects (Blok et al., 1997). This finding is consistent with the location of the ‘L region’ in cats, proposed to control PFM nuclei. The connections serve the coordinated inclusion of PFM into ‘sacral’ (LUT; anorectal and sexual) functions. Individual PFM and sphincters need not only be neurally coordinated ‘within’ a particular function (e.g. with bladder activity), but the single functions need to be neurally coordinated with each other (e.g. voiding and defecation, voiding and erection).

The sacral function control system is proposed to be a part of the ‘emotional motor system’ derived from brain or brainstem structures belonging to the limbic system. It consists of the medial and a lateral component (Holstege, 1998). The medial component represents diffuse pathways originating in the caudal brainstem and terminating on (almost all) spinal grey matter, using serotonin in particular as its neurotransmitter. This system is proposed to ‘set the threshold’ for overall changes in muscle activity, such as for instance in muscle tone under different physiological conditions (e.g. sleeping).

The lateral component of the emotional motor system consists of discrete areas in the hemispheres and the brainstem responsible for specific motor activities such as micturition and mating. The pathways belonging to the lateral system use spinal premotor inter-neurons to influence motor neurons in somatic and autonomic spinal nuclei, thus allowing for confluent interactions of various inputs to modify the motor neuron activity.

PFM nuclei also receive descending corticospinal input from the cerebral cortex. PET studies have revealed activation of the superomedial precentral gyrus during voluntary PFM contraction, and of the right anterior cingulate gyrus during sustained PFM straining (Blok et al., 1997). Not surprisingly, PFM contraction can be obtained by electrical or magnetic transcranial stimulation of the motor cortex in man (Vodušek, 1996; Brostrom, 2003).

**Afferent pathways**

Because PFM function is intimately connected to pelvic organ function, it is proposed that all sensory information from the pelvic region is relevant for PFM neural control.

The sensory neurons are bipolar. Their cell bodies are in spinal ganglia. They send a long process to the periphery and a central process into the spinal cord where it terminates segmentally or – after branching for reflex connections – ascends in some cases as far as the brainstem (Bannister, 1995).

The afferent pathways from the anogenital region and pelvic region are divided into somatic and visceral. Somatic afferents derive from touch, pain and thermal...
Neural control of micturition

Centres in the pons (brainstem) coordinate micturition as such, but areas rostral to the pons (the hypothalamus and other parts of the brain including the frontal cortex) are responsible for the timing of the start of micturition. The pontine micturition centre (PMC) coordinates the activity of motor neurons of the urinary bladder and the urethral sphincter (both nuclei located in the sacral spinal cord), receiving afferent input via the periaqueductal grey matter. The central control of LUT function is organized as an on–off switching circuit (or a set of circuits, rather) that maintains a reciprocal relationship between the urinary bladder and urethral outlet.

Without the PMC and its spinal connections coordinated bladder/sphincter activity is not possible, thus patients with lesions of the PMC and its spinal connections demonstrate bladder sphincter discoordination (dyssynergia). Patients with lesions above the pons do not show detrusor–sphincter dyssynergia, but have urge incontinence (due to bladder overactivity) and demonstrate noninhibited sphincter relaxation and an inability to delay voiding to an appropriate place and time.

Voluntary micturition is a behaviour pattern that starts with relaxation of the striated urethral sphincter and PFM. Voluntary PFM contraction during voiding can lead to a stop of micturition, probably because of collateral connections to detrusor control nuclei. Descending inhibitory pathways for the detrusor have been demonstrated (de Groat et al., 2001). Bladder contractions are also inhibited by reflexes, activated by afferent input from the PFM, perineal skin and anorectum (Sato et al., 2000).

Neural control of anorectal function

Faeces stored in the colon are transported past the rectosigmoid ‘physiological sphincter’ into the normally empty...
rectum, which can store up to 300 ml of contents. Rectal distension causes regular contractions of the rectal wall, which is effected by the intrinsic nervous (myenteric) plexus, and prompts the desire to defecate (Bartolo and Macdonald, 2002).

Stool entering the rectum is also detected by stretch receptors in the rectal wall and PFM; their discharge leads to the urge to defecate. It starts as an intermittent sensation, which becomes more and more constant. Contraction of the PFM may interrupt the process, probably by concomitant inhibitory influences to the defecatory neural ‘pattern generator’, but also by ‘mechanical’ insistence on sphincter contraction and the propelling of faeces back to the sigmoid colon (Bartolo and Macdonald, 2002).

The PFM are intimately involved in anorectal function. Apart from the ‘sensory’ role of the PFM and the external anal sphincter function, the puborectalis muscle is thought to maintain the ‘anorectal’ angle, which facilitates continence, and has to be relaxed to allow defecation. Current concepts suggest that defecation requires increased rectal pressure coordinated with relaxation of the anal sphincters and PFM.

Pelvic floor relaxation allows opening of the anorectal angle and perineal descent, facilitating faecal expulsion. Puborectalis and external anal sphincter activity during evacuation is generally inhibited. However, observations by EMG and defecography suggest that the puborectalis may not always relax during defecation in healthy subjects (Fucini et al., 2001).

**Neural control of the sexual response**

The PFM are actively involved in the sexual response. Their activation has been mostly explored in males during ejaculation, where their repetitive activation during a several seconds interval is responsible for the expulsion of semen from the urethra, particularly by the bulbocavernosus muscles (Petersen et al., 1955). Little is known on PFM activity patterns during other parts of the human sexual response cycle.

It is assumed that apart from general changes in muscle tone set by the emotional motor system, the sacral reflex circuitry governs much of the PFM activity during the sexual response cycle. The bulbocavernosus reflex behaviour, as known from studies (Vodušek, 2002a) would allow for reflex activation of the PFM during genital stimulation. Tonic stimulation of the reflex is postulated to hinder venous outflow from penis/clitoris, thus helping erection. Reflex contraction of the PFM should conceivably contribute to the achievement of the ‘orgasmic platform’ (contraction of the levator ani and – in the female – the circumvaginal muscles). Climax in humans (in both sexes, and in experimental animals) elicits rhythmic contractions of the PFM/perineal muscles, which in the male drives the ejaculate from the urethra (assisted by a coordinated bladder neck closure).

**NEUROPHYSIOLOGY OF PELVIC FLOOR MUSCLES**

Muscle activity is thoroughly dependent on neural control. ‘Denervated’ muscle atrophies and turns into fibrotic tissue. Muscle – like every tissue – consists of cells (muscle fibres). But the functional unit within striated muscle is not a single muscle cell, but a motor unit. A motor unit consists of one alpha (or ‘lower’) motor neuron (from the motor nuclei in the spinal cord), and all the muscle cells this motor neuron innervates. The motor unit – in other words – is the basic functional unit of the somatic motor system; control of a muscle means control of its motor units. Thus, in discussing neural control of muscle, we really only need to consider the motor neurons in the spinal cord and all the influences they are exposed to.

The function of pelvic floor and sphincter lower motor neurons is organized quite differently from other groups of motor neurons. In contrast to the reciprocal innervation that is common in limb muscles, the neurons innervating each side of the PFM have to work in harmony and synchronously. Indeed, sphincters may be morphologically considered to constitute ‘one’ muscle – which is innervated by two nerves (left and right)!

By concomitant activity the PFM acts as the ‘closure unit’ of the excretory tracts, the ‘support unit’ for pelvic viscera and an ‘effector unit’ in the sexual response. In general, muscles involved in these functions from both sides of the body act in a strictly unified fashion as ‘one muscle’: this has been demonstrated for the pubococcygei muscles, but has not really been documented for the whole group of PFM and sphincters (Deindl et al., 1993). However, as each muscle in the pelvis has its own unilateral peripheral innervation, dissociated activation patterns are possible and have been reported between the two pubococcygei (Deindl et al., 1994) and between levator ani and the urethral sphincter (Kenton and Brubaker, 2002).

The differences in evolutionary origin of the sphincter muscles and levator ani furthermore imply that unilateral activation may be less of an impossibility for the PFM than for sphincters. It can be postulated that the neural mechanisms controlling the different muscles involved in sphincter mechanisms and pelvic organ support may not be as uniform as has been assumed. How much variability there is in normal activation patterns of PFM is not yet clarified. It is clear, however, that the coordination between individual PFM can definitively be impaired by disease or trauma.

**Tonic and phasic pelvic floor muscle activity**

The normal striated sphincter muscles demonstrate some continuous motor unit activity at rest as revealed by kinesiological EMG (Fig. 4.2). This differs between individuals
and continues also after subjects fall asleep during the examination (Chantaine, 1973). This physiological spontaneous activity may be called tonic, and depends on prolonged activation of certain tonic motor units (Vodušek, 1982).

The ‘amount’ of tonic motor unit activity can in principle be assessed counting the number of active motor unit potentials or analysing the interference pattern by EMG; this has so far not been much studied. Thus, little is known about the variability and the normal range of tonic activity in normal subjects, and the reproducibility of findings; this makes it difficult to assess the validity of results from the few studies reporting activity changes accompanying LUT, anorectal or sexual dysfunction.

As a rule, tonic motor unit activity increases with bladder filling, at the same time depending on the rate of filling. Any reflex or voluntary activation is mirrored first in the firing frequency of these motor units. Recruitment of additional motor units can be seen on reflex manoeuvres (thin arrows: pinpricks at anal wedge; full arrows: strong cough), and on command to contract (empty arrows). The additionally recruited motor units have larger PMEs.

With any stronger activation manoeuvre (e.g. contraction, coughing), and only for a limited length of time, new motor units are recruited (see Fig. 4.2). These may be called ‘phasic’ motor units. As a rule, they have potentials of higher amplitudes and their discharge rates are higher and irregular. A small percentage of motor units with an ‘intermediate’ activation pattern can also be encountered (Vodušek, 1982). It has to be stressed that this typing of motor units is electrophysiological, and no direct correlation to histochemical typing of muscle fibres has so far been achieved.

Figure 4.2 Kinesiological EMG recording from anal sphincter muscle. Concentric needle electrode recording in a 40-year-old continent woman. Note ongoing sparse firing of motor unit potentials, which is called the ‘tonic activity’ (on this time scale the motor unit potentials/PME/are just thin perpendicular lines, as seen in the uppermost ray – before the thin arrows indicate reflex excitation). Tonic motor units as a rule have small amplitudes (small thin perpendicular lines). Tonic activity can also be seen after the voluntary contraction (last two rays). Recruitment of additional motor units can be seen on reflex manoeuvres (thin arrows: pinpricks at anal wedge; full arrows: strong cough), and on command to contract (empty arrows). The additionally recruited motor units have larger PMEs.

With regard to tonic activity, sphincters differ from some perineal muscles; tonic activity is encountered in many but not all detection sites for the levator ani muscle (Vodušek, 1982; Deindl et al., 1993) and is practically never seen in the bulbocavernosus muscle (Vodušek, 1982). In the pubococcygeus of the normal female there is some increase of activity during bladder filling, and reflex increases in activity during any activation manoeuvre performed by the subject (e.g. talking, deep breathing, coughing).

On voiding, inhibition of the tonic activity of the external urethral sphincter – and also the PFM – leads to relaxation. This can be detected as a disappearance of all EMG activity, which precedes detrusor contraction. Similarly, the striated anal sphincter relaxes with defeication and also micturition (Read, 1990).

**Reflex activity of pelvic floor muscles**

The human urethral and anal striated sphincters seem to have no muscle spindles; their reflex reactivity is thus intrinsically different from the levator ani muscle complex, in which muscle spindles and Golgi tendon organs have been demonstrated (Borghi et al., 1991). Thus, PFM have the intrinsic proprioceptive ‘servo-mechanism’ for adjusting muscle length and tension, whereas the sphincter muscles depend on afferents from skin and mucosa. Both muscle groups are integrated in reflex activity, which incorporates pelvic organ function.

The reflex activity of PFM is clinically and electrophysiologically evaluated by eliciting the bulbocavernosus and anal reflex. The bulbocavernosus reflex is evoked on non-painful stimulation of the glans (or – electrically – the dorsal penile/clitoral nerve). As recorded electromyographically, it is a complex response: its first component is thought to be an oligosynaptic and the later component, a polysynaptic reflex (Vodušek and Janko, 1990). The polysynaptic anal reflex is elicited by painful (pinprick) stimulation in the perianal region.

The constant tonic activity of sphincter muscles is thought to result from the characteristics of their ‘low-threshold’ motor neurons and the constant ‘inputs’ (either of reflex segmental or suprasegmental origin). It is supported by cutaneous stimuli, by pelvic organ distension and by intra-abdominal pressure changes.

Sudden increases in intra-abdominal pressure as a rule lead to brisk PFM (reflex) activity, which has been called the ‘guarding reflex’; it is organized at the spinal level. It needs to be considered that ‘sudden increases in intra-abdominal pressure’, if caused by an intrinsically driven manoeuvre (i.e. coughing), include feed forward activation of the PFM as part of the complex muscle activation pattern. The observed PFM activation in the normal subject (e.g. during coughing) is thus a compound ‘feed-forward’ and ‘reflex’ muscle activation.
Another common stimulus leading to an increase in PFM activity is pain. The typical phasic reflex response to a nociceptive stimulus is the anal reflex. It is commonly assumed that prolonged pain in pelvic organs is accompanied by an increase in ‘reflex’ PFM activity, which would indeed be manifested as ‘an increased tonic motor unit activity’. This has so far not been much formally studied. Whether such chronic PFM overactivity might itself generate a chronic pain state and even other dysfunctions may be a tempting hypothesis, but has not been well demonstrated so far.

To correspond to their functional (effector) role as pelvic organ supporters (e.g. during coughing, sneezing), sphincters for the LLT and anorectum, and as an effector in the sexual arousal response, orgasm and ejaculation, PFM have also to be involved in very complex involuntary activity, which coordinates the behaviour of pelvic organs (smooth muscle) and several different groups of striated muscles. This activity is to be understood as originating from so-called ‘pattern generators’ within the central nervous system, particularly the brainstem. These pattern generators (‘reflex centres’) are genetically inbuilt.

**Awareness of Muscle**

The sense of position and movement of one’s body (in most instances mostly dependent on muscle activity) is referred to as ‘proprioception’, and is particularly important for sensing limb position (stationary proprioception) and limb movement (kinaesthetic proprioception).

Proprioception relies on special mechanoreceptors in muscle tendons and joint capsules. In muscles there are specialized stretch receptors (muscle spindles) and in tendons there are Golgi tendon organs, which sense the contractile force. In addition, stretch-sensitive receptors signalling postural information are in the skin. This cutaneous proprioception is particularly important for controlling movements of muscles without bony attachment (lips, anal sphincter). By these means of afferent input the functional status of a striated muscle (or rather: a certain movement) is represented in the brain. Indeed, muscle awareness reflects the amount of sensory input from various sites. Typically, feedback to awareness on limb muscle function (acting at joints) is derived not only from input from muscle spindles, and receptors in tendons, but also from the skin, and from visual input, etc. The concept of ‘awareness’ thus in fact overlaps with the ability to voluntarily change the state of a muscle (see below).

In contrast to limb muscles, the PFM (and sphincters) lack several of the above-mentioned sensory input mechanisms and therefore the brain is not ‘well informed’ on their status. Additionally, there may be a gender difference, inasmuch as pelvic floor muscle awareness in females seems to be, in general, less compared to males. (The author concludes this on the basis of long personal experience with PFM EMGs in both genders; there seems to be no formal study on PFM activation patterns in man apart from ejaculation.)

Healthy males have no difficulties in voluntarily contracting the pelvic floor, but up to 30% of healthy women cannot do it readily on command. The need for ‘squeezing out’ the urethra at the end of voiding and the close relationship of penile erection and ejaculation to PFM contractions may be the origin of this gender difference. The primarily weak awareness of PFM in women seems to be further jeopardized by vaginal delivery.

**Voluntary activity of pelvic floor muscles**

Skilled movement of distal limb muscles requires individual motor units to be activated in a highly focused manner by the primary motor cortex. By contrast, activation of axial muscles (necessary to maintain posture, etc.) – while also under voluntary control – depends particularly on vestibular nuclei and reticular formation to create predetermined ‘motor patterns’.

The PFM are not, strictly speaking, axial muscles, but several similarities to axial muscles can be proposed as regards their neural control. In any case, PFM are under voluntary control (i.e. it is possible to voluntarily activate or inhibit the firing of their motor units). EMG studies have shown that the activity of motor units in the urethral sphincter can be extinguished at both low and high bladder volumes even without initiating micturition (Sundin and Petersen, 1975; Vodušek, 1994).

To voluntarily activate a striated muscle we have to have the appropriate brain ‘conceptualization’ of that particular movement, which acts as a rule within a particular complex ‘movement pattern’. This evolves particularly through repeatedly executed commands and represents a certain ‘behaviour’.

Proprioceptive information is crucial for striated muscle motor control both in the ‘learning’ phase of a certain movement and for later execution of overlearned motor behaviours. It is passed to the spinal cord by fast-conducting, large-diameter myelinated afferent fibres and is influenced not only by the current state of the muscle, but also by the efferent discharge the muscle spindles receive from the nervous system via gamma efferents. To work out the state of the muscle, the brain must take into account these efferent discharges and make comparisons between the signals it sends out to the muscle spindles along the gamma efferents and the afferent signals it receives from the primary afferents.

Essentially, the brain compares the signal from the muscle spindles with the copy of its motor command (the ‘corollary discharge’ or ‘efferents copy’) which was sent to the muscle spindle intrafusal muscle fibres by the CNS via gamma efferents. The differences between the two signals are used in deciding on the state of the muscle. The experiments were
carried out in limb muscles (McCloskey, 1981), but it has been suggested (Morrison, 1987) that similar principles rule in bladder neurocontrol.

NEUROMUSCULAR INJURY TO THE PELVIC FLOOR DUE TO VAGINAL DELIVERY

Many studies using different techniques have demonstrated neurogenic and structural damage to the PFM and sphincter muscles as a consequence of vaginal delivery (Vodušek, 2002b). Other lesion mechanisms, such as muscle ischaemia, may also be operative during childbirth. As a consequence, the PFM would become weak; such weakness has indeed been demonstrated (Verelst and Leivseth, 2004). The sphincter mechanisms and pelvic organ support become functionally impaired, with SUI and prolapse being a logical consequence.

Although muscle weakness may be a common consequence of childbirth injury, there seem to be further pathophysiological possibilities for deficient PFM function; it is not only the strength of muscle contraction that defines its functional integrity.

Normal neural control of muscle activity leads to coordinated and timely responses to ensure appropriate muscle function as required. Muscular ‘behavioural’ patterns have been studied by kinesiological EMG recording (Deindl et al., 1993). Changes in muscular behaviour may originate from minor and repairable neuromuscular pelvic floor injury (Deindl et al., 1994).

In nulliparous healthy women two types of behavioural patterns, named as tonic and phasic pattern, respectively, can be found:

- the tonic pattern consists of a crescendo–decrescendo type of activity (probably derived from grouping of slow motor units) that may be the expression of constant (‘tonic’) reflex input parallel to the breathing pattern;
- the phasic pattern, probably related to fast-twitch motor unit activation, is motor unit activity seen only during activation manoeuvres, either voluntary contraction or coughing.

With respect to these muscle activation patterns, parous women with SUI are subject to a number of possible changes (Deindl et al., 1994), such as a significant reduction of duration of motor unit recruitment, unilateral recruitment of reflex response in the pubococcygeal muscle and paradoxical inhibition of continuous firing of motor units in PFM activation on coughing.

The reasons for such persisting abnormalities are not clear and are difficult to explain by muscle denervation (which has been amply studied) alone. Although not directly proven in studies, it is reasonable to assume that motor denervation is accompanied also by sensory denervation of the PFM. In addition to denervation injury there may be some further temporary ‘inhibitors’ of PFM activation, such as periods of pain and discomfort after childbirth (e.g. perineal tears, episiotomy), increased by attempted PFM contraction.

All these above-mentioned factors may lead to a temporary disturbance of PFM activation patterns after childbirth. This, in combination with a particularly vulnerable pelvic floor neural control (which only evolved in its complexity phylogenetically after the attainment of the upright stance), might become persistent, even if the factors originally leading to the problem disappear.

CONCLUSION

The PFM are a deep muscle group that have some similarities in their neural control with axial muscles. They are under prominent reflex and relatively weak voluntary control, with few and poor sensory data contributing to awareness of the muscles. Furthermore their neural control mechanism is fragile due to its relative phylogenetic recency, and is exposed to trauma and disease due to its expansive anatomy (from frontal cortex to the entrapment of spinal cord, and extensive peripheral innervation, both somatic and autonomic).

Vaginal delivery may lead to structural and denervation changes in the PFM, but also to secondary changes in their activation patterns. Dysfunctional neural control induced by trauma, disease, or purely functional causes may manifest itself by over- or underactivity, and/or by discoordination of PFM activity. Often these disturbances are not ‘hard-wired’ into the nervous system, but only a problem of neural control ‘software’ (which can be ‘re-programmed’). Therefore, physical therapy should in many patients provide an appropriate, and even best available, treatment.

REFERENCES


Measurement of pelvic floor muscle function and strength, and pelvic organ prolapse

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5.1 Introduction

Kari Bø

CLASSIFICATION AND DEFINITIONS

The International Classification of Impairments, Disabilities and Handicaps (ICIDH) (1997), more recently changed to the International Classification of Functioning, Disability, and Health (ICF) (2002), is a World Health Organization (WHO)-approved system for classification of health and health-related states in rehabilitation science. According to this system, the causes of a non-optimally functioning pelvic floor (e.g. muscle and nerve damage after vaginal birth) can be classified as the pathophysiological component. Nonfunctioning pelvic floor muscles (PFM) (reduced force generation, incorrect timing or coordination) are the impairment component, and the symptom of pelvic floor dysfunction (e.g. urinary leakage, faecal incontinence, or pelvic organ prolapse) is a disability. How the symptoms and conditions affect the woman’s quality of life and participation in fitness activities is an activity or participation component.

Physical therapists (PTs) working to prevent or treat pelvic floor dysfunction aim to improve disability and activity/participation components by improving PFM function. Hence, it is important to measure all ICF components. In this chapter we deal only with the pathophysiological and impairment component, with a focus on assessment of ability to contract the PFM and measurement of PFM strength, resting activity and ability to relax.

The main reasons for physical therapists to conduct high-quality assessment of ability to contract the PFM and different aspects of PFM function are as follows.

1. Without proper instruction, many women are unable to volitionally contract PFM on demand. This may be because the muscles are situated at the floor of the pelvis and are not visible from the outside. In addition, the muscles are seldom used consciously. Several studies have shown that more than 30% of women do not contract their PFM correctly at their first consultation, even after thorough individual instruction (Kegel, 1948; Benvenuti et al., 1987; Bø et al., 1988; Bump et al., 1991). The most common errors are to contract the gluteal, hip adductor, or abdominal muscles instead of the PFM (Bø et al., 1988). Some women also stop breathing or try to exaggerate inspiration instead of contracting the PFM. Some studies have demonstrated that many women strain, causing PFM descent, instead of actively squeezing and lifting the PFM upward (Bø et al., 1990; Bump et al., 1991). For proper contraction of the PFM, it is mandatory that women receive precise training with appropriate monitoring and feedback. Hay-Smith et al. (2001) found that in the reports of only 15 of 43 RCTs they reviewed did the authors state that a correct PFM contraction was checked before training began.

2. In intervention studies evaluating the effect of PFM training, the training is the independent variable meant to cause a change in the dependent variable (e.g. stress urinary incontinence [SUI] or pelvic organ prolapse [POP]) (Thomas et al., 2005). Thus, measurement of PFM function and strength before and after training is important to determine whether the intervention has made significant changes. Even in the presence of tissue pathology (e.g. neuropathy), if there is no change in PFM function or strength after a training programme commensurate with that pathology, the training programme has been of insufficient dosage (intensity, frequency or duration of the training period) or the participants have had inadequate adherence (Bouchard et al., 1994). It is
likely that such programmes have not followed muscle training recommendations.

In this chapter we describe different measurement tools such as clinical observation, vaginal palpation, electromyography (EMG), vaginal squeeze pressure measurement (manometry), urethral pressure measurement (stationary and ambulatory), dynamometry, ultrasonography and magnetic resonance imaging (MRI) in use for assessment of the PFM. This can be either assessment of unconscious co-contraction of the PFM during an increase in abdominal pressure or ability to volitionally perform a correct contraction. A correct voluntary contraction is described as an elevation and squeeze around the pelvic openings (Kegel, 1948).

**Muscle strength** has been defined as ‘the maximum force that can be exerted against an immovable object (static or isometric strength), the heaviest weight which can be lifted or lowered (dynamic strength), or the maximal torque which can be developed against a preset rate-limiting device (isokinetic strength)’ (Frontera and Meredith, 1989). Maximum strength is often referred to as the maximum weight the individual can lift once. This is named the one repetition maximum or 1RM (Wilmore and Costill, 1999).

Maximum strength is measured through a **maximum voluntary contraction.** Maximum voluntary contraction refers to a condition in which a person attempts to recruit as many fibres in a muscle as possible for the purpose of developing force (Knuttgen and Kraemer, 1987). The force generated is dependent on the cross-sectional area of the muscle and the neural components (e.g. number of activated motor units and frequency of excitation; Wilmore and Costill, 1999). Hence, PFM strength is a surrogate for underlying factors that will change with regular strength training.

**Muscle power** is the explosive aspect of strength and is the product of strength and speed of movement [power = (force × distance)/time] (Wilmore and Costill, 1999). Muscle force is reduced with speed of the contraction. Power is the key component of functional application of strength. Speed, however, changes little with training, thus power is changed almost exclusively through gains in strength (Wilmore and Costill, 1999).

**Muscular endurance** can be classified as:

1. ability to sustain near maximal or maximal force, assessed by the time one is able to maintain a maximum static or isometric contraction;
2. ability to repeatedly develop near maximal or maximal force determined by assessing the maximum number of repetitions one can perform at a given percentage of 1RM (Wilmore and Costill, 1999).

Muscle strength measurement may be considered an indirect measure of PFM function in real-life activities. Women with no leakage do not contract voluntarily before coughing or jumping. Their PFM contraction is considered to be an automatic co-contraction occurring as a quick and effective activation of an intact neural system.

Other important factors for a quick and effective contraction are the location of the pelvic floor within the pelvis, the muscle bulk, stiffness/elasticity of the pelvic floor and intact connective tissue.

A stretched and weak pelvic floor may be positioned lower within the pelvis compared with a well-trained or noninjured pelvic floor (Bø, 2004). The time for stretched muscles to reach an optimal contraction may be too slow to be effective in preventing descent against increased abdominal pressure (e.g. sneeze), thereby allowing leakage to occur.

In general, when measuring muscle strength it can be difficult to isolate the muscles to be tested, and many test subjects need adequate time and instruction in how to perform the test. In addition, the test situation may not reflect the whole function of the muscles, and the generalizability from the test situation to real-world activity (external validity) has to be established (Thomas et al., 2005). Therefore, when reporting results from muscle testing, it is important to specify the equipment used, position during testing, testing procedure, instruction and motivation given, and what parameters are tested (e.g. ability to contract, maximum strength, endurance). When testing the PFM, additional challenges are present because muscle action and location are not easily observable.

Whether a measurement tool should be used in clinical practice or in research depends on its responsiveness, reliability and validity. These terms are used slightly different in different research areas and have somewhat different definitions in different textbooks of research methodology. The definitions given below are the ones we have chosen to use in this textbook.

- **Responsiveness:** the degree or amount of variation that the device is capable of measuring; the ability of a tool to detect small differences or small changes (Currier, 1990).
- **Reliability:** consistency or repeatability of a measure. The most common way to establish stability of a test is to perform a test–retest. **Intra-test** reliability is conducted by one researcher measuring the same procedure in the same subjects twice. **Inter-test** reliability is conducted when two or more clinicians or researchers are conducting measurement of the same subjects (Currier, 1990).
- **Validity:** degree to which a test or instrument measures what it is supposed to measure.
  - **Logical (face) validity:** condition that is claimed when the measure obviously involves the performance being measured (e.g. squeeze and elevation of the PFM can be felt by vaginal palpation).
  - **Content validity:** condition that is claimed when a test adequately samples what it should cover (few methods measure both squeeze pressure and elevation of the PFM).
  - **Criterion validity:** the degree to which the scores on a test are related to some recognized standard,
Evidence-Based Physical Therapy for the Pelvic Floor

or criterion (e.g. clinical observation of inward movement of the perineum during attempts to contract the PFM compared with ultrasonography).

- **Concurrent validity**: involves a measuring instrument being correlated with some criterion administered at the same time or concurrently (e.g. simultaneous observation of inward movement of the PFM strength with manometers and dynamometers).

- **Predictive validity**: degree to which scores of predictor variables can accurately predict criterion scores.

- **Diagnostic validity**: ability of a measure to detect differences between those having a diagnosis/problem/condition/symptom with those not.

- **Sensitivity**: the proportion of positives that are correctly identified by the test.

- **Specificity**: the proportion of negatives that are correctly identified by the test (Currier, 1990; Altman, 1997; Thomas et al., 2005).

It is important for PTs who treat patients with pelvic floor dysfunction to understand the qualities and limitations of the measurement tools they use (Bo and Sherburn, 2005). This chapter will provide the information needed for PTs to understand the application of each tool to the measurement of the PFM. In many instances the PT may need thorough supervised instruction from other professionals before starting to use new equipment. In most cases, when available, receiving results from assessment of the PFM from other professionals (e.g. radiologists) provides the best results.

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5.2 Visual observation and palpation

Kari Bø

VISUAL OBSERVATION

A correct contraction can be observed clinically (Kegel, 1948), by ultrasound (Beco et al., 1987; Dietz et al., 2002; Petri et al., 1999) or with dynamic magnetic resonance imaging (MRI) (Bø et al., 2001; Stoker et al., 2001).

In 1948, Kegel described a correct PFM contraction as a squeeze around the urethral, vaginal and anal openings, and an inward lift that could be observed at the perineum (Kegel, 1948, 1952). He estimated the inward movement in the lying position to be 3–4 cm (Kegel, 1952). However, newer research visualizing lifting distance inside the body with MRI and ultrasound has not supported his estimation, which was based on visual observation. Bø et al. (2001) demonstrated a mean inward lift during PFM contraction to be 10.8 mm (SD 6.0) in 16 women using dynamic MRI in a sitting position. This corresponded with an inward lift of 11.2 mm (95% CI: 7.2–15.3) measured with suprapubic ultrasound in a supine position (Bo et al., 2003).

Most PTs would use visual observation of the PFM contraction as a starting point for measurement of ability to contract. In spite of this, there is a paucity of research on responsiveness, reliability and validity of this method.

Bø et al. (1990) used observation of movement of a vaginal catheter, vaginal palpation and vaginal squeeze pressure to measure PFM function and strength. They registered the ability to contract from visual observation as:

- correct (inward movement of the catheter);
- no contraction (no movement);
- straining (outward movement).

There was 100% agreement between observation and the vaginal palpation test in women who either contracted correctly or were not able to contract according to the palpation test. The observation classified six who were straining and were not detected on the palpation test. Hence observation of movement may be more sensitive to straining and Valsalva manoeuvre than palpation.

Responsiveness

No studies have been found evaluating the responsiveness of visual observation.

Intra- and inter-rater reliability

Devreese et al. (2004) developed an inspection scale for the PFM and abdominal muscles to be used in crook lying, sitting and standing position. Contractions were inspected during both voluntary contraction and reflex contraction during coughing. They classified the contraction of the PFM as either ‘coordinated’ (inward movement of 1 cm of the perineum and a visible contraction of the deep abdominal muscle) or ‘not coordinated’ (downward movement of the pelvic floor and/or an outward movement of the abdominal wall. The results of inter-tester reliability showed kappa coefficients between 0.94 and 0.97. Slieker et al. (2009) tested intra- and inter-observer reliability of observation and found inter-rater K(w) (weighted kappa) values of only 0.33 during coughing and 0.013 during straining. Kw for inter-rater reliability for visible co-contraction was 0.52. Intra- and inter-observer reliability for visible co-contraction was 0.48 and 0.52, respectively. There was high intra- and inter-observer reliability in observation of incontinence, relaxation and inward movement during PFM contraction.

Validity

Shull et al. (2002) stated that by visual observation one is generally observing superficial perineal muscles. From this observation, researchers assume that the levator ani is responding similarly. It may, however, not be the case.

Observing the inward movement of a correct PFM contraction is the starting point for measurement of PFM function, and has the advantage of being a simple, non-invasive method. However, the inward lift may be created by contraction of superficial muscle layers only, and have no influence on urethral closure mechanism. Conversely, there may be palpable PFM contraction with no visible outside movement. A correct lift can be difficult to observe from the outside, particularly in obese women. Also it is questionable whether it is possible to grade centimetres of inward movement from the outside of the body. In the future ultrasound may take over the role of visual observation, and would also serve as a biofeedback and teaching tool.

Whether the muscle action observed by visual observation or ultrasound is sufficiently strong to increase urethral closure pressure can only be measured by urodynamic assessment in the urethra and bladder. Interestingly, Bump et al. (1991) found that, although contracting correctly, only 50% of a population of continent and incontinent women were able to voluntarily contract the PFM with enough force to increase urethral pressure.
and compared continent and incontinent women with blinded investigators. The results showed that continent women exhibited significantly better coordination between the pelvic floor and lower abdominal muscles during coughing in all three positions (crook lying, sitting and standing). Amaro et al. (2005) compared 50 women with SUI and 50 continent women. They found that there was a negative observation of ability to contract in 25.5% of the SUI group compared to 0 in the nonincontinent group.

**Conclusion**

Visual observation can be used in clinical practice to give a first impression about ability to contract. Further estimation about the amount of the inward movement is not recommended. Visual observation should not be used for scientific purposes because MRI and especially ultrasound are more responsive, reliable and valid methods to assess movement during contraction, straining and physical exertion.

**CLINICAL RECOMMENDATIONS**

**PFM assessment using observation**

- Inform and explain the procedure to the patient.
- Teach the patient how to contract the PFM by use of models, anatomical drawings and imagery.
- After the patient has undressed, ask the patient to lie down on the bench with hips and knees bent and shoulder width apart (crook lying). Cover pelvic area with a towel. Support legs of patient (one leg against the wall, the other leg support with one hand).
- Allow some time for patient to practise before observing the contraction.
- Ask the patient to breathe normally and then lift the perineum inwards and squeeze around the openings without any movement of the pelvis or visible co-contraction of the gluteal or hip-adductor muscles. A small drawing in of the lower abdomen with the PFM contraction is accepted. Observe the patient’s attempt to contract and register how the contraction was performed (correct, no contraction, inconclusive, straining).
- If there is an observable contraction, give positive feedback and explain that you will palpate to register action of the deeper muscles, and coordination and strength of the contraction. If you are not able to observe inward movement, explain that this is common at the first attempt, and that it is not always easy to assess from the outside, and that you need to conduct a vaginal palpation to be sure whether there is a contraction or not.

**VAGINAL PALPATION**

Vaginal palpation (Fig. 5.1) is used to:

1. assess the ability of the patient to contract and relax the PFM correctly;
2. measure PFM muscle strength via a maximal occlusive and lifting force (assessing the person’s attempt to conduct a maximum voluntary contraction), ability to sustain a contraction (endurance) or perform a number of repeated contractions (endurance);
3. assess other elements of PFM, such as resting tension/muscle activity, the ability to fully relax after a contraction, coordination with lower abdominal muscles, symmetry of right and left PFM contraction, scarring and adhesions, and the presence of pain, major PFM injuries, speed and sequence of recruitment of levator ani with the perineal muscles, and transverse and anteroposterior diameters of the urogenital hiatus.

Figure 5.1 During vaginal palpation the physical therapist instructs the patient about how to perform a contraction correctly ("Squeeze around my finger and try to lift the finger inwards") and tells her how well she is able to do it and also about coordination skills and strength. With encouragement, most patients are able to contract harder.
The ICS Clinical Assessment Group (see www.icsoffice.org) has proposed qualitative scales of measurement for some of these parameters (absent, partial, full). Slieker et al. (2009) found that in general the intra-rater reliability was much higher than the inter-rater reliability. Moderate to substantial inter-rater reliability was found for palpation of pain, levator closure, and voluntary contraction. Endurance, fast contraction and voluntary relaxation had a Kw value of 0.37, 0.47 and 0.17, respectively. Palpation of involuntary contraction during coughing and movement of the perineum had an inter-rater reliability of 0.33 and 0.03, respectively, and palpation of involuntary relaxation during straining had a Kw of 0.15 only.

Kegel described vaginal palpation as a method to evaluate the ability to perform a correct contraction (Kegel, 1948, 1952). He placed one finger in the distal one-third of the vagina and asked the woman to lift inwards and squeeze around the finger. Kegel did not use this method to measure PFM strength. He classified the contraction qualitatively as correct or not. In addition, he developed the ‘perineometer’, a pressure manometer, to measure PFM strength through vaginal squeeze pressure (Kegel, 1948).

Van Kampen et al. (1996) reported that after Kegel first described vaginal palpation as a method to evaluate PFM function, more than 25 different palpation methods have been developed. Some examiners use one, and others two fingers.

Worth et al. (1986) and Brink et al. (1989) have evaluated pressure, duration, muscle ‘ribbing’, and displacement of the examiner’s finger in a specific scoring system. This system has mainly been used by American nurses. There has been no systematic research to determine the best method of palpation to assess ability to contract, or any of the parameters of muscle strength, endurance, or power.

Laycock has developed the modified Oxford grading system (Box 5.1) to measure PFM strength (British Medical Research Council, 1943; Laycock, 1994), and this seems to be the system mostly used by PTs to assess PFM strength in clinical practice.

### Box 5.1 The modified Oxford grading scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>no contraction</td>
</tr>
<tr>
<td>1</td>
<td>flicker</td>
</tr>
<tr>
<td>2</td>
<td>weak</td>
</tr>
<tr>
<td>3</td>
<td>moderate (with lift)</td>
</tr>
<tr>
<td>4</td>
<td>good (with lift)</td>
</tr>
<tr>
<td>5</td>
<td>strong (with lift)</td>
</tr>
</tbody>
</table>

The modified Oxford grading scale is a 6-point scale where half numbers of + and − can be added when a contraction is considered to fall between two full grades, so it expands to a 15-point scale when both + and − are used:

- 0 = no contraction
- 1 = flicker
- 2 = weak
- 3 = moderate (with lift)
- 4 = good (with lift)
- 5 = strong (with lift)

### Responsiveness

The Oxford grading system has been modified from the Medical Research Council scale (1943), which suffers from poor responsiveness and nonlinearity (Beasley, 1961).

One of the difficulties of measurement using the modified Oxford scale is that it produces one value for two elements (occlusion and lift) in the one scale. The palpating fingers may not be sensitive enough to differentiate the proportions of occlusion versus lift. To separate these two elements, manometers or dynamometers can be used to evaluate occlusion, and ultrasound to measure the lift component. When the responsiveness of this scale is tested against vaginal squeeze pressure, it should be recognized that only one element, occlusion, is being compared.

Bo and Finckenhagen (2001) questioned the responsiveness of the original scale (without + and −) because they did not find that the scale could separate between weak, moderate, good, or strong when comparing measurement of vaginal squeeze pressure. This was supported by Morin et al. (2004) comparing vaginal palpation and dynamometry in continent and incontinent women. They found that important overlaps were observed between each category of vaginal palpation. Mean force values differed significantly only between nonadjacent levels in palpation assessment (e.g. between 1 and 3, 1 and 4, 1 and 5, 2 and 4, and 2 and 5 (p <0.05).

Frawley et al. (2006) found that the Oxford grading scale using + and − had lower kappa values in intra-test reliability testing and recommended using the original six-point scale in research.

### Intra- and inter-rater reliability

The results from studies evaluating intra- and inter-rater reliability of vaginal palpation for strength measurement are conflicting (McKey and Dougherty, 1986; Hahn et al., 1996; Isherwood and Rane, 2000; Bo and Finckenhagen, 2001; Jeyaseelan et al., 2001; Laycock and Jerwood, 2001; Frawley et al., 2006; Jean-Michel et al., 2010).

Isherwood and Rane (2000) found high inter-rater reliability whereas Jeyaseelan et al. (2001) concluded that inter-tester reliability should not be assumed, and needs to be established when two or more clinicians are involved in pre- and post-treatment assessment.

Bo and Finckenhagen (2001) using the six-point scale and Laycock and Jerwood (2001) using the 15-point scale found agreement between testers in only 45% and 46.7% of the tested cases, respectively. The latter was supported by Jean-Michel et al. (2010) reporting that test–retest values for the Oxford muscle grading system were unacceptably poor within and between examiners. However, no data were reported in the study.

Devreeze et al. (2004) developed a new vaginal palpation system assessing muscle ‘tone’, endurance, speed of contraction, strength, lift (inward movement) and coordination, and evaluated both superficial and deep PFM.
They found high agreement in inter-observer reliability in tone (95–100% agreement) and reliability coefficients between 0.75 and 1.00 for measurements of the other parameters above. The scoring system developed is qualitative and open to personal interpretation, but it was a first step towards standardizing a measurement system for observation and palpation.

Frawley et al. (2006) found 79% complete agreement in both crook lying and supine using the six-point scale but this dropped to 53% and 58%, respectively, using the 15-point scale. They tested intra-tester reliability of vaginal digital assessment and found good to very good kappa values of 0.69, 0.69, 0.86 and 0.79 for crook lying, supine, sitting and standing positions, respectively. In addition, they compared vaginal palpation with vaginal squeeze pressure measurement with the Peritron and found that the Peritron was more reliable than vaginal palpation (Frawley et al., 2006).

Muscle ‘tone’ requires a universally acceptable definition to establish a reliable measurement system, and to differentiate ‘tone’ from ‘stiffness’, ‘contracture’ and ‘spasm’. Simons and Mense (1998) have proposed that ‘muscle tone’ specific to a muscle rather than generalized tone can be defined as ‘the elastic and viscoelastic stiffness of a muscle in the absence of motor unit activity’. The elastic component or ‘elastic stiffness’ is measured qualitatively by pressing or squeezing a muscle. However, measurement of the viscoelastic component is more complex and is dependent on the speed at which the muscle is moved using pendular, oscillatory and resonant frequency measurements (Simons and Mense, 1998). These viscoelastic measurements are not possible for the PFM because the PFM do not pass over a joint to allow elongation then shortening. If the PFM are elongated using vaginal palpation to stretch the muscle fibres, the muscle belly is actually being compressed by the examining digit and elastic stiffness is again being measured. Tension may be a better terminology than tone.

One can also discuss how one can assess that there is no motor unit activity. At least for the PFM, there is always electromyographic (EMG) activity, except before and during voiding (Fowler et al., 2002). Dietz and Shek (2008) proposed a new scale for resting tone from 0 (muscle not palpable) to 5 (hiatus very narrow, no distension possible, ‘woody’ feel, possibly with pain: vaginismus) and found a Kw of 0.55 (CI: 0.44–0.66). A low resting tone was associated with pelvic organ prolapse.

MRI and ultrasound are commonly used to assess injuries to the PFM. Dietz et al. (2006) and Dietz et al. (2012) compared vaginal palpation and 4D pelvic floor ultrasound and concluded that palpation of the pubovisceral muscle correlated poorly with 3/4D ultrasound in detecting levator ani trauma. In another study (Dietz et al., 2012), vaginal palpation was compared with 3/4D ultrasound analysed with either render or multi-slice imaging and kappa values between 0.35 and 0.56 were found. Kruger et al. (2013) assessed 72 women ≥60 years and found that the predictive ability of the digital assessment varied from poor to moderate. The variable ‘width between insertions’ performed best. Palpation could not distinguish between uni- and bilateral trauma.

Validity

Several investigators have studied criterion validity of vaginal palpation comparing vaginal palpation and vaginal squeeze pressure (McKey and Dougherty, 1986; Hahn et al., 1996; Isherwood and Rane, 2000; Bø and Finckenhaen, 2001; Jarvis et al., 2001; Kerschan-Schindel et al., 2002).

Isherwood and Rane (2000) compared vaginal palpation using the Oxford grading system and compared it with an arbitrary scale on a perineometer from 1 to 12. They found a high kappa of 0.73. In contrast, Bø and Finckenhagen (2001) found a kappa of 0.37 comparing the Oxford grading system with vaginal squeeze pressure. Heitner (2000) concluded that lift was most reliably tested with palpation, and that all other measures of muscle function were better tested with EMG.

Hahn et al. (1996) found that there was a better correlation of vaginal palpation and pressure measurement in continent than in incontinent women (r = 0.86 and 0.75, respectively). This was supported by Morin et al. (2004) comparing vaginal palpation with dynamometry, finding r = 0.73 in continent and r = 0.45 in incontinent women, respectively.

Lying, sitting or standing?

PFM function and strength is often measured in a supine position, despite the fact that urinary leakage is more common in the upright position with gravity acting on the PFM. Very few studies have addressed measurement in different positions.

Devreese et al. (2004) investigated inter-rater reliability of clinical observation and vaginal palpation in crook lying, sitting and standing positions. They found high inter-tester reliability in all positions, but did not report whether there were differences in measurement values in the different positions.

Frawley et al. (2006) found that vaginal palpation of PFM contraction had moderate to high intra-test reliability in crook lying, supine, sitting and standing position. Both Bø and Finckenhaen (2003) and Frawley et al. (2006) found that PTs and patients preferred testing using vaginal palpation and vaginal squeeze pressure in lying positions. Bø and Finckenhaen (2003) found that the testing procedure was easiest to standardize when the patient was supine, and therefore recommend this in clinical practice.

For scientific purposes the position of the patient should be chosen according to the research question.

One or two fingers?

There is a discussion whether one or two fingers should be used for vaginal palpation (Shull et al., 2002; Bø et al.,
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2005) and this may depend on factors such as whether the patient is nulliparous and has a narrow vaginal introitus and urogenital hiatus, or whether there is introital discomfort or pain.

Hoyte et al. (2001) reported increased diameters from nonsymptomatic parous women, to parous women with pelvic organ prolapse (POP). In parous women, vaginal birth may have stretched the PFM and its investing fascia. However, time and PFM training may normalize this in many women. When palpating, the anterior and posterior vaginal walls are always in apposition and in contact with the finger. The lateral vaginal walls expand in the upper vagina at the level of the fornices and above the level of the levator ani. At the PFM level, the lateral diameter of the urogenital hiatus marks the medial borders of the levator ani and these borders may be palpated through the intervening vaginal mucosa.

Ghetti et al. (2005) stated that intra- and inter-rater reliability of vaginal palpation to assess the diameter of the hiatus needs to be done. In addition, criterion validity between magnetic resonance imaging (MRI)/ultrasound and vaginal palpation of the hiatus has to be established.

Putting a muscle on stretch makes it more difficult to perform a maximal contraction (Frontera and Meredith, 1989). Therefore the aim of palpation should be to gain maximum sensation for the palpation with no stretch. This must not be confused with the fact that a quick stretch can be used to facilitate the stretch reflex. Quick stretch is one technique used by PTs to facilitate a correct PFM contraction if the patients are unable to contract (Brown, 2001).

**Sensitivity and specificity**

There are few studies comparing measurement of PFM function and strength in continent and incontinent women using vaginal palpation.

Hahn et al. (1996) compared 30 continent and 30 incontinent women using vaginal palpation and found that the group of incontinent women had lower scores on palpation test (1.0 ± 0.1) compared to the group of continent women (1.9 ± 0.1) (p < 0.001).

Devreese et al. (2004) found a significant difference in favour of continent women in speed of contraction, maximum strength and coordination of both superficial and deep PFM, and inward movement of the superficial, but not the deep PFM, assessed with vaginal palpation. Amaro et al. (2005) found normal function assessed by palpation in 18% of incontinent women versus 90% in the continent group. Thompson et al. (2006a, 2006b) also confirmed stronger PFM in continent compared to incontinent women.

**Conclusion**

Today most PTs use vaginal palpation to evaluate PFM function because both squeeze pressure and lift can be registered, though with poor discrimination. It is a low-cost method and is relatively easy to conduct.

Vaginal palpation of PFM contraction is recommended as a good technique for PTs to understand, teach and give feedback to patients about correctness of the contraction. Position of the patient, instruction given and the use of one or two fingers have to be standardized and reported. However, whether palpation is robust enough to be used for scientific purposes to measure muscle strength is questionable. Palpation as a method to detect morphological abnormalities is still under discussion.

**CLINICAL RECOMMENDATIONS**

**Palpation procedure**

Following perineal observation, with the patient in the crook lying position:

- Explain the palpation procedure to the patient and obtain consent.
- Prepare examination gloves, gel and tissues, and check with the patient for latex and gel allergy. Use vinyl gloves for preference.
- Wash hands, put on gloves and apply a little gel on the palpating gloved finger(s).
- Gently part the labia and insert one finger in the outer one-third of the vagina.
- Ask the patient whether she feels comfortable.
- If appropriate, insert the second finger.
- Ask the patient to lift in and squeeze around the finger(s) and observe or control the action so that the pelvis is not moving or the hip adductor or gluteal muscles are not contracted.
- Give feedback of correctness, performance and strength.
- Record whether PFM contraction is:
  - correct;
  - only possible with visible co-contraction of other muscles:
  - not present;
  - in the opposite direction (straining or Valsalva).
- To record the maximum voluntary contraction (MVC) request a 3–5 s maximum effort PFM contraction after one or two submaximal ‘practice’ contractions. If you do not have a sensitive, reliable and valid tool to measure strength, use the Oxford grading scale to record the MVC. Separately record the lift component as absent, partial or complete.
- Note the voluntary relaxation after these contractions and record this as absent, partial or full.
- If no further vaginal measurements are to be made, discard the examination gloves into the appropriate waste disposal and allow the patient privacy for dressing.
REFERENCES


**5.3 Electromyography**

David B Vodušek

**INTRODUCTION**

Electromyography (EMG) is the extracellular recording of bioelectrical activity generated by muscle fibres. The term indeed stands for at least two different clinically used methods, which are quite distinct and as a rule performed in different settings (laboratories), for different purposes. On the one hand EMG can reveal the ‘behaviour’ (i.e., patterns of activity) of a particular muscle, or it can also be used to demonstrate whether a muscle is normal, myopathic or denervated/reinnervated. The former is focused on the muscle as a whole and can be called ‘kinesiological EMG.’ The latter is focused on the muscle constituents (muscle fibres and motor units) and can be called ‘motor unit’ EMG. The terminology specified here is not much in use, but will be used in this chapter for clarity. (The division in the use of the EMG signal is recognized by experts but usually not specifically underlined; both ‘types of EMG’ are thus just called ‘EMG’, and this can confuse the uninitiated when reading the literature on EMG.)

In clinical neurophysiology, EMG techniques are combined with conduction studies to assess involvement of the neuromuscular system by trauma or disease (Aminoff, 2012).

**MUSCLE FIBRE, MOTOR UNIT, MUSCLE**

A single muscle fibre (cell) does not contract on its own, but rather in concert with other muscle fibres that are part of the same motor unit (i.e., innervated by the same motor neuron). Its axon reaches the muscle via a motor nerve. Within the muscle the motor axon tapers and then...
branches to innervate muscle fibres, which are scattered throughout the muscle. Fibres that are part of the same motor unit are not adjacent to one another. Bioelectrical activity generated by the concomitant activation of muscle fibres from one motor unit is ‘summed’ by the recording electrode as a ‘motor unit action potential’ (MUP). As many motor units are active within a contracting muscle and the recording surface of the EMG electrode is adjacent to muscle fibres from several motor units, several MUPs are recorded by the recording electrode. This produces an ‘interference pattern’ of MUPs in a given time interval of recording. If the activation of a normal muscle is strong, most motor units are activated and the interference pattern is ‘full’ (Vodušek and Fowler, 2004; Podnar and Vodušek, 2012).

**KINESIOLOGICAL EMG**

Prolonged recording of bioelectrical activity of a muscle provides a qualitative and quantitative description of its activity over time, thus characterizing its ‘behaviour’ during particular manoeuvres (see Chapter 4, Fig. 4.2). It should be borne in mind that kinesiological EMG does not provide information on the ‘state’ of the muscle (i.e., whether its motor units have been changed due to neuropathy or myopathy). A special analysis of the EMG signal is necessary to provide that information. Meaningful kinesiologic EMG can, of course, be obtained only from innervated muscle.

When the pattern of activity of an individual muscle is of interest, the EMG should ideally provide a selective recording, uncontaminated by neighbouring muscles on one hand, and a faithful detection of any activity within the source muscle on the other hand. Both objectives are difficult to achieve simultaneously. Overall detection from the bulk of a muscle can only be achieved with nonselective electrodes, selective recordings from small muscles can only be made with intramuscular electrodes with small detection surfaces. Nonselective recordings carry the risk of contamination with activity from other muscles; selective recordings may fail to detect activity in all parts of the source muscle. Meaningful recordings from deep muscles can be accomplished only by invasive techniques.

Considering the above, truly selective recording from sphincter muscles can probably be obtained only by intramuscular electrodes. In clinical routine the concentric needle electrode is used as a rule. Needle electrodes, however, may produce some pain on movement, and can be dislodged. Instead, two thin isolated/bare tip wires (with a hook at the end) can be introduced into the muscle with a cannula, which is then withdrawn, and the wires stay in place (Deindl et al., 1993). The advantage of this type of recording is good positional stability and painlessness once the wires are inserted, though their position cannot be much adjusted.

To make EMG recording less invasive various surface-type electrodes have been devised – also for special use in the perineum. Small skin-surface electrodes can be applied to the perineal skin. Other special intravaginal, intrarectal or catheter-mounted recording devices have been described. Recordings with surface electrodes are more artefact prone and furthermore the artefacts may be less easily identified.

Critical online assessment of the ‘quality of the EMG signal’ is mandatory in kinesiological EMG, and this requires either auditory or oscilloscope monitoring of the raw signal. Integration of high-quality EMG signals by the software of modern recording systems may help in quantification of results. It should be borne in mind that kinesiological EMG needs some concomitant event markers to make it a valid indicator of muscle activity correlated with specific manoeuvres or other physiologic events (e.g. detrusor pressure).

**EMG METHODS TO DIFFERENTIATE NORMAL FROM PATHOLOGICAL MUSCLE**

EMG may help to differentiate between normal, denervated, and reinnervated and myopathic muscle. In pelvic floor muscles (PFM) and sphincter muscles, ‘neurogenic’ changes are sought as a rule because only patients with suspected denervation injury are routinely referred for assessment. One or several muscles may be examined, according to the clinical problem in the individual patient. The levator ani, anal and urethral sphincter, and bulbocavernosus are the muscles routinely examined, but if a rather equal involvement of PFM is suspected, examination of the external anal sphincter (on one or both sides) suffices (Podnar et al., 1999).

**Concentric needle EMG**

Single-use disposable concentric needle EMG (CN EMG) electrodes are used as a rule to diagnose striated muscle denervation/reinnervation. The CN EMG electrode records spike (or ‘near’) activity from about 20 muscle fibres in the vicinity of its active recording surface at the bevelled tip (Vodušek and Fowler, 2004). The number of motor units recorded depends both upon the local arrangement of muscle fibres within the motor unit and the level of contraction of the muscle.

CN EMG can provide information on insertion activity, abnormal spontaneous activity, MUPs and interference pattern (Podnar and Vodušek, 2012).

In healthy skeletal muscle initial placement of the needle (and any movement of the tip) elicits a short burst of ‘insertion activity’ due to mechanical stimulation of excitable membranes. Absence of insertion activity with
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Chapter 5

Measurement of pelvic floor muscle function and strength, and pelvic organ prolapse

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an appropriately placed needle electrode usually means a complete denervation atrophy of the examined muscle (Podnar and Vodušek, 2012). At rest, tonic MUPs are the only normal bioelectrical activity recorded.

In partially denervated sphincter muscle there is – by definition – a loss of motor units, but this is difficult to estimate. Normally, MUPs should intermingle to produce an ‘interference’ pattern on the oscilloscope during strong muscle contraction, and during a strong cough. The number of continuously active MUPs during relaxation can be estimated by counting the number of continuously firing low-threshold MUPs (Podnar et al., 2002a).

In patients with lesions of peripheral innervation, fewer MUPs fire continuously during relaxation. In addition to continuously firing low threshold (‘tonic’) motor units, new motor units (‘phasic’) are recruited voluntarily and reflexly. It has been shown that the two motor unit populations differ in their characteristics: reflexly or voluntarily activated ‘high-threshold’ MUPs being larger than continuously active ‘low-threshold’ MUPs (Podnar and Vodušek, 1999).

Using the standard recording facilities available on all modern EMG machines, individual MUPs can be captured and their characteristics determined (Fig. 5.2). Typically MUP amplitude and duration are measured.

To allow identification of MUPs and to be certain the ‘late’ MUP components of complex potentials are not due to superimposition of several MUPs, it is necessary to capture the same potential repeatedly. MUPs are mostly below 1 mV and certainly below 2 mV in the normal urethral and anal sphincter; most are less than 7 ms in duration, and few (less than 15%) are above 10 ms; most are bi- and triphasic, but up to 15–33% may be polyphasic. Normal MUPs are stable – their shape on repetitive recording does not change (Vodušek and Light, 1983; Fowler et al., 1984; Rodi et al., 1996) (Fig. 5.3).

There are indeed two approaches to analysing quantitatively the bioelectrical activity of motor units: either individual MUPs are analysed (Podnar et al., 2002a; Podnar et al., 2002b), or the overall activity of intermingled MUPs (the ‘interference pattern’ – IP) is analysed (see Fig. 5.3) (Aanestad et al., 1989; Podnar et al., 2002a; Podnar et al., 2002b).

Generally three techniques of MUP analysis (‘manual-MUP’, ‘single-MUP’ and ‘multi-MUP’) and one technique of IP analysis (turn/amplitude – T/A) are available on advanced EMG systems. By either method a relevant sample of EMG activity needs to be analysed for the test to be valid.

In the small half of the sphincter muscle collecting 10 different MUPs has been accepted as the minimal requirement for using single-MUP analysis. Using manual-MUP and multi-MUP techniques sampling of 20 MUPs (standard number in limb muscles) from each EAS poses no difficulty in healthy controls and the majority of patients (Podnar et al., 2000b; Podnar et al., 2002b). Normative data obtained from the external anal sphincter (EAS) muscle by standardized EMG technique using all three MUP analysis techniques (multi-MUP, manual-MUP, single-MUP) have been published (Podnar et al., 2002b). There are several technical differences in the methods. The template based multi-MUP analysis of MUP is fast, easy to apply and allows little examiner bias (see Fig. 5.3).

Use of quantitative MUP and IP analyses of the EAS is facilitated by the availability of normative values (Podnar et al., 2002b) that can be introduced into the EMG system software. It has been shown that normative data are not significantly affected by age, gender (Podnar et al., 2002a), number of uncomplicated vaginal deliveries (Podnar et al., 2000a), mild chronic constipation (Podnar and Vodušek, 2000), and the part of the external anal sphincter

Figure 5.2 Schematic representation of the motor unit potential (MUP) to demonstrate different components, and parameters analysed. Modified from Podnar, Mrkaic et al., 2002 and Podnar, Vodušek et al., 2002.
Figure 5.3 Comparison of normal (above) and pathological (below) motor unit potentials (MUPs) sampled by multi-MUP analysis from the right halves of the subcutaneous parts of the external anal sphincter (EAS) muscles. On the right, logarithm amplitude vs duration plots of the MUPs are shown; the inner rectangle presents the normative range for mean values, and the outer rectangle for outliers. Below the MUP samples, values are tabulated. Three plots on the bottom were obtained by turn/amplitude analysis of the interference pattern (IP) in a patient with a cauda equina lesion. Delineated areas (clouds) present the normative range, and dots individual IP samples. The normal subject was a 45-year-old woman. Results of MUP and IP analysis were normal. The pathological sample was obtained from a 36-year-old man with cauda equina lesion caused by central herniation of the intervertebral disc 13 months before the examination, with perianal sensory loss. Mean values for MUP amplitude and area are above the normative range, and polyphasicity is increased. In addition, for all MUP parameters shown, individual values of more than 2 MUPs are above the outlier limits. Note that IP analysis in the patient is within the normative range despite marked MUP abnormalities.
**CN EMG findings due to denervation and reinnervation**

In PFM and perineal muscles, complete or partial denervation may be observed after lesions to its nerves. The changes occurring in striated muscles after denervation are in principle similar. After complete denervation all motor unit activity ceases and there may be electrical silence for several days; 10–20 days after a denervating injury, ‘insertion activity’ becomes more prolonged and abnormal spontaneous activity in the form of short biphasic spikes, ‘fibrillation potentials’, biphasic potentials with prominent positive deflections and ‘positive sharp waves’ appear. With successful axonal reinnervation MUPs appear again; first short bi- and triphasic, soon becoming polyphasic, serrated and of prolonged duration (Vodušek and Fowler, 2004; Podnar and Vodušek, 2012). In partially denervated muscle some MUPs remain and mingle eventually with abnormal spontaneous activity. In longstanding partially denervated muscle a peculiar abnormal insertion activity appears, so-called ‘repetitive discharges’. This activity may be found in the striated urethral sphincter without any other evidence of neuromuscular disease (Podnar and Vodušek, 2012).

In partially denervated muscle, collateral reinnervation takes place. Surviving motor axons will sprout and grow out to reinnervate denervated muscle fibres. This will result in a change in the arrangement of muscle fibres within the motor unit. Following reinnervation several muscle fibres belonging to the same motor unit come to be adjacent to one another; this is reflected in changes of MUPs (increased duration and amplitude).

In the late stage, after reinnervation has been completed, CN EMG as a rule finds a reduced number of remaining motor units (i.e., the IP of MUPs is reduced). The MUPs are of higher amplitudes, and longer duration, and the percentage of polyphasic MUPs is increased. Such a finding may be taken as proof of previous denervation and successful reinnervation. The function of the reinnervated muscle will depend on the number (and size) of remaining motor units. The relative amount of remaining motor units can only be estimated (Podnar et al., 2002a; Podnar et al., 2002b; Vodušek and Fowler, 2004).

**Single fibre electromyography**

The single fibre electromyography (SFEMG) electrode has similar external proportions to a CN EMG electrode, but instead of having the recording surface at the tip, it is on the side above the tip and its recording surface is much smaller. Because of the arrangement of muscle fibres in a normal motor unit, a SFEMG needle will record only 1–3 single muscle fibres from the same motor unit.

The SFEMG parameter that reflects motor unit morphology is fibre density (FD), which is the mean number of muscle fibres belonging to an individual motor unit per detection site. To measure FD, recordings from 20 different detection sites within the examined muscle are necessary and the number of component potentials to each motor unit recorded and averaged. The normal fibre density for the anal sphincter is less than 2.0 (Neill and Swash, 1980; Vodušek and Janko, 1981).

Due to its technical characteristics a SFEMG electrode is able to record changes that occur in motor units due to reinnervation, but is less suitable to detect changes due to denervation itself (i.e., abnormal insertion and spontaneous activity). The SFEMG electrode is also suitable for recording instability of motor unit potentials, the ‘jitter’ (Stalberg and Trontelj, 1994). This parameter has not been much used in PFM.

SFEMG has been and can be used in research. It is not widely used; its use in diagnostics in general clinical neuropsychological laboratories is restricted to investigation of pathology of the neuromuscular transmission. The recording needles are very expensive, and disposable needles are not available. For this reasons the principles of SFEMG recording are now often applied to recording with concentric needle electrodes (using filters cutting off low frequencies of the EMG signal to achieve higher selectivity of recording).

**USEFULNESS OF EMG IN CLINICAL PRACTICE AND RESEARCH**

The validity, reliability, responsiveness and sensitivity/specificity of EMG have to be discussed separately for the particular physiological information sought from EMG, and for various EMG techniques, types of recording and applications. EMG is often falsely understood as ‘one method’, and as a method precisely measuring muscle ‘function’. Muscle function is, however, complex and different EMG techniques address different aspects of it, but never really cover all of it. Indeed, motor unit EMG techniques, for instance, are more useful in diagnosing denervation and reinnervation (i.e., helping in diagnosing a neurological lesion) than in diagnosing the functional deficit (i.e., quantifying the number of motor units and thus providing data that would be directly functionally relevant).

It has to be distinguished whether EMG is used to detect the pattern of muscle activity, or rather to detect muscle denervation/reinnervation. EMG methods are reasonably
Validated the EMG signal

(Kinesiological) EMG has good logical validity (i.e., it measures the presence/absence of striated muscle activity), but technical expertise is required. EMG recording has to be differentiated from artefacts, which is very straightforward for intramuscular recordings (particularly if also amplified as an acoustic signal). In surface recordings, the artefacts are much more difficult to sort out. With surface electrodes there may be a problem of changing contact quality, particularly with prolonged recordings, creating a variability of recording quality. For detection with surface EMG there are studies claiming sound reliability and clinical predictive validity for intra-anal electrodes (Glazer et al., 1999).

Content validity of kinesiological EMG recording implies a continuous recording from the same defined source; needle electrodes may become dislodged, therefore intramuscular wire electrodes are much more intrinsically reliable for long-term recordings. The content validity of recordings with surface-type electrodes depends on the type of electrode, and the possibility of their movement (displacement). A particular problem is content validity of repeated EMG recordings, which should sample the same source; this is intrinsically better for surface recordings, which are less selective. On the other hand, content validity of surface recordings may be questioned if the source of EMG activity is claimed to be only one of several muscles in the vicinity of the electrodes. With surface-type electrodes the overall anatomical source of the EMG signal in the pelvic region is often uncertain – is the EMG really derived only from the muscle that is claimed as the source? The other relevant issue is the question of representativeness – is the EMG signal really representative for the muscle or muscle group for which it is being claimed to be representative? In other words, for all electrode types, content validity needs to be established for the physiological relevance of the particular source recording. Thus, for example, anal sphincter recording may not be conclusive for urethral sphincter behaviour.

The kinesiological EMG (as obtained during polygraphic urodynamic recording) has accepted diagnostic validity to detect detrusor/striated sphincter dyssynergia, but this has not been formally much researched and probably holds true particularly for intramuscular recordings for the urethral sphincter. Indeed, the test has not yet been standardized. Its sensitivity and specificity are not known, but are far from ideal.

The logical and content validity of CN EMG to diagnose muscle reinnervation is good, but is usually not discussed in these terms. The diagnostic validity of CN EMG to detect striated muscle denervation and reinnervation is generally accepted. CN EMG sensitivity and specificity to detect moderate to severe denervation and reinnervation is accepted as good. These statements are supported by a large body of experience with nerve and muscle lesions as defined clinically, electrophysiologically and histopathologically (see Aminoff, 2012). The sensitivity and specificity to diagnose changes of reinnervation may vary for different types of CN EMG signal analysis (Podnar et al., 2002b).

SFEMG has good logical and content validity, and good diagnostic validity to detect changes due to reinnervation, but does not seem to be used clinically for PFM.

Responsiveness

A technically good EMG recording is capable of demonstrating absence of electrical activity in a nonactive muscle (‘electrical silence’) and a graded response to increasing muscle activation. There is no difficulty in detecting even small differences in EMG activity from a given source with a good (technically reliable) technique.

Reliability

The consistency and reproducibility (Engstrom and Olney, 1992) of results of diagnostic EMG (quantitative techniques using concentric and single fibre needle electrodes) is accepted as good if performed by experienced testers (see Aminoff, 2012). Extensive experience is needed for either method, possibly even more for CN EMG.

Straightforward parameters from surface EMG recordings (presence/absence of muscle activation) are much easier to interpret than CN EMG, and the consistency and reproducibility of such recordings (if technical issues are solved) are good. The overall consistency and reproducibility of results of the kinesiological EMG as a tool to investigate physiology lies more with the reproducibility of the ‘physiology’ that is being assessed (i.e., reproducibility of muscle ‘behaviour’).

USE OF KINESIOLOGICAL EMG AND CN EMG IN PARTICULAR PATIENT GROUPS

Kinesiological EMG recordings of sphincter and PFM are used in research, and diagnostically in selected patients with voiding dysfunction to ascertain striated muscle behaviour during bladder filling and voiding, and in
selected patients with anorectal dysfunction. The method
is not standardized.

The demonstration of voluntary and reflex activation of
PFM is indirect proof of the integrity of respective (central
and peripheral) neural pathways. The demonstration of
a normal PFM behaviour pattern (i.e., striated sphincter
nonactivity during voiding) is indirect proof of integrity
of the relevant central nervous system centres for lower uri-

nary tract neural control.

Kinesiological EMG as a tool (if a sound technique is
used) is not controversial, but there is little knowledge
on behavioural patterns of PFM in health and disease.
Therefore, short intervals of EMG in a particular patient
may be misinterpreted as indicating significant pathology,
whereas it only may represent normal variability of muscle
behaviour or some nonspecific muscle response to the ex-
perimental setting. Kinesiological EMG is also used as a
therapeutic tool in biofeedback.

CN EMG is performed particularly in neurologi-
cal, neurosurgical and orthopaedic patients with (sus-
pected) lesions to the conus, cauda equina, the sacral
plexus or the pudendal nerve, and only rarely in uro-
logical, urogynaecological and proctological patients
with suspected ‘neurogenic’ uro-ano-genital dysfunc-
tion (Vodušek, 2011).

Pelvic floor muscle denervation has been implicated
in the pathophysiology of genuine stress incontinence
(Snooks et al., 1984) and genitourinary prolapse (Smith
et al., 1989); different EMG techniques have been used in
research to identify sphincter injury after childbirth. The
usefulness of CN EMG in routine investigation of women
after vaginal delivery and/or with urinary incontinence
is, however, minimal and seems to be restricted in prac-
tice to the rare cases of severe sacral plexus involvement
(Vodušek, 2002).

Isolated urinary retention in young women was tradi-
tionally thought to be due either to multiple scler-
osis or psychogenic factors (Siroky and Krane, 1991).
Profuse complex repetitive discharges and ‘decelerat-
ing burst activity’ in the urethral sphincter muscle have,
however, been described by CN EMG in such patients
(Fowler et al., 1988). It was proposed that this pathologi-
cal spontaneous activity leads to sphincter contraction,
which endures during micturition and causes obstruction
to flow (Deindl et al., 1994). The syndrome was asso-
ciated with polycystic ovaries (Fowler and Kirby, 1986)
and is now referred to as Fowler’s syndrome. Because CN
EMG will detect both changes of denervation and rein-
nervation that occur with a cauda equina lesion, as well
as abnormal spontaneous activity, it has been argued that
this test is mandatory in women with urinary retention
(Fowler et al., 1988). The specificity of CN EMG path-
ological changes in women in retention has, however,
been questioned.

Pelvic floor muscle denervation has been implicated in
the pathophysiology of anorectal dysfunctions; but EMG
in routine investigation of anorectal dysfunction is also
limited to selected patient groups (Vodušek and Enck,
2006).

The CN EMG electrode can be employed at the same
diagnostic session for recording motor evoked responses
and/or reflex responses for a more comprehensive evalu-
aton of the nervous system (Vodušek and Fowler, 2004;

In conclusion, both ‘kinesiological’ and ‘motor unit’
EMG have contributed significantly to our understanding
of pelvic floor, lower urinary tract, anorectal and sexual
function in health and disease, but there is still much re-
search to be done.

EMG is helpful in diagnosing selected patients with sus-
ppected neurogenic PFM dysfunction, either to demon-
strate dysfunction of detrusor–sphincter coordination (kine-
siological EMG) or to prove denervation/reinnervation in
striated PFM and sphincters. In the case of mild to moder-
ate partial denervation EMG is very limited in providing
data on muscle strength (which is logically impaired due
to denervation).

**CLINICAL RECOMMENDATIONS**

- Any EMG method should be, as a rule, used by
  properly trained examiners. Even surface EMG
  recordings – noninvasive and apparently easy
to use – need training and experience. There are
many technical pitfalls, and not all are apparent
to the unsuspicious and uninformed, untrained
examiner. Close collaboration of PTs with
neurologists and clinical neurophysiologists is
recommended.
  - At present, the only widely accepted diagnostic
use of kinesiological EMG is to diagnose detrusor–
striated sphincter dyssynergia. Any other use of
kinesiological EMG recordings should follow
a protocol, after determining the validity and
reliability of the recordings, and minutely
describing all aspects of the technique in
publications. Standardization of the
method in different settings should be
strived for.
  - CN EMG is the electrophysiologic method of choice
in the routine examination of skeletal muscle
suspected to be denervated/reinnervated. CN EMG
of PFM and sphincter muscles is an optional method
in incontinent patients with suspected peripheral
nervous system involvement. Extrapolation from
EMG data to muscle strength, power and endurance
should be undertaken cautiously.
  - CN EMG should be performed only by properly
trained examiners who are licensed by the relevant
national authority.
REFERENCES


5.4 Vaginal squeeze pressure measurement

Kari Bø

Measurement of squeeze pressure is the most commonly used method to measure pelvic floor muscle (PFM) maximum strength and endurance. The patient is asked to contract the PFM either as hard as possible – maximum voluntary contraction (MVC) or maximum strength – to sustain a contraction (endurance), or repeat as many contractions as possible (endurance). The measurement can be done either in the urethra, vagina or rectum.

Kegel (1948) developed a vaginal pressure device connected to a manometer (named the perineometer) showing the pressure in millimetres of mercury as a measure of PFM strength. He did not report any data about responsiveness, reliability or validity for his method. The term ‘perineometer’ is somewhat misleading because the pressure-sensitive region of the probe of the manometer is not placed at the perineum, but in the vagina at the level of the levator ani. Currently several types of vaginal pressure devices are available to measure vaginal squeeze pressure, all with different device sizes and technical parameters (Dougherty et al., 1986; Bø et al., 1990a; Laycock and Jerwood, 1994; Sigurdardottir et al., 2009) (Figs 5.4–5.6). The tools measure pressure in mmHg, cmH₂O or hPa.

RESPONSIVENESS

In most studies describing measurement tools, data on responsiveness are not reported. However, in a newer type of apparatus, a specialized balloon catheter connected to a fibroptic microtip and strain gauge pressure transducer has shown high responsiveness (Kvarstein et al., 1983; Abrams et al., 1986; Dougherty et al., 1986; Svenningsen and Jensen, 1986; Bø et al., 1990a). In the apparatus of Bø et al. (1990a) (Camtech AS, Sandvika, Norway), the transducer’s measurement range is 0–400 cmH₂O, with linearity of 0.5–1%, hysteresis less than 0.5%, thermal baseline drift less than 0.5% (typically 0.2 cmH₂O per °C), and thermal sensitivity drift less than 0.1% per °C (Kvarstein et al., 1983; Svenningsen and Jensen, 1986).
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INTRA- AND INTER-TESTER RELIABILITY

Several authors (Dougherty et al., 1986; McKey and Dougherty, 1986; Bø et al., 1990a; Wilson et al., 1991; Frawley et al., 2006; Sigurdardottir et al., 2009) have shown that vaginal squeeze pressure can be measured with satisfactory reliability. However, Dougherty et al. (1991) reported a within-subjects mean of 15.5 mmHg (SD 3.9) and a between-subjects mean of 132.4 mmHg (SD 11.5) in healthy subjects with an age range from 19 to 61 years. A significant variation was confirmed by Bø et al. (1990a), who also showed that at the first attempt some women needed some time to find and recruit motor units, whereas other women fatigued, causing the strength to drop considerably after only a few attempts. However, comparing the results of the whole group of women on two different occasions 14 days apart, reproducible results were found. Wilson et al. (1991) also found a significant difference between first and last contractions. They did not find a significant difference between measurements obtained with a full or empty bladder or during the menstrual cycle. Dougherty et al. (1991) did not find a significant difference when muscle strength was measured on different days, at different times of the day, or during stress.

Kerschan-Schindl et al. (2002) tested intra-tester reliability of the Peritron perineometer and found that the absolute difference in maximal contraction force and mean contraction force within 5 s was less than 3.3 mmHg and 4.5 mmHg, respectively. Frawley et al. (2006) tested intra-tester reliability of the Peritron perineometer and found ICC (intra-class correlation) values for squeeze pressure readings to be 0.95, 0.91, 0.96 and 0.92 for crook lying, supine, sitting and standing positions, respectively. The ICC values for endurance testing in the same positions were much lower: 0.05, 0.42, 0.13 and 0.35. ICC values for resting pressure were 0.74, 0.77, 0.47 and 0.29. They concluded that there were high values of reliability of maximal voluntary contraction measured by the Peritron. However, endurance testing was unreliable, and so also was resting pressure in sitting and standing position. High intra-tester reliability of strength measurement was also confirmed by Chehrehrazi et al. (2009) while Rahmani and Mohseni-Bandpei (2011) found high intra-tester ICC values for both strength and endurance of the Peritron – 0.95 for strength and 0.94 for endurance – with somewhat lower values for between-day comparisons – 0.88 and 0.83, respectively. Sigurdardottir et al. (2009) found an inter-rater ICC value of 0.97, p <0.001 and a CV (coefficient of variation) of 11.1% testing MVC by the Myomed 932.

VALIDITY

Of the three pelvic canals, measurement within the urethra has the best face and content validity for measuring urethral closure pressure caused by the force of muscle contraction. This is where the increased pressure created by the PFM contraction is required to prevent urinary leakage. However, because of the risk of infection and the lack of availability of equipment in most physical therapy clinics, this method has mostly been used for research purposes (Benvenuti et al., 1987; Lose, 1992). Rectal pressure may not be a valid measure of the PFM in relation to urinary incontinence because it also includes contraction of the anal sphincter muscle. However, in men rectal pressure is the only practical option. In contrast to men, most women would have little sense of where the urethra is located, and most women probably would have the optimal sense of PFM contraction in the vagina. Therefore, vaginal squeeze pressure is the most commonly used method clinically.

PLACEMENT OF THE DEVICE

Size of the vaginal probe differs between devices. Some devices cover the full length of the vagina and placement of the probe is therefore not a problem. Using smaller devices (Dougherty et al., 1986; Bø et al., 1990a), location of the probe in the vagina creates both a reliability and validity problem because the balloon may be located outside the anatomical location of the PFM. The balloon or transducer has to be placed at the same anatomical level and at the level where the PFM are located. Kegel (1948, 1952) suggested that the PFM were located in the distal one-third of the vagina, and Bø (1992) found that most women had the highest pressure rise when the balloon was placed with the middle of the balloon 3.5 cm inside the introitus. However, individual differences were found.

SIZE AND SHAPES OF THE DEVICE

Results reported from different squeeze pressure and electromyography (EMG) apparatus cannot be compared due to differences in the diameter of the vaginal devices. There is discussion regarding the optimum diameter of vaginal devices (Schull et al., 2002). It is unknown whether a wide-diameter vaginal device stretches the PFM, inhibiting its activity or, conversely, increasing activity by providing firm proprioceptive feedback. In a study by Bø et al. (2005), measurement of PFM maximum strength was compared using two commonly used apparatuses with a different size of the vaginal probe. Significant differences were found, and it was concluded that measurements obtained with different methods cannot be compared.
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Squeeze pressure measurements obtained from all three canals can be invalid because an increase in abdominal pressure will increase the measured pressures. The PFM form one wall of the abdominopelvic cavity, and all rises in abdominal pressure will increase the pressure measured in the urethra, vagina and rectum.

Both Bø et al. (1988) and Bump et al. (1991) have shown that straining is a common error when women attempt to contract their PFM, and therefore an erroneous measurement can be registered. However, because a correct contraction involves an observable inward movement of the perineum or the instrument, and straining creates a downward movement, some authors (Bø et al., 1990b; Bø et al., 1991) have suggested that a valid measurement can be ensured by simultaneous observation of inward movement of the perineum.

Some researchers (Cammu and Van Nylen, 1998) have tried to avoid co-contraction of the abdominal muscles interfering with measurement of PFM strength by use of surface EMG on the rectus abdominis muscle to train subjects to relax their abdominal muscles or by simultaneous abdominal pressure measurement. Performance of a near-maximal PFM contraction is important to achieve the best training effect (Komi, 1992; Wilmore and Costill, 1999). Several researchers (Bø et al., 1990b; Dougherty et al., 1991; Sapsford et al., 2001; Neumann and Gill, 2002), however, have shown that there is a co-contraction of the deep abdominal muscles (lower transversus abdominis and internal oblique) during attempts at a correct, maximal contraction. Neumann and Gill (2002) also reported that during a maximum PFM contraction the mean abdominal pressure was 9 mmHg (range 2–19). The abdominal pressure rose to a mean of 27 mmHg (range 11–34) with a head and leg lift from supine while performing a PFM contraction, and 36 mmHg (range 33–52) during forced expiration and PFM contraction when supine, two activities that require diaphragmatic and outer abdominal muscles (external oblique and rectus abdominis) activity. A normal co-contraction of the lower abdominal wall, therefore, can be allowed because abdominal pressure rise is small with this co-contraction.

Dougherty et al. (1991) allowed an increase in abdominal pressure of 5 mmHg only, to ensure the least abdominal pressure interference with the measurement results. Bø et al. (1990b) standardized the testing by not allowing any movement of the pelvis during measurement. Further investigation is required to assess how subtle changes in postural activity might affect vaginal pressure measurements.

Contraction of other muscles, such as the hip adductor and external rotator muscles and gluteals, also alters intravaginal pressure measurement (Bø et al., 1990b; Peschers et al., 2001). Bø and Stien (1994) showed with concentric needle (CN) EMG in women without urinary incontinence that contraction of these other muscles increased muscle activity in both the striated urethral wall muscle and the PFM. However, when analysing the whole group of women, contraction of the other pelvic muscles did not give a higher pressure response than contraction of the PFM alone. Caution has to be exercised though, because for some individuals this may occur. Because the gross motor pattern of gluteal and adductor activity is not part of the normal neuromuscular action of the PFM and lower transversus abdominis synergy, co-contractions of the outer pelvic muscles are discouraged when measuring PFM action and strength.

Jean-Michel et al. (2010) developed an interesting new device; the Colpexin pull test. This instrument measures the pull-out resistance or force required to remove the sphere from the vagina during active contraction, which represents the strength of the PFM. This is a promising new device measuring eccentric contraction of the PFM with no influence of abdominal pressure. However, testing is required of responsiveness, reliability and validity.

Sensitivity and Specificity

Several case–control studies comparing PFM strength with vaginal squeeze pressure in continent and incontinent women have demonstrated that continent women have better strength than incontinent women (Hahn et al., 1996; Mørkved et al., 2004; Amaro et al., 2005; Thompson et al., 2006), and that there is an association between improvement in muscle function or strength and reduction in urinary incontinence (Bo, 2003). However, some studies have not found an association between increase in muscle strength and improvement in incontinence (Elser et al., 1999), which may be explained by the fact that there was no improvement in muscle strength following the low-dosage exercise protocol.

Conclusion

Because all increases in abdominal pressure will affect urethral, vaginal and rectal pressures, squeeze pressure cannot be used alone. With simultaneous observation of inward movement of the perineum, it is likely that a correct contraction is measured. Cautious teaching of the patient, standardization of instruction and motivation, and standardization of the patient’s position and performance are mandatory. If the aim is to measure the ability to close the urethra, urethral pressure should be measured. If overall PFM strength is the aim of the investigation, vaginal squeeze pressure (pressure manometry or dynamometric force) is preferred because this is the least invasive method with a low risk of infection in women.
CLINICAL RECOMMENDATIONS

Measurement of vaginal squeeze pressure is difficult, and clinical skills and experience are important factors in achieving reliable and valid results. The method has to be used with caution. However, when used in accordance with knowledge from research in this area, measurement of PFM contraction can give important information and feedback to both the patient and therapist (Fig. 5.7).

- Fully inform the patient about the test procedure and gain consent.
- Give the patient privacy to undress and a drape to place over herself on the examination couch.
- Always start the instruction with observation and palpation of PFM contraction.
- If the patient is unable to contract, strains or uses other muscles instead of the PFM, pressure measurement is not possible.
- The patient can be supine, crook lying, sitting or standing. Use the same position for each assessment for that patient.
- The PT should be in a position to be able to observe the perineum.
- Prepare the measuring device before washing hands and putting on examination gloves.
- Follow local infection control guidelines with regard to covering the probe or use a single-use apparatus.
- Gently insert the probe or ask the patient to do it.
- Once the probe is comfortably in place, instruct the patient to relax and breathe normally before the PFM contraction.
- Support the device to keep it in the same intravaginal position.
- Instruct the patient to contract the PFM as hard as possible with no visible co-contraction of hip adductor, gluteal or rectus abdominis muscles (pelvic tilt) and then to relax without pressing the perineum downwards.
- A small indrawing or ‘hollowing’ using internal abdominals with maximum contraction and no tilting of the pelvis is allowed.
- Resting pressure, holding time and repeated contractions can also be registered depending on the device parameters.
- Only contractions with simultaneous visible inward movement of the perineum or the measurement device can be considered valid measurements of PFM strength.
- Register at least three contractions and use the maximum or mean of the three contractions.
- Other aspects of muscle performance, such as holding time and number of repeated

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**Figure 5.7** Measurement of resting pressure, pelvic floor muscle maximal strength, attempts of holding, and repeated contractions at first time consultation in two nulliparous female sports students. Both were able to contract the PFM as assessed by vaginal palpation. The first had proven urodynamic stress urinary incontinence (SUI) with 43 g of leakage on ambulatory urodynamics. The second had no symptoms of pelvic floor dysfunction.
contractions (endurance), onset of contraction, slope and area under the curve, and resting pressure (relaxation) can be measured if the equipment used provides for this.

- Gently remove the probe, and either dispose of the intravaginal component or wash it according to local guidelines before sterilization.
- Allow the patient privacy to dress before discussing the results.

REFERENCES


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5.5 Pelvic floor dynamometry

Chantale Dumoulin, Mélanie Morin

INTRODUCTION

Precise, quantitative measurements of strength are critical in order to determine the clinical progression of neuromuscular weakness and to assess a patient’s response to an intervention aimed at increasing muscle strength. Dynamometers accurately, and independent of an evaluator’s subjective judgment, measure muscle function (i.e., strength, endurance, contraction speed and tone). Although widely used for more than 50 years by PTs to evaluate trunk as well as upper- and lower-extremity muscles (Bohannon, 1990), dynamometric technology has only been applied to pelvic floor muscle (PFM) assessment in the last two decades. To date, more than 12 dynamometers, of varying shapes and technical properties, have been described in the literature.

Caufriez (1993, 1998) and Rowe (1995) were the first to report the use of dynamometers to measure PFM function. However, these were only reported in non-peer-reviewed manuscripts (Caufriez, 1993, 1998) and in a brief conference abstract (Rowe, 1995). Caufriez’s dynamometer (known as the ‘pince tonimétrique’) was initially designed to assess PFM tone and consists of two speculum branches that can be opened, in an angular excursion to increase the vaginal aperture, by pressing on two handles. Rowe’s dynamometer comprises a probe with a movable rigid-window section against which the PFMs press during a contraction. The studies by Sampselle et al. (1998) and Howard et al. (2000) were the first to indicate the use of a pelvic floor dynamometer, the Michigan dynamometer, in clinical trials. A patent document published in 2002 described the dual-branch speculum used in these trials in more detail (Ashton-Miller et al., 2002).

In 2003, Dumoulin designed and developed the Montreal Dynamometric Speculum to measure anteroposterior PFM forces (Dumoulin et al., 2003). The speculum comprises two aluminium branches (Fig. 5.8): the upper branch is fixed and the lower one can be slowly adjusted through the turn of a screw. PFM forces are measured by two paired sets of strain gauges affixed to each side of the dynamometer’s lower branch. Since its initial design, several of the dynamometer’s properties have been improved upon: such as (1) the base supporting the speculum now enables insertion to follow the natural angle of the vagina (Morin et al., 2008b; Morin et al., 2010b); (2) the
mechanism that widens the vaginal opening was modified to create a smoother opening and a numerical linear-position transducer was incorporated to provide real-time measurement of the distance between both branches during a dynamic stretch (Morin et al., 2008b; Morin et al., 2010b); (3) a third branch, distally positioned in the vagina, was added to verify whether the recorded PFM forces are being minimally influenced by intra-abdominal pressure (Morin et al., 2006); and (4) the size of the branches was reduced to that of a paediatric spectrum to enable the assessment of women with vaginal atrophy and vulvovaginal pain (Morin et al., 2010a) (see Fig. 5.9).

A comprehensive assessment of PFM function can be undertaken with the Montreal Dynamometric Speculum, which measures PFM parameters such as strength, endurance, speed of contraction and tone (i.e., passive properties) during static and dynamic stretches.

Another PFM dynamometer was developed by Verelst and Leivseth (2004b). The uniqueness of this instrument lies in its ability to measure PFM forces in the transverse direction of the urogenital hiatus. The Verelst and Leivseth dynamometer comprises two semi-rounded parallel branches; one branch contains a metal plate to which a strain gauge is affixed. During a PFM contraction, the metal plate is deformed (depressed) and the resulting forces are measured. Both branches can be opened, enabling 30–50 mm measurements of the transverse opening. The sensor is also connected to a signal processing system.

The Kolpexin Pull Test dynamometer differs from the others in both shape and methodology. Developed by Guerette et al. (2004), it comprises a 36 mm Kolpexin ball (also referred to as a sphere), connected to a digital tensiometer, that is inserted into the vagina. The force required to remove the sphere from the vagina is recorded at rest and during a maximal PFM contraction.

In 2007, Constantinou and coworkers (Constantinou and Omata, 2007; Constantinou et al., 2007) designed a four-sensor probe. Each sensor is mounted on a leaf spring that, when inserted, can be expanded to make contact with the vaginal wall and then retracted for ease of removal. This configuration enables the assessment of the spatial distribution of forces as well as the positioning of the sensors in each quadrant (anterior, posterior, left and right) during a PFM contraction. A positioning system was added to the probe handle to track the orientation/angulation of the probe during a PFM assessment (Peng et al., 2007).

Similarly, Saleme et al. (2009) designed a multidirectional PFM measuring tool to evaluate the spatial distribution of PFM forces; however, the sensors are not mounted on extractible leaf springs but are mounted directly on the probe instead.

Recently, three additional dynamometric prototypes were developed; all of them use a conventional gynaecological two-branch speculum to which strain gauges are affixed and connected to a computer (Parezanovic-Ilic et al., 2009; Nunes et al., 2011; Romero-Culleres et al., 2013). The support system for Nunes’ speculum can be adjusted to evaluate the PFM forces in both the anteroposterior and transverse directions. Information about the two remaining prototypes is limited as the Parezanovic–Ilic speculum was described only in an article written in Serbian and the Romero-Culleres speculum has been presented only in a conference abstract (Romero-Culleres et al., 2013).

Lastly, the Elastometer, developed by Kruger et al. (2013), was designed to assess PFM passive forces in order to investigate their role in predicting delivery-related trauma. The device consists of a hand-held device comprising two aluminium branches with detachable acetylplastic speculum tips (Fig. 5.10). A load-cell amplifier and a displacement transducer have been integrated into the handpiece, providing force and branch-separation measurements to a central computerized unit. The Elastometer is innovative because the two branches can be separated by pressing a button that activates a motor incorporated into the speculum. This enables the evaluator to apply a controlled stretch, at a constant speed, to the PFMs in a transverse direction in order to assess PFM passive properties.
IN VITRO CALIBRATION STUDIES

Five investigators have reported in vitro calibration studies for their PFM dynamometers: the Rowe, Michigan, Montreal, Verelst and Saleme dynamometers (Rowe, 1995; Dumoulin et al., 2003; Verelst and Leivseth, 2004b; Miller et al., 2007; Saleme et al., 2009).

Rowe reported that his dynamometer exhibited good linearity with a quantification accuracy of 0.07 Newtons (N), a maximum experimental error of 0.3 N, and a minimum hysteresis (i.e., the area between the lengthening and the shortening curve) for a range of forces between −5 N and +5 N. Output was found to drift by less than 0.14 N in 2 hours of continuous service and to be repeatable within and between days as well as at both room and body temperatures (Rowe, 1995).

The calibration methodology for the Michigan dynamometer was reported by Miller et al. (2007). A sensitivity of 0.401 Volt (V)/N was found when five different loads were applied at two locations on the branches. The coefficient of determination for the linear regression line (R²) relating output voltage to input force was R² = 0.99, which indicates a good linearity. A thermal drift rate in the output of the force transducer of ±0.14 N/min was reported when the speculum was warmed from ambient room temperature to body temperature for 5 minutes.

The Montreal dynamometer was assessed for linearity, repeatability, ability to measure the resulting force independent of its point of application on the speculum branch, and hysteresis (Dumoulin et al., 2003). Linearity was excellent for a range of forces from 0 to 15 N, with regression coefficients close to unity (R² = 0.999). To evaluate the repeatability, the dynamometer was loaded using the same loading technique twice. Neither the slopes nor intercepts of the regression lines were significantly different between the two loading trials, indicating a high reliability for these in vitro measurements. To verify that force was being measured independent of the exact site of application on the lower branch of the speculum, successive loading using the same loading technique was done at distances of 2.5, 3.5 and 4.5 cm from the tip of the lower branch. The slopes and intercepts of the regression lines in the three loading trials were not significantly different, confirming that force measurement was independent of the force-application site. Finally, hysteresis was calculated by dividing the maximum difference in voltage output between two loading conditions by the maximum scale output recorded with the highest load. The device exhibited a minimal hysteresis of 0.00006%. Furthermore, the output for the strain gauges was found to drift by less than 0.003 N in 1 hour of continuous service at ambient temperature (Dumoulin et al., 2003).

Verelst and Leivseth (2004b) reported very good linearity for their dynamometer, up to 60 N with a nonlinearity quantification of ±2% of rate output at temperatures ranging between 15 and 50 °C and a resolution of 0.06 N.

For Saleme’s dynamometer probe (2009), the calibration process was performed by applying six load cycles (loads varied from 2 N to 45 N). The relationships between the voltage resistances and the forces were assessed using a second-order regression analysis; a coefficient of R² = 0.98 was reported.

No details regarding calibration techniques were provided for the other dynamometer publications.

TEST–RETEST RELIABILITY STUDIES

Six dynamometers (Verelst, Michigan, Montreal, Nunes and Romero-Culleres dynamometers as well as the Elastometer) have undergone test–retest reliability of their dynamometric measurements. PFM maximal strength was the most studied parameter (Dumoulin et al., 2004; Verelst and Leivseth, 2004b; Miller et al., 2007; Nunes et al., 2011; Romero-Culleres et al., 2013). In two studies of the Montreal dynamometer the assessment of PFM function was also extended to study the reliability of other parameters such as passive properties, contraction speed and endurance (Morin et al., 2007b; Morin et al., 2008b). The Elastometer reliability study only focused on an assessment of passive properties as this was the purpose for which it was specifically designed (Kruger et al., 2013).

The test–retest reliability of the Michigan dynamometer was studied on 12 nulliparous continent women (Miller et al., 2007). During each visit, maximal strength was assessed during two contraction trials for a total of three visits, each about 1 week apart. A coefficient of reliability (CR), defined as ±2 times the standard deviation of the difference, was calculated to assess reliability. The CR for the within-visit (i.e., between the two contractions) variability ranged from ±3.8 N to ±4.2 N; the highest was observed during the third visit (Table 5.1). To facilitate
Table 5.1 Reliability of PFM maximal strength measurements using different dynamometers

<table>
<thead>
<tr>
<th>Dynamometer</th>
<th>Sample</th>
<th>Method</th>
<th>Results</th>
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</thead>
<tbody>
<tr>
<td>Michigan (Miller et al., 2007)</td>
<td>12 continent, nulliparous</td>
<td>3 visits ±1 week apart, with 2 trials/visit</td>
<td>Within-visit variability&lt;br&gt;C: ±3.8 to ±4.2 N&lt;br&gt;Corresponding ICC for the lowest&lt;br&gt;C: 0.83, SEM: 0.86 (CV 13.9%)&lt;br&gt;Between-visit variability (test–retest)&lt;br&gt;C: ±5.5 N to ±8.2 N</td>
</tr>
<tr>
<td>Montreal (Dumoulin et al., 2004)</td>
<td>29 SUI, parous</td>
<td>Measurements taken at 19, 24 and 29 mm vaginal aperture (anteroposterior diameter)&lt;br&gt;3 visits ±4 week apart, 3 trials/aperture</td>
<td>Between-visit variability (test–retest)&lt;br&gt;19 mm: Φ 0.71, SEM 1.22 N (CV 30%)&lt;br&gt;24 mm: Φ 0.88, SEM 1.49 N (CV 21%)&lt;br&gt;29 mm: Φ 0.76, SEM 2.11 N (CV 24%)</td>
</tr>
<tr>
<td>Verelst (Verelst and Leivseth, 2004b)</td>
<td>20 continent, parous</td>
<td>Measurements at 30, 35, 40, 45 and 50 mm (transverse diameter). Repeated at 2- to 4-day intervals</td>
<td>No significance within-subject day-to-day variability&lt;br&gt;Most favourable opening: 40 mm&lt;br&gt;30 mm, CV 22%; 35 mm, CV 15%; 40 mm, CV 11%; 45 mm, CV 10%; 50 mm, CV 8%</td>
</tr>
<tr>
<td>Nunes (Nunes et al., 2011)</td>
<td>17 continent, nulliparous</td>
<td>Measurements taken at apertures measured 4.9 N of passive forces&lt;br&gt;3 visits ±3 weeks apart, 3 trials/visit</td>
<td>Between-visit variability (test–retest)&lt;br&gt;Anteroposterior ICC: 0.71–0.89, SEM: 1.96 N (CV 70.2%)&lt;br&gt;Transverse ICC: 0.46–0.72, SEM: 1.86 N (CV 44.29%)</td>
</tr>
<tr>
<td>Romero-Culleres (Romero-Culleres et al., 2013)</td>
<td>122 SUI</td>
<td>2 visits (the second visit implicated two different raters), 2 trials/visit</td>
<td>Within-visit variability (intra-rater reliability)&lt;br&gt;ICC: 0.93&lt;br&gt;Inter-rater variability&lt;br&gt;ICC: 0.92</td>
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</table>

CR, coefficient of repeatability; CV, coefficient of variation; ICC, intra-class correlation coefficient; SEM, standard error of mean; SUI, stress urinary incontinence.

comparisons to other studies, the authors also recalculated the reliability of this parameter using other statistics the result of which included an intra-class correlation coefficient (ICC) of 0.83, a standard error of mean (SEM) of 0.86, and a coefficient of variation (CV) of 13.9%. In terms of the between-visit reliability, the best was found between the second and third visits with a CR of ±5.5 N, which was based on the best within-visit trials per visit.

Three studies of the Montreal dynamometer were undertaken to evaluate the test–retest reliability of the dynamometric measurements of PFM strength, contraction speed, endurance and passive properties among women suffering from stress urinary incontinence (SUI) (Dumoulin et al., 2004; Morin et al., 2007b; Morin et al., 2008a). In the Dumoulin et al study, there were 29 primipara or multipara women between 23 and 42 years of age presenting different severity levels for SUI (Dumoulin et al., 2004). The PFM strength assessment was carried out during three visits separated by 4-week intervals. The participants were instructed to contract their PFMs as hard as they could for 10 seconds. Maximal strength values were recorded for three dynamometer openings: at a distance of 0.5, 1.0 and 1.5 cm between the two dynamometer branches. These speculum openings correspond to vaginal apertures (anteroposterior diameters) of 19, 24 and 29 mm, respectively. At each aperture, women had to perform three maximal PFM contractions. The generalizability theory (Shavelson, 1988) was applied to estimate the reliability of the PFM measurements. The reliability was quantified by the Index of Dependability (Φ) (a statistic similar to the ICC type 2), the corresponding Standard Error of Measurement (SEM) for the mean of three strength-measurement trials performed in one session, and the CV. Results are presented in Table 5.1 above. The largest coefficient of dependability, a value of 0.88, was obtained at the 24 mm aperture. The corresponding SEM reached 1.49 N (CV 21%) (Dumoulin et al., 2004).

In the Morin et al study (Morin et al., 2007b), 19 primipara or multipara women between 23 and 41 years of
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age with SUI participated in two sessions, at an 8-week interval, in which a single evaluator assessed their contraction speed and endurance. For the endurance test, the women were asked to maintain a maximal contraction for 90 seconds; the normalized area under the force curve was taken as the endurance parameter: [area under the curve/maximal strength]×100. For the contraction speed measurements, the women were instructed to contract their PFMs maximally and relax, repeating this sequence as fast as possible for 15 seconds. The contraction speed was quantified by the rate-of-force development of the first contraction and the number of contractions performed during the 15-second period. Similar to the Dumoulin et al study (2004), data reliability was evaluated using the generalizability theory. The normalized area under the force curve showed good reliability with a coefficient (Φ) of 0.81, a SEM of 298% and a CV of 11%. The range of observed coefficients of dependability for the contraction speed measurements, from 0.79 to 0.92, indicates a good to very good test–retest reliability. The associated SEMs for the rate-of-force development was 1.39 N/s (CV 22%) and 1.4 contractions (CV 11%).

A test–retest reliability study of the Montreal dynamometer to evaluate its ability to measure PFM passive properties was conducted by Morin et al. (2008b) using 32 postmenopausal women with SUI. It was conducted in two sessions separated by a 2-week interval. PFM passive properties were evaluated in the anteroposterior direction under four conditions. (1) PFM forces were recorded with the speculum closed, that is, at its minimal opening (passive resistance at minimal aperture). (2) PFM forces were recorded at the maximal vaginal aperture (passive resistance at maximal aperture); the maximal stretching amplitude was determined by either the participant’s tolerance limit or an increase in EMG activity as recorded by electrodes on the lower branch. (3) To assess PFM passives properties, the PFMs and surrounding tissues were stretched during five lengthening and shortening cycles conducted at a constant speed; passive forces and passive elastic stiffness (PES) (ΔF/Δaperture) were calculated at different vaginal apertures. The area between the lengthening and the shortening curve (hysteresis) was also computed. (4) The percent of passive-resistance loss after 1 minute of sustained stretching was calculated. The means for the two sessions were analysed for each parameter. The dependability index, SEM and CV results, presented in Table 5.2, indicate a good to excellent reliability for PFM passive properties, except for the measurement of forces at minimal aperture, which indicate lower reliability.

To assess their dynamometer, Verelst and Leivseth (2004b) completed a test–retest reliability study of PFM strength measurements in the transverse direction of the urogenital hiatus. Twenty healthy parous women with no history of urinary incontinence participated in this study. Dynamometric measurements were taken in the transverse plane at consecutively increasing diameters of 30, 35, 40, 45 and 50 mm and were repeated at 2- to 4-day intervals. The within-subject effect for day-to-day variability for all dynamometer openings was nonsignificant, indicating that the measurements were reliable. The 40 mm dynamometer opening being the most favourable based on the Bland–Altman plot. The CVs, presented in Table 5.2, indicate good reliability. Interestingly, both the Dumoulin et al. (2004) and Verelst and Leivseth (2004b) studies observed that the vaginal aperture influences the PFM strength assessment. Both found a PFM strength–length relationship, suggesting that an increase in vaginal aperture (length) resulted in higher strength measures.

The test–retest reliability of the Nunes speculum was tested on 17 continent nulliparous women during three sessions at 7-day intervals. Maximal strength was evaluated in both anteroposterior and transverse directions and three maximal contractions were performed during each visit. The speculum opening was defined as the point at which each woman attained 4.9 N for passive forces. The mean aperture of the device across the three visits was 20.60 mm (SD 1.78). ICC and SEM were used to assess the reliability. Similar to the Miller et al. (2007) study, the reliability values were found to be better between the second and third visits. ICC values ranging from 0.49 to 0.89 indicate an acceptable to excellent reliability, with the anteroposterior directions being the most reliable (Table 5.1).

The reliability of the Romero-Culleres dynamometer was evaluated in 122 women with SUI. To assess the intra-rater within-visit variability, PFM dynamometric strength was assessed during two PFM maximal contractions. Then, during a second visit, two evaluators performed the dynamometric assessment in order to assess the inter-rater reliability. The intra-rater and inter-rater reliability was evaluated using ICC; the results indicated good to excellent reliability (Table 5.2).

Kruger et al. (2013) recently published the Elastometer reliability results for PFM passive properties. Twelve continent women were tested twice, 3–5 days apart using the same protocol. They were encouraged to remain relaxed during the assessment of their PFM passive forces, which were taken during dynamic stretches. Data acquisition was automated with the device opening in 20 stepwise increments from 30 to 50 mm over a 60-second period. Three trials were performed, with the first discounted as a preconditioning and familiarization process. The between-visit reliability was found to be excellent, with an ICC of 0.92 and 0.86 for the second and third trials respectively (Table 5.2).

Overall, the test–retest reliability of dynamometric measurements has been extensively studied for various PFM functions and in different populations; the results suggest acceptable reliability hence their integration into and contribution to PFM rehabilitation programmes should be investigated. However, it should be pointed out that the inter-rater reliability of dynamometric measurements has only been evaluated once (by Romero-Culleres et al., 2013), and thus deserves further investigation.
<table>
<thead>
<tr>
<th>Dynamometer</th>
<th>Sample</th>
<th>Method</th>
<th>Results</th>
</tr>
</thead>
</table>
| Montreal (Morin et al., 2008b) | 32 SUI postmenopausal       | 2 visits ± 2 weeks apart, 2 trials/visit    | **Between-visit variability (test–retest)**  
1. Passive resistance at minimal aperture  
Φ 0.51, SEM 0.22 N (CV 87%)  
2. Passive resistance at maximal aperture  
Φ 0.82, SEM 0.57 N (CV 24%)  
3. Five lengthening and shortening cycles  
Forces at max aperture: Φ 0.85, SEM 0.67 N (CV 20%)  
PES at maximal aperture: Φ 0.75, SEM 0.10 N/mm (CV 23%)  
Forces at mid-point aperture: Φ 0.86, SEM 0.05 N (CV 15%)  
PES at common aperture (20 mm): Φ 0.93, SEM 0.03 N (CV 13%)  
Hysteresis: Φ 0.88, SEM 2.20 N/mm (CV 28%)  
4. Percentage of loss in passive force after a 1 m stretch: Φ 0.66, SEM 6.37% (CV 20%) |
| Elastometer (Kruger et al., 2013) | 12 continent                | 2 visits ± 3–4 days apart, 3 trials/visit   | **Between-visit variability (test–retest)**  
Trial 2 (visit 1 vs 2) ICC: 0.92  
Trial 3 (visit 1 vs 2) ICC: 0.86 |

PES, passive elastic stiffness; SUI, stress urinary incontinence.
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ACCESSION

Two studies (Dumoulin et al., 2004; Morin et al., 2010b) assessed the acceptance of the dynamometric procedures. Dumoulin and coworkers (2004) evaluated the acceptance of passive and active force measurements among young and middle-aged women during the course of their test–retest reliability study. The subjects’ unanimous appreciation for the Montreal dynamometer when asked to comment on the measurement procedures, implied that the instrument was acceptable and that the measuring procedures were not painful (Dumoulin et al., 2004). Morin et al. (2010b) also confirmed the acceptance of the procedures related to the passive properties assessment, including dynamic stretches (shortening cycles) among young and middle-aged continent women.

VALIDITY STUDIES

Validity studies for the PFM measurements were reported for only seven of the previously described dynamometers: the Montreal dynamometer (Dumoulin et al., 2003), the Kolpexin ball (Guertet et al., 2004), Saleme’s multidirectional PFM measuring tool (Saleme et al., 2009), Verelst’s intravaginal probe (Verelst and Leivseth, 2004b), Constantinou’s four-sensor probe (Constantinou and Omata, 2007), Nunes’ speculum (Nunes et al., 2011) and Miller’s instrumented speculum (Ashton-Miller et al., 2002).

Validity criterion

To date, there is no recognized gold standard for evaluating PFM function hence it is impossible to evaluate the validity criterion of individual dynamometric instruments. Consequently, validation of PFM dynamometers must rely on the construct validity, which Dunn (1989) has defined as the extent to which a test can be proven to measure a hypothetical construct, in this case, PFM function (Dunn, 1989). Various studies need to be performed to support the construct’s validity, specifically convergent validity (i.e., correlation with another instrument) and the Known Groups Method (Nunnally and Bernstein, 1994; Portney and Watkins, 2000).

Convergent validity

Two convergent validity studies were conducted for the Montreal dynamometer (Morin et al., 2004b; Morin et al., 2006). First, a study was carried out to correlate dynamometric measurements with a digital assessment of the pelvic floor (Morin et al., 2004b). Thirty continent women and 59 women with SUI, aged between 21 and 44, participated in the study. Spearman’s rho correlation coefficients were calculated to assess the correlation between the dynamometric measurements and the modified Oxford grading system (Laycock, 1992). Significant correlations were found between the two measurements with coefficients of $r = 0.727$, $r = 0.450$ and $r = 0.564$ for continent, incontinent and all women, respectively ($p < 0.01$).

Further, in another study by the same investigator, the point of maximum-force application on the lower branch of the Montreal dynamometer was recorded and correlated to the location of highest PFM EMG activity as recorded by a series of electrodes placed on the dynamometer (Morin et al., 2006). Ten women (PTs specialized in pelvic floor therapy) who had no symptoms of urinary incontinence were asked to complete a 10-second maximal contraction task; this participant group was specifically selected because they knew how to contract and isolate their PFMs. The location of the resulting PFM force corresponded to the highest EMG amplitude recorded by a pair of closely-located electrodes on the lower branch of the dynamometer, suggesting that the forces recorded by the Montreal dynamometer originate from the PFMs (Morin et al., 2006).

Finally, in the same 2006 study, a parallel convergent validity study of the Montreal dynamometer also evaluated to what extent intra-abdominal pressure did or did not influence dynamometric measurements during maximum contraction and Valsalva manoeuvre. The same 10 female PTs participated. With the participant supine, the dynamometer was inserted in the vagina and an intra-rectal balloon was positioned in the rectum in order to monitor changes in intra-abdominal pressure (IAP). Women were asked to complete a 10-second maximal contraction task, to do a 10-second Valsalva manoeuvre at 50 cmH$_2$O (as measured by the rectal balloon) and a 10-second Valsalva at 100 cmH$_2$O. To determine the influence of the IAP forces on the PFM strength measurements during PFM voluntary contractions and Valsalva, the forces recorded with the dynamometer were compared to those predicted by the pressure recorded by the intra-rectal balloon. For an 11.35 N PFM maximal contraction, 0.54 N was attributed to IAP, corresponding to 6.8% of the overall contraction force. During a Valsalva manoeuvre, up to 14.2% of the forces recorded by the dynamometer were attributable to IAP. Therefore, overall IAP influence on PFM strength measurements was considered small during a PFM voluntary contraction and Valsalva, again confirming the validity of the Montreal Dynamometric Speculum.

The convergent validity of the Kolpexin ball was evaluated through a comparison of its force measurement with
that of a digital PFM assessment using the Brink’s scale (Guerette et al., 2004). Twenty-one women with urinary incontinence or pelvic organ prolapse (POP) symptoms, ranging from 36 to 85 years of age, participated in the study. A 36 mm Kolpexin ball was inserted into the vagina above the levator plate and connected to a digital tensiometer/force gauge. The force required to remove the sphere was recorded during three resting trials and three PFM maximal contractions followed by a digital assessment using the Brink’s scale. There were positive correlations between the strength of the maximum contraction as measured by the Kolpexin ball and the digital PFM assessment (adjusted \( R^2 = 0.52; p < 0.001 \)), and the maximum force minus resting force as measured by the Kolpexin ball and the digital PFM assessment (adjusted \( R^2 = 0.54; p < 0.001 \)), indicating good convergent validity (Guerette et al., 2004).

A study of the Saleme multidirectional PFM strength measurement tool (Saleme et al., 2009), confirmed the positioning of the probe sensors at the level of the PFM mass. The study was conducted on one participant and visualized through a nuclear magnetic resonance imaging test (NMRI) during which a polymer model of the tool’s probe was inserted in the vaginal canal of the study participant. The NMRI revealed that there was an important muscular mass around the vaginal probe’s sensors, indicating that the device’s dimensions and sensors were correctly positioned to measure PFM strength, thereby collaborating convergent validity (Saleme et al., 2009).

**Known Groups Method**

This type of construct validity focuses on the ability of a new instrument to discriminate between groups that are known to be different (Dunn, 1989; Portney and Watkins, 1993). In other words, if a dynamometer proved to be capable of differentiating between the PFM function of continent and stress incontinent women or women with and without pelvic organ prolapse, this capacity would support its construct validity. The following section presents Known Groups Method studies for SUI vs continent women, POP vs controls and provoked vestibulodynia vs no pain controls.

**PFM functional differences between continent and incontinent women**

Four research groups presented data on continent and incontinent women. In the Morin study (Morin et al., 2004a), 30 continent women and 59 women with SUI, aged between 21 and 44, were recruited. A 20-minute pad test was performed to confirm continence in the asymptomatic women and to determine the severity of incontinence in the women who reported leakages. The Montreal dynamometer was used to assess the following static PFM parameters: (1) passive force; (2) maximal strength in a self-paced effort; (3) rate-of-force development (rapidity of contraction) and number of contractions during a protocol of rapidly-repeated 15-second contractions; and lastly, (4) absolute endurance recorded over a 90-second period during a sustained maximal contraction. An analysis of covariance was used to control for the confounding variables of age and parity during comparisons of PFM function in continent and incontinent women. The incontinent women demonstrated both lower passive force and absolute endurance than the continent women (\( p \leq 0.05 \)). In the protocol for rapidly-repeated contractions, the rate-of-force development and number of contractions were both lower among the SUI participants (\( p = 0.01 \)) (Morin et al., 2004a).

Further, in another study, Morin et al. (2007a) studied the involuntary PFM response during coughing between 31 continent women and 30 women with symptoms of SUI. The Montreal Dynamometric Speculum was inserted in the vaginal cavity to evaluate the involuntary PFM response during coughing. Participants were instructed to perform two maximal coughs. There was a significant difference, favouring continent women, for the maximal rate-of-force development (rapidity to contract prior to a cough) (\( p = 0.032 \)) and a strong tendency for PFM peak force prior to a cough.

Finally, Morin compared PFM passive properties in 34 postmenopausal continent and 34 SUI women (Morin et al., 2008a). Using the Montreal dynamometer, the PFM passive properties assessed included: (1) resting forces at a 15 mm vaginal aperture; (2) resting force at maximal aperture; (3) passive resistance at a 25 mm vaginal aperture; and (4) passive elastic stiffness (PES) during five lengthening and shortening cycles. SUI women demonstrated lower passive forces at minimal, mean and maximal apertures (\( p < 0.05 \)) as well as lower PES at maximal aperture (\( p = 0.038 \)). The lower initial passive resistance and higher contribution of passive forces to total voluntary strength in continent women support the role of passive properties in maintaining continence (Morin et al., 2008a).

In conclusion, these three studies with the Montreal dynamometer suggest that PFM dynamometric parameters differ between continent and incontinent women at rest, during maximal voluntary contraction and during a cough.

Verelst and Leivseth (2004b) used their intravaginal probe to determine whether there is a difference between continent and SUI women in terms of (1) PFM fatigue, (2) pre-activation times between pelvic floor and abdominal muscles during coughing and (3) PFM maximal contractile force. Twenty-six continent and 20 SUI parous women were examined. Fatigue was measured with the intravaginal device and the time-to-fatigue was defined as the time it took for a 10% decline in the initial maximal-reference force. Simultaneous recordings of the force development in the levator ani muscle and the electromyographic
activity in the external oblique abdominal muscles were undertaken to determine whether a PFM contraction precedes activity in the abdominal muscles during coughing. Time-to-fatigue was identical in the two groups (10.5 seconds in the continent and 11.5 seconds in the incontinent group). Only normalized force was significantly different between the groups, being lower in the incontinent group (p = 0.013). It is likely, according to the Verelst and Leivseth, that reduced normalized force, as found in the incontinent group, is an important contributing factor to urinary incontinence.

In a second study, Verelst and Leivseth (2007) compared passive and active forces as measured in continent and incontinent parous women. Twenty-four parous continent and 21 parous incontinent women were examined using their intravaginal device. Passive and active force/stiffness were measured by increasing the transverse diameter of the vagina. To allow a more accurate comparison between groups, measured forces were normalized with respect to bodyweight. No difference was found between the groups according to passive forces but active force was significantly higher (p = 0.030) in the continent group when normalized for body weight. Further, normalized active stiffness was significantly reduced in the incontinent group (p = 0.021). Both active force development and active stiffness in the PFM were significantly reduced in SUI women.

Peng (Peng et al., 2007; Shishido et al., 2008), using the Constantinou four-sensor probe, studied the vaginal pressure profile (VPP) along the vaginal wall between 23 continent women and 10 women with symptoms of SUI. The continent group had significantly greater maximum pressure than the stress SUI group on the posterior vaginal side at rest (mean 3.4 + 0.3 vs 2.01 + 0.36 N/cm²) and during PFM contraction (4.18 + 0.26 vs 2.25 + 0.41 N/cm²). The activity pressure difference between the posterior and anterior vaginal walls in the continent women’s group was significantly increased when the pelvic floor muscles contracted vs that at rest (3.29 + 0.21 vs 2.45 + 0.26 N/cm²). However, the change observed in the SUI group was not significant (1.85 + 0.38 vs 1.35 + 0.27 N/cm²). Voluntary PFM contraction imposes significant closure forces along the vaginal wall of continent women but not in SUI women and discriminates between these two groups.

Chamochumbi et al. (2012), using the Nunes speculum, studied active and passive PFM forces in 16 continent and 16 SUI middle-aged women. Evaluation of PFM passive and active forces were completed in the anteroposterior and left–right directions. The anteroposterior active strength was significantly higher in the continent women (0.3 + 0.2 N) compared to the SUI women (0.1 + 0.1 N). All other parameters did not differ between continent and SUI women, implying that SUI women had a lower anteroposterior active strength than continent women (Chamochumbi et al., 2012).

In conclusion, the ability of the Montreal dynamometer, the Verelst’s intravaginal probe, the Constantinou four sensor probe and the Nunes speculum to discriminate different aspects of PFM function between SUI and continent women confirmed further aspects of their construct validity.

**PFM functional differences between women with and without provoked vestibulodynia**

One study (Morin et al., 2010b) used dynamometry to compare 56 asymptomatic and 56 symptomatic women suffering from provoked vestibulodynia. Five parameters for PFM function were assessed using the Montreal Dynamometric Speculum: (1) passive force at minimal aperture; (2) passive force at maximal aperture; (3) maximal strength; (4) speed of maximal voluntary contraction; and (5) endurance. Women suffering from vestibulodynia demonstrated higher passives forces (also called tonicity/tension) at minimal aperture (p < 0.05). Resistance at maximal aperture was higher for the asymptomatic group because it was evaluated at larger apertures (p < 0.009). Women with vestibulodynia had significantly lower strength and endurance (p < 0.018). Moreover, they had less speed of contraction (p < 0.001). These results suggest that women with provoked vestibulodynia show diverse PFM dysfunctions compared to asymptomatic women, thereby confirming further aspects of construct validity.

**PFM functional differences between women with and without pelvic organ prolapse**

One study (Delancey et al., 2007) compared PFM function among women with and without pelvic organ prolapse (POP). PFM function was measured in a case–control study in which groups were matched for age, race and hysterectomy status among 151 women with prolapse (cases) and 135 with normal support (controls). Vaginal-closure force at rest and during a PFM maximal contraction was measured with an instrumented vaginal speculum (Ashton-Miller et al., 2002). The women with a prolapse generated less vaginal-closure force during a PFM contraction than the controls (2.0 N compared with 3.2 N, p < 0.001). These results suggest that women with POP show PFM dysfunction and confirm aspects of construct validity for the tool.

**PFM dynamometry in pelvic floor rehabilitation**

Previously in this section the use of PFM dynamometry to identify PFM dysfunction has been demonstrated in different patient populations (i.e., women with urinary
Evidence-Based Physical Therapy for the Pelvic Floor (Dumoulin et al., 2010) evaluated, in the secondary analysis of an RCT on PFM rehabilitation in young postpartum women, the relationship between pre-physical therapy PFM function and treatment outcome. The relationship between potential predictive-PFM-function variables, as measured by a PFM dynamometer, and physical therapy success was studied using forward stepwise multivariate logistic regression analysis. Forty-two women (74%) were classified as treatment successes; 15 (26%) were not. Treatment success was associated with lower pre-treatment PFM passive force and greater PFM endurance (p <0.05); although the association with the latter was barely statistically significant. These results, the first using dynamometry, contribute new information on predictors of success for an 8-week PFM physical therapy programme in women with persistent postpartum SUI. More research is needed in this area, using dynamometry, to identify for pre-treatment those women with PFM dysfunction (UI, POP or pain) who are more likely to respond to physical therapy.

CONCLUSION

The PFM dynamometers are reliable, valid and objective instruments that can directly measure PFM passive forces, active forces and forces during effort, such as a Valsalva manoeuvre or a cough. Different dynamometric units allow for the evaluation of PFM forces in different directions (anteroposteriorly and latero-laterally); at different vaginal apertures; during rest, maximal PFM contraction and effort; and using a multitude of parameters (strength, endurance, rapidity of contraction, etc.). Further, measurements can be taken in the supine position and, with some units, in sitting or standing positions.

Clearly, dynamometry has multiple clinical applications: first, it can inform clinicians about a patient’s specific PFM dysfunction in terms of passive strength, maximum strength, rapidity of contraction, coordination and endurance. These PFM dysfunctions need to be rehabilitated and their progress monitored. Second, although fairly recent and, as of yet, reported in only one trial, dynamometry has been used to predict treatment outcome.

Although dynamometer appears to be a highly promising tool for assessing PFM function, commercial units are not yet available. Further, although psychometric evaluations have progressed a lot over the past 10 years, in some cases (with some units) validation studies are still required. More importantly, the tasks used by researchers to measure PFM parameters and function must be standardized in order to advance our understanding of PFM dysfunction in different populations with different PFM dysfunctions. Moreover, normative data on PFM function is still relatively unknown and requires further study. In the future, dynamometers will enable PTs to tailor PFM training to an individual’s specific needs as well as monitor progress. Finally, further dynamometric study could lead to the development and validation of clinical prediction rules for PFM physical therapy. As not all women respond to treatment, being able to predict responsiveness to physical therapy could enable practitioners to determine which women are best suited for PFM physical therapy, thereby improving the efficiency of PFM rehabilitation. A comprehensive clinical prediction rule would allow women to be directed to their most effective treatment option – physical therapy, pharmacological or surgery – thereby saving money in lost time and ineffective treatments.

CLINICAL RECOMMENDATIONS
(based on the Montreal dynamometer)

- Explain the tool and the procedures to the patient.
- After the patient has undressed, ask her to adopt a supine position, hips and knees flexed and supported, feet flat on a treatment table.
- Prior to the insertion of the dynamometer’s probe/speculum, give detailed instructions about contracting the pelvic floor musculature using anatomical models, drawings or vaginal palpation.
- Prepare the dynamometer by covering each branch of the probe/speculum with a condom lubricated with a hypo-allergen gel.
- Bring the two branches of the measuring device to their minimum aperture and gently insert the dynamometer into the vaginal cavity in an anteroposterior axis to a depth of 5 cm.
- Gently separate the two branches to obtain the appropriate aperture.
- Allow some time for the woman to adjust to the sensation of the probe/speculum inside her vagina; time that can be used for practising the required manoeuvre before recording a task (e.g. PFM maximal contraction).
- For a PFM maximal contraction, ask the patient to breathe normally and then to squeeze and lift the pelvic floor musculature as if to prevent the escape of flatus and urine. Record the results.
- Give positive feedback while taking measurements such as strength, endurance and coordination during active tasks or encourage relaxation during passive force measurements.
- After the evaluation session, discard the condoms and disinfect the dynamometer.


5.6 Urethral pressure measurements

Mohammed Belal, Paul Abrams

Continence depends on the intramural and extramural forces that maintain urethral closure while the bladder is filling. Stress leakage may occur if the urethral resistance is overcome by abdominal forces, therefore resulting in a vesical pressure that is higher than urethral pressure (Barnes, 1961). An understanding of urethral function is vital in incontinence.

Urethral pressure measurements are a common method of measuring urethral function. They assess the ability of the urethra to prevent urinary incontinence. They can be measured at single points in the urethra or most commonly over the entire length of the urethra (urethral pressure profile). We begin with the definitions of urethral pressure parameters, followed by the different methods and techniques used to obtain urethral pressures. The advantages and disadvantages of the different methods and techniques will be discussed.

**DEFINITIONS**

Urethral pressure is defined as the fluid pressure needed to just open a closed (collapsed) urethra (Griffiths, 1985). This definition implies that the urethral pressure is similar to an ordinary fluid pressure (i.e., is a scalar [does not have a direction] quantity with a single value at each point along the length of the urethra; Lose et al., 2002).

From the definition, it is apparent that the introduction of catheters changes the properties of the closed urethra but the effect on the urethral pressure measurement was considered to be small (Griffiths, 1985). Urethral pressure measures the intra- and extramural forces that cause apposition of the urethral walls, and associated definitions are as follows (Fig. 5.11).

- **Urethral pressure profile (UPP)** is a graph indicating the intraluminal pressure along the length of the urethra.
- **Urethral closure pressure profile** is given by the subtraction of intravesical pressure from urethral pressure.
- **Maximum urethral pressure (MUP)** is the maximum pressure of the measured profile.
- **Maximum urethral closure pressure (MUCP)** is the maximum difference between the urethral pressure and the intravesical pressure. This is the reserve pressure of the urethra to prevent leakage. The calculation of MUCP ($p_{ucp}$) requires the simultaneous recording of both intrauurethral ($p_{ura}$) and intravesical ($p_{ves}$) pressure. The calculation is as follows: $p_{ucp} = p_{ura} - p_{ves}$.
- **Functional urethral length (FUL)** is the length of the urethra along which the urethral pressure exceeds intravesical pressure in women.

![Figure 5.11](image-url)
METHODS OF MEASURING URETHRAL PRESSURE PROFILOMETRY

There are currently three methods of measuring urethral pressure profilometry:

- fluid perfusion technique or the Brown Wickham technique (Brown and Wickham, 1969);
- microtip/fibreoptic catheters;
- balloon catheters.

A summary of the advantages and disadvantages of the different methods is shown in Table 5.3.

Fluid perfusion technique

The fluid perfusion technique measures the pressure needed to perfuse the catheter, which is withdrawn at a constant speed, at a constant rate. The constant rate of infusion is usually provided by a syringe driver. The measured quantity can be very close to the local urethral pressure, provided that the urethra is highly distensible (Griffiths, 1980). Several factors affect the technique, as discussed below.

Catheter size

Catheter sizes from 4- to 10-French gauge are satisfactory to use in the fluid perfusion technique (Harrison, 1976). Large-sized catheters give a falsely higher reading because they record urethral elasticity as well as the urethral closure pressure (Lose, 1992).

Catheter eyeholes

Two opposing side holes 5 cm from the tip of the catheter are satisfactory (Abrams et al., 1978). A larger number of holes does not improve accuracy. The orientation is not important.

Perfusion rate

A perfusion rate of 2–10 ml/min will give an accurate measurement of closure pressure (Abrams et al., 1978). A syringe driver is preferable to a peristaltic pump.

Catheter withdrawal speed

The catheter should preferably be withdrawn continuously with the optimal withdrawal speed of less than 7 mm/s (Hilton, 1982). The usual rate of withdrawal is between 1 and 5 mm/s.

Response time

Response time is dependent on the rate of perfusion and the rate of catheter withdrawal. The perfusion method is able to record a maximum rate of change between 34 and 50 cm H\textsubscript{2}O/s.

Microtip/fibreoptic catheters

The microtransducer catheters have the ability to measure rapid changes in pressure. However, they appear to have several disadvantages. First there is a significant degree of positional dependence (Hilton and Stanton, 1983a). For example, if the catheter microtransducer is facing anteriorly, then the MUP is greater and FUL shorter than posteriorly (Abrams et al., 1978). Secondly, bending in urethral wall tissue may lead to a superimposition of local urethral tissue and transducer interactions on the urethral pressure: this requires the catheters to be very flexible. If used, it is recommended that the transducer faces laterally (Anderson et al., 1983).

Balloon catheters

The advantages of balloon catheters in measuring urethral pressures are that they avoid orientation dependence. However, in the past technical problems meant that the

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<th>Table 5.3 Advantages and disadvantages of the different methods of measuring urethral pressures</th>
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<td><strong>Fluid perfusion technique</strong></td>
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balloons were too large, causing a dilatation effect on the urethra. This results in an overestimation of the urethral pressure. Additionally the length of the balloon is also important. If the balloon is too long this averages out the pressure variations along the length of the urethra. Recent balloon catheters have overcome these difficulties (Pollak et al., 2004).

**FACTORS AFFECTING MAXIMUM URETHRAL CLOSURE PRESSURES**

Urethral pressure measurement can be carried out at different bladder volumes and in different subject positions at rest, during coughing or straining, and during voiding.

**Bladder volume**

Urethral closure pressure measurement in women depends on bladder volume. In continent women the urethral closure pressure increases with increasing volume. However, in women with stress incontinence it tends to decrease with increasing volume (Awad et al., 1978).

**Patient position**

Position also has an effect on urethral closure pressure: continent women show an increase in urethral closure pressure on standing whereas women with stress incontinence show a decrease in pressure on standing (Henriksson et al., 1977; Hendriksson et al., 1979). However, there is poor reproducibility of the urethral closure pressure in the standing position, thus limiting clinical use (Dorflinger et al., 2002).

**Pelvic floor activity**

Pelvic floor muscle (PFM) activity is always active except before and during voiding. However, a failure of relaxation of the voluntary pelvic floor contraction increases urethral closure pressure. This can usually be overcome by repeating the urethral pressure profilometry twice more or until a reproducible pattern is obtained, and if need be, over a longer period of time. Conversely, the effect of pelvic floor activity on the urethra can be assessed during measurement of urethral pressure. The catheter is placed at the point of MUP and the patient is asked to contract the pelvic floor voluntarily as if trying to stop themselves from passing urine. In normal women an increment above the MUCP is seen. A value of less than 10 cmH₂O above the MUCP denotes a poor pelvic floor squeeze in the fluid perfusion technique (Table 5.4).

The variation of urethral closure pressures depends on the method used and the position, and to facilitate reliable recordings, recommendations on the standardization of urethral pressure measurements have been made. Below are some of the recommendations of the International Continence Society (ICS) sub-committee on the standardization of urethral pressure measurements (Lose et al., 2002).

**STANDARDIZATION OF URETHRAL PRESSURE MEASUREMENTS**

The investigator is asked to specify:
1. type of measurement (point–profilometry–ambulatory);
2. period of time over which the measurement was recorded;
3. constant (given by the probe) or variable cross-sectional area of the urethra (i.e., inflation of a balloon);
4. patient position;
5. bladder volume;
6. manoeuvres (coughing, Valsalva, other);
7. withdrawal speed (for profilometry);
8. infusion medium and rate of infusion (for fluid-perfused catheters);
9. type of catheter;

| Table 5.4 Comparison of clinical PFM strength assessment by experienced clinicians and urethral pressure assessment (fluid perfusion technique) |
|-------------------------------------------------|-------------|-------------|-------------|
| **PFM strength as assessed by urologist/urogynaecologist** | Normal | Reduced | Absent |
| Number of patients | 2757 | 3399 | 485 |
| Urethral pressure assessment (cmH₂O) | 18.1 | 8.8 | 3.6 |
| (SD) | (15.5) | (11.1) | (7.5) |

A large series from the Bristol Urological Institute over a period of 15 years. PFM, pelvic floor muscle.
10. size of catheter;
11. catheter material – flexibility;
12. orientation of a directional sensor;
13. sensor position fixation (for point pressures or during coughing/straining);
14. zeroing of pressure sensors:
   - external transducers (and fluid-filled catheters) – superior edge of the symphysis pubis (piezometric) for pressure reference height; to correct for viscous pressure losses within the catheter, zero of pressure should be set as the reading in air when the fluid is flowing (zero reference point is atmospheric pressure)
   - microtip transducers calibrated to atmospheric pressure, but no pressure reference height is needed for catheter-mounted transducers; when calculating closure pressure using multisensor microtips, any difference in vertical height between the ‘bladder’ transducer and urethral transducer(s) should be taken into account;
15. recording apparatus:
   - describe type of recording apparatus – the frequency response of the total system should be stated; equipment with a sampling rate of 18 Hz can satisfactorily record cough-produced pressure changes in the urethra (Thind et al., 1994).

**NORMAL URETHRAL PRESSURE PROFILES**

There are sex differences between men and women in the range of normal urethral pressure values. In men, MUP does not significantly decrease with age (Abrams, 1997), whereas in women, after the menopause, MUP decreases. Prostatic length tends to increase with age in men; however, urethral length tends to decrease in women. A rough guide to MUP in women is a value of 92 minus age (cmH2O), using values obtained from the fluid perfusion technique (Edwards and Malvern, 1974).

**Urethral pressure profile shape**

**Men**

Certain features are seen in the male UPP; there are two peaks – the presphincteric peak followed by the prostatic plateau and then the sphincteric peak (Fig. 5.12). Abnormalities in the presphincteric prostatic plateau can be due to bladder neck hypertrophy or prostatic enlargement. The sphincteric peak in men can be too high, as seen in some neurogenic patients, or too low in male patients with iatrogenic causes of stress incontinence, such as after prostate surgery.

**Women**

The female urethral pressure profile tends to be symmetrical in shape, as seen in Figure 5.11.

Normal and abnormal urethral pressure profiles are shown in Figures 5.13 and 5.14, respectively. Women can also have low or high urethral pressures. A high urethral pressure sometimes denotes Fowler’s syndrome, a condition in which idiopathic sphincter overactivity causes voiding difficulties (Fowler et al., 1988). A low urethral pressure may denote intrinsic sphincter deficiency, which usually results from childbirth and may lead to stress urinary incontinence (SUI). A biphasic pressure wave may suggest a urethral diverticulum.

The measurement of resting UPPs has several uses:
- In post-prostatectomy incontinence; there is a close association between sphincter damage and a reduction in the MUCP (Hammerer and Huland, 1997).
- There is some evidence that a low MUCP is associated with a poor outcome for surgery in women for SUI (Hilton and Stanton, 1983b).
- Urethral pressure measurements may provide an answer to unexplained incontinence in women.
- When considering patients for urinary diversion surgery the MUCP gives an indication as to whether an artificial sphincter is necessary. An MUCP greater than 50 cmH2O would not require a sphincter if a good-volume, low-pressure reservoir is created (Abrams, 1997).

**Urethral pressure profile and incontinence surgery**

Several studies have suggested that female patients with a low urethral closure pressure and urethral length have a worse outcome after incontinence surgery (Bhatia and Ostergard, 1982; Hilton and Stanton, 1983b; Hilton, 1989). Some have not shown any difference (Sand et al., 2000; Harris et al., 2011).
Figure 5.13 Normal urethral pressures in women: the diagram shows two urethral pressure profiles (UPPs) with the shorter higher peak being the artefact recorded when the catheter is passed though the sphincter area to perform the second UPP seen on the right. Pucp, urethral closure pressure; Pura, intraurethral pressure; Pves, intravesical pressure.

Figure 5.14 Reduced urethral pressures: two urethral pressure profiles are recorded with a short artefact between them (see Fig. 5.13). Pucp, urethral closure pressure; Pura, intraurethral pressure; Pves, intravesical pressure.)
RESTING URETHRAL PRESSURE PROFILES

Responsiveness
The microtip catheters have a high frequency response of over 2000 Hz, which is more than adequate to record physiological events in the lower urinary tract. The fluid perfusion technique has a reduced responsiveness in comparison.

Reliability
The reproducibility and repeatability of the fluid perfusion technique and the microtip catheter have been shown to be reasonable (Abrams et al., 1978; Hilton, 1982; Wang and Chen, 2002). The standard deviation of measurements made on a single occasion using the fluid perfusion technique and the microtip catheter is shown to be approximately 5 and 3 cmH\textsubscript{2}O, respectively (Abrams et al., 1978; Hilton and Stanton, 1983a). The inter-test measurements show a standard deviation of 3.5–5 cmH\textsubscript{2}O depending on menstrual status (van Geelen et al., 1981; Hilton, 1982). Recently, balloon catheters have shown reasonable correlations with microtip catheters (Pollak et al., 2004).

Validity
The validity of the measurements of resting urethral pressure profilometry depends on the technique used. The measured quantity can be very close to the local urethral pressure, provided that the urethra is highly distensible (Griffiths, 1980) in the case of the fluid perfusion technique. Microtip catheters measure the stress of the urethral wall, not the pressure. The validity of urethral pressure measurements in assessing PFM strength is high.

Sensitivity and specificity
Female patients with SUI generally have significantly lower mean values of MUP than are seen in continent women (Hilton and Stanton, 1983a). The MUP is lowest in those women (and men) with increasingly severe SUI. However, there is a large overlap between the MUPs of normal and incontinent patients. Therefore urethral pressure profilometry does not have the diagnostic accuracy for SUI to be used alone (Versi, 1990).

STRESS URETHRAL PRESSURE PROFILES

This method assesses the pressure transmission from the abdominal cavity to the proximal urethra. Decreased conductance of abdominal pressure is associated with stress incontinence. Essentially a UPP is performed, preferably with a microtip catheter with the patient coughing. If the urethral closure pressures become negative on coughing, then leakage is likely and represents a positive test. Ideally the stress UPP should be carried out in the erect position with a full bladder. This poses practical issues. A lack of specificity with the test has limited its use (Versi, 1990). Stress UPPs are no longer used regularly in clinical practice.

URETHRAL REFLECTOMETRY

There is some interest in urethral reflectometry. The technique involves placing a thin polyurethane bag in the urethra. A pump applies preselected pressures stepwise to the bag. For every step the cross-sectional area is measured by acoustic reflectometry. Measurements are made both during inflation and deflation (Klarskov and Lose, 2007a). The initial results are comparable to urethral pressure profilometry (Klarskov and Lose, 2007b). However, to date all publications come from a single centre.

CONCLUSION

Resting urethral pressure measurements can be made using several techniques and the results are influenced by the technique used and biological factors. Urethral pressure measurements are static measurements that do not reflect the forces exerted on the urethra at leakage. Increases in abdominal pressure compress the urethra and result in reflex activation of the peri-urethral muscles, which are not assessed by resting urethral pressures.

CLINICAL RECOMMENDATIONS

- Urethral pressure measurements should be performed under the supervision of the urologist or urogynaecologist.
- The urologist or urogynaecologist should refer to the ICS standardization report on urethral pressure measurements.
- An understanding of the limitations of the different urethral pressure measurements is required before embarking on research.
- Multidisciplinary collaboration is required when PTs perform research in this area with the urologist or urogynaecologist providing the urethral pressure measurements.
- Urethral pressure measurements, if performed correctly, are good valid measurements for assessing PFM strength.
References


Ultrasound in the assessment of PFM and pelvic organ descent

Hans Peter Dietz

INTRODUCTION

Ultrasound is increasingly used for the morphological and functional assessment of the muscles of the pelvic floor. Recent developments have greatly simplified the direct demonstration of the inferior parts of the levator ani by ultrasound. The advent of 3D ultrasound has given us access to the axial plane while using noninvasive techniques. 4D ultrasound allows real-time imaging of the effect of manoeuvres such as cough, Valsalva and pelvic floor muscle contraction (PFMC), in any arbitrarily defined plane (Dietz, 2004b). Most recently, modern image processing techniques, both on- and offline, have enabled us to reach resolutions equivalent to magnetic resonance imaging in all three dimensions for most of the structures we are interested in, while delivering temporal resolution far above anything possible on MRI today.

This discussion will be limited to translabial or transperineal ultrasound, the only sonographic imaging modality to allow direct assessment of both levator structure and function. While transabdominal ultrasound has been used to describe levator activity (Thompson and O’Sullivan, 2003), such an assessment is necessarily indirect and very limited. Endovaginal imaging can demonstrate static anatomy and biometric measures, but the presence of an instrument within the vagina severely limits the assessment of function via manoeuvres such as Valsalva and pelvic floor muscle contraction.

TECHNIQUE

Translabil or perineal ultrasound (Koelbl and Hanzal, 1995; Schaer, 1997; Dietz, 2004a) is performed by placing a transducer (usually a 3.5–5, 4–8 or 6–9 MHz curved array) on the perineum (Fig. 5.15), after covering the instrument with a glove or thin plastic wrap for hygiene reasons. Powdered gloves can markedly impair imaging quality due to reverberations and should be avoided. Imaging can be performed in dorsal lithotomy, with the hips flexed and slightly abducted, or in the standing position. Bladder filling should be specified; for some applications prior voiding is preferable. The presence of a full rectum may impair diagnostic accuracy and sometimes requires repeat assessment after defecation. Parting of the labia can improve image quality. The transducer can generally be placed quite firmly against the symphysis pubis and the perineum without causing significant discomfort, unless there is marked atrophy. The resulting image includes the symphysis anteriorly, the urethra and bladder neck, the vagina, cervix, rectum and anal canal (see Fig. 5.16). Posterior to the anorectal junction a hyperechogenic area indicates the central portion of the levator plate, i.e., the puborectalis/pubococcygeus or pubovisceral muscle. The cul de sac may also be seen, filled with a small amount of fluid, echogenic fat or peristalsizing small bowel. Parasagittal or transverse views may yield additional information, e.g. enabling assessment of the puborectalis muscle and its insertion on the inferior pubic ramus.

Figure 5.15 Transducer placement (A) and field of vision (B) for translabial/perineal ultrasound, midsagittal plane.
From Dietz 2010 with permission.
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Chapter 5

Measurement of pelvic floor muscle function and strength, and pelvic organ prolapse

**BLADDER NECK POSITION AND MOBILITY**

Bladder neck position and mobility can be assessed with a high degree of reliability. Intra- and inter-observer variability have been published, with a test–retest series on 50 young nulliparous women seen after a minimum interval of 4 weeks showing an intraclass correlation of 0.77 (Dietz *et al.*, 2005a). In order to obtain valid and reproducible results, however, it is essential to ensure an adequate Valsalva manoeuvre, which ought to be sustained for at least 5 seconds (Orejuela *et al.*, 2012). The patient has to be coached to breathe in, hold her breath, and ‘push as if you had to push a baby out’ or ‘push as if you had to pass a hard motion’ in order to achieve adequate abdominal pressures. At the same time, one should ensure that the patient does not produce a concomitant levator contraction which will result in artificially low values for pelvic organ descent. This is most common in young women with good pelvic floor muscle function (Oerno and Dietz, 2007) and is evident as a reduction in the anteroposterior diameter of the levator hiatus, and as a posterior displacement of the prepubic subcutaneous tissues, seen inferior or caudal to the inferior surface of the symphysis pubis, due to contraction of the superficial perineal muscles. Pressure on the transducer has to be avoided during a Valsalva manoeuvre in order to allow full descent of pelvic organs.

Points of reference are the central axis of the symphysis pubis (Schaer, 1997) or its inferoposterior margin (Dietz, 2004c). The former may potentially be more accurate as measurements are independent of transducer position or movement; however, due to calcification of the interpubic disc the central axis is often difficult to obtain in older women, reducing accuracy. There have been no comparative studies on repeatability of measurements to date, however.

Measurements of bladder neck position are generally performed at rest and on maximal Valsalva manoeuvre. The difference yields a numerical value for bladder neck descent (see Fig. 5.16). On Valsalva, the proximal urethra is usually seen to rotate in a posteroinferior direction. The extent of rotation can be measured by comparing the angle of inclination between the proximal urethra and any other fixed axis. This is often accompanied by an opening of the retrovesical angle (see Fig. 5.16), although none of those changes in functional anatomy are diagnostic of urodynamic stress incontinence (Nazemian *et al.*, 2013).

Figure 5.16 illustrates how pelvic floor ultrasound can be used to quantify descent not just of the bladder neck and urethra, but also of the most dependent part of a cystocele, central and posterior compartment (Dietz *et al.*, 2001a).

The aetiology of increased bladder neck descent is likely to be multifactorial. The wide range of values obtained in young nulliparous women suggests a congenital component, and a twin study has confirmed a high degree of

![Figure 5.16 Determination of bladder neck descent and retrovesical angle: Ultrasound images show the midsagittal plane at rest (panels A, C) and on Valsalva (panels B, D). S = symphysis pubis; U = urethra; B = bladder; Ut = uterus; V = vagina; A = anal canal; R = rectal ampulla; L = levator ani. The lower images demonstrate the measurement of distances between inferior symphyseal margin and bladder neck (vertical, x; horizontal, y), and the retrovesical angle at rest (rva-r) and on Valsalva (rva-s).](image-url)
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heritability for anterior vaginal wall mobility (Dietz et al., 2005a). Vaginal childbirth (Peschers et al., 1996; Meyer et al., 1998; Dietz and Bennett, 2003) is probably the most significant environmental factor, with a long second stage of labour and vaginal operative delivery being associated with increased postpartum descent (Dietz and Bennett, 2003). This association between increased bladder descent and vaginal parity is also evident in older women with symptoms of pelvic floor dysfunction (Dietz et al., 2002a). While the pelvic floor is undoubtedly affected by pregnancy and childbirth, labour and delivery are in turn affected by pelvic floor characteristics: Anterior vaginal wall mobility on Valsalva has been found to be a potential predictor of delivery mode (Balmforth et al., 2003; Dietz et al., 2003).

LEVATOR ACTIVITY

Perineal ultrasound has been used for the quantification of pelvic floor muscle activity, in both women with stress incontinence and continent controls (Wijma et al., 1991), as well as before and after childbirth (Peschers et al., 1997; Dietz, 2004c). A cranioventral shift of pelvic organs imaged in a sagittal midline orientation is taken as evidence of a levator contraction (Dietz, 2004c). The resulting displacement of the internal urethral meatus is measured relative to the inferoposterior symphyseal margin (see Fig. 5.18). Another means of quantifying levator activity is to measure reduction of the levator hiatus in the midsagittal plane, or the change in the main hiatal plane axis relative to the central symphyseal axis (Fig. 5.18). Ultrasound can also be utilized for pelvic floor muscle exercise teaching by providing visual biofeedback (Dietz et al., 2001b). The technique has helped validate the concept of ‘the Knack’, i.e., of a reflex levator contraction immediately prior to increases in intraabdominal pressure such as those resulting from coughing (Miller et al., 1996). Correlations between cranioventral shift of the bladder neck on the one hand and palpation/perineometry on the other hand have been shown to be good (Dietz et al., 2002). Direct visualization of a levator contraction and shortening of fibres is possible on 2D ultrasound using an oblique parasagittal plane (see Fig. 5.19), which can also be used to demonstrate levator trauma (Dietz and Shek, 2009).

In addition, it is possible to observe reflex activation of the levator ani and the bulbospongious/bulbocavernous muscles, which is manifested as a reduction in the anteroposterior hiatal diameter and as a dorsal displacement of the clitoral area. While childbirth seems to have some effect on such reflexes (Dietz et al., 2012a), clinical utility appears to be limited (Dietz et al., 2012b).

PROLAPSE QUANTIFICATION

Translabial ultrasound can demonstrate uterovaginal prolapse (Dietz et al., 2001a). The inferior margin of the symphysis pubis serves as a line of reference against which the maximal descent of bladder, uterus, cul de sac and rectal ampulla on Valsalva manoeuvre can be measured (see Fig. 5.17). Findings have been validated against clinical staging and the results of a standardized assessment according to criteria developed by the International Continence Society, with good correlations shown for the anterior and central compartments (Dietz et al., 2001a; Dietz and Lekskulchai, 2007). While there may be poorer correlation between posterior compartment clinical assessment and ultrasound, it is possible to distinguish between ‘true’ and ‘false’ rectocele, i.e., a true fascial defect of the rectovaginal septum, and perineal hypermobility without fascial defects (Dietz and Steensma, 2005). Hopefully the ability to differentiate between different forms of posterior compartment descent will allow better surgical management in the future, not the least because enterocele can easily be distinguished from rectocele. The technique is now used to complement and in some cases replace defecation proctography (Beer-Gabel, 2002; Perniola et al., 2008; Steensma et al., 2010), and it can also demonstrate the external and internal anal sphincters (Dietz, 2012).

Disadvantages of the method include incomplete imaging of the bladder neck, cervix and vault with large rectoceles and the possible underestimation of severe prolapse due to transducer pressure. Needless to say, procidentia or complete vaginal eversion makes translabial imaging more difficult, especially if the prolapse is irreducible. Occasionally, apparent anterior vaginal wall prolapse will turn out to be due to a urethral diverticulum, a vaginal cyst such as a Gartner duct cyst (cystic remnant of the mesonephric or Wolffian ducts), a cyst due to epithelial inversion after repair surgery, or even a vaginal fibroma.

Figure 5.17 Prolapse assessment by translabial ultrasound, midsagittal plane. S = symphysis pubis; B = bladder; U = uterus; Cx = cervix; POD = pouch of Douglas; R = rectum; A = anal canal. There is a cystocele to about 3.5 cm and uterine prolapse to about 3 cm below the symphysis pubis.
3D/4D PELVIC FLOOR IMAGING

3D and 4D pelvic floor ultrasound is currently performed using systems that have evolved around transducers that allow motorized acquisition. The first such motorized probe was developed in 1974, and by 1987 transducers for clinical use were commercially available (Gritzky and Brandl, 1998). The first system platform, the Kretz Voluson, was developed around such a ‘fan scan’ probe. With such a transducer, automatic image acquisition is achieved by rapid oscillation of a group of elements.

3D imaging

The widespread acceptance of 3D ultrasound in obstetrics and gynaecology was helped considerably by the development of such transducers since they do not require any movement relative to the investigated tissue during acquisition. A single volume obtained at rest with an acquisition angle of 70 degrees or higher will include the entire levator hiatus with symphysis pubis, urethra, paravaginal tissues,
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the vagina, anorectum and pubovisceral muscle from the pelvic sidewall to the posterior aspect in the area of the arcus tendineus of the levator ani (ATLA) to the posterior aspect of the anorectal junction (see Fig. 5.20). The levator hiatus as seen on translabial 3D/4D ultrasound or MRI is the plane of minimal dimensions between the symphysis pubis/pubic rami anteriorly and the pubovisceral or puborectalis muscle laterally and posteriorly. Hiatal dimensions can be measured both in the axial plane (Dietz et al., 2005b) and in a rendered volume (Dietz et al., 2011c). The latter method is easier and at least as reproducible and may be preferable, given the non-Euclidean (warped) nature of the plane of the hiatus (Kruger et al., 2010).

The main advantage of volume ultrasound for pelvic floor imaging is access to the plane of the levator hiatus, i.e. the axial or transverse plane. Up until recently, pelvic floor ultrasound was limited to the midsagittal plane. Parasagittal (see Fig. 5.19) and coronal plane imaging (see Fig. 5.20, top right, for an example) may at times be helpful, although there are no obvious points of reference. Imaging planes on 3D ultrasound can be varied in a completely arbitrary fashion in order to enhance the visibility of a given anatomical structure, either at the time of acquisition or offline at a later time. The three orthogonal images (i.e., three planes at right-angles to each other – sagittal, transverse and axial) are complemented by a ‘rendered image’, i.e., a semitransparent representation of all volume pixels (voxels) in an arbitrarily definable ‘box’. The bottom right-hand image in Figure 5.20 shows a standard surface rendered image of the levator hiatus, with the rendering direction set from caudally to cranially, which is most convenient for imaging of thelevator muscle. Midsagittal, axial and coronal views of the levator hiatus are given in the ‘orthogonal’ images in the top row and bottom left.

4D imaging

4D imaging implies the real-time acquisition of volume ultrasound data, which can be represented in orthogonal planes or rendered volumes. Modern systems now generally allow the storing of cine loops of volumes, which is of major importance in pelvic floor imaging as it allows enhanced documentation of functional anatomy. Avulsion of the levator muscle from the inferior pubic ramus is often more evident on Valsalva or levator contraction, and most significant pelvic organ prolapse is not visible at rest in the supine position. Fascial defects such as those defining a true rectocele (Dietz, 2004a) usually only become visible on Valsalva.

The ability to perform a real-time 3D (or 4D) assessment of pelvic floor structures makes the technology superior to MRI imaging as the absence of real-time observation of manoeuvres means that patient compliance with instructions during MRI acquisition is very difficult to verify. Therefore, ultrasound has potential advantages when it comes to describing prolapse, especially when associated with fascial or muscular defects, and in terms of defining functional levator anatomy.

**CLINICAL RESEARCH USING 3D/4D PELVIC FLOOR ULTRASOUND**

Over the past 10 years, a substantial body of research employing 3D/4D translabial ultrasound has been accumulated in the literature. We do know what a normal, healthy pelvic floor in a nulligravid young woman looks like and how it functions (Dietz et al., 2005b; Yang et al., 2006; Kruger et al., 2008), and that there is substantial variation...
between individuals (Svabik et al., 2009). There have been several studies documenting findings in late pregnancy and after a first delivery (Dietz and Lanzarone, 2005; Dietz et al., 2005b; Valsky et al., 2009; Cassado Garriga et al., 2011; Albrich et al., 2012; Chan et al., 2012).

Hiatal depth and area measurements (see Fig. 5.21) seem highly reproducible (Dietz et al., 2005b; Hoff Braekken et al., 2008). Depth, width and area of the hiatus correlate strongly with pelvic organ descent, both at rest and on Valsalva (Dietz et al., 2008). While this is not surprising for the correlation between hiatal area on Valsalva and descent (as downwards displacement of organs may displace the levator laterally), it is much more interesting that hiatal area at rest is associated with pelvic organ descent on Valsalva. This data constitutes the first real evidence for the hypothesis that the state of the levator ani is important for pelvic organ support (DeLancey, 2001), even in the absence of levator trauma. Excessive hiatal distension or ‘ballooning’ (see Fig. 5.26 for an example) has been defined as a hiatal area of 25 cm² or more on maximal Valsalva which seems to be the optimal cut-off on ROC statistics (Dietz et al., 2008) and, incidentally, represents the mean plus 2 standard deviations in young nulliparous women (Dietz et al., 2005b).

The typical form of levator trauma, a unilateral avulsion of the levator ani muscle off the pelvic sidewall, is clearly related to childbirth (see Figs 5.22–5.25 and is palpable as an asymmetrical loss of substance in the anteromedial portion of the muscle. Digital evaluation for morphological abnormalities (Figs 5.23 and 5.24) requires significant operator experience, but it is within the reach of every practitioner caring for women with pelvic floor disorders (Dietz and Shek, 2008b). It appears that those components of the levator ani which form the hiatus, i.e., those subdivisions that insert on the inferior pubic ramus, are most critical, as judged by the results of mathematical modelling on the basis of tomographic (multislice) imaging (Dietz, 2007; Dietz et al., 2011a) (as seen in Fig. 5.22). Hence, we rate an avulsion as present if we are unable to palpate contractile tissue attached to the inferior pubic ramus. Partial trauma is common in the form of generalized or irregular thinning, or as slits or holes in the muscle insertion on the inferior pubic ramus, but this seems to be of less relevance than complete pubic avulsion (Dietz et al., 2011a).

In general, an avulsed muscle generates less force, resulting in reduced strength grading (Dietz and Shek, 2008a), but sometimes there is hypertrophy of more cranial aspects of the levator ani, partially compensating for the trauma. Avulsion also causes enlargement and asymmetry of the levator hiatus (Abdool et al., 2009; Dietz et al., 2011b), and both can sometimes be detected by simple observation of the vulva and introitus during a Valsalva manoeuvre. Enlargement of the hiatus (‘ballooning’) is usually measured on axial plane imaging (see Fig. 5.26), but it has recently been shown that simple clinical measurement (see Fig. 5.27) of the genital hiatus (gh) and perineal body (pb), as in the prolapse quantification system of the International Continence Society (ICS POP-Q), is strongly correlated with hiatal area as well as symptoms and signs of prolapse (Gerges et al., 2012; Khunda et al., 2012). Measurements of 7 cm or more are defined as ‘clinical ballooning’ (Gerges et al., 2012).

The last few years have also resulted in a clarification of risk factors and clinical consequences of avulsion. It is likely that factors such as birthweight, length of second stage, size of the fetal head and forceps delivery increase the probability of avulsion injury (Dietz and Lanzarone, 2005; Krofta et al., 2009; Valsky et al., 2009; Kearney et al., 2010; Shek and Dietz, 2010; Blasi et al., 2011; Cassado Garriga et al., 2011; Albrich et al., 2012; Chan et al., 2012). The clear association between forceps delivery and avulsion suggests that primary forceps should probably be regarded as obsolete, except in situations of imminent fetal demise. However, most currently defined ‘predictors’ of trauma are of very limited use since they are not available prior to the onset of labour. In order to prevent levator avulsion, we would need predictors that can be determined during pregnancy. It is plausible that the risk of trauma to the insertion of the pubourethralis muscle will depend not just on the required dissection, but also on the biomechanical properties of muscle and muscle–bone interface, which are hitherto undefined.
Figure 5.22  Tomographic translabial imaging of the puborectalis muscle. This representation is obtained by multislice imaging of a volume on pelvic floor muscle contraction, with slices placed at 2.5 mm slice intervals, from 5 mm below to 12.5 mm above the plane of minimal hiatal dimensions. A complete defect of the puborectalis muscle is evident on the patient’s right (the left side of any single slice) and marked with a (*).  
*From Dietz 2007, with permission.*

Figure 5.23  Typical right-sided levator avulsion on tomographic ultrasound (A), shown in a palpation model (B) and documented in a drawing. 
*Panel C is modified from Dietz et al., 2011c.*
Figure 5.24 Demonstration of the palpatory assessment of the levator ani for the diagnosis of avulsion. On the intact side (A) the vaginal fornix, i.e., the space between the urethra medially, the inferior pubic ramus superiorly and the puborectalis muscle laterally, admits one finger only. On the abnormal side (B) there is much more room in that space, and no contractile tissue can be palpated on the inferior pubic ramus.

Figure 5.25 Major levator avulsion behind large vaginal tear immediately after a normal vaginal delivery at term (left panel). Despite attempts at repairing the defect, it is still evident 3 months later on US (middle) and MRI (right).

Figure 5.26 Determination of minimal hiatal dimensions in the midsagittal (left) and in the axial plane (right) in a patient with a large cystocele. The hiatus at maximal Valsalva is indicated by the dotted line. S, symphysis pubis; C, cystocele; U, uterus; R, rectal ampulla; A, anal canal; L, levator ani.
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In some studies, avulsion seems associated with maternal age at first delivery (Dietz and Lanzarone, 2005; Kearney et al., 2006; Dietz and Simpson, 2007), implying that ageing has an impact on pelvic floor biomechanics even during the reproductive years. Finally, there is some evidence suggesting that it is usually the first vaginal delivery that causes by far the most morphological and functional alteration, both in terms of actual tears as well as in terms of levator distensibility or pelvic organ support (Dietz et al., 2002a; Dickie et al., 2010; Horak et al., 2012; Kamisan Atan et al., 2012).

Unsurprisingly, avulsion has substantial medium- and long-term consequences. The puborectalis muscle is the main determinant of intravaginal pressures (Jung et al., 2007) and has been termed the ‘love muscle’ in the popular press. It is not surprising that women notice the effect of avulsion on pelvic floor muscle strength (Dietz et al., 2012e) and sexual function. The latter seems to primarily manifest as reduced tone and ‘vaginal laxity’ (Thibault-Gagnon et al., 2012). Considering the popularity of cosmetic genitoplasty procedures aimed at ‘tightening’ the vagina, this may become an important consideration in the future.

In the long term, the most substantial consequence of levator trauma is female pelvic organ prolapse, especially of the anterior and central compartments (Dietz and Simpson, 2008). The larger a defect is, both in width and depth, the more likely are symptoms and/or signs of prolapse (Dietz, 2007). The effect of avulsion on prolapse seems largely independent of ballooning (Dietz et al., 2012c) or abnormal distensibility of the levator hiatus, which also is associated with prolapse (Dietz et al., 2008). One of the more intriguing recent findings is an association between rectal intussusception, an early form of rectal prolapse, and avulsion as well as ballooning (Rodrigo et al., 2011). The most important issue for the surgeon is that both avulsion and ballooning seem to be risk factors for prolapse recurrence both on ultrasound (Dietz et al., 2010; Model et al., 2010; Wong et al., 2011; Weemhoff et al., 2012) and on MRI (Morgan et al., 2011). This implies that such findings are likely to become useful for surgical planning.

The effect of avulsion on urinary and faecal incontinence is much less clear. We often assume that urinary incontinence is a sign of a weak pelvic floor, which may be a misconception. There is some evidence that major levator avulsion defects may be negatively associated with stress urinary incontinence (SUI) and urodynamic stress incontinence (USI) (Dietz et al., 2009; Morgan et al., 2010). How can this be explained in view of the fact that pelvic floor muscle training (PFMT) is a proven therapeutic intervention in women with stress urinary incontinence (Wilson et al., 2005)? If the puborectalis muscle is part of the urinary continence mechanism, shouldn’t it matter if this muscle is disconnected from the inferior pubic ramus? One could point out that the therapeutic success of PFMT does not prove a role of the levator ani muscle in stress urinary continence. The intervention trains not just the levator muscle but likely all muscle innervated by the pudendal nerve and associated pelvic nerves arising from S2–S4. And of course there are other mechanisms by which childbirth might affect urinary continence. Denervation is one (Allen et al., 1990), damage to the urethral rhabdosphincter or the longitudinal smooth muscle of the urethra may be another. And finally there is the issue of pressure transmission, likely mediated through the pubourethral ligaments and/or suburethral tissues. As regards anal, or faecal, incontinence, some studies have identified no significant association with levator trauma (van de Geest and Steensma, 2010; Chantarasorn et al., 2011), others found levator avulsion to be an independent risk factor for faecal incontinence after primary obstetric anal sphincter tear repair (Shek et al., 2012).

OUTLOOK

The ready availability of axial plane imaging is likely to have a significant impact on conservative and surgical treatment paradigms for pelvic floor disorders. Since the 19th century, gynaecologists and surgeons have attempted to cure prolapse and incontinence by reducing organs in a
cranial direction, using a vaginal approach. Since the mid-20th century, suspending those organs by means of sutures and/or mesh has become popular, with the Burch colposuspension, sacrospinous colpopexy and sacrocolpopexy the main examples. Since the mid-1970s, the defect-specific approach blames all prolapse on distinct fascial defects and sets out to repair these discrete defects. Neither concept is entirely satisfactory, as evidenced by the recent development of mesh techniques.

Now largely forgotten, a rather different approach to prolapse surgery was developed by Bob Zacharin of Melbourne, Australia, in the 1960s and 1970s. He appreciated the central role of the levator ani in pelvic organ support long before the advent of modern cross-sectional imaging and proposed focusing on levatorplasty as the primary means of treating female pelvic organ prolapse (Zacharin, 1980). Unfortunately, Zacharin’s approach was highly invasive and morbid, which ensured that the method never gained wider popularity. However, it seems obvious to the observer of severe levator ballooning on Valsalva that poor levator resting tone and marked distensibility will not be cured by a Burch colposuspension or an abdominal vault suspension. Such women are destined for recurrence of prolapse, often in another location, but recurrence all the same. They undergo a vaginal hysterectomy with repairs, come back with incontinence, have a Burch colposuspension, return with a rectoenterocele, have a sacrocolpopexy or sacrospinous fixation, and then come back with a large high cystocele or anterior enterocele, get an anterior mesh repair, which then erodes or causes chronic pain, until they either give up on us, or we give up on them.

A focus on (and understanding of) functional levator anatomy may change all that. Clearly, in some women the levator ani muscle has to be the target of our therapeutic efforts, at least in an adjunctive sense. This may not have to involve the morbidity and technical difficulty of the original Zacharin procedure. Conservative treatment, as discussed in later chapters, very likely has a role to play. Our goal should be to increase resting tone and bulk of the levator ani muscle, reducing downwards displacement of pelvic organs and improving pressure transmission to the urethra – and there may be other ways of achieving this than with conventional physical therapy. An increase in resting tone or stiffness and a reduction in hiatal dimensions may in theory be achievable by direct electrophysiological or even pharmacological means, by scarification or through injection of bulking substances. Axial plane imaging will hopefully deliver the means of optimizing treatment regimens in order to achieve these goals.

As regards surgical treatment, there have been several attempts at direct repair of avulsion. This seems likely to fail immediately post partum (Dietz et al., 2007) but is feasible and moderately effective in the context of prolapse surgery (Dietz et al., 2013). However, in many women there is partial trauma and/or ‘microtrauma’, that is, traumatic overdistension of the hiatus without macroscopic tears (Shek and Dietz, 2010). In those women, other approaches will be necessary, such as a minimally invasive levatorplasty, using the ischiorectal fossa for access (Dietz et al., 2012d).

It is now very clear that a substantial minority of women suffer significant pelvic floor trauma in labour, be it due to overdistension, avulsion or denervation of the levator ani muscle and due to trauma to the external and internal anal sphincters. In the future, we may be able to identify those women most at risk of such injury and intervene to prevent such damage from occurring in the first instance. It is conceivable that antenatal use of a vaginal balloon device may reduce the likelihood of levator trauma, likely by altering pelvic floor biomechanical properties (Shek et al., 2011), and that epidural analgesia may be protective, probably due to partial paralysis of the muscle (Shek and Dietz, 2010). In the meantime, the increasing popularity of vacuum and the ever-rising caesarean section rates are counteracting the effect of delayed childbearing on the likelihood of major pelvic floor trauma in labour.

**CONCLUSIONS**

Ultrasound imaging, and in particular translabial or transperineal ultrasound, has become an important research tool for assessing the levator ani. This development has had positive consequences for the development of clinical examination skills, especially as regards palpation of morphological abnormalities of the levator ani (‘avulsion’), and as regards excessive distensibility of this muscle (‘ballooning’). While both diagnoses were first defined with the help of imaging, they can be obtained by simple clinical examination on PFMC and on Valsalva manoeuvre.

Much information on pelvic floor muscle morphology and function can be gleaned easily and cheaply using 2D ultrasound systems. However, direct demonstration of the inferior aspects of the levator is much simplified by axial plane imaging, i.e. 3D/4D ultrasound. The availability of this technology is increasing rapidly, with hundreds of thousands of such systems now installed worldwide. Most tertiary obstetric/gynaecology units in the developed world (and increasingly in the developing world) have access to 3D-capable systems, enabling them to obtain a functional and morphological assessment of the pelvic floor muscle with minimal discomfort to the patient and at very low cost. Physical therapists, urologists and gynaecologists are in the process of discovering the usefulness of such systems for their field. Undoubtedly, pelvic floor imaging by ultrasound provides a superior tool for research and clinical assessment. It will alter our perception of pelvic floor morbidity and hopefully enhance our means of treating the same.

There is currently no evidence to prove that the use of modern imaging techniques improves patient outcomes. However, this limitation is true for many diagnostic modalities in clinical medicine. Due to methodological issues,
situation is unlikely to improve soon. In the meantime, it has to be recognized that any diagnostic method is only as good as the operator behind the machine, and diagnostic ultrasound is well known for its operator-dependent nature. Teaching is therefore of paramount importance to ensure that imaging techniques are used appropriately and effectively.

**CLINICAL RECOMMENDATIONS**

Pelvic floor imaging is unlikely to become a routine intervention in the hands of each and every clinical practitioner providing pelvic floor re-education, but it already is a very useful tool for research and the most convenient imaging method currently available. Below is a list of recommendations for the clinical use of ultrasound equipment in assessing pelvic floor function via the translabial route.

**Equipment**

- Realtime B mode-capable diagnostic ultrasound system
- Cine loop function
- 3.5–5–6 MHz curved array transducers with a footprint of at least 6 cm
- B/W videoprinter, or other recording device
- Nonpowdered gloves
- Ultrasound gel
- Alcoholic wipes for disinfection of probes between patients
- [Optional] 3D/4D capability, tomographic/multislice imaging, speckle reduction.

**Examination**

- Position patient supine (lithotomy), with feet close to buttocks, lower abdomen and legs covered with sheet for privacy.
- Examine after voiding (and defecation if possible).
- Cover contact surface of transducer with gel, then with glove/transducer cover while avoiding bubbles between transducer and cover.
- Place transducer in midsagittal plane after parting labia (if necessary).
- Ask patient to cough to clear air bubbles/detritus.
- Perform at least three manoeuvres (Valsalva, PFMC) each and watch for incorrect manoeuvres such as levator activation with Valsalva and vice versa.
- Observe presence/absence of ‘the Knack’, i.e., reflex muscle activation, in the sense of a dorsocaudal movement of the prepubic fat pad and ventral movement of the posterior aspect of the levator ani muscle on coughing.
- Provide biofeedback teaching.
- Compare images and measurements at rest and on manoeuvre.
- Obtain hiatal dimensions (axial plane) at rest and on Valsalva.

**Documentation for assessment of PFMC**

- Position of bladder neck or midsagittal hiatal diameter at rest and on PFMC
- Need for teaching/biofeedback, and success of teaching
- Presence of reflex contraction on coughing (‘the Knack’)
- Assessment of levator integrity using axial plane tomographic imaging
- Assessment of hiatal distensibility on maximal Valsalva using axial plane rendered volumes or single plane measurements.

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5.8 MRI of intact and injured female pelvic floor muscles

John OL DeLancey, James A Ashton-Miller

INTRODUCTION

Pelvic striated muscle activity is critical to normal continence and pelvic organ support. Three portions of the levator ani muscle support the pelvic organs, as described in Chapter 4. These muscles must constantly adjust to the widely varying stresses placed on the pelvic floor during daily activities that may range from sitting and reading, to jumping on a trampoline, to forcefully sneezing. This chapter will focus on the levator ani muscle damage seen after vaginal delivery and the implications of this damage for muscle rehabilitation.

Each muscle in the body has its own specific action. Knowing the functional loss that occurs when a muscle is injured is important to understanding the dysfunction that arises from muscle injury. When one of the levator ani muscle elements is damaged, knowing how pelvic muscle training is influenced by muscle injury type has relevance to clinical therapy. If one muscle in the shoulder, for example, is damaged, there is a characteristic impairment that results. Damage to the pectoral muscle, for example, would limit forward motion of the arm, while not limiting its backward movement. Now that MRI can show us evidence of localized muscle injury in an individual it will be possible to better understand the relationship between injury to a specific part of the muscle and specific female pelvic floor problems.

The mechanism of injury to a muscle may also influence its rehabilitation. If a muscle is weak it can be strengthened. If a portion of the muscle is partially denervated then the remaining muscle parts can be recruited to compensate for its muscle loss. If, on the other hand, an entire muscle is lost through avulsion from its attachment and subsequent atrophy or is lost through complete denervation, then it may not be possible to improve the function of the missing muscle. In the past, knowing how a given type of pelvic floor muscle injury would respond to treatment has not been possible because it has not been possible to visualize and locate the injury. Now, with the advent of modern imaging, we can directly see the pelvic floor muscles and their injuries. There is the very real possibility that failure rates with muscle training will decline as patients are more appropriately selected for treatment based on each individual's specific situation.

MRI ANATOMY OF THE NORMAL LEVATOR ANI MUSCLE STRUCTURE

The levator ani muscle consists of several parts. Each has its own origin and insertion. The suggested terms for these components, along with their origin/insertion and function, are listed in Table 5.5 based on a review of anatomical descriptions available in the literature (Kearney et al., 2004). These are shown in Figs 5.28 and 5.29. Although these parts are simple and are described consistently by authors who have personally studied the muscle, a profusion of conflicting terms that have historically applied to this region makes it somewhat complicated to interpret the literature, as described in Kearney et al. (2004).

The iliococcygeal muscle is a thin sheet of muscle that spans the pelvic canal from the tendinous arch of the levator ani to the midline iliococcygeal raphe where it interdigitates with the muscle of the other side and connects with the superior surface of the sacrum and coccyx.

Arising from the pubic bone and passing beside the pelvic organs is the pubovisceral muscle. This muscle has previously been called the pubococcygeal muscle, but we favour Lawson’s term ‘pubovisceral’ (Lawson, 1974) because it describes the origin and insertion accurately, whereas the older term is based on evolutionary considerations rather than human anatomy. Within the
### Table 5.5 Overview of the nomenclature and functional anatomy of the levator ani

<table>
<thead>
<tr>
<th>Terminologia anatomica</th>
<th>Origin</th>
<th>Insertion</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pubococcygeal (we favour ‘pubovisceral’)</td>
<td>Pubis</td>
<td>Perineal body</td>
<td>Tonic activity pulls perineal body ventrally toward pubis</td>
</tr>
<tr>
<td>Puboperineal</td>
<td>Pubis</td>
<td>Vaginal wall at the level of the mid-urethra</td>
<td>Elevates vagina in region of mid-urethra</td>
</tr>
<tr>
<td>Pubovaginal</td>
<td>Pubis</td>
<td>Intersphincteric groove between internal and external anal sphincter to end in the anal skin</td>
<td>Inserts into the intersphincteric groove to elevate the anus and its attached anoderm</td>
</tr>
<tr>
<td>Puboanal</td>
<td>Pubis</td>
<td>Sling behind rectum</td>
<td>Forms sling behind the rectum forming the anorectal angle and closing the pelvic floor</td>
</tr>
<tr>
<td>Iliococcygeal</td>
<td>Tendinous arch of the levator ani</td>
<td>Two sides fuse in the iliococcygeal raphe</td>
<td>The two sides form a supportive diaphragm that spans the pelvic canal</td>
</tr>
</tbody>
</table>

**Figure 5.28** Schematic view of the levator ani muscles from below after the vulvar structures and perineal membrane have been removed showing the arcus tendineus levator ani (ATLA); external anal sphincter (EAS); puboanal muscle (PAM); perineal body (PB) uniting the two ends of the puboperineal muscle (PPM); iliococcygeal muscle (ICM); puborectal muscle (PRM). Note that the urethra and vagina have been transected just above the hymenal ring.


**Figure 5.29** The levator ani muscle seen from above looking over the sacral promontory (SAC) showing the pubovaginal muscle (PVM). The urethra, vagina and rectum have been transected just above the pelvic floor. PAM, puboanal muscle; ATLA, arcus tendineus levator ani; ICM, iliococcygeal muscle. (The internal obturator muscles have been removed to clarify levator muscle origins.)

pubovisceral muscle are parts that attach to the perineal body (puboperinealis), and a part that inserts into the anal canal and skin (puboanal). The vaginal wall is attached to this mass of muscle and those fibres to which the vaginal wall is attached belong to the pubovaginal portion of the pubovisceral muscle. Arising near the perineal membrane and coursing lateral to the remainder of the levator ani muscle is the puborectal muscle. It forms a sling behind the rectum and is distinct from the pubovisceral muscle. While the puborectal muscle creates an angulation in the rectum, the pubovisceral muscle elevates the anus, perineal body and vagina. (Lawson includes this muscle within the pubovisceral muscle complex, but we prefer a separate designation because it has a very different muscle fibre direction.) Each of these different origin/insertion pairs has its unique mechanical action. Injury to one component may have different mechanical effects than damage to another. For example, loss of the pubovaginal muscle would prevent elevation of the anterior vaginal wall (and urethra), while loss of the puborectal muscle would prevent kinking of the rectum in the post-anal angle. Therefore knowing their subdivisions will make a difference.

Magnetic resonance imaging (MRI) is a new and exciting investigative tool that provides anatomical detail in the pelvic floor. It has, for the first time, allowed the detailed anatomy and integrity of the levator ani muscles to be examined. Not only has this technique revealed important insights about normal anatomy, but it also allows investigators to study muscle damage while providing permanent records of muscle morphology that can be evaluated by researchers blinded to the subject’s clinical status, minimizing potential observer bias. Systematic studies concerning repeatability of these techniques, their validity and their responsiveness to change are yet to be carried out. However, the detailed anatomical information that can be gained from these techniques has already established their use in research, and data concerning the performance of these measures are certain to be forthcoming.

**MRI APPEARANCE OF THE LEVATOR ANI MUSCLES**

Damage to the levator ani muscle has been described in cadavers with pelvic organ prolapse for 100 years (Halban and Tandler, 1907). Matched cross-sections of a cadaver pelvis and MR images clarified the anatomy of the levator ani muscles in cross-sectional imaging (Strohbehn et al., 1996). Recent advances in MRI and 3D ultrasound have allowed the muscles to be examined and demonstrated the anatomy of the muscles in serial 2D images (Fig. 5.30) and in 3D reconstructions (Fig. 5.31) (Margulies et al., 2006; Shobeiri et al., 2009). These scans show considerable variation in the normal thickness and configuration of the muscle from one individual to another (Tunn et al., 2003) (Fig. 5.32). As is true in other parts of the body, this variation in muscle bulk is likely attributable to a combination of genetic factors, daily demands and exercise. The amount of muscle that an individual has should have implications for pelvic floor function and injury. A woman with a naturally bulky set of muscles may lose half of her muscle bulk due to injury or atrophy and still have the same amount of muscle as a woman with naturally delicate muscles. The consequences of these variations and damage remain to be determined.

**BIRTH IS A MAJOR EVENT CAUSING PELVIC FLOOR DYSFUNCTION**

Vaginal birth increases the likelihood that a woman will have pelvic floor dysfunction (Mant et al., 1997; Rortveit et al., 2003) and vaginal birth has been identified as a cause of damage to the muscle (DeLancey et al., 2003). The levator ani muscles and pelvic floor undergo remarkable changes during the second stage of labour to dilate sufficiently for the fetal head to be delivered. Understanding how injury can occur and how recovery does or does not proceed are central to understanding the role of rehabilitation.

**Recovery after vaginal birth**

Pelvic muscle training is a mainstay of recovery after vaginal birth, decreasing incontinence and improving muscle function more rapidly than occurs without regular exercise (Sampselle et al., 1998; Mørkved et al., 2003). Imaging has allowed us to study the process of normal recovery and has given insight into changes the muscle must undergo to return to its normal healthy state.

Soon after delivery the pelvic floor sags and the urogenital hiatus is wider than normal (Fig. 5.33) (Tunn et al., 1999; Krofta et al., 2009). Muscle recovery results in resumption of the near-normal position in most women over the course of the first 6 months, the time when normal pelvic muscle strength also returns to normal (Sampselle et al., 1998). Chemical changes in the muscle where there is increased fluid from oedema in certain muscle parts early in the recovery reveal the changes in muscular tissue during the normal healing process (Fig. 5.34).

**Injury from vaginal birth**

Injury to the levator ani muscle has been reported in between 13% (Shek and Dietz, 2010) and 36% (Dietz and Lanzarone 2005) of women. These injuries involve the pubovisceral muscle and occasionally the iliococcygeal muscle (DeLancey et al., 2003) (Fig. 5.35). Examination of women both before and after vaginal birth using 3D ultrasound confirms that these types of injury occur during
Figure 5.30 Axial and coronal images from a 45-year-old nulliparous woman. The urethra (U), vagina (V), rectum (R), arcuate pubic ligament (A), pubic bones (PB) and bladder (B) are shown. Black arrows point to the levator ani muscles. The arcuate pubic ligament is designated as zero for reference, and the distance from this reference plane is indicated in the lower left corner. Note the attachment of the levator muscle to the pubic bone in axial 1.0, 1.5 and 2.0. Coronal images show the urethra, vagina and muscles of levator ani and obturator internus (OI).

Measurement of pelvic floor muscle function and strength, and pelvic organ prolapse

Chapter

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Video delivery (Dietz and Lanzarone, 2005) and are not due to the pregnancy itself. Several factors indicating a difficult birth are associated with increased risk of injury; namely forceps, increased second stage length and larger head circumference. Forceps delivery is associated with an injury rate of 63% (Krofta et al., 2009), with an odds ratio of 3.8 for muscle avulsion and increased second stage of labour length is associated with increased levator hiatus area (Shek and Dietz, 2010). The independent risk of forceps over and above prolonged second stage is supported by the observation that women delivered with forceps for a prolonged second stage have a higher rate of levator injury (63%) compared to those delivered with forceps for fetal distress (42%) (Kearney et al., 2010b). Head circumference over 35.5 cm was associated with an odds ratio of 3.3 (Valsky et al., 2009). Vacuum-assisted vaginal birth and epidural are not associated with increased risk.

Among the women with injury to the pubovisceral muscle the amount of muscle injury varies from one individual to another. Some of these injuries involve complete bilateral loss of pubovisceral muscle bulk (see Fig. 5.35) while others have only unilateral loss (Fig. 5.36). There is also variation in the amount of architectural distortion that occurs. Some individuals show major changes in the overall architecture (Fig. 5.37) while others have intact spatial relationships (Fig. 5.38). Whether this represents the difference between a muscle rupture that distorts muscle appearance or denervation that simply results in loss of muscle without deformity, remains to be determined.

What are the mechanisms of levator injury?

There are several injury mechanisms that have been hypothesized. Neuropathy, muscle tearing or stretch, and compression have all been suggested. The earliest studies of muscle injury used electrophysiological techniques that demonstrated that birth causes changes in mean motor
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Unit duration after vaginal birth (Allen et al., 1990b) as well as neuropathic changes outside the normal range in electromyographic examination turns/amplitude data in 29% of women at 6 months post partum (Weidner et al., 2006). Abnormal tests have been seen in women with both prolapse and stress incontinence (Weidner et al., 2000). Although the pudendal nerve innervates the voluntary urethral and anal sphincters, it does not innervate the levator ani muscles, which receive their own nerve supply from the sacral plexus (Barber et al., 2002). These techniques, however, cannot distinguish between mechanisms for the visible abnormality seen in imaging.

Recently, techniques used in musculoskeletal MRI that allow injury mechanisms to be determined were applied to study the muscle in women at high-risk for injury after vaginal birth (Miller et al., 2010). The injury mechanisms were evaluated using fluid-sensitive and anatomical sequences made in the early (7 weeks) and late (7 months) postpartum period. Levator injuries were seen in 7 of 19 women. Focal tears at the muscle’s insertion into the pubic bone were seen in all. Delayed atrophy, where the muscle is relatively normal early and shows loss of muscle bulk

Figure 5.34 Changes in muscle appearance following birth showing the left side of the pelvis at different time points after delivery. The urethra (U), vagina (V) and levator ani (LA) can be seen. Notice the increasing definition of the structures post partum, especially the medial portion of the levator ani muscle adjacent to the vagina, which is quite pale 1 day after delivery, but recovers its signal by 6 months.
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Figure 5.35 (A) Axial proton density MRI shows normal pubococcygeal muscle with the muscle outlined at the level of the mid-urethra. (B) A similar image from a woman with complete loss of the pubococcygeal muscle (expected location of pubococcygeal muscle shown by outline). PB, pubic bone; R, rectum; U, urethra, V, vagina.
Figure 5.36 Axial and coronal images from a 34-year-old incontinent primiparous woman showing a unilateral defect in the left pubovisceral portion of the levator ani muscle. The arcuate pubic ligament (A), urethra (U), vagina (V), rectum (R) and bladder (B) are shown. The location normally occupied by the pubovisceral muscle is indicated by the open arrowhead in axial and coronal images 1.0, 1.5 and 2.0.

Figure 5.37 Axial and coronal images of a 38-year-old incontinent primiparous woman are shown. The area where the pubovisceral portion of the levator ani muscle is missing (open arrowhead) between the urethra (U), vagina (V), rectum (R) and obturator internus muscle (OI) is shown. The vagina protrudes laterally into the defects to lie close to the obturator internus muscle. A, arcuate pubic ligament.

late, was not seen. Oedema could arise either from compression or muscle stretch. If it was compression, it would involve the internal obturator muscle that shares the space between the fetal head and pubic bone. Oedema was seen in all subjects in the levator ani muscle but never in the obturator, indicating that oedema was caused by muscle stretch rather than compression.

Computer models have studied the stretching that occurs in the levator ani muscle. Those parts of the muscle that are stretched the most are those parts that are seen to be injured (Lien et al., 2004). Using a computer model of the levator ani muscle based on anatomy from a normal woman, the degree to which individual muscle bands are stretched could be studied (Fig. 5.39). This analysis revealed that the muscle injured most often, the pubovisceral (pubococcygeal) portion, was the portion of the muscle that underwent the greatest degree of stretch, and the second area of observed injury, the iliococcygeal muscle, was the second most stretched muscle. Furthermore, when the portion of the muscle at risk was identified in cross-sections cut in the same orientation as axial MRI scans, the pattern of predicted injury matched the injury seen in MRI (Fig. 5.40). Further studies that include the viscoelastic properties of the muscle have revealed that the pubovisceral muscle enthesis and the muscle near the perineal body are the regions of greatest strain thereby placing them at highest risk for stretch-related injury. Decreasing perineal body tissue stiffness significantly reduced tissue stress and strain, and therefore injury risk, in those regions (Jing et al., 2012).

Figure 5.38 Levator ani defect in a 30-year-old incontinent primiparous woman with loss of muscle bulk but preservation of pelvic architecture. The area where the levator is absent in this woman is shown (open arrowhead) in the axial images and the coronal images 1.5 and 2.0. Note that in contrast to Figure 5.30, where the vagina lies close to the obturator internus (OI), it has a normal shape. The normal appearance of the levator ani muscle is seen in coronal images 2.0 and 2.5 (arrows). A, arcuate pubic ligament; R, rectum; U, urethra; V, vagina.

**Figure 5.39** (A) On the left is a computer model of selected levator ani muscle bands before birth, with muscle fibres numbered and the muscle groups identified; the figure on the right demonstrates muscle band lengthening present at the end of the second stage of labour. (B) A graphic representation of the original and final muscle lengths (top) and the stretch ratio (bottom), indicating the degree to which each muscle band must lengthen to accommodate a normal-sized fetal head. Note that the pubococcygeal muscle fascicles labelled ‘PC2’ undergo the greatest degree of stretch and would be the most vulnerable to stretch-induced injury. From *Lien et al.*, 2004, with permission. © Biomechanics Research Laboratory 2003.

**Figure 5.40** (A) Normal anatomy in an axial mid-urethra proton density MRI showing the pubovisceral muscle (*) (see Fig. 5.28 for orientation). (B) Woman who has lost a part of the left pubovisceral muscle (displayed on the right side of the image, according to standard medical imaging convention) with lateral displacement of the vagina into the area normally occupied by the muscle. The arrow points to the expected location of the missing muscle. The puborectalis is left intact bilaterally. OI, obturator internus; PB, pubic bone; R, rectum; U, urethra; V, vagina. (C) Axial, mid-urethral section of the model through the arch of the pubic bone (see pubic symphysis [PS], top) and the model levator ani muscles corresponding to those from the patients shown in (A) and (B). Intact muscles are shown in dark shading. Simulated PC2 muscle atrophy is illustrated by the light shading of the left-side PC2 muscle. This location is shown to correspond with the location of muscle atrophy demonstrated in Figure 5.33. R, rectum; U, urethra; V, vagina. From *Lien et al.*, 2004, with permission. © Biomechanics Research Laboratory 2003.
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Visible injury to the levator ani muscle is highly associated with pelvic organ prolapse. In a case–control study comparing the occurrence of major levator ani muscle defects that involve loss of more than 50% of the pubovisceral portion of the levator ani muscle, major defects were found in 16% of women with normal support and 55% of women with prolapse (DeLancey et al., 2007). Among women attending a clinic for urogynecologic problems, prolapse was seen in 150/181 (83%) women with avulsion and in 265/600 (44%) women without avulsion (Dietz and Simpson, 2008). There is also evidence that levator defects are associated with faecal incontinence. In a study of women with anal sphincter tears at birth, women who had a levator tear seen in association with the sphincter tear tended towards more faecal incontinence (35.3%) compared to women who had sphincter laceration but intact levator ani muscles (16.7%, p = 0.10) (Heilbrun et al., 2010). Not all pelvic floor disorders, however, are similarly associated with levator ani muscle injury. As mentioned earlier in this chapter, visible injury is more common in women with stress incontinence 9 months after vaginal birth; however, this is not true of women at mid-life who present for care with stress incontinence in whom injury is seen in 13% compared with 18% seen in continent women (DeLancey et al., 2008).

There are potentially both direct and indirect ways in which birth-induced levator injury may influence pelvic floor function. Pelvic organ support is provided by the combined action of the levator ani muscles and the endopelvic fascia. The levator ani closes the vagina by creating a high-pressure zone (Guaderrama et al., 2005) similar to the high-pressure zones created by the urethral and anal sphincter muscles. Women with levator defects have a lower vaginal closure force (2.0 Newtons) compared to women without defects (3.1N, p < 0.001) (DeLancey et al., 2007). There is also an increase in levator hiatus area of 28% with injury compared to 6% in women without injury (Shek and Dietz, 2009). The muscle and ligaments must resist the downward force applied on the pelvic floor by the superincumbent abdominal organs and the forces that arise from increases in abdominal pressure during cough, sneeze or from inertial loads placed on them when landing from a jump (for example). This normal-load sharing between the adaptive action of the muscles and the energy efficient action of static connective tissues is part of the elegant load-bearing design of the pelvic floor. When injury to one of these two components occurs, the other must carry the increased demands placed on it. When the muscle is injured, the connective tissue is subjected to increased load. If this load exceeds the strength of the pelvic tissues, they may be stretched or broken and prolapse may result. This forms a causal chain of events by which pelvic muscle injury may influence pelvic organ prolapse or urinary incontinence. In addition, there is accumulating evidence that women operated on for pelvic organ prolapse or urinary incontinence have higher postoperative failure rates if they have levator ani muscle impairment assessed by biopsy (Hanzal et al., 1993), muscle function testing (Vakili et al., 2005) and ultrasound detected levator damage (Dietz et al., 2010) than women who have normal muscles. There are early differences in pelvic organ support after surgery depending on whether or not a defect is present (Morgan et al., 2011). Muscle avulsion is seen more commonly in women with anatomical recurrence at 2 years compared to women with no avulsion (Weemhoff et al., 2012).

Birth-induced levator ani muscle injury may be accompanied by other types of injury that occurred during vaginal birth. A birth that was sufficiently difficult that it resulted in injury to the levator ani muscle may have also created injury to the connective tissue supports. This hypothesis is supported by observations made on the function of the urethral sphincter in women with injured levator ani muscle and also those with intact muscles (Miller et al., 2004). In this study of 28 women with normal muscles and 17 women with complete bilateral pubovisceral muscle loss, women with intact muscles generated a greater increase in urethral pressure during a maximal pelvic muscle contraction than those with absent pubovisceral muscles (14 ± 11 vs 6 ± 9 cmH₂O) (Table 5.6). This difference in the ability to increase pressure came from the fact that more of the

<table>
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<th>Table 5.6 Urethral closure pressure data in 28 women with intact pubovisceral muscles and 17 women with absent pubovisceral muscles</th>
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<tbody>
<tr>
<td><strong>Pubovisceral muscle intact</strong></td>
</tr>
<tr>
<td>Pressure increase &gt;5 cmH₂O (%)</td>
</tr>
<tr>
<td>Mean MUCP (SD)</td>
</tr>
<tr>
<td>Mean volitional MUCP pressure increase (SD)</td>
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</tbody>
</table>

MUCP, maximum urethral closure pressure.
Adapted from Miller et al., 2004
women (86%) were able to elicit a measurable increase (>5 cmH₂O) in urethral closure than those with missing muscles (41%); among women that could increase urethral closure pressure, the increase in urethral closure pressure was the same. Women with complete levator muscle loss can volitionally elevate urethral pressure in the absence of the pubovisceral muscle (presumably using their still-intact striated urethral sphincter muscle), but fewer women are able to do this, suggesting the occurrence of sphincter injury as well in a subset of women in this group. This indicates that some women who are unable to contract their levator ani muscles, due to muscle (or nerve) injury, escape injury to the urethral sphincter (or pudendal nerve), whereas others do not, and that this phenomenon occurs more often in women with muscle problems.

**ISSUES IN REHABILITATION**

'The injured patient is entitled to know at the outset, in general terms, what [her] injuries are, what the immediate treatment will be, and what may be the expected result' (Committee on Trauma, ACS 1961: 16) and 'The fate of the injured person depends to a large extent upon the initial care that [her] injuries receive. Skilled competent care may salvage function in seemingly hopeless situations; inept care for even a trivial injury may end in disaster' (Committee on Trauma, ACS 1961: 1).

This statement made over 40 years ago articulates an enduring truth about injury management; that is, knowing the type of injury is an important guide to proper treatment. Imaging has now demonstrated specific evidence of localized muscle loss revealing a great variety of injury patterns in different women. At present, without specific testing, we do not know whether birth-induced muscle injury is caused by neurological injury or by muscle rupture. Whether there should be similar treatment of these two types of injury remains to be determined. Further research is needed to develop effective strategies to answer this question in individual women.

In addition, the nature of a woman's defect later in life may influence the type of therapy selected. Pelvic muscle training can have two effects. First, it can improve a woman's skill in using her muscles and, second, it can improve the contractile force. Whether exercise changes resting urethral function is not known. If the ability to contract a normally-innervated pelvic floor muscle during a cough is lost, for example, a woman can be taught to purposefully contract the muscle. Second, the muscles can be exercised to become stronger through hypertrophy. Therefore, if the normally occurring muscle contraction occurs, but is not strong enough, this muscle can be strengthened and continence improved. Most of a person's time is not spent coughing or jumping. Most of the time, there should be normal 'tone' in the muscle. This tonic activity is similar to the action of postural muscle in the back in that it automatically adjusts to the loads placed upon it. Whether this can be improved is unknown.

At present, the success of muscle training in women with different types of levator ani muscle injury is not clear. If the pubovisceral muscle is missing, then the connections between the pubic bone and the vagina or perineal body are missing. Although the iliococcygeal and puborectal muscles remain, there are presently no data to know whether the success of pelvic muscle training is similar in women with and without muscle injury. This should be a fertile field for research as new imaging modalities make the detection of muscle injury routine.

**REFERENCES**


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ACKNOWLEDGEMENT

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6.1 Motor learning

Ability to contract the pelvic floor muscles
Practical teaching of correct PFM contraction
References

6.2 Strength training

Introduction to the concept of strength training for pelvic floor muscles

ABILITY TO CONTRACT THE PELVIC FLOOR MUSCLES

Before starting a training programme of the pelvic floor muscles (PFM) one has to ensure that the patients/clients are able to perform a correct PFM contraction. A correct PFM contraction has two components: squeeze around pelvic openings and inward (cranial) lift (Kegel, 1952). Several research groups have shown that more than 30% of women are not able to voluntarily contract the PFM at their first consultation even after thorough individual instruction (Kegel, 1952; Benvenuti et al., 1987; Bø et al., 1988; Bump et al., 1991). In a study of 343 Austrian women aged 18–79 years attending routine gynaecological visits, 44.9% were not able to contract the PFM. It was reported that involuntary contraction was present before increase in intra-abdominal pressure in only 26.5% (assessed by palpation) (Talasz et al., 2008). Common mistakes when trying to perform a PFM contraction are listed in Table 6.1. Bø et al. (1988) and Bø et al. (1990a) found that many women contracted other muscles in addition to the PFM, and nine out of 52 were straining instead of lifting. Bump et al. (1991) found corresponding results in an American population, with as many as 25% of women straining instead of squeezing and lifting. These findings were later supported by Thompson and O’Sullivan (2003) in a population of Australian women.

There may be several explanations why a voluntary PFM contraction is difficult to perform:

- the PFM have an invisible location inside the pelvis;
- neither men nor women have ever learned to contract the PFM and most people would be unaware of the automatic contractions of the muscles;
- the muscles are small and, from a neurophysiological point of view, therefore more difficult to contract voluntarily;
- the common awareness of these pelvic and perineal areas of the body may be associated with voiding and defecation, and straining at toilet is common.
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Tries (1990) suggests that there may be a lack of sensory feedback during PFM training (PFMT) in some women, causing:

- problems with feedback from the correct muscles because other muscles are used instead of the PFM;
- insufficient kinaesthetic feedback due to low intensity contractions in weak PFM;
- lack of or reduced sensation, which may limit the sensory incentive that normally leads to a motor response or reflex preventing leakage.

Motor re-learning depends on sensory feedback (Tries, 1990). Following Gentile (1987), learning is in general facilitated by the use of feedback, and the physical therapist (PT) should give external feedback as ‘knowledge of results’ (KR) as a part of the intervention. KR may compensate for a loss of normal sources for internal feedback in patients with central- or peripheral nerve injuries (Winstein, 1991). Although many women have reduced innervations in the pelvic floor (e.g. after injury related to pregnancy and delivery), the use of KR may be useful in learning correct PFM contraction.

Our reason for attempting to isolate the PFM contraction from outer pelvic muscles when training the muscles is not because we do not appreciate that all muscles in the body act together and never work in isolation (Bo et al., 1990a; Bo et al., 1999; Mørkved and Bo, 1997; Mørkved et al., 2002; Mørkved et al., 2003; Overgård et al., 2008; Stafne et al., 2012). However, such simultaneous contractions of outer and more commonly used larger muscle groups outside the pelvis may mask the awareness and strength of the PFM contraction. The person erroneously believes he or she is performing a strong contraction, but the PFM are not doing the job. Most importantly, to train and build up a muscle or muscle group’s strength and volume it is mandatory to work specifically with the targeted muscle.

More concerning than the contraction of outer pelvic muscles simultaneously with PFM contraction, is straining. If patients are straining instead of performing a correct contraction, the training may permanently stretch, weaken and harm the contractile ability of the PFM. In addition, straining may stretch the connective tissue of fasciae and ligaments, thereby potentially increasing the risk of development of pelvic organ prolapse. Proper assessment of ability to contract the PFM and feedback on performance is therefore mandatory before starting a training programme.

### Table 6.1 Common errors in attempts to contract the pelvic floor muscles

<table>
<thead>
<tr>
<th>Error</th>
<th>Observation</th>
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<tbody>
<tr>
<td>Contraction of outer abdominal muscles instead of the PFM</td>
<td>The person is curving the back, or starts the attempt to contract by ‘hollowing’/tucking the stomach inwards (note that a small ‘hollowing’ can be seen in a correct contraction with the transverse abdominal muscle co-contracting)</td>
</tr>
<tr>
<td>Contraction of hip adductor muscles instead of the PFM</td>
<td>A contraction of the muscles of the inner thigh can be seen</td>
</tr>
<tr>
<td>Contraction of gluteal muscles instead of the PFM</td>
<td>The person is pressing the buttocks together, lifting up from the bench</td>
</tr>
<tr>
<td>Stop breathing</td>
<td>The person closes her/his mouth and holds their breath</td>
</tr>
<tr>
<td>Enhanced inhaling</td>
<td>The person takes a deep inspiration often accompanied by contraction of abdominal muscles, and tries erroneously to ‘lift up’ the pelvic floor by the inspiration</td>
</tr>
<tr>
<td>Straining</td>
<td>The person presses downwards. When undressed, the perineum can be seen pressing in a caudal direction. If the person has pelvic organ prolapse, the prolapse may protrude</td>
</tr>
</tbody>
</table>

**PRACTICAL TEACHING OF CORRECT PFM CONTRACTION**

The steps of learning a correct muscle contraction can be separated into five levels.

1. **Understand** – the patient needs to understand where the PFM are located and how they work (cognitive function).
2. **Search** – the patient needs time to put this understanding into her or his body. Where is my pelvic floor?
3. **Find** – the patient must find where the PFM are, but often needs reassurance from the PT of the location.
4. **Learn** – after having found the PFM, the patient needs to learn how to perform a correct contraction of the PFM. Feedback from the PT is mandatory.
5. **Control** – after having learned to contract, most subjects still strive for a while to perform controlled and coordinated contractions recruiting as many motor units as possible during each contraction; most people are unable to hold the contraction, perform repetitive contractions or conduct contractions of high velocity or strength during their first attempts of training. 
Basically, four teaching tools can be used to facilitate skill acquisition (Gentile, 1972): the therapist can try to verbally indicate key aspects of the task or performance, supplementary visual input can be provided, direct physical contact with the learner might be employed, and the therapist can structure the environmental conditions under which practice is to take place.

**Teaching tools**

To facilitate correct PFM contractions the PT can use different teaching tools.

Verbal instructions should be based on knowledge of the function of the PFM, namely to form a structural support and to ensure a fast and strong contraction during abrupt increase in abdominal pressure. One example of a training command is ‘squeeze and lift’.

To teach patients, the PT might use drawings and anatomical models of the pelvic floor to show the patient where the muscles are located anatomically (Fig. 6.1). We also recommend the PT demonstrates a correct PFM contraction in standing position, showing that there should be no movement of the pelvis or thighs visible from the outside. The patient can also palpate the PT’s buttocks to feel the difference between gluteal muscle contraction and the relaxed position these muscles should hold during PFM contraction. Allow the patient to ask questions and practise a few contractions for her/himself.

One way to help patients understand the action of the PFM is to use imagery such as describing the contraction as a lift starting with closure of the doors (squeeze) and from there the elevator is moving upstairs (lift). Another way is to explain the action as eating spaghetti or the action of a vacuum cleaner. Many patients may have general low body awareness and sometimes it is necessary first to focus on the pelvic area and make the patient move the pelvis in different directions by use of outer pelvic muscles (Fig. 6.2). When the patient is familiar with the pelvic area, one can start to focus on the internal pelvic muscles (the PFM).

One way of visualizing where the PFM are located and how they work is to use a skeleton and place the patient’s hand as if it was the pelvic floor inside the pelvis. Then the PT presses the hand towards the ‘pelvic floor’ to make the patient understand the role of the PFM as a structural support for all the pelvic organs and how it should resist increases in abdominal pressure (Fig. 6.3).

Direct physical contact may be used to enhance sensory stimulation and proprioceptive facilitation. An effective position to teach a correct PFM contraction is having the patient sit on an armrest or at the edge of a table with legs in abduction, feet on the floor, straight back and hip flexion. In this position, the patient gets exteroceptive, and for some maybe proprioceptive, stimulus on the perineum/PFM. The patient is then instructed to squeeze and lift away from the chair without rising up, and then relax again (Fig. 6.4). After this instruction the patient is allowed to go to the toilet to empty the bladder. Observation and vaginal palpation then takes place. Figure 6.5 shows the relationship between the PT and patient with vaginal palpation during attempts to contract the PFM. Both PT and patient give verbal feedback to each other during the contraction. In addition, proprioceptive facilitation may be used during vaginal palpation to enhance contraction of the PFM. The palpation (rectal for men) is also important to give feedback of the strength of the contraction and to make the patient understand that although he or she is contracting correctly it is possible to work much harder. Gentile (1987) claims that in general one of the most important roles of the instructor is to keep the patient’s motivation high because practice/training is a premise for learning. A distinction must be made between feedback aiming at giving information about

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**Figure 6.1** Use of anatomical models or illustrations to teach anatomy and physiology of the pelvic floor. Place the anatomical model in front of the patient’s pelvis so she can see the correct position of the organs as they are located inside her.
performance or results, and verbal comments to motivate the patient to adherence.

It can be explained to men that if they perform a correct PFM contraction they feel and see a lift of the scrotum. If appropriate, a mirror can be used for both men and women to see the inward lifting movement. However, some people feel uncomfortable observing their genitalia, and the PT must show tact before suggesting this method.

Another way of facilitating learning may be to structure the environmental conditions under which practice is to take place. We emphasize a situation during PFMT, both at home and training groups, that allows thorough concentration. One consequence of this is that during group training classes we do not use music when teaching the PFM contractions.

Although as many as 30% may not be able to conduct a correct PFM contraction at the first consultation, we have experienced that most women learn to contract if they are given advice to practise on their own at home for a week. It is important not to strain the patient at the first consultation if she is not able to contract. Ask the patient to exercise on an armrest at home and also ask her or him to try to stop the dribble at the end of the voiding. However, stopping the urine stream is not recommended in a training protocol, as it may disturb the fine neurological balance between bladder and urethral pressures during voiding. There should be no PFM activity just before (opening of the urethra) and during voiding. Stopping the dribble at the very end of the voiding is therefore only recommended as a test of the ability to contract, and many patients have reported that they have learned to contract the PFM with this method. Another way to improve the awareness of a correct PFM contraction is to contract other circular muscles (e.g. those surrounding the mouth; Liebergall-Wischnitzer et al., 2005).

Two experimental studies evaluating the effect of contracting the circular muscles on the PFM contractions did not support that such contractions facilitated or augmented the effect of voluntary PFM contraction using surface EMG and ultrasound (Bø et al., 2011; Resende et al.,

Figure 6.2 First teach the patient where the pelvis is by practising movements of the pelvis in an anteroposterior direction (A) and sideways (B).
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If the patient is still unable to contract the PFM after 1 week of rehearsal on her own, the PT may try general muscle facilitation techniques to stimulate awareness of the PFM. Methods such as fast stretch of the PFM, tapping on the perineum or muscles, pressure/massage techniques or electrical stimulation can be tried (Brown, 2001). However, no studies have yet been found evaluating the effect of such techniques to increase awareness of the PFM or ability to contract. These recommendations are therefore based on clinical experience only.

Studies using concentric needle electromyography (EMG) in the urethral sphincter and the PFM have demonstrated that there is a co-contraction of the PFM with the use of outer pelvic muscles (gluteal, hip adductor and rectus abdominis) in healthy volunteers (Bø and Stien, 1994). In addition, Sapsford and Hodges (2001) used surface EMG and found that there was a co-contraction of the PFM during transversus abdominis (TrA) contraction in healthy volunteers. Therefore, many PTs recommend contractions of outer pelvic muscles and hope for a co-contraction of the PFM if the patient is not able to perform a correct PFM contraction. However, we do not know whether there are such simultaneous co-contractions in persons with pelvic floor dysfunction, and there are no studies showing the effect of interventions for different symptoms of pelvic floor dysfunction using contraction of other muscles
than the PFM. If some of the outer pelvic muscles are to be used instead of the PFM, we recommend hip adductor and gluteal muscle contractions and not TrA or other abdominal muscle training because contraction of all the abdominal muscles may increase abdominal pressure (Hodges and Gandevia, 2000). In addition, Bø et al. (2003) showed that when contracting the TrA, 30% of trained female PTs showed descent of the PFM. In another study using perineal ultrasonography it was shown that a PFM contraction was significantly more effective than a TrA contraction in reducing the levator hiatus area and that in some women the levator hiatus opened up during TrA contraction (Bø et al., 2009). Therefore, if there is no co-contraction of the PFM with the abdominal muscle contractions this may strain and weaken the PFM.

High-quality studies in the area of PFM awareness and motor learning should be of high priority and are strongly encouraged in future research. However, it is important that PTs are aware that some subjects may never be able to perform a voluntary PFM contraction. In a study by Bø et al, four of 52 patients were still unable to contract after 6 months of PFMT (Bø et al., 1990a). Inability to contract the PFM may be due to severe muscle, nerve and connective tissue damage or inability to learn this specific task due to a general low body/muscle/movement awareness. These patients should not spend a lot of time and money with the PT, but should be referred back to their treating general practitioners, urologists or gynaecologists for other treatment options as soon as possible.

Figure 6.5 Vaginal palpation is mandatory to give immediate feedback on correctness of the attempt to contract the pelvic floor muscles.

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The pelvic floor muscles (PFM) are regular skeletal muscles and will therefore adapt to strength training in the same way as other muscles (Fig. 6.6). The aim of a strength training regimen is to increase strength and change muscle morphology by increasing the cross-sectional area, improve neurological factors by increasing the number of activated motor neurons and their frequency of excitation, and improve muscle ‘tone’ or stiffness (DiNubile, 1991) (Fig. 6.7).

Specific changes are dependent on the type of exercise and the training programme used, but response to a specific training programme also depends on genetics and hereditary factors (Haskel, 1994). However, whenever starting to activate any muscle in the body, physiological changes will occur within the activated muscles. Table 6.2 gives a list of some of the physiological adaptations in the muscle fibre following regular strength training.

Connective tissue is abundant within and around all skeletal muscles including the epimysium, perimysium and endomysium. These connective tissue sheaths provide the tensile strength and viscoelastic properties (‘stiffness’) of muscle and provide support for the loading of muscle (Fleck and Kraemer, 2004). There is evidence that strength
training can increase connective tissue mass, change the mechanical properties and that intensity of training and load bearing are major factors for effective training (Arampatzis et al., 2007). Magnusson et al. (2007) found in their study that the adaptability of tendon to loading differs in men and women. Tendons in women might have a lower rate of new tissue formation, respond less to mechanical loading and have a lower mechanical strength. Increased estradiol levels may slow down the rate of collagen synthesis. This may increase the risk of injury compared to men, and be something to take in consideration in training progression.

The theoretical rationale for intensive strength training of the PFM is that strength training may build up the structural support of the pelvis by elevating the levator plate to a permanent higher location inside the pelvis and by enhancing hypertrophy and stiffness of the PFM and connective tissue. This would facilitate a more effective co-contraction of the PFM and prevent descent during

**Table 6.2 Muscle fibre adaptation with resistance training**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Muscle’s adaptational response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Muscle fibre myofibrillar protein content</td>
<td>↑</td>
</tr>
<tr>
<td>Capillary density</td>
<td>↔↓</td>
</tr>
<tr>
<td>Mitochondrial volume density</td>
<td>↓</td>
</tr>
<tr>
<td>Myoglobin</td>
<td>↓</td>
</tr>
<tr>
<td>Succinate dehydrogenase</td>
<td>↔↓</td>
</tr>
<tr>
<td>Malate dehydrogenase</td>
<td>↔↓</td>
</tr>
<tr>
<td>Citrate synthase</td>
<td>↔↓</td>
</tr>
<tr>
<td>3-hydroxyacyl-CoA dehydrogenase</td>
<td>↔↑</td>
</tr>
<tr>
<td>Creatine phosphokinase</td>
<td>↑</td>
</tr>
<tr>
<td>Myokinase</td>
<td>↑</td>
</tr>
<tr>
<td>Phosphofructokinase</td>
<td>↔↓</td>
</tr>
<tr>
<td>Lactate dehydrogenase</td>
<td>↔↑</td>
</tr>
<tr>
<td>Stored ATP</td>
<td>↑</td>
</tr>
<tr>
<td>Stored PC</td>
<td>↑</td>
</tr>
<tr>
<td>Stored glycogen</td>
<td>↑</td>
</tr>
<tr>
<td>Stored triglycerides</td>
<td>↑?</td>
</tr>
<tr>
<td>Myosin heavy chain composition</td>
<td>Slow to fast</td>
</tr>
</tbody>
</table>

ATP, adenosine triphosphate; PC, phosphocreatine. From Kraemer and Fry, 1995.
increases in abdominal pressure. In an assessor blinded randomized controlled trial of 6 months of PFMT to prevent and treat pelvic organ prolapse Brækken et al. (2010) found a significant increase in PFM thickness of 15.6%, a reduction in the levator hiatus area of 6.3%, reduction in muscle length of 6.3% and a lift of the position of the bladder neck and rectal ampulla of 4.3 and 6.7 mm respectively compared to the control group. In addition, the levator hiatus area and muscle length were reduced during Valsalva, indicating increased PFM stiffness and improvement of automatic function. The pelvic floor can be considered as a trampoline with its position inside the pelvis. If the trampoline is stretched and sagging down, it is difficult to jump. However, a firm trampoline gives a quicker response and an effective ‘push’ upwards (Fig. 6.8). Increased stiffness in the connective tissue/tendons is probably important in all movements where we try to develop strength as fast as possible. Arampatzis et al. (2007) found that to increase stiffness and hypertrophy in the Achilles tendon, the loading/strain in the strength training regimen should be high (90% of MVC).

As most individuals starting a PFMT regimen would be untrained, some improvements would probably occur regardless of the type of training programme applied (Kraemer and Ratamess, 2004). Because all PFMT studies have used different training dosage and different outcome measures, it is not possible to compare effects and conclude which training programme is the most effective. It may be considered much easier to improve quality of life (QoL) compared to reducing amount of urinary leakage or increasing muscle hypertrophy. Both general and disease-specific QoL parameters will most likely change because of factors other than the actual training programme (e.g. as a result of information that the condition can be improved or cured, care, support, comfort and motivation). On the other hand, a change in morphological muscle factors must be due to actual training. In addition, there is a huge difference between a report of ‘feeling better’ and measurement of cure on a pad test with standardized bladder volume. We will argue that proper training is needed to make a measurable change in muscle morphology and cure symptoms of pelvic floor dysfunction (Brækken et al., 2010).

Figure 6.8 With its location in the bottom of the pelvis, the pelvic muscles should act as a trampoline when abdominal pressure is increased. A stiff trampoline gives a quick response to load and pushes upwards.
TERMINOLOGY AND DEFINITIONS

**Muscle strength**

Muscle strength is 'the maximal amount of force or torque a muscle or muscle group can generate in a specific movement pattern at a specific velocity of movement' (Knuttgen and Kraemer, 1987; Knuttgen and Komi, 2003). To include different muscle actions it has also been defined as 'the maximum force which can be exerted against an immovable object (static/isometric strength), the heaviest weight which can be lifted or lowered (concentric and eccentric dynamic strength), or the maximum torque which can be developed against a pre-set rate limiting device (isokinetic strength)' (Frontera and Meredith, 1989).

A repetition is one complete movement of an exercise (e.g. one contraction of the PFM). It normally consists of two phases: the concentric muscle action and the eccentric muscle action (Fleck and Kraemer, 2004).

A set is a group of repetitions performed continuously without stopping or resting. Sets typically range from 1 to 15 repetitions (Fleck and Kramer, 2004).

**Maximum voluntary contraction**

Knuttgen and Kraemer (1987) described a maximum voluntary contraction as 'a condition in which a person attempts to recruit as many fibres in a muscle as possible for the purpose of developing force'. They focus on the importance of the word 'voluntary' because inhibitory mechanisms in the central nervous system (CNS) can limit the recruitment of motor units, and the total number of muscle fibres that will produce the force. An important part of strength training is to diminish the inhibitory mechanisms during maximal effort and allow the person to come as close as possible to total recruitment. The resistance at which the subject can perform only one lift of a free weight (dynamic contractions) and not be able to repeat it is termed the one repetition maximum (1RM) (Bompa and Carrera, 2005). For an isometric contraction the max strength is often referred to as maximum voluntary contraction (MVC) (Tan, 1999). The mass of the free weight that limits the person to 10 repetitions would be termed 10RM (Knuttgen and Kraemer, 1987). Performing voluntary maximal muscular actions means that the muscles involved must contract against as much resistance as its present fatigue level will allow. This is often referred to as overloading the muscle.

**Local muscle endurance**

Local muscle endurance is usually defined as either number of repetitions conducted, or duration of sustaining a contraction. Number of repetitions is inversely related to the percentage of 1RM, and varies with training status, sex and amount of muscle mass needed to perform the exercise (Hoeger et al., 1990). Fatigue is a necessary component of local muscle endurance training (Kraemer and Ratamess, 2004), and increases in maximum strength usually increase local muscle endurance, while muscle endurance training does not improve maximum strength. Training to increase muscle endurance requires the performance of a high number of repetitions and minimizing recovery between sets.

**Muscle power**

Muscle power is the explosive aspect of strength, the product of strength and speed of movement (force x distance)/time (Wilmore and Costill, 1999). Power is the functional application of both strength and speed (distance/time), and is the key component of most performances. Knuttgen and Kraemer (1987) explain the interrelationship between power and strength emphasizing that high power development means less than maximal force and maximal force development means low power. Maximum strength is the highest force we are able to develop during contractions of slow-velocity or isometric contractions, while power is the ability to develop much force during high-velocity contractions.

The order in which motor units are recruited is relatively constant and according to the size principle. This means that in light movements using low force, the smaller motor units (low-threshold), motor neurons innervating slow-twitch, type I, muscle fibres, are always recruited first. With increasing loads the muscle demands more force and progressively higher threshold motor units (type II muscle fibres) are recruited (Fleck and Kraemer, 2004). This also applies when the load is constant, but the speed of contraction increases. At higher shortening velocities submaximal forces can be maximum or at least close to maximum (Åstrand et al., 2003). Exceptions from the recruiting hierarchy have been found during explosive muscle actions, where probably all motor units activate at once, and during pure eccentric muscle actions, where activity has been seen in big type II-fibre units, and no activity in smaller type I-fiber units (Christova and Kossev, 2000).

Training to increase muscular power requires two general loading strategies:

1. Moderate to high training loads are needed to recruit high-threshold fast-twitch motor units for strength, but this implies moderate to slow velocity contractions.
2. Incorporation of light to moderate loads performed at an explosive lifting velocity.

These two loading strategies were used in the PFMT programme developed by Bø et al. (1990). The patient is asked to contract as close to maximum as possible, try to hold the contraction and then to add 3–4 fast contractions on top of the holding period (fig. 6.9).
There are several determinants of muscle strength.

- **Anatomy.** There is an individual difference in joint angle and lever arm in different muscles. The longer the lever arm the more work the muscle can produce (work = force × lever arm). The most optimal lever arm is difficult to establish in the PFM. A sagging pelvic floor may be more difficult to lift voluntarily, and the expected automatic co-contraction during increased abdominal pressure may be too slow to stop excessive downward movement. Also total number of muscle fibres within a muscle, the cross-sectional area, the distribution of type I and type II muscle fibres (especially in fast dynamic contractions), and the internal muscle architecture are determinants of muscle strength (Åstrand et al., 2003). This differs between individuals.

- **Length–tension.** There is an optimal length at which muscle fibres generate maximal force. The total amount of force generated depends on the total number of myosin cross-bridges interacting with active sites on the actin. If a sarcomere or a muscle is stretched or shortened beyond the optimal length, less force can be developed (Fleck and Kraemer, 2004).

- **Force–velocity.** As the velocity of a movement increases, the maximal force a muscle can produce concentrically decreases. Conversely, as the velocity of movement increases, the force that a muscle can develop eccentrically increases (Fleck and Kraemer, 2004).

- **Muscle volume.** There is a highly significant positive correlation between cross-sectional area and maximum strength, especially for experienced athletes (Brechue and Abe, 2002). The connection is less pronounced if untrained and in complex exercises because differences in technique can explain a bigger part of the result (Carroll et al., 2001).

- **Neural control (motor unit recruitment and rate of firing) is an important component of muscle strength and a prerequisite for development of muscle hypertrophy (Fleck and Kraemer, 2004).**

- **Metabolic component (the rate at which myosin splits ATP) (Fleck and Kraemer, 2004).**

The two most important factors that can be influenced by strength training are neural adaptations and muscle volume (hyptertrophy) (Fleck and Kraemer, 2004).

**Neural adaptations**

Neural factors can be listed as neural drive (recruitment and rate of firing) to the muscle, increased synchronization of the motor units, increased activation of agonists, decreased activation of antagonists, coordination of all motor units and muscles involved in a movement, and inhibition of the protective mechanisms of the muscle (e.g. Golgi tendon organs) (Fleck and Kraemer, 2004). When a person attempts to produce a maximal contraction, all available motor units are activated. Force can be increased by recruiting more motor units and an increase in motor unit firing rate. It has been suggested that untrained individuals are not able to voluntarily recruit the highest threshold motor units or maximally activate their muscles (Kraemer et al., 1996).

An important part of training adaptation is therefore developing the ability to recruit all motor units in a specific exercise. This is especially important for PFMT because so few people are aware of the PFM or have ever tried to contract the PFM voluntarily. Another important neural adaptation to training is a reduction in antagonist activation. For the PFM it is difficult to say which muscles can be considered antagonists. However, abdominal contraction without a PFM contraction may be an antagonist contraction. An automatic co-contraction of the PFM to counteract any increase in abdominal pressure or the increase from ground reaction force may be considered a goal for training.

The initial quick gains in strength seen after strength training seem to be due to neural adaptation (Sale, 1988).
A 50% increase in muscle strength within only weeks of training is common. This strength gain is much greater than can be explained by muscle hypertrophy (Fleck and Kraemer, 2004). After approximately 8 weeks of regular training, muscle hypertrophy becomes the predominant factor in strength increase, especially in young men. However, muscle hypertrophy also reaches a maximum and plateaus. Then the participants again need to work on neural factors to increase maximum force. Despite minimal changes in muscle fibre size during long-term training in competitive Olympic weightlifters, strength and power increases have been described (Kraemer et al., 1996).

Greater loading is needed to increase maximal strength as one progresses from intermediate to advanced levels of training and loads greater than 80–85% of 1RM are needed to produce further neural adaptations during advanced resistance training (Kraemer and Ratamess, 2004). This is important because maximizing strength, power and hypertrophy may be accomplished only when the maximal numbers of motor units are recruited.

Some of the strength exercises are more difficult to coordinate than others and put the nervous system under greater demands. The potential for neural adaptations to influence the result are therefore higher during such exercises (Chilibeck et al., 1998). The more complex bench and leg press movements compared to the not-so-coordinated difficult arm curl exercise may delay hypertrophy in the trunk and legs (Chilibeck et al., 1998).

According to Shield and Zhou (2004) there is in general only a small room for improvement in fully activating the muscles in healthy people. The amount differs with type of contraction (isometric, dynamic), muscle groups, injuries, degenerations and complexity of movements. But there are still disagreements in the amount of activation, and the effect of strength training. Most studies imply a full activation of most muscles is measured with early twitch interpolation techniques, whereas newer more sensitive techniques reveal that even healthy adults routinely fail to fully activate a number of different muscles despite maximal effort (Shield and Zhou, 2004). Other factors that can only be explained by neural factors are the cross-education effect seen in unilateral training (Munn et al., 2004), and the effect where strength is increased after imagined contractions (Åstrand et al., 2003).

**Hypertrophy**

One of the most prominent adaptations to strength training is muscle enlargement. The growth in muscle size is primarily due to an increase in the size of the individual muscle fibre (Fleck and Kraemer, 2004). According to Fleck and Kraemer (2004), humans have a potential to hyperplasia, but it does not happen on a large scale and is far from the dominating cause of hypertrophy. An increase in the number of muscle fibres has been shown in birds and mammals, but there are limited data to prove this in humans.

The increase in cross-sectional area is attributed to increased size and number of the contractile proteins (actin and myosin filaments) and the addition of sarcomeres within existing muscle fibres. An increase in non-contractile proteins has also been suggested.

Satellite cells and myonuclei may indicate cellular repair after training and the formation of new muscle cells, and the proportion of satellite cells that appear morphologically active, increase as a result of resistance training (Fleck and Kraemer, 2004).

Muscle fibre hypertrophy has been found in both type I and type II fibres after strength training. However, most studies show greater hypertrophy in type II and especially IIA fibres (Kraemer et al., 1995; Green et al., 1999). Genetic factors decide whether a person has predominantly type I or type II muscle fibres, and though transitions from type IIB (now named IIX) to type IIA have been found (Adams et al., 1993; Green et al., 1999; Campos et al., 2002), such changes only seem to happen within fibre type (e.g. not from type II to type I; Fleck and Kraemer, 2004). Cessation of training leads to transitions back from IIA to IIB. But even if most studies fail to find changes in the amount of type I fibres, some strength and sprint studies do. Kadi and Thornell (1999) found that there was a significant increase in the amount of MyHC Ila protein and a significant decrease of the amount of MyHC I and IIB in the trapezius muscle of women in the strength group.

Different muscles have different distributions of fibre types and the total number of muscle fibres varies between individuals. Because the number and distribution of muscle fibres does not seem to be the dominant factor for hypertrophy, and it is impossible to evaluate the number and distribution of muscle fibres in an individual without biopsies, types of muscle fibres should be disregarded as a factor when prescribing PFMT. The aim is to target as many motor units as possible in each contraction.

Greater hypertrophy has been associated with high-volume compared to low-volume programmes (Kraemer and Ratamess, 2004). Short rest intervals have also been shown to be beneficial for hypertrophy and local muscle endurance (Kraemer and Ratamess, 2004). Some studies suggest that a fatigue stimulus with metabolic stress factors has an influence on optimal strength development and muscle growth, even if the mechanisms are unknown. Rooney et al. (1994) found that a 30-second rest between each lift gave a significant lower strength increase than the same amount of repetitions and loads with no rest between the lifts. Maximal hypertrophy may be best attained by a combination of strength and hypertrophy training. One study showed greater increases in cross-sectional area and strength when training was divided into two sessions a day rather than one (Kraemer and Ratamess, 2004).

With the initiation of a strength training regimen, changes in the types of muscle proteins start to take place within a couple of workouts. This is caused by increased protein synthesis, a decrease in protein degradation, or a
combination of both. Protein synthesis is significantly elevated up to 48 hours after exercise (Fleck and Kraemer, 2004). However, to demonstrate significant muscle fibre hypertrophy, a longer training time is required (>8 weeks) (Fleck and Kraemer, 2004). As studies are demonstrating an elevated muscle protein synthesis after an acute strength training bout (Biole et al., 1995; MacDougall et al., 1995; Phillips et al., 1997), the discrepancy seen between increased strength and muscle growth early in a strength training regimen may be more due to methodological problems in measuring small changes in muscle cross-sectional area rather than the traditionally assumed effect of neural adaptation. Another contributing factor that can explain why the role of neural adaptation may be overestimated at the beginning of a strength training programme is an increase of muscle fibre girth at the expense of extracellular spaces (Astrand et al., 2003). In most training studies increase in muscle fibre cross-sectional area ranges from about 20% to 40% (Fleck and Kraemer, 2004). In an uncontrolled PFMT trial Bernstein (1996) found an increase in levator ani thickness of 7.6% at rest and 9.3% during contraction, while the increase in PFM thickness was 15.6% in the randomized controlled trial by Brækken et al. (2010). Is a voluntary PFM contraction a concentric or isometric muscle action? MRI studies (Bo et al., 2001) have shown that there is a movement of the coccyx during PFM contraction. Hence, the contraction is concentric. However, this movement is small and there therefore must be an isometric component of PFMT. It has been suggested that 6s is necessary to reach maximum contraction. However, holding times between 3 and 10s are recommended for isometric contractions (Fleck and Kraemer, 2004). Daily isometric training is superior to less frequent training, but three training sessions per week will bring significant increases in maximal strength. Isometric training alone with no external weights has been shown to increase protein synthesis 49% and muscular hypertrophy of both type I and type II muscle fibres. Twelve weeks of training increased knee extensor cross-sectional area 8% and muscle isometric strength 41% (Fleck and Kraemer, 2004). PFM action is eccentric during increases in abdominal pressure.

DOSE–RESPONSE ISSUES

Dose–response issues deal with how much (or how little) exercise is needed to make a measurable training response (Bouchard et al., 1994; Bouchard, 2001). The dosage can be divided into mode of exercise, frequency, intensity, volume and duration of training. A training response is a progressive change in function or structure that results from performing repeated bouts of exercise, and is usually considered to be independent of a single bout of exercise. However, there is increasing evidence that one bout of exercise can give acute biological responses (Bouchard et al., 1994).

Mode of exercise

Mode of exercise refers to type of training (e.g. strength training, flexibility training, cardiovascular training, and all types of specific exercises for different muscle groups). There is only one way to conduct a PFM contraction (squeeze around the pelvic openings and a lift inwards/forwards). However, the exercises can be conducted in different positions and they can be performed as isometric, concentric and eccentric contractions (Fig. 6.10).

Frequency

Frequency of exercise is usually defined as number of training sessions per week in which a certain muscle group is being trained or a particular exercise performed (Fleck and Kraemer, 2004). Training with heavy loads increases the recovery time needed before subsequent sessions. Most resistance training studies have used frequencies of 2–3 alternating days per week in previously untrained individuals. Power lifters typically train 4–6 days per week (Kraemer and Ratamess, 2004).

Intensity

Intensity of strength training is most often defined as the percentage of maximum (e.g. any given percentage of maximum or different RM resistances for the exercise; Fleck and Kraemer 2004). Intensity is by far the most important factor for effective and quick response to a strength-training programme (Fatouros et al., 2005; American College of Sports Medicine, 2009). Intensity is also one of the most important factors in maintaining the effects of resistance training (Fatouros et al., 2005).

Training intensity has traditionally been synonymous with training load, but it might be the degree of activation and not the training resistance itself that determines the training intensity in strength training (Burd et al., 2012). The minimal intensity that has been shown to increase strength in young healthy individuals is 60–65% of 1RM. However, 50–60% of 1RM has been shown to increase strength in special populations (e.g. older women) (American College of Sports Medicine, 2009).
The following recommendations for intensity were given by Garber et al. (2011):

Muscular endurance: 15–20 repetitions of <50% of 1RM
Power: 8–12 repetitions of 20–50% of 1RM and lighter for older people
Strength: 40–50% of 1RM (or 10–15 RM) for novice older adults and novice sedentary adults, 8–12 repetitions of 60–70% of 1RM for novice/intermediate exercisers and ≥80% of 1RM for experienced strength trainers.

It is important to notice that all of these recommendations were based on studies on extremity muscles and not on the abdominals, back or pelvic floor muscles.

Single sets are recommended for novice and older adults to start with; 2 sets to improve strength and power and ≤2 sets for muscular endurance. Two- to three-minute breaks are recommended between sets.

Performing many repetitions with very light resistance will result in no or minimal strength gain. This is quite contradictory to the recommendations given by Kegel (1956). Although he emphasized to train against resistance, he advised performing at least 500 contractions per day, and for long this was the dominating recommendation for PFMT. Today, however, it is important to use modern evidence-based training principles to gain the best effect. Fewer contractions take less time and may therefore also be much more motivating. Hence exercise adherence may increase.

**Duration**

The duration of the training period (e.g. whether it is 3 weeks or 6 months) influences the results. According to the American College of Sports Medicine (1998), it is reasonable to believe that short-term exercise studies conducted over a few weeks have certain limitations. Several studies have shown that increasing the duration of the exercise period adds substantial improvement in muscle strength.

In a randomized controlled trial (RCT) of PFM strength training to treat female stress urinary incontinence (SUI), Bø et al. (1990) demonstrated an increasing PFM strength in the intensive training group throughout the 6-month training period (Fig. 6.11). Short training periods may therefore not elicit the true effect of exercises. The American College of Sports Medicine (1998) recommends that to evaluate the efficacy of various intensities, frequencies and durations of exercise on fitness variables, a 15–20-week duration is an adequate minimum standard.

**Training volume** is a measure of the total amount of work (joules) performed in a training session, in a week of training, in a month of training or in some other period of time (Fleck and Kraemer, 2004). The simplest method to estimate training volume is to summate the number of repetitions performed in a specific time period or the total amount of weight lifted. More precisely, it can be determined by calculating the work performed (e.g. total work in a repetition is the resistance multiplied by the vertical distance a weight is lifted).

**Periodization** is the planned variation in the training volume and intensity (Fleck and Kraemer, 2004). Variation is extremely important for continued gains in strength and other training outcomes. For improvements to occur, the programme used should be systematically altered so that the body is forced to adapt to changing stimuli. Variation can be achieved by altering muscle actions (isometric, concentric, eccentric), positions, repetitions, load, resting periods and types of exercises.

Adherence (in medical literature often termed compliance) is the extent to which the individual follows the exercise prescription. Adherence is the most important factor influencing outcome and should be reported in all exercise programmes. For theories of adherence and strategies to increase adherence, see Chapter 7.

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**HOW TO INCREASE MUSCLE STRENGTH AND UNDERLYING COMPONENTS**

Four main principles are important in achieving measurable effects of strength training and underlying components: specificity, overload, progression and maintenance.

**Specificity**

The effect of exercise training is specific to the area of the body being trained (American College of Sports Medicine,
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There have been some suggestions that regular physical activity may enhance PFM strength (Bø, 2004). However, the prerequisite for this is that the load put on the pelvic floor by the increased abdominal pressure or ground reaction force is counteracted by an adequate response from the PFM. Obviously, in women with pelvic floor dysfunction the PFM are not co-contracting in adequate time or with enough strength to counteract the increased load. In such cases the PFM are not trained but overloaded and stretched. There therefore needs to be a balance between the degree of loading and the cocontraction of the PFM. A gymnast may have adequate response from the PFM during coughing and light activities. However, landing from a summersault on the bar may be too much load and risk urinary leakage. A small increase in abdominal pressure may therefore be an adequate stimulus for a co-contraction and thereby a ‘training effect’, while a huge increase may cause PFM descent and stretch and weaken the PFM.

Although studies have shown that there are co-contractions of the PFM with hip adductor, gluteal and different abdominal muscle contractions in healthy subjects (Bø and Stien, 1994; Sapsford and Hodges, 2001; Neumann and Gill, 2002) such contractions may not occur in persons with PFM dysfunction and may be weaker than a specific PFM contraction. One should therefore focus on specific PFMT. In addition, Graves et al. (1988) have shown that resistance training should be conducted through a full range of motion for maximum benefit.

Overload

Muscular strength and endurance are developed by the progressive overload principle (e.g. by increasing more than normal the resistance to movement or frequency and duration of activity; American College of Sports Medicine, 2009). Muscular strength is best developed by using heavier weights/resistance (that require maximum or near maximum tension development) with few repetitions, and muscular endurance is best developed by using lighter weights with a great number of repetitions (American College of Sports Medicine, 2009). There are several ways to overload a muscle or muscle group:

- add weight or resistance;
- sustain the contraction;
- shorten resting periods between contractions;
- increase speed of the contraction;
- increase number of repetitions;
- increase frequency and duration of workouts;
- decrease recovery time between workouts;
- alternate form of exercise;
- alternate range to which a muscle is being worked.

The PT can manipulate all the above-listed factors when training the PFM. However, certain important factors are difficult to apply for PFMT (e.g. to add weight and resistance). Plevnik (1985) invented vaginal-weighted cones to make a progression of overload to the PFM (Fig. 6.12). Vaginal cones come in different shapes and weights and are placed above the levator muscle. The patient is asked to start with a weight that she can hold for 1 minute in standing position. The actual training is to try to stay in an upright position with the cone in place for 20 minutes. When the woman is able to walk around with a weight in place for 20 minutes, a heavier weight should replace the one used to make progression in workload. Although correct from a theoretical exercise science point of view this method can be questioned from a practical point of view. In addition, holding a contraction for a long time may decrease blood supply, cause pain and reduce oxygen consumption (Bø, 1995). Many women report that they are unable to hold the cones in place and adherence may be low (Cammu and Van Nylen, 1998; Bø et al., 1999).

Any magnitude of overload will result in strength development, but heavier resistance loads to maximal or near maximal will elicit a significantly greater training effect (American College of Sports Medicine, 2009). Heavy resistance training may cause an acute increase in systolic and diastolic blood pressure, especially when a Valsalva manoeuvre is evoked (American College of Sports Medicine, 2011). This is of importance for PFMT because many women tend to erroneously perform a Valsalva manoeuvre when attempting to perform a PFM contraction. Ferreira et al. (2013) assessed heart rate during PFMT sessions and blood pressure before and after each training session in pregnant women. Heart rate significantly increased during training, but only for a limited time. Increase in blood pressure and heart rate during the training period was within normal ranges. Anecdotally, some women report slight headache, dizziness and discomfort during their first PFMT sessions, and this may be due to an increase in blood pressure or inadequate breathing. Normal breathing during attempts to perform maximum contractions.
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is almost impossible. Therefore, an emphasis on normal breathing between each contraction is important.

Eccentric (lengthening) exercises are effective in increasing muscle strength (Fleck and Kraemer, 2004). However, the potential for skeletal muscle soreness and muscle injury is increased when compared to concentric (shortening) or isometric contractions, particularly in untrained individuals (Fleck and Kraemer, 2004; American College of Sports Medicine, 2009). Eccentric contractions are also more difficult to perform (require more motor skill and muscle awareness) than concentric or isometric contractions, and are therefore not recommended at the beginning of a PFMT programme.

**Progression**

The three principles of progression are overload, variation and specificity.

Progressive overload is defined as ‘continually increasing the stress placed on the muscle as it becomes capable of producing greater force or has more endurance’ (Fleck and Kraemer, 2004: 7). One of the first reports of progression in strength training is from ancient Greece where Milo, an Olympic ‘wrestler’, lifted a calf each day until it reached full growth (DiNubule, 1991).

The American College of Sport Medicine (2002, 2009) recommends that both concentric, eccentric and some isometric muscle actions are used in strength training programmes. For initial training it is recommended that loads corresponding to 8–12 repetitions (60–70% of RM) are used for novice training. For intermediate to advanced training the recommendation is to use a wider range, from 1–12 repetitions (80–100% of 1 RM) in a periodized fashion, with eventually an emphasis on heavy loading (1–6 RM) with rest periods of at least 2–3 minutes between sets, and with a moderate to fast contraction velocity. Higher volume and emphasis on 6–12 RM is recommended for maximizing hypertrophy.

In practice, the principle of progressive overload is the most difficult factor to overcome in PFMT. It is difficult to put weight on the pelvic floor, and therefore other methods need to be used. In most cases the PT has tried to encourage the woman to contract the PFM as close to maximum as possible. This can be done simultaneously with vaginal palpation (feedback) and with any measurement tool in situ (biofeedback). Using biofeedback to reach a maximum contraction can be important from an exercise science point of view. Strong verbal encouragement and motivation seem to be very important in reaching maximum effort. However, the PT should always ensure that the patient is performing a correct contraction and not involving other muscles or increasing abdominal pressure too much. Leaving a patient to train alone is likely to result in loss of the overload and progression because only a few individuals can motivate themselves for maximal efforts. Follow-up, either individual training with the PT or in a class, seems to be a prerequisite for effective training.

Bø et al. (1990) have developed a method for progression in PFMT (see Fig. 6.9). First the patient learns to contract as hard as possible with no holding period, then the patient is encouraged to hold as long as possible, and the third step is to add 3–4 fast contractions on top of the sustained contraction. After this has been accomplished the PT encourages the patient to contract as hard as possible in each contraction.

One way to produce progression is to ask the patient to contract against progressively increasing gravity going from a lying to a standing position (Fig. 6.13). Clinical experience has shown that most women find that PFM contractions are more difficult to conduct in the squatting position (Fig. 6.14). However, it is important that patients choose a position in which they are able to perceive the contraction, and also choose a position in which they feel a certain difficulty when training. In this way they stimulate the CNS and hopefully recruit an increasing number of motor units. In a group training setting the different positions are also used for variation in the training pro-

**Figure 6.13** In the standing position the pelvic floor muscles must contract against gravity, which is more difficult than in a supine or prone position.
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grammé (Bø et al., 1990, 1999). So far, there are no studies comparing the effect of different positions on the development of PFM strength.

Another method to increase progression of the contraction is to use vaginal or rectal devices and ask the patient to hold back when the PT or the patient him- or herself withdraws the device. This method implies eccentric muscle contraction and may be a very effective method to increase strength. However, to date there are no studies comparing this training programme with no exercise or other exercise regimens and one should be aware of the increased risk of injuries and development of muscle soreness in untrained individuals.

There is a need for more research evaluating different ways of adding progressive overload to PFMT.

Initial training status plays an important role in the rate of progression during strength training. Trained individuals have shown much slower rates of improvement than untrained individuals. Kraemer and Ratamess (2004) report that a literature review showed that muscular strength increased approximately 40% in ‘untrained’, 20% in ‘moderately’ trained, 16% in ‘trained’, 10% in ‘advanced’ and 2% in ‘elite’ over periods of 4 weeks to 2 years. The only study looking at the development of PFM strength (see Fig. 6.11) showed a 100% increase after 1 month of exercise. This may be explained by the PFM being totally untrained, and shows a huge potential for improvement. In a meta-analysis Rea et al. (2003) confirmed statistically greater effect sizes in untrained compared to resistance-trained individuals with respect to training intensity, frequency and volume on progression. As the person approaches his or her genetic ceiling, small changes in strength require large amounts of training time.

Maintenance

Maintenance training is work to maintain the current level of muscular fitness. Cessation of exercise training is often termed ‘detraining’. Fleck and Kraemer (2004) described detraining as ‘a deconditioning process that affects performance because of diminished physiological capacity’. Detraining from a muscle strengthening programme will reduce muscle girth, muscle fibre size, short-term endurance and strength/power, whereas capillary density, fat percentage, aerobic enzymes and mitochondrial density will increase (Fleck and Kraemer, 2004). However, following a shorter period of detraining most individuals would still have higher values for these variables than untrained subjects, and physiological functions return quickly with retraining after the detraining period. Strength may be maintained for up to 2 weeks of detraining in power athletes and in recreationally trained individuals strength loss has been shown to take as long as 6 weeks. However, eccentric force and power seem to be more sensitive to detraining effects over a few weeks (Fleck and Kraemer, 2004).

In general, strength gains decline at a slower rate than strength increases due to training. There are few studies, however, investigating the minimal level of exercise necessary to maintain the training effect. A 5–10% loss of muscle strength per week has been shown after training cessation (Fleck and Kraemer, 2004). Greater loss has been shown in the elderly (65–75-year-olds) compared to younger people (20–30-year-olds), and for both groups most strength loss was from week 12–31 after cessation of training.

The rate of strength loss may depend on the duration of the training period before detraining, training intensity, type of strength test used and the specific muscle groups examined. Graves et al. (1988) showed that when strength training was reduced from 3 or 2 days a week to at least 1 day a week, strength was maintained for 12 weeks of reduced training. Reducing training frequency therefore does not seem to adversely affect muscular strength as long as intensity is maintained (Fleck and Kraemer, 2004; Fatourus et al., 2005). In one study, 24 weeks of heavy resistance training three times a week increased vertical jump ability 13%. Twelve weeks of detraining decreased the ability, but it was still 2% above the pretraining value (Fleck and Kraemer, 2004). It is suggested that the ability to perform complex skills involving strength components may be lost if not included in the training programme (Fleck and Kraemer, 2004).

Figure 6.14 Squatting is reported to be a difficult position for contracting the pelvic floor muscles and can therefore be used as a progression in loading.
Electromyography (EMG) studies have shown a change in motor unit firing rate and motor unit synchronization, and that this may cause the initial strength loss in the detraining period. Type II fibres may atrophy to a greater extent than type I fibres during short detraining periods in both men and women (Fleck and Kraemer, 2004). Fleck and Kraemer (2004) concluded that research has not yet indicated the exact resistance, volume and frequency of strength training or the type of programme needed to maintain the training gains. However, studies indicate that to maintain strength gains or slow strength loss the intensity should be maintained, but the volume and frequency of training can be reduced: 1–2 days a week seems to be an effective maintenance frequency for those individuals already engaged in a resistance training programme (Kraemer and Ratamess, 2004).

Only one follow-up study measuring PFM strength after cessation of PFMT has been found. Bø and Talseth (1996) showed that there was no reduction in PFM muscle strength in the intensive training group 5 years after cessation of a RCT. 70% of the women in this group reported strength training of the PFM at least once a week.

RECOMMENDATION FOR EFFECTIVE TRAINING DOSAGE FOR PELVIC FLOOR MUSCLE TRAINING

The American College of Sports Medicine has given the following recommendations for general strength training for (novice) adults (American College of Sports Medicine, 2009):

- target major muscles;
- perform 8–12 slow and moderate velocity, close-to-maximum contractions (even fewer repetitions better to optimize strength and power);
- perform 1–3 sets per exercise;
- exercises should be conducted 2–3 days a week;
- it is difficult to improve at the same rate for long-term periods, e.g. > 6 months, without manipulation of programme variables.

Table 6.3 shows more specific recommendations for strength training regimens to effectively improve muscle strength, power and hypertrophy (Kraemer and Ratamess, 2004). Developing from untrained to intermediate and advanced, the progression is to get closer to maximum contraction and to add more training days per week.

**CLINICAL RECOMMENDATIONS**

- Make sure the patient is able to perform a correct contraction.
- Ask the patient to contract as hard as possible.
- Progress with sustained contractions, and add contractions with higher velocity as a progression.
- Holding time should be 3–10s.
- Recommend PFMT every day.
- Encourage and motivate patients to get as close to maximum contraction as possible. Use strong verbal encouragement.
- Advance to eccentric contractions if possible (no data on effect of eccentric training for the PFM).
- Inform the patient that strength training develops in steps and that the largest improvements come during the first training period. After that the patient needs to work harder to achieve further improvement.

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<thead>
<tr>
<th>Muscle action</th>
<th>Strength</th>
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<td></td>
<td>Eccentric and concentric</td>
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<td>Exercise selection</td>
<td>Single and multiple-joint</td>
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<td>Exercise order</td>
<td>High before low intensity</td>
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<td>Loading</td>
<td>60–70% 1 RM</td>
<td>60–70% for strength 30–60% for velocity/technique</td>
<td>60–70% 1 RM</td>
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<tr>
<td>Volume</td>
<td>1–3×8–12 repetitions</td>
<td>1–3×8–12 repetitions</td>
<td>1–3 sets×8–12 repetitions</td>
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<tr>
<td>Rest intervals</td>
<td>1–2 min</td>
<td>2–3 min for core 1–2 min for others</td>
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<td>Slow to moderate</td>
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<td>2–3 days per week</td>
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Table 6.3 Recommendations for progression of training for strength, power and hypertrophy in novice participants

From Kraemer and Ratamess, 2004.
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7.1 Female stress urinary incontinence

Prevalence, causes and pathophysiology

Jacques Corcos

The most fascinating aspect of medicine is its constant evolution over time. Its progression is based on new findings in our laboratories, new data from clinical research, new imaging techniques, and new views and theories. Looking back at 25 years of urology, it is not very apparent that major advances have been made in understanding the pathophysiology of most of the diseases seen in urology: stones, cancer, sexual and voiding dysfunction, infertility, benign prostate hyperplasia, and even hydrocele. Obvious changes have been introduced in the treatment of these diseases, with sophisticated techniques, such as shock wave lithotripsy, robotic surgery, advanced radiation therapy, precise sperm retrieval and utilization, lasers, mid-urethral slings and new drugs. The overall result of new treatments (associated with better nutrition, prevention, hygiene, etc.) is that, according to the World Health Organization, life expectancy in the Western world has increased significantly over the last quarter century. In other words, we are improving patient care without really understanding our
patient’s diseases. Nevertheless, while we do not have a solid grasp of the pathophysiology of these diseases, we have at least eliminated some old concepts and beliefs that have served their time but were never really supported by high-level evidence from animal and human studies. This is particularly true in the field of female incontinence. We therefore propose to revisit the classical pathophysiology of female incontinence through the critical eyes of a long-standing researcher and clinician who has spent hours listening to others and who is trying today to synthesize what we presently know and do not know, and what we should focus on in research in the next decade.

**PREVALENCE OF SUI**

Most published surveys on the prevalence of incontinence have evaluated it as a whole. Two important studies on urinary incontinence (UI) in Europe and the United States defined it as any leakage occurring in the past 30 days (Kinchen et al., 2003; Hunskaar et al., 2004). Their overall results were congruent, showing average UI prevalence of 35% and 37%, respectively. At 37% and 42%, respectively, stress urinary incontinence (SUI) seemed to be the most prevalent type in these studies, whereas mixed urinary incontinence (MUI) was found in 33% and 46%. Similar numbers were obtained in earlier surveys (Yarnell et al., 1981; Burgio et al., 1991; Hannestad et al., 2000). However, Hampel et al. (1997) undertook a meta-analysis of 48 reports and arrived at a slightly higher SUI incidence of 49%, with only 29% of MUI. Age was an important parameter in prevalence, severity, ‘bothersomeness’ and other variables studied. SUI occurred mainly in young and perimenopausal women (Hunskaar et al., 2004). MUI increased beyond menopause and has become the most ubiquitous type of incontinence in the 7th decade of life.

Difficulties in differentiating between types of incontinence are related to the fact that most epidemiological studies were based on telephone or direct interviews, making the diagnosis of UI incomplete, as urodynamic tests were not performed. This is also due to the fact that most of the patients interviewed understood poorly the classification of incontinence used in these studies.

The main questions are: What is really important to clinicians? Do they need classification? If so, can classification be made easier for patients to understand? If we agree that it is important for physicians to arrive at the right diagnosis and offer the best treatment, it is of little interest to them (in comparison to payers and the pharmaceutical industry) to know the real prevalence of each incontinence type. In other words, simple estimation of the number of people losing urine should be enough. However, it is important for caregivers to know about the ‘bothersomeness’ of incontinence. Sandvik et al. (2000) determined that only 20% of incontinent women were ‘suffering’ from bothersome and serious incontinence.

Finally, the most relevant prevalence for caregivers should be the number of sufferers who seek treatment, although this number may vary according to the type of treatment offered and may also be a good argument for the development of awareness about treatment modalities. Probably, incontinent patients will more readily accept a non-invasive approach, such as pelvic floor exercises or injectables, rather than a surgical procedure, and the number of sufferers seeking therapy may depend on the invasiveness of the intervention proffered. Future epidemiological studies should keep these important considerations in mind, as they explain why a large number of incontinent patients do not consult physicians.

**CAUSES AND PATHOPHYSIOLOGY OF SUI**

From a very simplistic viewpoint, urinary incontinence occurs when bladder pressure rises higher than urethral pressure. Physiologically, this does not happen since bladder compliance on the one hand and adaptability of urethral pressure in response to increased bladder pressure on the other hand makes the equation impossible. Thus, incontinence is always secondary to urethral incapacity to overcome bladder pressure either because there is a limit to what urethral pressure can resist physiologically or because it is weak.

UI is more frequent in women than in men (Hunskaar et al., 2004). Many reasons could account for this gender difference: dissimilarities in the anatomy of pelvic floor muscles and ligaments supporting the bladder and sphincter, the effect of childbirth and maternal injury on pelvic structures and the sphincter, as well as hormones that have receptors in the bladder, sphincter and vaginal area. Finally, genetic factors that are not yet well studied could explain racial and familial incontinence trends.

**General and specific causes**

The pathophysiology of secondary SUI is generally easy to understand.

**Congenital anomalies.** These mainly involve the central nervous system (e.g. myelomeningocele, sacral agenesis, severe scoliosis). Most of these lesions produce neurogenic overactive bladder. However, the lowest lesions, involving the bottom segments of the spinal cord, may evoke cauda equina syndrome with pelvic floor weakness and/or areflexic bladder. Other congenital anomalies (e.g. bladder extrophy) affect the bladder itself and its sphincter mechanism, which is often only partially developed (Koelbl et al., 2002).

**Nervous system injuries and diseases.** These include, e.g. multiple sclerosis, lipomas and other benign or malignant tumours. In a similar line of thought, incontinence in these
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situations is mainly due to neurogenic overactive bladder, but lower lesions, such as disc compression, sacral tumours, sacral injuries and neuropathies (e.g. diabetes mellitus or toxins), are associated with pelvic floor weakness and hypo-functional bladder. With all these lesions, incontinence is related to detrusor overactivity, leading to urge incontinence, overflow in the case of detrusor hypocontractility, or SUI if the pelvic floor is hypofunctional.

**Detrusor anomalies and innervation.** Connective tissue is not an important component of the normal detrusor because smooth muscle cells are arranged closely together (Gosling, 1997). Connective tissue is increased in obstructed bladders, indicating that some smooth muscle fibres convert from a contractile to a synthetic collagen phenotype. Bladder collagen transformation is not seen with ageing. Denervation is observed in both these models, but to a much lesser extent in ageing bladders. Except for the usual changes secondary to ageing, there are no structural bladder wall alterations in women with pure SUI.

**Effect of pregnancy and delivery** on the lower urinary tract. Very little is known about the relationship between delivery, pelvic floor changes and SUI. It is widely recognized that SUI may be a consequence of pregnancy/delivery and that pregnancy usually worsens pre-existing SUI (Hojberg et al., 1999). Transient SUI is relatively common for a few weeks after normal vaginal delivery.

According to Koelbl et al. (2002), vaginal delivery might produce SUI via four major mechanisms:

1. Injury to connective tissue supports by the mechanical process of vaginal delivery.
2. Vascular damage to pelvic structures as a result of compression by the presenting part of the fetus during labour.
3. Wounds to pelvic nerves and/or muscles from trauma during parturition.
4. Direct harm to the urinary tract during labour and delivery. The physiological changes accompanying pregnancy may make women more susceptible to these pathophysiological processes.

Pelvic floor muscle strength decreases after delivery. According to some authors, it returns to the normal range a few weeks later (Peschers et al., 1997). To others, weakness is persistent (Dumoulin et al., 2004). Incontinence seems to be linked to several parameters (e.g. forceps use, labour duration, number of deliveries, pre-existing bladder neck mobility). It also appears that a close relationship exists between pudendal analgesia during labour and the severity of pelvic floor injuries (Francis, 1960; Cutner and Cardozo, 1992). Episiotomies are often reported to worsen post-partum pelvic floor dysfunction. However, evidence is seldom available, and its relationship to SUI is unproven (Hong et al., 1988).

**Ageing.** Incontinence at large is more frequent in the elderly. However, SUI prevalence is relatively decreased because of increased MUI. Ageing in women qualitatively modifies the pelvic floor muscles. Proportional numbers of slow- and fast-twitch muscle fibres change with age, as reported by Koelbl et al. (1989) who biopsied the pelvic floor of elderly, incontinent women. Also, the response to electrical stimulation is decreased and electromyography modified by ageing (Smith et al., 1989). These findings are consistent with the two main classical causes of incontinence: intrinsic sphincter deficiency (ISD) and bladder neck/urethral hypermobility. We strongly believe that SUI in women is always associated with sphincteric deficiency. In our opinion, pelvic floor relaxation eliciting different degrees of prolapse is not the only cause of sphincteric dysfunction; vascular, neurological and myogenic causes are also to blame. This is supported by the fact that numerous women with pelvic prolapse and/or bladder neck hypermobility are not incontinent and therefore have competent sphincters. This viewpoint is shared by Chaikin et al. (1998) and Kayigil et al. (1999).

**Bladder neck and urethral hypermobility**

To be fully functional, the urethra must be supported by a ‘non-elastic’ structure, originally the urethral pelvic ligament, which provides a backboard against increasing abdominal forces compressing the urethra. This is the basis of the ‘hammock theory’ popularized by DeLancey (1994). Such loss of support results in what is classically called urethral hypermobility or rotational descent of the urethra around the pubic bone. For a long time, this defect was considered to be the main cause of SUI. It was also the basis behind the pressure transmission theory (Enhorning, 1960; Athanassopoulos et al., 1994), and the later development of ‘slings’ for the treatment of women with SUI. Lax urethral support could be ascribed to numerous factors, including childbirth, strenuous exercises, pelvic denervation after surgery or trauma, and probably genetic elements that remain to be proven.

The theory of urethral hypermobility is easy to understand, and explains the success of surgery in SUI repair. However, SUI can occur without urethral hypermobility, and failure of surgery is not always associated with recurrence of hypermobility, leaving plenty of room for ISD.

**Intrinsic sphincter deficiency**

The female urethra is a short but complex organ intimately connected to the bladder and pelvic floor structures. Anatomically, it can be isolated and described very precisely (DeLancey et al., 2002), but its functionality cannot be studied separately (Corcos and Schick, 2001).

Besides its proximal smooth muscle sphincteric component and its mid-urethra rhabdosphincter, the urethral wall comprises an outer muscle coat and an inner epithelial membrane continuous with the bladder urothelium. The outer smooth muscle coat extends throughout the
length of the urethra and is essentially made up of longitudinal fibres, whereas circular fibres are rare. Innervation of this coating is mainly parasympathetic, and its function appears to be to shorten and open the urethral lumen during micturition (Ek et al., 1977).

The urethral lamina propria covers the entire length of the urethra. It is lined by the urethral urothelium and lies on a rich layer of vascular plexus and mucous glands, which separates it from the smooth muscle layers. The vascular plexus is important for normal continence and has been shown to be highly sensitive to hormone levels in women (Dokita et al., 1991; Persson and Andersson, 1992). A defect in one of these entities elicits poor closure of the sphincteric urethra and SUI. Loss of sphincteric mass has been clearly demonstrated by different imaging modalities: electromyography, ultrasound and magnetic resonance imaging (Yang et al., 1991; Schaer et al., 1995; Masata et al., 2000).

However, it is hard to believe that urethral sphincter mechanisms, in continuous engagement during a lifetime, can spontaneously become anatomically incompetent. Ageing, through nerve and vascular ‘injuries’, can weaken the sphincter (Koelbl et al., 1989). Nerve and vascular injuries, provoked by declining hormone levels (menopause), pelvic surgery, radiation therapy, neuropathies (e.g. diabetes mellitus, toxins), are the most common causes of sphincteric weakness. Furthermore, a relationship probably exists between hypermobility and ISD. Repeated elongation of muscular fibres of the sphincter and surrounding tissues, including the nerves, may be responsible for sphincteric damage.

**Mixed urinary incontinence**

MUI is even more difficult to understand and, presently, only paper- and pencil-based theories try to explain concomittance of the overactive bladder and defective sphincter. It is even difficult at times to understand a patient who gives hesitant answers to questions, which are supposed to help discriminate between stress and urge incontinence. We have already noted that pelvic floor training can be successful in treating stress and urge incontinence as well as MUI. In addition, we know that two out of three patients with MUI become free of symptoms after surgery directed solely against the stress component. If we add to this the epidemiological picture that stress incontinence is more common in younger women than MUI and urge incontinence (Hannestad et al., 2000), and if we include the fact that the more pronounced stress incontinence they have, the more likely it is that they also have a component of urge incontinence (Bump et al., 2003; Teleman et al., 2004), then the new, emerging picture seems easier to interpret but still does not give an answer about actual causes.

**CONCLUSION**

In conclusion, SUI pathophysiology proposes a model based mainly on two mechanisms: bladder neck mobility and ISD. We believe that ISD is by far the most important component of both conditions, bringing some sense to physical therapy in the treatment of SUI as far as sphincter innervation is preserved. Sphincteric weakness can be evoked by several factors, among which pregnancy/delivery and ageing are generally the most important. Albeit imperfect, this theory has the advantage of clarity and ease of understanding. However, too many elements remain unclear, leaving room for more research on muscle physiology and the effects of ageing and other factors responsible for SUI.

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Pauline Chiarelli

MODIFIABLE FACTORS ASSOCIATED WITH URINARY INCONTINENCE

In examining the relationship between lifestyle factors and pelvic floor dysfunction, available evidence most commonly refers to associations between lifestyle factors and urinary incontinence. However, it seems reasonable to assume that lifestyle factors shown to have a strong association with urinary incontinence might also impact on other symptoms of pelvic floor dysfunction.

Epidemiological studies have shown urinary incontinence to be associated with a number of lifestyle factors or risk factors, some of which might be considered modifiable. Being modifiable, these factors should be of interest to healthcare professionals when developing behavioural interventions aimed at reducing the symptoms of pelvic floor dysfunction. The fact that various peak bodies internationally recommended the inclusion of behavioural interventions aimed at modifying relevant lifestyle factors is testament to the importance of their inclusion within continence promotion.
interventions (NICE, 2006; Landefeld et al., 2008; Abrams et al., 2009).

This section explores the strength of the association between a number of lifestyle factors and pelvic floor disorders and the evidence currently available to support the inclusion of lifestyle changes within continence promotion interventions. The text also outlines some of the principles of behaviour change/health promotion and how these might best be incorporated within continence promotion interventions to help patients adopt relevant behaviours to maximize prescribed lifestyle changes.

Several epidemiological studies have shown a strong association between self-reports of urinary incontinence and lifestyle factors such as obesity (Chiarelli and Brown, 1999; Hunskaar, 2008; Townsend et al., 2008a), physical activity (Nygaard et al., 1994; Bø and Borgen, 2001; Maserejian et al., 2012), smoking (Tampakoudis et al., 1995; Hannestad et al., 2003; Tahtinen et al., 2011) and dietary factors (Brown et al., 1999; Dallosso et al., 2004; Gleason et al., 2013).

EVIDENCE TO SUPPORT THE IMPACT OF LIFESTYLE CHANGES ON SYMPTOMS OF PELVIC FLOOR DYSFUNCTION

The Fourth International Consultation on Incontinence (Abrams et al., 2009) examined the evidence relating to the conservative treatment for urinary incontinence in women, including lifestyle interventions (Hay-Smith et al., 2009). Systematic reviews of the literature pertaining to lifestyle interventions included lifestyle factors: obesity, physical forces (exercise, work) smoking and dietary factors including caffeine, alcohol and fluid intake as well as constipation. Strong evidence in favour of health behaviours or lifestyle changes to reduce pelvic floor muscle dysfunction was not available in most cases. The International Consultation on Incontinence (ICI) was recently updated in the Fifth International Consultation (Abrams et al., 2013). Committee 12 again reviewed the evidence related to lifestyle issues and urinary incontinence (Moore et al., 2013) but included few studies different to those discussed herein.

A summary of the findings of the ICI committee examining several lifestyle interventions and their impact on the management of urinary incontinence are provided here (Hay-Smith et al., 2009). Using the same search strategy, inclusion and exclusion criteria as implemented by the initial reviewers, an update of the relevant literature examining lifestyle interventions from 2005 to the present has been added (Table 7.1).

In preparing the systematic review, levels of evidence and grades of recommendation were decided for each lifestyle factor reviewed.

Levels of evidence

Abbreviated levels of evidence and grades of recommendations used within the ICI recommendations are as follows:

- Level 1: usually involves one well-designed randomized controlled trial (RCT).
- Level 2: includes at least one good-quality prospective cohort study.
- Level 3: good-quality retrospective case–control study.
- Level 4: includes good-quality case series.

Table 7.1: Trials included in the review of lifestyle factors and urinary incontinence

<table>
<thead>
<tr>
<th>Author and lifestyle factor</th>
<th>Litman et al., 2007: the relationship between LUTS and lifestyle and clinical factors</th>
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<tbody>
<tr>
<td>Design</td>
<td>An epidemiological survey – population-based, randomized stratified sample: cluster cells defined by age, gender and race</td>
</tr>
<tr>
<td>Sample size and inclusion criteria</td>
<td>5506 randomly selected community dwelling adults, aged 30–79</td>
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<tr>
<td>Response rate/drop-out Measures</td>
<td>None reported</td>
</tr>
<tr>
<td>Covariates: age, BMI, smoking status, physical activity, alcoholic drinks, depressive symptoms and other self-reported comorbidities</td>
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<tr>
<td>Results</td>
<td>LUTS increased significantly p &lt; 0.001 with age</td>
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<td></td>
<td>Those aged 50–59 years had greatest odds for increased LUTS</td>
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<td></td>
<td>In women there was a significant increase in the odds of LUTS with BMI &gt;30 kg/m² compared with &lt;25 kg/m² in women p = 0.009. BMI was not associated with LUTS in men</td>
</tr>
<tr>
<td></td>
<td>Increased physical activity was associated with a significant decrease in the odds of LUTS (p = 0.003)</td>
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<td>Depressive symptoms were the only factor significantly associated with an increase in the odds of LUTS across gender and racial groups. OR 2.4 (95% CI: 1.9–3.2)</td>
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<tr>
<td>Level of evidence</td>
<td>2</td>
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<table>
<thead>
<tr>
<th>Author and lifestyle factor</th>
<th>Design</th>
<th>Sample size and inclusion criteria</th>
<th>Response rate/drop-out</th>
<th>Measures</th>
<th>Results</th>
<th>Level of evidence provided</th>
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</table>
| Auwad et al., 2008: weight loss | Pilot study, Prospective cohort design | n = 64; BMI >30 kg/m²; urodynamically proven UI | 65% response rate 42 women achieved weight loss >5%; 5 women did not lose the required 5% body weight 17 women dropped out | BMI = weight loss ≥5%; waist circumference; body composition analysis; 24 hour pad-test; KHQ; 3-day FVC; incontinence severity; bladder neck mobility | Mean weight loss = 8.8 kg (SD 5.5)  
Median difference other parameters (CI):  
Body composition analysis 4.7 (4.05–5.55)  
Reduced waist circumference 4 cm (3.0–4.75)  
Pad weight 19 g (13–28)  
Bladder neck mobility: 2.44 cm (1.66–3.34)  
Other measures:  
KHQ: Significant change on all nine parameters.  
Incontinence severity: significant improvement Wilcoxon’s signed rank test p < 0.001  
3-day FVC: nocturia significantly reduced but not frequency  
Significant correlation between pad-test improvement and reduced waist circumference as well as bladder neck mobility  
No correlation between reduced bladder neck mobility and waist circumference | Level 2 |
| Wasserberg et al., 2009: surgically induced weight loss | Prospective cohort study | 82 women who filled out a pre-surgery questionnaire before undergoing bariatric surgery consequently losing more than a 50% of their excess body weight 46 (56%) women agreed to repeat the questionnaires | Pelvic floor distress inventory; pelvic floor impact questionnaire | Prevalence of any pelvic floor dysfunction (mainly urinary symptoms) was 87% before surgery and decreased to 65% after surgery (p = 0.02)  
The trend towards reductions in the prevalence of pelvic organ prolapse and colorectal symptoms did not reach statistical significance | Level 2 |
| Townsend et al., 2008: BMI and waist circumference | Population-based longitudinal study | Data from 1634 women were entered in the analysis – these were a random subset of 1939 women randomly chosen from among 6790 incontinent women from a sample of 35 754 women reported to be continent at baseline 2 years previously | Continence and type, severity and frequency of leaking; BMI; waist circumference | Highly significant linear trends of increasing risk of any, frequent and severe urinary incontinence with both increasing BMI and increasing waist circumference (p for trend ≤0.001)  
In the reference group (women with BMI 21–22.9 kg/m²) the risk of developing at least weekly urinary incontinence was reduced by 19% (CI: 4–32%)  
Significant elevations in risk of 125% (CI: 83–175%) in women with BMI ≥35 kg/m²  
Comparing extreme quintiles of waist circumference (≥37.5 inches vs ≤29 inches) multivariable RR were 2% (CI: 1.65–2.44) for frequent UI and 2.09 (CI 1.51–2.89) for severe UI | Level 2 |
| Subak et al., 2009: weight loss | RCT with a 2-to-1 ratio of assignment between intervention and control. Intensive 6-month behavioural weight loss programme intervention designed to produce an average loss of 7–9% of initial body weight within the first 6 months of the programme | | | |
### Table 7.1 Trials included in the review of lifestyle factors and urinary incontinence—cont’d

| Sample size and inclusion criteria | 338 overweight and obese women aged at least 30 years with a BMI of 25–50 and able to walk unassisted 4 m to approximately 270 m without stopping, with at least 10 UI episodes per week as measured on a 7-day voiding diary |
| Response rate/drop-out | 226 assigned to the weight loss intervention with 5 drop-outs; 112 assigned to the control group with 15 drop-outs |
| Measures | BMI; 7-day voiding diary designed to identify incontinence episodes as predominantly stress and urge or ‘other’ |
| Results | At the 6-month visit, compared with women in the control group the women in the weight loss group had: Significant mean weight loss of 8% of body weight (p <0.001) Mean decrease in the total number of incontinence episodes per week of 47.4% vs 28.1% (p=0.01) Reduction in stress incontinence episodes (p=0.009) Reduction in urge incontinence episodes (p=0.04) |
| Level of evidence provided | Level 1 |

#### Design
A 2-year longitudinal study

#### Sample size and inclusion criteria
2355 older (54–79 years) or women who were continent at survey 1, then incontinent at survey 2

#### Response rate/drop-out
Additional UI information was requested of 80% of incontinent women (n=1193) and returned by 84% of these

#### Measures
Incontinence and its type; physical activity categorized as metabolic equivalent task hours

#### Results
After adjusting for potential confounding factors, increasing levels of total physical activity were associated with decreasing incidence of urinary incontinence (test for trend p<0.01) Women with the highest levels of activity had a 15–20% lower risk of developing urinary incontinence compared with women in the lowest levels of activity

| Level of evidence provided | Level 2 |

#### Author and lifestyle factor
Danforth et al., 2007: physical activity

#### Design
Longitudinal population-based study

#### Sample size and inclusion criteria
70,712 women aged 37–54 years who returned full-length versions of NHS II questionnaire in 2001 and 2003

#### Response rate/drop-out
Supplementary questionnaire sent to 1058 women reporting incident frequent UI with 79.6% response rate

#### Measures
Calculation of long-term activity levels; UI and the amount of leaking

#### Results
Mean age of women was 45.9 years. The median level of physical activities was 17.0 MET hours per week, roughly equal to 5.7 hours per week of walking at an average pace. Among women in the highest quintile of physical activity, the RR for incident incontinence was 0.80 (95% CI: 0.7–0.89). Higher levels of physical activity are associated with 25–30% reduction in the risk of developing stress incontinence

| Level of evidence provided | Level 2 |

#### Author and lifestyle factor
Maserejian et al., 2012: physical activity, smoking and alcohol consumption

#### Design
Longitudinal observational study, with randomly selected participants interviewed face-to-face at baseline and 5 years later

#### Sample size and inclusion criteria
2301 men and 3201 women aged 30–79 years from three racial/ethnic groups

#### Response rate/drop-out
Completed follow-up interviews obtained for 1610 men and 2535 women. Response rate 80.5%

#### Measures
The AUA symptom index (AUA-SI); BMI and waist circumference; physical activity for the elderly; smoking status; alcohol consumption

(Continued)
### Results

7.7% of men and 12.7% of women with no reported LUTS at baseline, reported LUTS at follow-up. A low level of physical activity was associated with a 2–3 times greater likelihood of LUTS. No significant association between LUTS and physical activity in men.

Storage symptoms were twice as likely to develop in women who were current smokers (OR 2.15; 95% CI: 1.30, 3.56; \( p = 0.003 \)) compared to never smokers. No association between smoking and LUTS in men.

There was a no significant association with alcohol consumption and LUTS in men or women.

### Level of evidence provided

Level 2

### Author and lifestyle factor

Tahtinen et al., 2011: smoking in women

### Design

Postal survey, randomly selected participants from the Finnish Population Register

### Sample size and inclusion criteria

2002 women aged 18–79

### Response rate/drop-out

67% response rate

### Measures

Case definitions for stress urinary incontinence (SUI), urgency, and urge urinary incontinence (UUI) were ‘often’ or ‘always’ based on reported occurrence (never, rarely, often, always). Case definitions for urinary frequency were based on reporting of longest voiding interval as less than 2 hours and for nocturia reporting of at least two voids per night.

Current smoking status

### Results

Frequency reported by 7.1%, nocturia 12.6%, SUI 11.2%, urgency 9.7% and UUI 3.1%.

Current smoking was significantly associated with:

- Urgency: OR 2.7 (95% CI: 1.7–4.24, current smokers), and OR 1.8 (CI 95% CI: 1.2–2.9, former smokers) when compared to never smokers.
- Frequency: OR 3.0 (95% CI: 1.8–5.0, current smokers, and OR 1.7 (95% CI: 1.0–3.1, former smokers).

There was no association found between smoking and nocturia. Current heavy smoking compared with light smoking was associated with an additional risk of urgency (OR 2.1, 95% CI: 1.1–3.9) and frequency (OR 2.2, 95% CI: 1.2–4.3).

Suggestion of a dose–response relationship

### Level of evidence provided

Level 2

### Author and lifestyle factor

Tettamanti et al., 2011: coffee and tea consumption

### Design

A population-based study using the Swedish Twin Register and a Web-based survey

### Sample size and inclusion criteria

14094 of 42852 female twins born between 1959 and 1985 and with information related to one urinary symptom and coffee and tea consumption

### Response rate/drop-out

Response rate 66%, \( n = 14094 \)

### Measures

Lower urinary tract conditions based on recommendations from the International Continence Society. Data on coffee and tea consumption categorized into three groups: 0 cups daily, 1 or 2 cups daily, and 3 or more cups daily.

Relevant covariates were age, smoking, parity, BMI.

### Results

Prevalence of overall SUI, UUI and MUI showed a near dose–response relationship with increasing age and with increasing BMI. All types of UI were more prevalent among women with the largest consumption of coffee. Significant associations between coffee intake and all incontinence subtypes except nocturia and OAB. Significant associations between the highest daily tea intake and nocturia (\( p = 0.05 \)) and OAB (\( p = 0.04 \)).

Smokers had lower rates of urinary tract dysfunction compared with non-smokers except for nocturia.

Suggestion of a dose–response relationship

### Level of evidence provided

Level 2

### Author and lifestyle factor

Jura, Townsend et al., 2011: caffeine intake

Townsend, Jura et al., 2011: fluid intake

### Design

Prospective cohort study during 4 years of follow-up

### Sample size and inclusion criteria

65176 women aged 37–79 years without incontinence at baseline

### Response rate/drop-out

Of women who responded to the UI questions at baseline, 93% provided UI information on at least one follow-up questionnaire.
Rating of randomized controlled trials

Methodological quality of RCTs was further rated using the PEDro scale (Maher and Sherrington, 2003) (Table 7.2).

Grades of recommendation

Grades of recommendation are (Abrams et al., 2002):

- Grade A: consistent level 1 evidence, the recommendation being considered mandatory for placement within a clinical care pathway.
- Grade B: based on consistent level 2 or 3 studies or ‘majority’ evidence from RCTs.
- Grade C: based on level 4 studies or most evidence from level 2/3 studies.
- Grade D: evidence is inconsistent/inconclusive or non-existent.

Obesity

Risk-based rationale for including obesity within the review

It seems reasonable to assume that increased body mass index (BMI) might necessarily translate into increased abdominal forces acting upon the bladder itself as well as the pelvic floor. However, increases in measured waist circumference rather than BMI show greater significant association with increased intravesical pressure, supporting the theory that improvements in urinary incontinence after weight loss might be due more specifically to a reduction in the amount of abdominal fat, rather than an overall decrease in BMI (Auwad et al., 2008). The urodynamic characteristics of incontinent, overweight or obese women also support this concept. While increased waist circumference is significantly associated with both increased abdominal pressure and increased intravesical pressure, increased BMI has been shown to be significantly associated with increased abdominal pressure only (Richter et al., 2008).

A systematic review undertaken by Hunskaar (2008) found high-level evidence to support the view that moderate weight loss should be seen as an adequate first-line therapy for urinary incontinence in women. This review highlighted the dose–response effect of increased body mass index and increased waist–hip ratio that results predominantly in symptoms of stress urinary incontinence (including mixed incontinence) rather than symptoms of urge incontinence or overactive bladder (Hunskaar, 2008).

A systematic review that explored community-based prevalence studies using bivariate or multivariate analysis to explore the association of urinary incontinence and increased BMI agreed that there was a clear dose–response effect of weight on urinary incontinence and suggested that for each
5 unit increase of BMI there was an approximate 20–70% increase in urinary incontinence risk (Subak et al., 2009a). Increased BMI has also been implicated in the development of pelvic organ prolapse. Women with increased BMI are more likely to undergo surgery for prolapse than women with a normal BMI (Jelovsek et al., 2007).

ICI summary and recommendation

The Committee determined obesity to be an independent risk factor for incontinence and recommended that massive weight loss significantly decreases urinary incontinence in morbidly obese women (level of evidence: 2), and found level 1 evidence to support the effect of weight loss in women who are moderately obese. Given the evidence of increasing obesity among women, recommendation was also made that weight loss advice should be included within continence promotion interventions. The prevention of weight gain was recommended as having a high research priority (Hay-Smith et al., 2009) (grade of recommendation: A).

Supporting evidence: obesity reduction as a management strategy

A search of the literature related to the impact of reducing BMI or waist circumference on pelvic floor disorders retrieved numerous papers describing interventions aimed at reducing BMI to improve continence status.

In a study exploring the relationships between gender and lifestyle factors related to lower urinary tract symptoms, a significant increase in such symptoms was found in women with a BMI of 30 kg/m² or greater when compared to women with a body mass index of less than 25 kg/m². However, BMI was not associated with any increase in the odds of lower urinary tract symptoms among men (Litman et al., 2007) (level of evidence: 2).

Auwad et al. (2008) undertook a study in 64 obese women with urologically proven urinary incontinence. The study was initially designed to be a randomized controlled trial (RCT). However, when many of the women in the control group independently began dieting and lost weight, the study design, of necessity, became a longitudinal cohort study of the impact of weight loss on the incontinence symptoms of participant women. Participants’ urine loss was measured by pad weight testing and BMI as well as waist circumference were calculated. Forty-two women (65%) achieved a weight loss of at least 5% of initial body weight. A weak but statistically significant correlation was found between reductions in pad-test weight, decreased waist circumference and bladder neck mobility. However, there was no correlation between reduction in BMI and reductions in urinary incontinence measured by pad-test weight (Auwad et al., 2008) (level of evidence: 2).

In a large longitudinal study of women in the United States aged between 54 and 79 years, study participants provided measures of height, weight and waist circumference in the baseline study. For the follow-up study 2 years later women provided the same information along with information related to the onset of incontinence. Of the 35,754 women in this study, 34% were overweight, 17% were obese and a multivariate analysis showed

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<th>Study</th>
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<th>8</th>
<th>9</th>
<th>10</th>
<th>Total score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subak et al., 2009</td>
<td>+</td>
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<td>7</td>
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+, criterion is clearly satisfied; –, criterion is not satisfied; ?, not clear if the criterion was satisfied. Total score is determined by counting the number of criteria that are satisfied, except that ‘eligibility criteria specified’ score is not used to generate the total score. Total scores are out of 10.
highly significant linear trends of increasing the risks of any, frequent and severe urinary incontinence with both increasing BMI and increasing waist circumference. When BMI and waist circumference were included in the same model of analysis, only waist circumference remained a significant predictor of stress incontinence (Townsend et al., 2008a) (level of evidence: 2).

Wasserberg et al. (2009) explored the effect of surgically induced weight loss on pelvic floor disorders in morbidly obese women. A series of 178 women underwent bariatric surgery, 82 of whom achieved at least 50% reduction in excess body weight and 46 (56%) of these women provided follow-up data showing a significant reduction in urinary symptoms. As well as this, there was a non-significant trend toward decreased prevalence of pelvic organ prolapse and measured colorectal symptoms in participant women (Wasserberg et al., 2009) (level of evidence: 2).

A well-designed randomized controlled trial involving 338 overweight and obese women with at least 10 urinary incontinence episodes per week explored the impact of weight loss on symptoms of urinary incontinence. Women in the intervention group reduced the mean weekly number of incontinence episodes by 47% after 6 months on the weight loss programme. Women in the weight loss programme had a greater reduction in their urinary stress incontinence than their symptoms related to overactive bladder (OAB) (Subak et al., 2009b) (level of evidence: 1).

**Physical activity**

**Risk-based rationale for including physical activity within the review**

As increases in BMI must necessarily translate into increases in abdominal forces acting upon the pelvic floor as well as the bladder itself, it might be reasonably assumed that increases in abdominal pressure inherent with some sporting or work activities might also contribute to pelvic floor dysfunction and urinary incontinence.

An exploration of activity, sport and fitness levels among 82 incontinent women aged 28–80 years referred to a hospital gynaecology clinic for treatment of urinary incontinence concluded that women seeking treatment for urinary incontinence report similar levels of physical activity as continent women. Furthermore, the study reported that successful conservative or surgical cure of urinary incontinence did not result in increases in activity levels in the women cured of incontinence in the longer term (Stach-Lempinen et al., 2004).

**ICI summary and recommendation**

The review undertaken by the Fourth ICI concluded that strenuous exercise was likely to unmask symptoms of urinary incontinence in women and that moderate levels of regular physical activity are associated with lower levels of reported urinary incontinence. There was little evidence found to support any negative impact from repetitive occupational heavy lifting on pelvic floor dysfunction. The committee recommendation was for more research to underpin the assumptions that repeated heavy exertion contributes to urinary incontinence and whether alleviating such exertions reduces women’s experience of urinary incontinence or pelvic organ prolapse (Hay-Smith et al., 2009).

**Supporting evidence: changes to physical activity as a management strategy**

A longitudinal analysis of data available from the Nurses’ Health Study of women aged 54–79 years explored women’s activity levels and their risk of developing urinary incontinence and the type of urinary incontinence (Danforth et al., 2007). Activity levels were averaged across all sequential questionnaires and calculated as ‘metabolic equivalent task hours per week’ which were divided into five groups (quintiles). The analysis revealed total physical activity and walking were associated with significantly reduced odds of developing stress urinary incontinence but not symptoms of urge incontinence (Danforth et al., 2007; Townsend et al., 2008b). It seems reasonable to assume that the role played by physical activity in maintaining healthy body weight might make an important contribution to these findings (level of evidence: 2).

Similar results were found by Litman et al. (2007), who studied the association between physical activity and lower urinary tract symptoms. The study involved 2301 men and 3205 women and showed that physical activity directly decreased the odds of lower urinary tract symptoms, particularly in women when comparing study participants with high levels of physical activity against those with low levels of physical activity (Litman et al., 2007). The outcomes of this study are supported by other large prospective studies of the onset of incontinence and attributable factors (Townsend et al., 2008b) (level of evidence: 2).

Another large observational, US population-based longitudinal study among 3201 women and 2301 men again found low levels of physical activity to be associated with a 2–3 times greater likelihood of experiencing lower urinary tract symptoms. Women were 68% less likely to experience lower urinary tract symptoms if they reported high versus low levels of physical activity (Maserejian et al., 2012).

No studies have examined the effect on urinary incontinence of ceasing provocative activities, so the grade of recommendation remains at C.

**Smoking**

**Risk-based rationale for including smoking cessation within the review**

It is commonly held that smokers are more likely than non-smokers to have a chronic cough. Because cough is related to increases in abdominal pressure, coughing might be likely to contribute to the lower urinary tract symptoms.
Evidence-Based Physical Therapy for the Pelvic Floor
dysfunction usually associated with genuine SUI (Bump and McLish, 1994; Hannestad et al., 2003). However, the impact of nicotine on cholinergic detrusor pathways has also been implicated in animal studies (Koley et al., 1984) and a number of studies have associated past and current smokers with both stress urinary incontinence as well as symptoms of OAB (Nuotio et al., 2001; Danforth et al., 2006).

ICI summary and recommendation
The review of lifestyle interventions in relation to smoking concluded that while smoking might increase the chance of more severe urinary incontinence (level of evidence: 3) no studies were found to show that smoking cessation resolves or reduces urinary incontinence. The ICI 2009 suggests the need for prospective studies to determine the impact of smoking cessation on both the onset and the resolution of urinary incontinence (Hay-Smith et al., 2009) (grade of recommendation: C).

Supporting evidence: smoking cessation as a management strategy
A well-conducted, prospective longitudinal analysis of the relationship between a number of lifestyle factors and the onset of SUI and OAB longitudinally over a 1-year period provides a higher level of evidence supporting the effect of smoking on the development of SUI and OAB. Exploration of data from a large, longitudinal population study concluded that smoking might contribute to the development of urinary incontinence in women but not in men (Maserejian et al., 2012) (level of evidence: 2).

Further exploration of the relationship between smoking status, smoking intensity and bladder symptoms among 3000 Finnish women revealed urinary urgency and frequency to be three times more common among current than never smokers. The association found between symptom severity and smoking intensity in study participants suggests a dose–response relationship. This study found no association between smoking and stress urinary incontinence (Tahthinen et al., 2011) (level of evidence: 2).

No studies were found that examined the impact of smoking cessation on symptoms of urinary incontinence. Therefore the grade of recommendation remains at C.

Dietary factors
Risk-based rationale for including dietary factors within the review
A number of dietary factors are of interest with regard to the management of incontinence. These include caffeine, overall daily fluid intake, alcohol and diet as a whole. Each of these factors was reviewed individually by the 2009 ICI in relation to the conservative management of urinary incontinence in women.

Caffeine
Caffeine is the most widely consumed stimulant drug in the world and is well known for its diuretic and stimulant effects (Creighton and Stanton, 1990). The amount of caffeine in beverages varies considerably and daily consumption of highly caffeinated beverages is on the rise (Arya et al., 2000).

The impact of caffeine ingestion in normal, healthy people without lower urinary tract symptoms was studied in a randomized, double blind, placebo controlled trial involving 80 healthy participants who received a twice-daily, standardized dose of caffeine (calculated equivalent to 200 mg in a person weighing 70 kg). While the caffeine induced an initial diuresis in participants, there were no other significant or sustained effects on lower urinary tract in the normal healthy study participants (Bird et al., 2005).

In exploring the impact of caffeine reduction on urinary incontinence, an early RCT by Bryant et al. (2000) compared a bladder training protocol that included reduction of caffeine ingestion to 100 mg per day compared with bladder training protocol without reduction of caffeine intake. Participants in this study experienced a significant reduction in number of voids per 24 hours, reduced occasions of urgency per 24 hours, but the reduction in occasions of leakage per 24 hours did not reach significance (Bryant et al., 2000) (level of evidence: 2).

ICI summary and recommendation
The data related to caffeine intake and urinary incontinence is conflicting with large cross-sectional studies showing no association between caffeine ingestion and urinary incontinence (level of evidence: 3), while small clinical trials suggest caffeine restriction improves symptoms of urgency (level of evidence: 2). The committee recommended caffeine reduction should be included as part of an intervention to reduce bladder symptoms (Hay-Smith et al., 2009) (grade of recommendation: B).

Supporting evidence: caffeine restriction as a management strategy
Population studies related to the impact of caffeine intake on lower urinary tract symptoms provide conflicting results.

A population-based study of 14,031 Swedish twins explored the relationship between coffee and tea consumption and symptoms of urinary incontinence. Women with high coffee intake were found to have lower risk of urinary incontinence compared to women who did not drink coffee. Coffee intake was not found to be related to any specific subtypes of urinary incontinence. However, significant associations were found between high levels of tea consumption and the risk of OAB symptoms and nocturia (Tettamanti et al., 2011) (level of evidence: 2).

A large prospective cohort study involving 65,176 female nurses aged between 37 and 79 years was followed...
longitudinally over 4 years. Caffeine intake and symptoms of urinary incontinence were measured and findings suggest that high (but not low) coffee ingestion was associated with a modest but significantly increased risk of incontinence in women with the highest versus those with the lowest intake (>450 mg daily vs <150 mg daily). The attributable risk of urge incontinence associated with high caffeine intake was calculated to be 25%. It was estimated that the onset of urinary incontinence might be eliminated in 25% of the cases if the high caffeine intake were to be eliminated (Jura et al., 2011) (level of evidence: 2).

Longitudinal data from the same study mentioned above was also used to estimate the association between long-term caffeine intake and the progression of symptoms in 21,564 mildly incontinent women. Baseline caffeine intake and changes in caffeine intake over 4 years were measured, as were their symptoms of incontinence. The percentage of women with progressive symptoms of incontinence was similar across all categories based on the level of caffeine intake. It was therefore concluded that long-term caffeine intake over 2 years did not appear to be associated with the risk of the progression of women's urinary incontinence symptoms (Townsend et al., 2012) (level of evidence: 2).

No studies directly related to the impact of reduced caffeine ingestion on urinary incontinence were found.

While the level of evidence in support of caffeine reduction in the management of urgency, frequency and urge incontinence is strengthening, the recommendation remains at level B.

**Fluid intake**

The average fluid intake of healthy sedentary adults in temperate climates is estimated to be 1220 ml per person per day (Valtin, 2001). Incontinent people manipulate their fluid intake, reducing it in an attempt to prevent leakage episodes. Fluid intake is an important factor related not only to urinary incontinence, but also to bowel health, especially as an adjunct to the prevention of constipation.

**ICI summary and recommendation**

The review team concluded that fluid intake overall plays a minor role in the pathogenesis of urinary incontinence but since reduced fluid intake may lead to dehydration, urinary tract infections and constipation, fluid restriction as an intervention is recommended only in patients with abnormally high fluid intake. Allocated levels of evidence were 2–3 and the grade of recommendation was B (Hay-Smith et al., 2009).

**Supporting evidence: manipulation of fluid intake as a management strategy**

The prospective cohort study of 65,167 female nurses in the United States provided the opportunity to investigate associations between total fluid intake and incident urinary incontinence (including symptoms of stress, urge and mixed urinary incontinence) over 4 years. Comparing the group of women considered to have the highest fluid intake against women considered to have the lowest fluid intake, no significant risk of incident urinary incontinence was found, with higher fluid intake in women suggesting that women should not be encouraged to restrict their fluid intake to prevent the onset of any type of urinary incontinence (Townsend et al., 2011). The grade of recommendation remains at B.

**Alcohol**

**ICI summary and recommendation**

Following a review of a number of studies using unadjusted and adjusted analyses, there appears to be no association between alcohol consumption and urinary incontinence (Hay-Smith et al., 2009). In the face of these findings the ICI Committee allocated no levels of evidence or grade of recommendation.

**Supporting evidence: alcohol reduction as a management strategy**

A longitudinal study with follow-up over 4.8 years included 1610 men and 2535 women and explored lower urinary tract symptoms and a number of lifestyle factors, including alcohol intake, which was assessed by measuring both the type and amount of beverage consumed in the last 30 days. Women drinkers showed no association between alcohol intake and total lower urinary tract symptoms other than nocturia (Maserejian et al., 2012) (level of evidence: 2). (By comparison, men reported as moderate drinkers, i.e. less than one alcoholic drink daily, were more than twice as likely to develop lower urinary tract storage symptoms when compared to men who did not drink alcohol. Men who drank more than moderately were not seen to be at any further increased risk for lower urinary tract symptoms. Grade of recommendation related to men and lower urinary tract storage symptoms is B.)

**Diet**

Although diet might be seen to contribute to obesity and constipation, until now there has been scant evidence to support dietary manipulation in the management of urinary incontinence.

A meta-analysis assessed studies related to the effectiveness of cranberry and blueberry products in preventing symptomatic urinary tract infections and concluded that there was some evidence from four scientifically robust randomized controlled trials that cranberry juice may decrease the number of symptomatic urinary tract infections over a 12-month period, particularly in women with recurrent urinary tract infections (Jepson and Craig, 2007) (level of evidence: 1).
ICI summary and recommendation
The incidence of stress incontinence measured at baseline and followed up over one year was increased in women who consumed more total fat, saturated fatty acids and monounsaturated fatty acids, as well as those who consumed more carbonated beverages, zinc or vitamin B_{12} at baseline. The incidence of stress incontinence was seen to be reduced in women who ate more vegetables and bread and chicken at baseline. A higher intake of vitamin D, protein and potassium have also been associated with decreased onset of overactive bladder in women. (Hay-Smith et al., 2009). However the committee allocated no levels of evidence or grade of recommendation.

Supporting evidence: dietary manipulation as a management strategy
No further evidence related to dietary manipulation as a management strategy for the prevention or management of lower urinary tract symptoms was found.

Constipation
Epidemiological studies have shown associations between constipation and urinary incontinence (Chiarelli et al., 2000), and some early studies showed a clear association between straining at stool and pelvic floor dysfunction (Snooks et al., 1985; Lubowsi et al., 1988). Medical relief of constipation has been shown to significantly improve lower urinary tract symptoms in the elderly (Charach et al., 2001).

ICI summary and recommendation
Although there is evidence to support that chronic straining at stool is a risk factor for urinary incontinence and pelvic organ prolapse, there is no evidence from intervention trials to show that reducing constipation in incontinent patients actually reduces their experience of urinary incontinence (level of evidence: 3). The Committee recommended there was need of further research to explore the role of straining as a causative factor for urinary incontinence (Hay-Smith et al., 2009).

Supporting evidence: reducing constipation as a management strategy
No studies were found to support the resolution of constipation as a management strategy to prevent or reduce lower urinary tract symptoms.

Summary of lifestyle factors associated with urinary incontinence
In the light of available evidence, it seems reasonable that interventions aimed at improving lower urinary tract symptoms should include advice related to modifying relevant lifestyle risk factors. This might include advice about reducing waist circumference, constipation and caffeine consumption, while encouraging increased physical activity levels in incontinent women. Advice about decreasing alcohol consumption would seem to be relevant only for incontinent men.

MOTIVATING LIFESTYLE CHANGES
Just as there are models and theories used to predict and improve adherence to health behaviours, there are models and theories that address the processes of behaviour change. A commonly used definition of a theory is: ‘Systematically organized knowledge applicable in a relatively wide variety of circumstances, devised to analyse, predict or otherwise explain the nature of behaviour of a specified set of phenomena that could be used as the basis for action’ (VanRyn and Heaney, 1992).

Knowing about a problem is insufficient to motivate change. Healthcare professionals commonly believe that simply by telling patients about their condition and likely contributing health behaviours is sufficient to motivate individuals toward changing their health behaviours.

Evidence to the contrary would appear to have had little effect on the way healthcare professionals go about inducing behaviour change in their patients. It is well known that knowledge relating to health risks is not sufficient to encourage people to adopt health behaviours. If knowledge itself were enough, the rates of smoking in developed countries would be minimal as would the health risks associated with elevated BMI.

Individuals are bombarded with enormous amounts of information, which is interpreted through the filters of their past experiences, backgrounds, beliefs, values and attitudes. Human behaviour is complex, and understanding how to encourage behaviour change is even more complex. Many theories have been devised in an attempt to understand and promote changes in health behaviour. All such theories are based on the fact that health is mediated by some behaviour and that health behaviours have the potential to change.

Most behaviour change theories have emerged from the behavioural and social sciences, which in turn have borrowed from disciplines such as sociology, psychology, management and marketing. The theories derived from this variety of disciplines can be used to provide a framework or model that might be used to underpin the planning, adoption and evaluation of health behaviours.

Although some overlap of strategies might be observed, the models described in Table 7.3 are specifically related to health promotion – the adoption of specific health behaviours. In keeping with the evidence presented in relation to continence promotion, modifiable health behaviours
<table>
<thead>
<tr>
<th>Theory and authors</th>
<th>Health Belief Model (HBM) (Becker, 1974)</th>
</tr>
</thead>
</table>
| **Description**    | One of the earliest attempts to explain health behaviour  
The HBM extends the use of psychosocial variables to explain preventive health behaviour by delineating people's subjective perceptions or beliefs about their health  
Numerous studies of the HBM provide substantial empirical support for its usefulness in health education planning  
Evidence supports the effectiveness of this model in developing continence promotion programmes |
| **Key concepts**   | The HBM is based on three essential factors: the readiness of the individual to consider behaviour changes to avoid disease or minimize health risks; the existence of forces in the individual's environment that urge change (cues to action) and make it possible; the behaviours themselves  
The HBM asserts that to undertake a preventive health action, individuals must believe they are susceptible to the incontinence or that severity of present incontinence is likely to worsen; that incontinence and its sequelae are serious; that the action will be beneficial; and that the benefits will outweigh any costs or disadvantages  
Barriers to action  
Cues to action  
Self efficacy – confidence in performing the intervention |
| **Implications for practice** | The following concepts should be explored with the patient, and relevant information supplied:  
Patients’ perceptions of susceptibility, seriousness and progress of their condition. Corrected if unrealistic  
Patients’ understanding of the impact the health behaviour is likely to have on their condition  
Need to agree that the health behaviour will be beneficial and worthwhile  
Barriers to adoption of the health behaviour need to be explored, allowing the patient to suggest how perceived barriers might be overcome  
Reminders need to be instigated to encourage the behaviour  
Patient must demonstrate the required action  
Patient encouraged to set initial, attainable goals related to the behaviour |

<table>
<thead>
<tr>
<th>Theory and authors</th>
<th>Theory of Reasoned Action and Planned Behaviour (Ajzen and Fishbein, 1980)</th>
</tr>
</thead>
</table>
| **Description**    | Developed to explain behaviour that is able to be changed  
Assumes that people make rational, predictable decisions in well-defined circumstances  
Also assumes that the intention to act is the most important determinant of action and all factors relating to the particular action will need to be filtered through the initial intention  
If personal beliefs and social pressures are strong enough, the intention is likely to translate into action  
A person's intentions are likely to be greater if they feel they have enough personal control over the behaviour |
| **Key concepts**   | Attitude towards the behaviour  
Outcome expectations  
Value of outcome expectations  
Beliefs of others  
Motive to comply with others  
Perceived personal control over the behaviour |
| **Implications for practice** | Explore:  
The patient's attitudes to the required behaviour  
What the patient believes the outcome might be  
How important the expected outcome is to the patient  
What impact others might have on the behaviour (e.g. a family attitude to eating more vegetables)  
What the patient believes others will think  
How much control the patient feels in relation to the behaviour |

(Continued)
that might be discussed with patients include restriction of
caffeine, increasing physical activity and maintenance or
reduction of waist circumference. The attention to issues
surrounding BMI and waist circumference, must, of ne-
cessity, involve dietary manipulation as well as increased
activity levels. However, simply telling the patient that
weight loss is likely to improve their bladder symptoms
is unlikely to have any impact unless behaviour modifica-
tion strategies are implemented.

Behaviour modification strategies are based on a series of
evolving theoretical models. Among the theoretical models
that have been developed, some are intended to provide
understanding, whereas others are aimed more specifically
at developing effective intervention protocols. Those mod-
els are most used to develop strategies for use at an individual
level include the Health Belief Model, Theories of Reasoned
Action and Planned Behaviour, the Transtheoretical or

### Table 7.3 Theoretical models of behaviour change and their implications for practice—cont’d

<table>
<thead>
<tr>
<th>Theory and authors</th>
<th>Transtheoretical model (stages of change) (Prochaska and DiClemente, 1984)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Description</td>
<td>Integrates a number of principles and behaviours from other models</td>
</tr>
<tr>
<td></td>
<td>Based on the assumption that an intention to act (or behave) immediately precedes that action or behaviour</td>
</tr>
<tr>
<td></td>
<td>Looks closely at factors related to the intention to perform rather than the behaviour itself</td>
</tr>
<tr>
<td></td>
<td>Assessment of the stage a patient has reached can give an indication of the likelihood that they will comply with intervention requirements</td>
</tr>
<tr>
<td></td>
<td>Most patients seeking help have advanced through the initial stages of change and are in contemplation or preparation stage</td>
</tr>
<tr>
<td>Key concepts</td>
<td><em>Stages of change</em>:</td>
</tr>
<tr>
<td></td>
<td>Precontemplation: consciousness raising</td>
</tr>
<tr>
<td></td>
<td>Contemplation: recognition of the benefits of change</td>
</tr>
<tr>
<td></td>
<td>Preparation: identification of barriers</td>
</tr>
<tr>
<td></td>
<td>Action: the programme or intervention</td>
</tr>
<tr>
<td></td>
<td>Maintenance: recognition that relapse is a strong possibility</td>
</tr>
<tr>
<td>Implications for practice</td>
<td>Discuss the benefits of behaviour change</td>
</tr>
<tr>
<td></td>
<td>Discuss the consequences and progress likely if no changes are instigated</td>
</tr>
<tr>
<td></td>
<td>Allow the patient to identify barriers to behaviour change. Can the patient offer solutions to overcome the barriers?</td>
</tr>
<tr>
<td></td>
<td>Work out tailored intervention</td>
</tr>
<tr>
<td></td>
<td>Allow patient to repeat programme components in their own words to ensure understanding</td>
</tr>
<tr>
<td></td>
<td>Check self-efficacy</td>
</tr>
<tr>
<td></td>
<td>Monitor progress closely</td>
</tr>
<tr>
<td></td>
<td>Use patient-written records (e.g. diary) rather than self-reports for most variables</td>
</tr>
<tr>
<td></td>
<td>Discuss this with the patient and put strategies into place in readiness</td>
</tr>
</tbody>
</table>

- **Theory and authors**: Social Cognitive Theory (Bandura, 1977; Bandura, 1982)

<table>
<thead>
<tr>
<th>Description</th>
<th>Addresses underlying determinants of health behaviour as well as change methods</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Looks at continuous interplay between individual, environment and behaviour</td>
</tr>
<tr>
<td></td>
<td>Adds cognitions to the relationships</td>
</tr>
<tr>
<td></td>
<td>Organizes cognitive and behavioural elements of behaviour change</td>
</tr>
<tr>
<td></td>
<td>Recognizes behavioural reinforcement as external, internal, direct, observational or self-reinforcement</td>
</tr>
<tr>
<td></td>
<td>Healthcare professional seen more as an agent of change than an interventionist by developing patient’s personal competencies</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Key concepts</th>
<th><em>Expectations</em>:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Self control: goal-directed behaviour</td>
</tr>
<tr>
<td></td>
<td>Observational learning: observing the reward for a particular behaviour</td>
</tr>
<tr>
<td></td>
<td>Self efficacy: the belief in the ability to successfully perform the behaviour</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Implications for practice</th>
</tr>
</thead>
<tbody>
<tr>
<td>What does the patient see as a likely outcome from behaviour change?</td>
</tr>
<tr>
<td>Emphasize short-term, tangible benefits to begin with to booster the sense of self control</td>
</tr>
<tr>
<td>Explore the value placed on the outcome especially by peers</td>
</tr>
<tr>
<td>Patient must feel confident of self-control regardless of the environment</td>
</tr>
</tbody>
</table>
| Discuss coping strategies for situations when self-control might be less
Table 7.3 sets out the health behaviour theories and how they might be implemented to optimize continence promotion/behaviour change/lifestyle interventions.

From the table it is clear that the theories presented overlap on a number of issues and, in general, have more in common than not (Nutbeam and Harris, 2004).

In summary, the main points emphasized by the collected theories are as follows:

- Knowledge and beliefs about health. While advocating health education, all theories emphasize the role of individualization – personalizing the information so that it is seen by individuals as relevant and pertinent.
- A patient’s belief in their own ability to do what is asked. Exploring the patient’s feelings of competency in relation to the behaviour and encouraging repeated, well-supervised practice to improve self-efficacy and self-esteem.
- The importance of what is perceived as ‘normal’ by a patient in relation to the influences and values of their social group. The influence of the patient’s social group as a role model, family and peer influences.
- Patients move forward and back along a continuum of change or readiness to change.
- Awareness of the impact of socioeconomic and environmental factors on a patient’s ability to adopt specific behaviours.
- The importance of changing a patient’s environment or perceptions of the environment when it impacts on their progress (Nutbeam and Harris, 2004).

HOW MIGHT LIFESTYLE CHANGES BE ENCOURAGED IN CLINICAL PRACTICE?

Much of healthcare today involves helping patients manage conditions whose outcomes can be greatly influenced by lifestyle or behaviour change. However, healthcare professionals tend to make inappropriate assumptions about patients and behaviour change. These are likely to have a negative impact on the outcome of consultation and include such assumptions as: the patient ‘should’ and therefore ‘wants’ to change, that ‘now’ is the best time for the patient to change, that the healthcare professional is the ‘expert’ and knows what is best for the patient (Emmons and Rollnick, 2001).

To improve the interactions of healthcare professionals with patients related to behaviour change, an excellent technique for negotiating behaviour change in a clinical setting has been developed by Rollnick and Heather (1992). On close examination, this patient-centred interviewing technique appears to be underpinned by a number of the models described earlier. Originally developed to allow motivational interviewing (MI) related to substance abuse, the strategy is easily adaptable to suit any behavioural intervention related to lifestyle changes, and primary care clinicians have reported that the method is acceptable (Rollnick et al., 1997). This method of interviewing has been used successfully by various professions working in the fields of alcohol abuse, diabetes mellitus and tobacco smoking (Rollnick et al., 1999; Sellman et al., 2001) and a systematic review of the efficacy of the method shows it to be superior to other interviewing techniques (Dunn et al., 2001).

The technique is based on the concept of readiness to change and the fact that a patient’s decision to change behaviour is apt to move forward and back along a continuum (Prochaska and DiClemente, 1984; see Table 7.3 above). This ambivalence is one of the main reasons advice-giving has such limited effectiveness. Patients will only accept advice and act upon it when they are ready. They often experience feelings of ambivalence toward behaviour change and using MI techniques provides the opportunity to build rapport with the patient and to explore the perceived importance of behaviour change through their eyes, to provide information if necessary, and also to explore their feelings of confidence (self-efficacy) related to the change in behaviour.

MI requires interviewing skills that are commonly used by healthcare professionals, such as active listening and empathizing. The use of open and closed questioning is also an important component of MI (Emmons and Rollnick, 2001).

The theoretical base of the interview strategy places importance on concepts such as readiness (related to the Stages of Change Model), the importance of the behaviour (related to the Health Belief Model), the patient’s own concepts of beliefs and outcome expectations (related to the Theory of Planned Behaviour), and the patient’s confidence in their ability to change (related to self-efficacy).

In general terms, MI has been shown to work better than ‘usual’ or ‘traditional’ care. Systematic reviews support MI as effective in fields as diverse as diet and exercise, diabetes, hypertension, asthma and oral health (Knight et al., 2006; Martins and McNeil, 2009), each of which might be considered chronic conditions. Problems with bladder and bowel control are also considered chronic conditions but to date no studies are available providing evidence of increased effectiveness specific to continence promotion when MI is incorporated into the intervention. It seems reasonable to assume that healthcare practitioners well trained in MI techniques might easily incorporate them into continence promotion programmes.

The study by Alewijnse et al highlighted the fact that improved implementation of therapists’ counselling skills significantly improved women’s adherence to the pelvic floor muscle exercise programmes (Alewijnse et al., 2003). This confirms the fact that when the healthcare practitioners consciously implement specific counselling skills in which they have been trained, they are likely to optimize adherence to treatment programmes.
While the techniques involved in motivational interviewing may be appealing in their simplicity, practitioners tend to underestimate the complexity of MI and the need for adequate training (Mesters, 2009). Proficiency in effective utilization of MI techniques requires not only initial training but ongoing feedback (Miller and Mount, 2001).

While the focus of responsibility is changed when using MI, and the client is assigned more responsibility, this does not lessen the responsibility of the healthcare professional (Mesters, 2009). Using MI techniques can help both patients and practitioners to talk about behaviour change in less confrontational ways that are likely to stimulate behaviour change.

Proficient, effective MI techniques need to be practised, and well honed. Healthcare professionals willing to undertake training in MI techniques should realize that the training is just the beginning. As with any new skill – practice makes perfect.

**IS THERE EVIDENCE OF THE USE OF BEHAVIOUR MODELS WITHIN CONTINENCE PROMOTION?**

Healthcare practitioners use behavioural interventions on a daily basis without knowing it. Treatment protocols are regularly issued in ‘top down’ manner with the healthcare practitioners assuming that having been given the information, patients will know the importance of changing their behaviour and subsequently proceed to do so. Nothing could be farther from the truth (Rollnick and Heather, 1992).

Many continence promotion programmes incorporate behavioural techniques within their programmes in an ad hoc fashion, but it is important to examine the available supporting evidence within continence promotion.

Chiarelli and Cockburn (1999) used the Health Belief Model as a framework to underpin the development of a successfully implemented postnatal continence promotion programme. The study by Chiarelli and Cockburn also employed social marketing strategies in the development of materials used within the programme. There was a significantly positive trend shown in the proportions of women adhering with pelvic floor exercise protocols at adequate levels in the intervention group when compared with those in the control group (p = 0.001 Mantel Haenzel Chi Square).

There is little evidence, however, to show that other interventions have been based on any of the various models of behaviour change.

When new continence promotion programmes are under development, whether individual treatment protocols for use in a physical therapy practice or continence promotion programmes for use in postnatal women or an aged care setting, it seems rational that they be based on a proven framework such as that provided by the various models. In developing programmes aimed at behaviour change, further formative exploration is necessary to determine various beliefs and perceptions that underlie attitudes, motivation and behaviour. When this has been achieved, more effective health/continence promotion programmes might follow.

**CLINICAL RECOMMENDATIONS**

The following is the menu of strategies suggested as a framework for the interviewing technique that might easily be used within a continence promotion consultation (Rollnick and Heather, 1992; Emmons and Rollnick, 2001; Rollnick et al., 2008).

- **Establishing rapport/introducing the subject.** This provides an understanding of the client’s concerns about the suggested change and allows deeper understanding of the behaviour in the context of the person. The use of open-ended questions demonstrates to the patient that you are concerned about ‘their story’. Explore what they know about the behaviour as it relates to them personally.

- **Raising the subject.** It is important here to check that the patient is happy to talk about the subject.

- **Assessing the patient’s readiness to change.** Ask patients directly how they feel about changing the behaviour. By using such phrases as ‘on a scale of 1 to 10, 1 being absolutely unwilling and 10 being ready, right now, to give it a go’, the patient’s readiness to change can easily be assessed.

- **Provide feedback and raise awareness of the consequences of the behaviour.** Objective data can be introduced at this point, the patient’s need for more information can be explored and their concerns can be discussed, along with their feelings of self-efficacy. Offers of more support should be made at this point, especially if the patient feels little confidence in their ability to achieve the required change. If there is little readiness to change – this should be acknowledged and questions such as ‘what are the things about … [the behaviour] … that concern you?’

- **If the patient seems undecided.** Describe how other patients have coped in the same situation, but be careful to emphasize that ‘the patient knows best’ and support them in whatever decision they make. In some instances, the subject is better postponed until the patient indicates more readiness to change.

The brief description is provided here to show how patients might be encouraged to become active collaborators in changing their health behaviours by using a method of empowerment that is underpinned by the most commonly used theories of behaviour change.
It is important that specialized healthcare providers realize the need for referral to other 'experts in the field'. For example, where weight loss is the desired outcome, brief motivational interviewing within a continence promotion consultation might move the patient toward this behaviour, but referral to a dietician might be in the best interests of the patient. Motivational interviewing has been used successfully in many fields of health promotion and is a powerful tool to enhance communication with patients and guide them in making choices to improve their health, from weight loss, exercise and smoking cessation, to medication adherence.

REFERENCES


Bladder training

Jean F Wyman

INTRODUCTION

Bladder training has been advocated for treatment of overactive bladder (OAB) symptoms (e.g., urgency, frequency, urge incontinence and nocturia) since the late 1960s (Jeffcoate and Francis, 1966). It has also been recommended as a treatment for mixed urinary incontinence and stress urinary incontinence in women (Fantl et al., 1996; Moore et al., 2013). Bladder training is used for highly motivated adult patients without cognitive or physical impairments (Hadley, 1986; Wallace et al., 2009). The goal of bladder training is to restore normal bladder function through a process of patient education along with a mandatory or self-adjustable voiding regimen that gradually increases the time interval between voiding. How bladder training achieves its effects on improving lower urinary tract symptoms is unclear. One hypothesis is that it strengthens the brain’s control over bladder sensations and urethral closure (Fantl et al., 1981, 1991). An alternative hypothesis is that individuals change their behaviour in ways that increase the ‘reserve capacity’ of the lower urinary tract system as they become more knowledgeable of circumstances that cause bladder leakage (Fantl et al., 1991; Wyman et al., 1998).

Bladder training offers the advantages of being simple, relatively inexpensive, and free from unpleasant side-effects (Wyman and Fantl, 1991). This makes it attractive for use in older adults, particularly those with high comorbidity who are already on multiple drug regimens and for whom OAB drug therapy, because of its anticholinergic properties, would place them at higher risk for adverse drug effects (Rowner et al., 2011). Bladder training can be used alone or in combination with drug therapy or with types of nonsurgical treatments such as pelvic floor muscle training (PFMT).

This chapter will describe the evidence base for the use of bladder training in the prevention and treatment of OAB in adults. Comment will be made on the systematic literature reviews of bladder training as well as the methodological quality of individual studies. Clinical recommendations on the use of bladder training in adults with OAB will be provided.

BLADDER TRAINING PROTOCOLS

Bladder training consists of three main components: (1) patient education about the bladder, how continence is maintained and urgency suppression strategies; (2) a scheduled voiding regimen that gradually extends the inter-voiding intervals; and (3) positive reinforcement techniques provided by a healthcare professional (Fantl et al., 1996). How these components are delivered varies considerably in practice. In early bladder training protocols, bladder training (also referred to as bladder discipline, bladder drill, bladder re-education and bladder retraining) was conducted through 5–13 days of hospitalization to ensure mandatory adherence to a strict voiding schedule; voiding off schedule was not permitted even if incontinence resulted (Jeffcoate and Francis, 1966; Jarvis and Millar, 1980; Ramsey et al., 1996). Patients were given anticholinergic drug therapy or sedatives to help cope with severe urgency. In a modification of this approach, Frewen (1979, 1980) found that patients with less severe symptoms could be treated in a 3-month outpatient programme.

A number of variations became incorporated in bladder training programmes as they evolved over the decades. Outpatient protocols became the norm, and they differ in duration from 6 to 12 weeks. The initial voiding interval is established based on the individual’s voiding pattern from their baseline voiding diary, and typically is set at 1 hour. Self-adjustable schedules permit patients to void off schedule with severe urgency if they perceive an incontinent episode is imminent (Wyman and Fantl, 1991). Education on urgency suppression strategies such as distraction and relaxation techniques and/or use of pelvic floor muscle contraction provides patients with specific methods to control urgency episodes. Self-monitoring of voiding behaviour using voiding diaries or logs is frequently used. Fluid and caffeine modifications might be recommended (Ramsey et al., 1996; Bryant et al., 2002); however, in clinical trials this generally has been avoided to test the effect of bladder training as a sole intervention. Advice on constipation prevention (Dougherty et al., 2002), high fibre diets or weight loss reduction diets if appropriate (Ramsey et al., 1996) might also be provided. Alternative delivery strategies have been incorporated into clinical practice or trials, such as a facsimile machine submission of voiding diaries with weekly telephone feedback (Visco et al., 1999), a simplification of the teaching method using a brief written instruction sheet (Mattiasson et al., 2003), use of a programmable electronic voiding timing device (Davila and Primozich, 1998) and group-based teaching methods (Sampselle et al., 2005).

PREVENTION

There are no studies testing bladder training as a sole intervention in the prevention of OAB in adults. Therefore, there is no evidence to base clinical practice decisions on the use of bladder training to prevent or delay OAB.
Evidence-Based Physical Therapy for the Pelvic Floor

TREATMENT

Overview

This section describes the evidence base for bladder training as a treatment for OAB symptoms in adults. Bladder training has been: (1) compared as a sole treatment to no treatment; and (2) compared as a sole treatment to another treatment (nonsurgical or pharmacological); and (3) compared as a sole treatment to its use in combination with a nonsurgical or pharmacological treatment. Comment will be made on the search strategy and selection criteria used in selecting studies included in the evidence base, the systematic literature reviews on bladder training, as well as the methodological qualities of the included studies as they relate to the type of comparison being made using the PEDro Quality Scale (www.pedro.fhs.edu.au).

The following criteria were used to distinguish levels of evidence based on a modification of criteria proposed by Berghmans et al. (2000):

- To conclude there was strong evidence for or against bladder training for OAB patients, at least three high-quality studies with a PEDro score of 6 or greater were needed.
- The conclusion of weak evidence for bladder training required at least three high-quality studies with inconsistent results (e.g., 25–75% considered positive), or at least three low-quality studies with PEDro scores less than 6 with consistent results in favour of bladder treatment.
- To conclude that there is weak evidence against bladder training, there needed to be at least three low-quality studies with consistent results on at least one outcome measure (e.g., urgency, urinary frequency, nocturia, or urge incontinence).
- The conclusion of insufficient evidence was based on low-quality studies with consistent results or with fewer than three studies of whatever quality.

The following computerized databases were searched (1980–2013): OVID MEDLINE, CINAHL, PSYCHINFO and Cochrane Collaboration, using keywords: urinary incontinence, urgency incontinence, overactive bladder, detrusor overactivity, detrusor hyperactivity, detrusor instability, urgency, frequency, nocturia, conservative management, nonsurgical treatment, bladder training, bladder retraining, behavioral therapy, behavioral techniques, adult, aged, randomized controlled trial and clinical trial.

Studies were included if they were randomized controlled trials and met the following criteria: bladder training in at least one treatment arm alone; comparison of bladder training with another treatment in one arm versus a comparison of the other treatment alone; results included for participants with urge incontinence, urodynamic detrusor overactivity (previously diagnosed as detrusor instability), OAB with or without urinary incontinence are reported exclusively or separately from those for participants with mixed urinary incontinence; published full length report; trial report published in English.

Systematic literature reviews

Several systematic reviews have been published that provide qualitative synthesis with evidence grading on bladder training in the treatment of urinary incontinence or urge urinary incontinence (Berghmans et al., 2000; Wallace et al., 2009; Moore et al., 2013). The International Consultation on Incontinence has recently updated its previous reviews (Moore et al., 2013). The Cochrane Collaboration (Wallace et al., 2009) and the Agency for Healthcare Quality and Research (Shamliyan et al., 2007, 2012) published quantitative analyses of randomized controlled trial (RCT) data. Each systematic review varies in its objectives, methodology and the number and type of studies included. These variations contribute to differences in the number of studies reviewed and the conclusions regarding the effect of bladder training.

Berghmans et al. (2000) focused their review on RCTs that assessed physical therapies including bladder training as well as other forms of conservative therapies used in the treatment of urge incontinence. Of the nine bladder training trials they located that met inclusion criteria, they concluded that there was only weak evidence to suggest that bladder training is more effective than no treatment, and that bladder training is better than drug therapy.

The Cochrane Collaboration (Wallace et al., 2009) published an edited review updated from their 2004 review that included quantitative analyses of 12 RCTs (n = 1473 participants) on five pre-specified primary outcomes: (1) participant’s perception of cure of urinary incontinence; (2) participant’s perception of improvement of urinary incontinence; (3) number of incontinent episodes; (4) number of micturitions; and (5) quality of life. Adverse events were also noted. Their review is limited to RCTs with participants who had urinary incontinence; OAB studies where it could not be determined that participants had urinary incontinence were excluded. However, subanalyses did examine urge incontinence as a variable. The review focused on testing three hypotheses:

- Bladder training is better than no bladder training for the management of urinary incontinence.
- Bladder training is better than other treatments (such as conservative or pharmacological).
- Combining bladder training with another treatment is better than the other treatment alone.

The Cochrane Group found there was inconclusive evidence to judge the effects of bladder training in both the short and long term. The results of the trials reviewed tended to favour bladder training in the treatment of urinary incontinence; however, the trials were of variable quality and small size. They found no evidence of adverse effects. They also found no evidence to determine whether first-line therapy should be bladder training or...
anticholinergic drug therapy or whether bladder training was useful as a supplement to another therapy.

The International Consultation on Incontinence (ICI) (Moore et al., 2013) recently updated its previous systematic reviews that addressed a broader set of questions than the Cochrane review:

- What is the most appropriate bladder training protocol?
- Is bladder training better than other treatments?
- Can any other treatment be added to bladder training to add benefit?
- Does the addition of bladder training to other treatments add any benefit?
- What is the effect of bladder training on other lower urinary tract symptoms (LUTS)?

In contrast to the Cochrane review, the ICI included RCTs with participants who had urinary incontinence (urge, stress and mixed incontinence) as well as participants who had OAB without urinary incontinence. Reviews were done separately for women and men, and included evidence from systematic reviews, full-length articles, as well as conference abstracts if there was sufficient information available. Seventeen trials involving 2462 women and five trials with 142 men were included. The ICI concluded that there was no evidence to suggest the most effective method or specific parameters of bladder training. They concluded that from the few trials available in women with urge urinary incontinence, stress urinary incontinence and mixed urinary incontinence, bladder training is more effective than no treatment, but there was insufficient evidence to draw conclusions on its effect in men. They also found that there was insufficient evidence to draw conclusions on the comparative effectiveness of bladder training and drug therapy for women with detrusor overactivity or urge urinary incontinence. Although either might be effective, they did recommend that bladder training may be preferred by some clinicians and women because it does not produce the adverse effects associated with drug therapy. The ICI also found that there was insufficient evidence to draw conclusions on the comparative effectiveness of bladder training and current drug therapy, and the additional benefit of combining drug therapy with bladder training and vice versa.

The Agency for Healthcare Research and Quality (AHRQ) published two recent reviews on urinary incontinence that included a meta-analysis on the effect of bladder training (Shamilyan et al., 2007, 2012). The first review examined the evidence to support specific clinical interventions (e.g., bladder training) in reducing the risk of urinary incontinence in adults residing in community and long-term care settings (Shamilyan et al., 2007). No prevention studies were located for bladder training alone and there was limited evidence on the effectiveness of bladder training in community-dwelling adults.

The second of the AHRQ reviews focused on nonsurgical treatments for urinary incontinence in women (Shamilyan et al., 2012), and addressed several questions on nonpharmacological treatments that were relevant to bladder training. These included how the nonpharmacological treatments affected incontinence, incontinence severity and frequency, and quality of life either alone or combined with drugs; what their effectiveness was in comparison to other treatments; what their harms were when compared to other treatments; and what patient characteristics modified the effects of treatment outcomes and harms. Several outcomes were used in this meta-analysis: urinary incontinence; improvement (50% or greater reduction of urinary incontinent episodes on a 3–7 day diary; 70% reduction improvement on a quality of life scale; or 60% improvement on a global improvement scale).

This review concluded there was a low level of evidence that indicated bladder training improved urinary incontinence when compared to usual care for urge incontinence. When bladder training is combined with PFMT for mixed urinary incontinence, a high level of evidence was found that indicates significant benefits on continence and improvement in urinary incontinence. However, the evidence was low for reducing the bother of urinary incontinence and it was insufficient for improving quality of life.

In comparing bladder training to other forms of treatment, the AHRQ review (Shamilyan et al., 2012) found that continence did not differ between bladder training alone versus bladder training combined with PFMT training for continence or improvement. Continence did not differ between bladder training and PFMT, nor did satisfaction with current urinary incontinence and feelings of no impact from urinary incontinence on quality of life measures. Adverse effects were uncommon. The specific characteristics of women associated with better benefits and compliance to bladder training are unclear.

**Trial comparisons**

Fourteen RCTs on bladder training were located; of these, only 13 (n = 1518; majority female) met the criteria for inclusion in this review. A summary of these trials is presented in Table 7.4 with a rating of their quality using the PEDro scale in Table 7.5. Overall, the PEDro rating of these studies ranged from 2 to 7. With the exception of four trials (Fantl et al., 1991; Wyman et al., 1998; Dougherty et al., 2002; Mattiasson et al., 2003), these trials included sample sizes with groups of fewer than 50 participants.

**Bladder training versus no treatment or control**

Three RCTs of sufficient methodological quality (PEDro scores ≥6) compared the effect of bladder training to no treatment (Dougherty et al., 2002; Fantl et al., 1991; Yoon et al., 2003). The results in two trials favoured bladder training in improving incontinent episodes and the symptoms of urge incontinence as well as urinary frequency, urgency and nocturia (Fantl et al., 1991; Yoon et al., 2003).
### Table 7.4 RCTs on bladder training to treat overactive bladder and/or urge urinary incontinence

<table>
<thead>
<tr>
<th>Author</th>
<th>Bryant et al., 2002</th>
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<tbody>
<tr>
<td><strong>Design</strong></td>
<td>2-arm RCT: BT, BT and caffeine reduction</td>
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<tr>
<td><strong>Sample size and age (years)</strong></td>
<td>95 women and men with urinary symptoms, mean age 57 (SD 17)</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>Clinical assessment time/volume/caffeine charts indicating urinary urgency, frequency, with or without UUI and ingested ≥100 mg caffeine/24 hours</td>
</tr>
<tr>
<td><strong>Training protocol</strong></td>
<td>4-weekly visits BT programme; increase voiding intervals, maintain or increase fluid intake to 2L/24h, urgency control techniques, cease ‘just in case’ voiding</td>
</tr>
<tr>
<td><strong>Drop-out</strong></td>
<td>Not assessed</td>
</tr>
<tr>
<td><strong>Results</strong></td>
<td>No difference between groups on reduction in incontinent episodes/24 h (p=0.219)</td>
</tr>
<tr>
<td></td>
<td>Caffeine reduction with BT led to significantly greater decreases than BT alone in urgency (p &lt;0.002) and urinary frequency (p=0.037)</td>
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<table>
<thead>
<tr>
<th>Author</th>
<th>Columbo et al., 1995</th>
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<tbody>
<tr>
<td><strong>Design</strong></td>
<td>2-arm RCT: BT; oxybutynin</td>
</tr>
<tr>
<td><strong>Sample size and age</strong></td>
<td>81 women aged &lt;65 with UUI aged 24–65 years; mean 48.5</td>
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<tr>
<td><strong>Diagnosis</strong></td>
<td>Clinical assessment; cystometry; cystoscopy; postvoid residual determination; voiding diary</td>
</tr>
<tr>
<td><strong>Training protocol</strong></td>
<td>6-week outpatient programme, initial interval based on maximal voiding interval, encouraged to hold urine 30 min beyond initial voiding interval, progressively increase interval every 4–5 days to reach goal of 3–4 h voiding interval; at appointments every 2 weeks, encouragement and BT advice provided</td>
</tr>
<tr>
<td><strong>Drop-out</strong></td>
<td>BT arm: 2/39 (5.1%); drug arm: 4/42 (9.5%)</td>
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<tr>
<td><strong>Adherence</strong></td>
<td>Not reported</td>
</tr>
<tr>
<td><strong>Results</strong></td>
<td>BT arm: 27/37 (73%) clinically cured (e.g., no UUI or pad use) vs drug arm 28/38 (74%) at 6 weeks</td>
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<td></td>
<td>At 6 months, there were fewer relapses with BT (1/27) vs drug arm (12/28)</td>
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<td></td>
<td>BT clinically cured 8/13 (62%) with detrusor overactivity, 6/8 (75%) with low compliance bladder, and 13/16 (81%) with OAB without detrusor overactivity vs drug arm: 13/14 (93%), 6/9 (67%) and 9/15 (60%) respectively</td>
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<tr>
<td></td>
<td>Significant increase in first desire to void</td>
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<td>BT resolved diurnal frequency 20/29 (69%) and nocturia in 11/18 (61%)</td>
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<td></td>
<td>BT 17/27 (63%) clinically cured with detrusor overactivity returned to stable bladder vs drug arm16/28 (57%)</td>
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<table>
<thead>
<tr>
<th>Author</th>
<th>Dougherty et al., 2002</th>
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<tbody>
<tr>
<td><strong>Design</strong></td>
<td>2-arm RCT conducted in 3 phases; Group 2: BT vs no treatment</td>
</tr>
<tr>
<td><strong>Sample size and age (years)</strong></td>
<td>Women mean age (SD) 67.7 (8.3) based on 218 women</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>Clinical assessment; urinalysis; voiding diary</td>
</tr>
<tr>
<td><strong>Training protocol</strong></td>
<td>6–8-week programme according to Fantl et al., 1991; gradually increasing voiding interval with no times given, constipation prevention education, fluid and caffeine advice if there was a problem noted, positive reinforcement and continence goals decided at onset</td>
</tr>
<tr>
<td><strong>Drop-out</strong></td>
<td>BT arm: 21%; control arm: 15%</td>
</tr>
<tr>
<td><strong>Adherence</strong></td>
<td>Not reported</td>
</tr>
<tr>
<td><strong>Results</strong></td>
<td>In 89 women who underwent BT, 48% reduction in grams urine lost on 24-h pad-test, and 57% reduction in incontinent episodes; unable to determine results compared to control group</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Author</th>
<th>Fantl et al., 1991</th>
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<tr>
<td><strong>Design</strong></td>
<td>2-arm RCT: BT, 6-week delayed treatment</td>
</tr>
<tr>
<td><strong>Sample size and age (years)</strong></td>
<td>131 women aged ≥55 with detrusor overactivity with or without genuine stress incontinence or stress incontinence alone, mean (SD) age 67 (8.5)</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>Clinical assessment; urodynamics; voiding diary ≥1 incontinent episode per week</td>
</tr>
<tr>
<td><strong>Training protocol</strong></td>
<td>6-week outpatient programme, initial voiding schedule based on voiding diary, typically set at 1-h interval during waking hours only; increased by 30 min depending on schedule tolerance; instructed in urgency control strategies; encouraged to avoid voiding off schedule but not prohibited; instructed to maintain usual fluid intake pattern and keep treatment log; at weekly appointments, positive reinforcement, support, and optimism in successful outcome provided</td>
</tr>
</tbody>
</table>
### Table 7.4 RCTs on bladder training to treat overactive bladder and/or urge urinary incontinence—cont’d

| Drop-out | 8/131 (6%) at 6 weeks  
|          | 20/131 (15.3%) at 6 months |
| Adherence Results | Not reported |
| At 6-week follow-up; 12% were continent and 75% had reduced their incontinence 50% or better on voiding diary with results maintained at 6 months |
| In OAB group: frequency – improved for those with ≥5 voids/24 h; nocturia – unchanged; IIQ scores – improved at 6 weeks and maintained at 6 months |

**Author** Jarvis, 1981

**Design** 2-arm RCT; inpatient BT, flavoxate + imipramine

**Sample size and age (years)** 50 women with detrusor overactivity aged 17–78, mean (SD) 46.5 (13.6)

**Diagnosis** Clinical assessment; cystometry; cystoscopy

**Training protocol** Inpatient BT programme (details not provided)

**Drop-out** 5/25 (20%) drug therapy group only

**Adherence** Not reported

**Results** Greater improvement in BT group: 84% became continent and 76% symptom-free vs 56% continent and 48% symptom-free in drug therapy group

Improvements: frequency 78%, nocturia 81%, urgency 84%, urgency incontinence 84%

**Author** Jarvis and Millar, 1980

**Design** 2-arm RCT: inpatient BT, control (e.g., advised that they should be able to hold urine 4 h, be continent and allowed home)

**Sample size and age (years)** 60 women aged 27–79 with detrusor overactivity

**Diagnosis** Clinical assessment; cystometry; urethral dilatation

**Training protocol** Inpatient BT programme; initial voiding schedule typically set at 1.5 h during waking hours only; schedule increases by 30 min daily until 4-h interval reached; instructed to wait to assigned time or be incontinent; encouraged to maintain usual fluid intake and keep a fluid intake record; introduced to patient successfully treated by BT

**Drop-out** None reported

**Adherence** Not reported

**Results** 27/30 (90%) became continent and 25/30 (83.3%) symptom-free

Improvements noted with frequency 83.3%, nocturia 88.8%, urgency 86.7%, and urgency incontinence 80%, which were significantly better than control group (p < 0.01)

**Author** Mattiasson et al., 2003

**Design** 2-arm multicentre RCT: BT + tolterodine, tolterodine alone

**Sample size and age (years)** 501 women and men (75% women) aged ≥18 with OAB with and without urinary incontinence, median age 63

**Diagnosis** Clinical assessment; voiding diary

**Training protocol** Brief written instruction sheet on BT, emphasize bladder stretching through delaying urination with goal to reduce urinary frequency to 5–7/24 h, urgency suppression techniques, keep voiding diary every other week to chart progress, no other training or follow-up by study personnel

**Drop-out** 391/505 (23%); ITT analysis

**Adherence** Subsample of the BT group (n = 95) 68% kept voiding diary for 1 day, 72% at 11 weeks, not reported at 23 weeks; 60% kept diary for 7 days at 1 week, 62% at 11 weeks, and 46/56 (82%) at 23 weeks

**Results** BT yielded greater reductions in number of voids/24 h (p < 0.001) and volume voided (p < 0.0001)

No difference in BT + tolterodine compared to tolterodine alone in number of urgency episodes/24 h, incontinent episodes/24 h, and patient perceptions of symptoms

**Author** Szonyi et al., 1995

**Design** 2-arm double-blinded RCT: oxybutynin + BT vs placebo + BT

(Continued)
In one study, bladder training also led to increased voided volumes (Yoon et al., 2003). One trial did not clearly report outcomes to judge the evidence (Dougherty et al., 2002). Overall, there is weak evidence that bladder training is more effective than no treatment (control) in reducing urinary incontinence and OAB symptoms.

### Bladder training versus other treatments

Six trials were located in which bladder training was compared to other treatments: pelvic floor muscle training (PFMT) in two studies of sufficient methodological quality (Wyman et al., 1998; Yoon et al., 2003), and drug therapy in two studies of sufficient methodological quality (Wiseman et al., 1991; Szonyi et al., 1995) and two studies of low quality (Jarvis, 1981; Columbo et al., 1995). There have been no published trials comparing bladder training to electrical stimulation, incontinence devices, or surgical management.

### Table 7.4 RCTs on bladder training to treat overactive bladder and/or urge urinary incontinence—cont’d

| Sample size and age (years) | 60 adults aged ≥70 (mean age 82.2) with OAB |
| Training protocol | Not specified |
| Drop-out | 8/30 (26.7%) BT+drug group; 5/30 (16.7%) placebo group |
| Adherence | Not specified for BT, 80% for drug and 80% in placebo group |
| Results | Greater reduction in diurnal frequency (p=0.003) and superior subjective benefit (86% vs 55%; p=0.02) in BT and drug therapy group; no difference in nocturia or incontinent episodes |

| Design | 2-arm double-blinded RCT: terodiline+BT vs placebo+BT |
| Sample size and age (years) | 37 adults aged ≥70 (mean age 80.4) with OAB due to detrusor overactivity |
| Diagnosis | Clinical assessment; cystometry; laboratory studies; urine culture; voiding diary |
| Training protocol | Asked to delay bladder emptying for as long as possible whenever they experienced the need to void |
| Drop-out | 1 BT+drug group; 2 BT+placebo group |
| Adherence | Not reported |
| Results | Both groups improved slightly but no difference between groups in micturition frequency and incontinent episodes |

| Design | 3-arm, 2-site RCT: BT, PFMT, BT+PFMT |
| Sample size and age (years) | 204 women aged ≥55 (mean age 61, SD 9.7) with detrusor overactivity with or without genuine stress incontinence or stress incontinence alone |
| Diagnosis | Clinical assessment; urodynamics; voiding diary≥1 incontinent episode per week |
| Training protocol | 12-week outpatient BT programme: 6 week visits (1st 6 weeks), 6 mailed-in logs (2nd 6 weeks). Same training protocol as Fantl et al., 1991 above |
| Drop-out | 11/204 (5.4%) at 12 weeks, 16/204 (7.8%) at 24 weeks |
| Adherence | 57% of clinic visits, 85% adherence to voiding schedule during treatment and 44% to voiding schedule at 24 weeks |
| Results | At 12 weeks, BT+PFMT had less incontinent episodes than BT alone (p=0.004), but by 24 weeks no difference noted between groups. No differences noted in treatment response by urodynamic diagnosis. Women with detrusor overactivity had less symptom distress (p=0.054) and greater improvement in life impact (p=0.03) than those with stress incontinence alone at 12 weeks; no differences at 24 weeks |

BT, bladder training; ITT, intention to treat analysis; IIQ, Incontinence Impact Questionnaire; UUI, urge urinary incontinence. For other abbreviations, see text.
they were much or somewhat better with PFMT than bladder training, the difference did not reach statistical significance after treatment or 3 months later. Similarly, although women in the PFMT group had fewer incontinent episodes per day than those in the bladder training group, the difference was not statistically significant at 3 or 6 months post-treatment (Wyman et al., 1998).

The results in a sufficient quality RCT that compared an 8-week outpatient bladder training programme to a biofeedback-assisted PFMT and a no-treatment control group (Yoon et al., 2003) are difficult to interpret because of low power and unclear reporting of incontinent episodes and between-group changes. No significant differences at post-treatment were found between groups on volume of urine leaked on a clinic pad-test. The bladder training group was found to have a significant decrease in micturition and nocturia, and a significant increase in voided volume; the other two groups did not change significantly. With only two trials, small sample sizes and a limited number of OAB participants, there is only weak evidence that bladder training is more effective than PFMT in the treatment for urge incontinence and OAB.

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+, criterion is clearly satisfied; −, criterion is not satisfied; ?, not clear if the criterion was satisfied. Total score is determined by counting the number of criteria that are satisfied, except that ‘eligibility criteria specified’ score is not used to generate the total score. Total scores are out of 10.

* Based on OAB symptoms (e.g., urinary frequency, nocturia, urinary incontinent episodes).

* Blinded to active vs placebo drug; no studies were able to blind for use of bladder training in protocol.
Four trials compared bladder training alone to bladder training with other treatments including placebo treatments (Wiseman et al., 1991; Szonyi et al., 1995; Wyman et al., 1998; Bryant et al., 2002). Two RCTs were of sufficient methodological quality (Wiseman et al., 1991; Wyman et al., 1998), whereas one RCT was low quality (Bryant et al., 2002).

One trial compared bladder training alone to bladder training with caffeine reduction in adults (combination therapy) with OAB and found that the combination intervention was more successful than bladder training in reducing urgency episodes (Bryant et al., 2002). Although the combination therapy group also had a greater reduction in incontinent episodes, this was not statistically significant and could have been affected by lower power. Overall, there was insufficient evidence to determine whether bladder training and caffeine reduction for individuals who consume more than 100 mg caffeine daily is superior to bladder training alone.

In one of the sufficient-quality RCTs with a relatively large sample size, Wyman et al. (1998) compared a bladder training programme to combination therapy of bladder training and biofeedback-assisted PFMT. Although 94 participants reported urge incontinence at baseline, much fewer actually had urodynamically diagnosed detrusor overactivity (n = 38). The combination therapy group had significantly greater improvements in incontinent episodes and quality of life scores at 12 weeks than the bladder training group; however, by 24 weeks there were no group differences. Because of the relatively small sample size with OAB symptoms alone, there are insufficient data to draw benefit of bladder training in this group, particularly after 24 weeks. It appears that the combination of bladder training and PFMT may lead to longer-term benefit, but additional research is needed.

Bladder training and drug therapy versus drug therapy or placebo
Two trials compared bladder training and oxybutynin to either bladder training and placebo or placebo alone. In a low quality trial, Columbo et al. (1995) found that a 6-week course of 5 mg oxybutynin chloride (immediate release, IR) three times a day had a similar clinical cure rate (e.g., self-reported total disappearance of urge incontinence, no protective pads, or further treatment) as bladder training. The relapse rate at 6 months was higher for the drug group while those in the bladder training group showed better maintenance of their results. In a sufficient quality RCT conducted with older adults, bladder training and oxybutynin (2.5 mg IR twice daily) was superior to a placebo group in reducing daytime frequency and producing subjective benefit (Szonyi et al., 1995). However, there were no differences between groups in reducing incontinent episodes or nocturia.

One large sufficient-quality RCT compared bladder training and tolterodine (2 mg twice daily) to the effect of the drug alone in adults with OAB with and without urge incontinence (Mattiasson et al., 2003). In this trial, bladder training significantly augmented drug therapy resulting in reduced voiding frequency and increased volume per void compared to drug alone. However, there was no difference between groups in their reduction of incontinence episodes and urgency episodes.

Overall, there is weak evidence that indicates augmenting OAB drug therapy with bladder training may be helpful for OAB symptoms. The results tend to favour that bladder training does improve urinary frequency; it is inconclusive, however, that it benefits urge incontinent or nocturia.

CONCLUSION
In summary, the evidence base on bladder training comprises relatively few studies, most with small sample sizes, and of moderate to good methodological quality. There is no evidence to judge the benefit of bladder training as a sole intervention in the prevention of OAB, and only weak evidence to judge its effectiveness in treatment. Bladder training may be helpful in the short-term treatment of OAB in women, but evidence regarding its long-term effects is inconclusive. There is insufficient evidence comparing bladder training to other nonsurgical treatments and current drug therapy. Few trials have reported on adverse events, and only one has reported on adherence. There is weak evidence to guide choices among bladder training, other nonsurgical treatments and current drug therapies. The additional benefits of combining bladder training with other treatments were inconsistent, although it may be beneficial to add caffeine reduction and PFMT to bladder training for patients with OAB. The benefits of combining bladder training with the newer drug therapies for OAB were not consistently noted on all OAB symptoms.

CLINICAL RECOMMENDATIONS
Bladder training has no known adverse effects and can be used safely as a first-line treatment for OAB in women. Its effects might be augmented by caffeine reduction for individuals who have difficulty with urinary urgency and who drink more than 100 mg of caffeine daily.

Bladder training programmes can be successfully implemented in outpatient settings. The ICI has recommended that bladder training be initiated by assigning an initial voiding interval based on the baseline voiding frequency (Moore et al., 2013). Typically, this is set at a 1-hour interval during waking hours, though a shorter interval (e.g., 30 minutes or less) may be necessary. The schedule is increased by 15–30 minutes per week depending on tolerance to the schedule (i.e., fewer incontinent episodes than the previous week, minimal interruptions to the schedule and the individual’s control over urgency). Ideally, the healthcare provider should monitor a patient’s progress on a weekly basis during the training period.
Patient education should be provided about normal bladder control and methods to control urgency, such as distraction and relaxation techniques including pelvic floor muscle contraction. Self-monitoring of voiding behavior using voiding diaries is a useful adjunct to treatment and can help the patient and clinician evaluate adherence to the schedule, evaluate progress and determine whether the voiding schedule should be changed.

Clinicians should monitor progress, determine adjustments to the voiding interval and provide positive reinforcement during the training period. If there is no improvement after 3 weeks of bladder training, the patient should be re-evaluated with consideration given to other treatment options.

REFERENCES


INTRODUCTION

In 1948 Kegel was the first to report pelvic floor muscle training (PFMT) to be effective in the treatment of female urinary incontinence. In spite of his reports of cure rates of over 84%, surgery soon became the first choice of treatment, and not until the 1980s was there renewed interest in conservative treatment. This interest may have developed because of higher awareness among women regarding incontinence and health and fitness activities, cost of surgery and morbidity, complications, and relapses reported after surgical procedures (Fantl et al., 1996).

Although several consensus statements based on systematic reviews have recommended conservative treatment and especially PFMT as the first choice of treatment for urinary incontinence (Fantl et al., 1996; Hay-Smith et al., 2009; Dumoulin and Hay-Smith, 2010; Immamura et al., 2010; Hay-Smith et al., 2011; Herderschee et al., 2011; Moore et al., 2013), many surgeons seem to regard minimally invasive surgery as a better first-line option than PFMT. The scepticism against PFMT may be based on inappropriate knowledge of exercise science and physical therapy, beliefs that there is insufficient evidence for the effect of PFMT, that evidence for long-term effect is lacking or poor, and that women are not motivated to regularly perform PFMT. The aim of this chapter is to report evidence-based knowledge about the above-mentioned points related to PFMT for stress urinary incontinence (SUI).

RATIONALE FOR PFMT FOR SUI

To date, there are two main theories of mechanisms on how PFMT may be effective in the prevention and treatment of SUI (Bo, 2004):

1. Women learn to consciously contract before and during an increase in abdominal pressure, and continue to perform such contractions as a behaviour modification to prevent descent of the pelvic floor.
2. Women are taught to perform regular strength training over time to build up ‘stiffness’ and structural support of the pelvic floor.

There is basic research, case–control studies and randomized controlled trials (RCTs) to support both hypotheses. In addition to these main theories, two other theories have been proposed:

3. Sapsford (2001, 2004) claimed that the PFM was effectively trained indirectly by contraction of the internal abdominal muscles, especially the transversus abdominal (TrA) muscle.

Also, many physical therapists (PTs) claim that there is a fourth theory, named ‘functional training’:

4. ‘Functional training of the PFM’ means that women are asked to conduct a PFM contraction during different tasks of daily living (Carriere, 2002).

Evidence for theory 1

By intentional contraction of the PFM before and during an increase in abdominal pressure there is a lift of the pelvic floor in a cranial and forward direction and a squeeze around the urethra, vagina and rectum (Kegel, 1948; DeLancey, 1990, 1994a, 1994b, 1997). Ultrasonography and MRI studies have verified a lift in a cranial direction and movement of the coccyx in a forward, anterior and cranial direction (Bo et al., 2001; Thompsen and O’Sullivan, 2003). Miller et al. (1998) named this voluntary counterbracing-type contraction ‘the Knack’, and in a single-blind randomized controlled trial (RCT) showed that the Knack performed during a medium and deep cough reduced urinary leakage by 98.2 and 73.3%, respectively. Cure rate in ‘real life’ was not reported. Also research on basic and functional anatomy research supports the Knack as an effective manoeuvre to stabilize the pelvic floor (Miller et al., 2001; Peschers et al., 2001c). However, to date there are no studies on how much strength is necessary to prevent descent during coughing and other physical exertions, and we do not know if regular counterbracing...
during daily activities is enough to increase muscle strength or cause morphological changes of the PFM.

Evidence for theory 2

Kegel (1948) originally described PFMT as physiological training or ‘tightening up’ of the pelvic floor. The theoretical rationale for intensive strength training (exercise) of the PFM to treat SUI is that strength training may build up the structural support of the pelvis by elevating the levator plate to a permanent higher location inside the pelvis and by enhancing hypertrophy and stiffness of the PFM and its connective tissue. This would facilitate a more effective automatic motor unit firing (neural adaptation), preventing descent during an increase in abdominal pressure. The pelvic openings and the levator hiatus may narrow and the pelvic organs are held in place during increases in abdominal pressure. In addition, a pelvic floor located at a higher level inside the pelvis may yield a much quicker and more coordinated response to an increase in abdominal pressure, closing the urethra by increasing the urethral pressure (Constantinou and Govan, 1981; Howard et al., 2000).

Ultrasound studies have shown that parous women have a more caudal location of the pelvic floor than nulliparous women (Peschers et al., 1997). Difference in anatomical placement has also been shown between continent and incontinent women (Miller et al., 2001; Peschers et al., 2001a).

In an uncontrolled study by Bernstein (1997), a significant increase in muscle volume after training was shown by ultrasound. However, due to the lack of a control group, more research is needed to provide conclusive evidence that muscle hypertrophies after PFMT.

None of the strength training RCTs on SUI has evaluated the effect of PFMT on PFM tone or connective tissue stiffness, position of the muscles within the pelvic cavity, their cross-sectional area or neurophysiological function. However, in an uncontrolled trial of PFMT for SUI, Balmforth et al. (2004) found that the position of the bladder neck was observed by ultrasound to be significantly elevated at rest, and during Valsalva manoeuvre and squeeze after 14 weeks of supervised PFMT and behavioural modifications. In an assessor blinded RCT of PFMT in women with pelvic organ prolapse (POP), Brøkken et al. (2010) found statistically significant changes in PFM strength, thickness, muscle length, levator hiatus area and position of the rectal ampulla and bladder neck in favour of the PFMT group compared to the control group. As the results were found in POP women there is a need for a similar study in women with SUI. However, since the results were consistent for all morphological changes in this more complicated group to treat, one may assume that even better results will be found in a group with SUI only.

In some studies the patients were tested both subjectively and objectively during physical activity, and had no leakage during strenuous tests after the training period (Bø et al., 1990a; Bø et al., 1999; Mørkved et al., 2002). Therefore, the effect most likely was due to improved automatic muscle function and not only ability to voluntarily contract before an increase in abdominal pressure. Brøkken et al. (2010) found that there was less increase in the levator hiatus area during Valsalva in the PFMT group compared to the control group and suggested that this may be due to increased automatic function of the PFM.

Evidence for theory 3

Sapsford (2001, 2004) suggests that the PFM can be trained indirectly by training the TrA muscle. This is based on an understanding that the PFM are part of the abdominal capsule surrounding the abdominal and pelvic organs. The structures included in this capsule (often referred to as the ‘core’) are the lumbar vertebrae and deeper layers of the multifidus muscle, the diaphragm, the TrA and the PFM (Sapsford, 2001, 2004).

Several studies have shown that different abdominal muscles co-contract during PFM contraction (Bø et al., 1990b; Bø and Stien, 1994; Neumann and Gill, 2002; Peschers et al., 2001b; Sapsford et al., 2001). In addition, some studies have shown that there is a co-contraction of the PFM during different abdominal muscle contractions in healthy volunteers. Bø and Stien (1994), using concentric needle EMG, found that there was a co-contraction of the PFM during contractions of the rectus abdominis in continent women. Sapsford and Hodges (2001) found that PFM surface electromyography (EMG) increased with TrA contractions in six healthy females, and this was supported by a study of four continent women by Neumann and Gill (2002). In continent women, Sapsford et al. (1998) found that a sustained isometric abdominal contraction termed ‘hollowing’ in which the TrA and internal obliques are contracted increased the urethral pressure as much as a maximal PFM contraction. However, they had also ensured that the women were simultaneously contracting the PFM. Based on these findings, Sapsford (2001, 2004) recommends that incontinence training should begin by training the TrA, rather than the PFM specifically.

To date there are no RCTs comparing the effect of indirect training of the PFM via TrA on SUI with either untreated controls, conscious precontraction of the PFM or strength training. However, Dumoulin et al. (2004) compared PFMT with PFMT and TrA training, and did not find any further benefit of adding TrA training to the protocol. In a systematic review Bø and Herbert (2013) analysed the effect of alternative exercise regimens for the PFM and found three RCTs for abdominal training, two RCTs for Pilates and two RCTs for the Paula method. None of the alternative exercises proved to be better than or yielded additional effect to PFMT. No RCTs were found for the effect of yoga, Tai Chi, balance, posture or respiration exercises to prevent or treat SUI in women.
Evidence for theory 4

In some physical therapy practices the PFMT protocol seems to include only teaching the patients to co-contract the PFM with low load during all daily activities and movements (Carriere, 2002). No specific strength training protocol or follow-up training is undertaken. This can be considered using the same theory as use of conscious pre-contraction, or the Knack. However, unlike the use of a conscious contraction, the idea is that by learning to contract, over time this may become an automatic function and by itself be enough to prevent SUI. Therefore, in ‘functional training’ the conscious contraction is further developed to be performed in all daily activities where leakage may occur. This means that the woman is asked to contract while lifting, doing housework, playing tennis, etc.

Because it is possible to learn to hold a hand over the mouth before and during coughing, it is perhaps possible to learn to precontract the PFM before and during simple and single tasks such as coughing, lifting and performing abdominal exercises. However, multiple task activities and repetitive movements such as running, playing tennis, or participating in dance and aerobic activities most likely cannot be conducted with intentional co-contractions of the PFM. To date, there are no basic studies, case–control studies, uncontrolled studies or RCTs to support the use of this kind of functional training of the PFM.

METHODS

Only outcomes from RCTs are included. Computerized search on PubMed, studies, data and conclusions from Clinical Practice Guideline (AHCPR, USA) (Fandl et al., 1996), the Fifth International Consultation on Incontinence (ICI) (Moore et al., 2013), and Cochrane library and other systematic reviews (Herbison and Dean, 2009; Dumoulin and Hay-Smith, 2010; Immamura et al., 2010; Herderschee et al., 2011; Hay-Smith et al., 2011) have been used as background sources. Physical therapy techniques to treat SUI include PFMT with or without biofeedback, electrical stimulation and cones. Because SUI and urge incontinence are different conditions that most likely need different treatment approaches, only studies including female SUI are presented here. Methodological quality of RCTs reporting cure rates assessing the condition with pad-tests are judged by use of the PEDro rating scale (Herbert and Gabriel, 2002).

EVIDENCE FOR PFMT TO TREAT SUI

Updated and comprehensive systematic reviews on PFMT in the treatment of SUI with detailed tables can be found in the Cochrane library (Herbison and Dean, 2009; Dumoulin and Hay-Smith, 2010; Herderschee et al., 2011; Hay-Smith et al., 2011) and the ICI consensus (Moore et al., 2013) so we will not repeat the same detailed tables of each RCT here. We refer to the same studies and newer studies found in our updated search in the text, and urge the reader to stay updated with new studies through the Cochrane library and the PEDro database.

It is difficult to make meaningful comparisons between studies and groups of studies in this area because there is a great heterogeneity between studies. This heterogeneity involves inclusion criteria of the studies (several studies include women with SUI and urge and mixed incontinence), different outcome measures, and different exercise regimens with a huge variety of training dosage. In addition, many researchers have used combined interventions (e.g. electrical stimulation and strength training, bladder training and strength training).

In this textbook, unlike what was done in most of the Cochrane systematic reviews, we have made different decisions with respect to inclusion and exclusion criteria and how to present data.

• First, the Cochrane group made a decision to combine studies with a diagnosis of SUI, urge and mixed incontinence, whereas we have chosen to attempt to report data by separate diagnosis.

• Second, the Cochrane group decided not to analyse data based on measurement of urine loss by pad-tests (except for the review by Dumoulin and Hay-Smith, 2010). In our point of view this excludes results from many high-quality studies, and abolishes the opportunity to look at cure rates at the disability level of the International Classification of Functioning, Disability and Health (ICF) (WHO, 2001).

• Third, in the Cochrane review there is no evaluation of the quality of the intervention. There is a dose–response relationship in exercise therapy. Therefore, a thorough discussion of the quality of the intervention is necessary to elaborate a correct cause–effect relationship found or not found in RCTs of PFMT.

One important flaw in PFMT studies is a lack of ability to contract the PFM. Several research groups have shown that over 30% of women are unable to voluntarily contract the PFM at their first consultation even after thorough individual instruction (Kegel, 1952; Benvenuti et al., 1987; Bo et al., 1988; Bump et al. 1991). Hay-Smith et al. (2001) reported that ability to contract PFM was checked before training in only 15 of 43 RCTs on the effect of PFMT for SUI, urge and mixed incontinence. Common mistakes are to contract other muscles such as abdominals, gluteals and hip adductor muscles instead of the PFM (Bo et al., 1988; Bo et al., 1990b). In addition, Bump et al. (1991) showed that as many as 25% of women may strain instead of squeeze and lift. If women are straining instead of performing a correct contraction, the training may harm and not improve PFM function. Proper assessment of ability to contract the PFM is therefore mandatory (see Fig.7.1 and Vaginal palpation in Chapter 5).
Female pelvic floor dysfunctions and evidence-based physical therapy

Chapter

The numerous reports by Kegel, with over 80% cure rate comprised uncontrolled studies with the inclusion of a variety of incontinence types and no measurement of urinary leakage before and after treatment. However, since then, several RCTs have demonstrated that PFM exercise is more effective than no treatment to treat SUI (Henalla et al., 1989, 1990; Hofbauer et al., 1990; Wong et al., 1997; Miller et al., 1998; Bø et al., 1999). In addition, a number of RCTs have compared PFMT alone with either the use of vaginal resistance devices, biofeedback or vaginal cones (Herbison and Dean, 2009; Herderschee et al., 2011; Hay-Smith et al., 2011; Moore et al., 2013). Of all the RCTs, only one did not show any significant effect of PFMT on urinary leakage (Ramsey and Thou, 1990). Interestingly, in this study there was no check of the woman’s ability to contract, adherence to the training protocol was poor, and the placebo group contracted gluteal muscles and external rotators of the hips; activities that may give co-contractions of the PFM (Bø et al., 1999b; Peschers et al., 2001b).

Combined improvement and cure rates

As for surgery (Dmochowski et al., 2013) and pharmacology studies (Andersson et al., 2013), a combination of cure and improvement measures is often reported. To date there is no consensus on what outcome measure to choose as the gold standard for cure (urodynamic findings of SUI, number of leakage episodes, ≤2 g of leakage on pad-test [tests with standardized bladder volume, 1-hour, 24-hour, and 48-hour], women’s report, etc.) (Blaivas et al., 1997; Hilton and Robinson, 2011). Subjective cure and improvement rates of PFMT reported in RCTs in studies including groups with SUI and mixed incontinence vary between 56% and 70% (Moore et al., 2013).

Cure rates for SUI

It is often reported that PFMT is more commonly associated with improvement of symptoms, rather than a total cure. However, short-term cure rates of 44–70%, defined as ≤2 g of leakage on different pad-tests, have been found after PFMT (Henalla et al., 1990; Wong et al., 1997; Bø et al., 1999; Mørkved et al., 2002; Dumoulin et al., 2004; Zanetti et al., 2007). Table 7.6 describes these studies and Table 7.7 gives the methodological quality of the same studies. The highest cure rates were shown in two single-blind RCTs of high methodological quality. The participants had thorough individual instruction by a trained PT, combined training with biofeedback (BF) or electrical stimulation (ES), and close follow-up once or every second week. Adherence was high, and drop-out was low (Dumoulin et al., 2004; Mørkved et al., 2002). Because biofeedback and electrical stimulation have not shown any additional effect to PFMT in RCTs and systematic reviews (biofeedback RCTs being flawed because of difference in training dosage in favour of the biofeedback intervention) (Herderschee et al., 2011; Moore et al., 2013), one could hypothesize that the key factors for success are most likely close follow-up and more intensive training.

Quality of the intervention: dose–response issues

Because of use of different outcome measures and instruments to measure PFM function and strength, it is impossible to combine results between studies, and it is difficult to conclude which training regimen is the more effective. Also the exercise dosage (type of exercise, frequency, duration and intensity) varies significantly between studies (Moore et al., 2013). Looking into the studies on SUI patients included in the Cochrane systematic review, duration of the intervention varies between 6 weeks and 6 months, intensity (measured as holding time) varies between 3 and 40 s, and number of repetitions per day between 36 and over 200. Frequency of training is every day in all RCTs (Hay-Smith et al., 2001a).
### Table 7.6 Cure rates reported as less than 2 g of leakage measured with a variety of pad-tests in RCTs of PFMT to treat stress urinary incontinence

<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td><strong>Design</strong></td>
<td>Randomized to PFMT, interference therapy, oestrogen, or control</td>
<td>Randomized to PFMT, oestrogen, control</td>
<td>Randomized to PFMT with PT or PFMT with biofeedback</td>
<td>Randomized to clinic-based PFMT or home-based PFMT</td>
</tr>
<tr>
<td><strong>Sample size and age (years)</strong></td>
<td>104 women; mean age with variance not reported</td>
<td>26 postmenopausal women, mean age 54 (49–64)</td>
<td>40 women, mean age 45 (range 40–48)</td>
<td>47 women, mean age 48.8 (SD 9.4)</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>Urodynamic stress incontinence</td>
<td>SUI on history</td>
<td>Urodynamic SUI</td>
<td>Urodynamic SUI</td>
</tr>
<tr>
<td><strong>Training protocol</strong></td>
<td>PFMT: vaginal palpation, contract PFM 5x/h, hold 5 s; 10 sessions once a week with PT</td>
<td>6-week intervention</td>
<td>4-week intervention; vaginal palpation; both groups asked to perform PFMT at home at least 3x/day</td>
<td>4-week training period</td>
</tr>
<tr>
<td></td>
<td>Interference: 10 sessions with PT, 0–100 Hz, 20 min</td>
<td>PFMT: protocol not explained</td>
<td>PFMT + PT: individual treatment with PT 3–4x/day</td>
<td>Clinic: 8 sessions plus daily PFMT</td>
</tr>
<tr>
<td></td>
<td>Oestrogen: Premarin vaginal cream each night for 12 weeks (1.25 mg)</td>
<td>Oestrogen: Premarin vaginal cream 2 g/night</td>
<td>PFMT + BF: individual treatment as above with addition of four times with BF</td>
<td>Home: daily PFMT at home</td>
</tr>
<tr>
<td><strong>Drop-out</strong></td>
<td>4/104: not reported from which groups</td>
<td>Not reported</td>
<td>Not reported</td>
<td>Not reported</td>
</tr>
<tr>
<td><strong>Adherence</strong></td>
<td>Not reported</td>
<td>Not reported</td>
<td>Not reported</td>
<td>Not reported</td>
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<tr>
<td><strong>Results</strong></td>
<td>65% cured or &gt;50% reduction</td>
<td>PFMT: 50% cured or &gt;50% reduction</td>
<td>PFMT + PT: 25%</td>
<td>PFMT: 50% cured or &gt;50% reduction</td>
</tr>
<tr>
<td></td>
<td>Oestrogen: 0</td>
<td>Control: 0</td>
<td>PFMT + BF: 5%</td>
<td>Oestrogen: 0</td>
</tr>
<tr>
<td></td>
<td>Control: 0</td>
<td>Oestrogen: 0</td>
<td>PFMT + BF: 58% cured</td>
<td>Control: 0</td>
</tr>
<tr>
<td></td>
<td>No difference between groups; 55% cured</td>
<td>No difference between groups; 55% cured</td>
<td>No difference between groups; 55% cured</td>
<td>No difference between groups; 55% cured</td>
</tr>
</tbody>
</table>
### Table 7.6  Cure rates reported as less than 2 g of leakage measured with a variety of pad-tests in RCTs of PFMT to treat stress urinary incontinence—cont’d

| Author          | Design                          | Sample size and age (years) | Diagnosis                        | Training protocol                                                                                                                                      | Drop-out | Adherence | Results                  |
|-----------------|---------------------------------|-----------------------------|----------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------|
| Bø et al., 1999 | Randomized to PFMT, ES, cones, or control | 107 women, mean age 49.5 (range 24–70) | Urodynamic SUI                   | 6-month intervention; vaginal palpation. PFMT: 3x 8–12 contractions per day at home. Training diary. Weekly 45-min exercise class. Individual assessment of muscle strength and motivation for further training once a month |
|                 |                                  |                             |                                  | Drop-out 8%                                                                                                                                          |          | 93%        | PFMT: 44% cured           |
|                 |                                  |                             |                                  | Control: 6.7% cured                                                                                                                                   |          |            |                          |
| Mørkved et al., 2002 | Randomized to PFMT or PFMT with BF | 103 women, mean age 46.6 (range 30–70) | Urodynamic SUI                   | 6-month intervention after vaginal palpation. Both groups: same amount of exercise and meeting with PT once a week for the first 2 months, then once every second week. Three sets of 10 contractions holding 6 s add 3–4 fast contractions on top at each visit. Home training: three sets of 10 contractions daily. BF: same programme with BF. |
|                 |                                  |                             |                                  | Drop-out 8.7%                                                                                                                                          |          |            | PFMT: 85.3%; PFMT + BF: 88.9%  |
|                 |                                  |                             |                                  | Control: 6.7% cured                                                                                                                                   |          |            | PFMT: 69% cured           |
|                 |                                  |                             |                                  | Control: 6.7% cured                                                                                                                                   |          |            | PFMT + BF: 67% cured       |
| Aksac et al., 2003 | Randomized to PFMT, PFMT with BF, or control group on oestrogen | 50 women, mean age 52.9 (SD 7.1); 20 in each training group, 10 in control group | Urodynamic diagnosis of SUI | 8 weeks of: PFMT: vaginal palpation, 10 contractions 3x daily, hold for 5 s, progressing to 10 after 2 weeks. Weekly clinic sessions + ‘regular’ home training. BF: weekly clinic sessions, use BF at home 3x/week, 20 min with 10 s hold and 20 s rest |
|                 |                                  |                             |                                  | Drop-out None                                                                                                                                         |          |            |                           |
|                 |                                  |                             |                                  | Control: 0% cured, 20% improvement                                                                                                                    |          |            | PFMT: 75% cure, 25% improvement |
|                 |                                  |                             |                                  | BF: 80% cured, 20% improvement                                                                                                                       |          |            | PFMT: 80% cured, 20% improvement |
| Dumoulin et al., 2004 | Randomized to multimodal PFMT, multimodal PFMT + abdominal training, or control | 64 women, mean age 36.2 (range 23.3–39) | Urodynamic SUI                   | (Continued)
Bø et al. (1990b) have shown that instructor-followed up training is significantly more effective than home exercise. In this study individual assessment and teaching of correct contraction was combined with strength training in groups in a 6-month training programme. The women were randomized to either an intensive training programme consisting of seven individual sessions with a PT, combined with 45 minutes weekly PFMT classes, and three sets of 8–12 contractions per day at home or the same programme except for the weekly intensive exercise classes. The results showed a much better improvement in both muscle strength (see Chapter 6, Fig. 6.11) and urinary leakage in the intensive exercise group: 60% were reported to be continent/almost continent in the intensive exercise group compared to 17% in the less intensive group. A significant reduction of urinary leakage, measured by pad-test with standardized bladder volume, was only demonstrated in the intensive exercise group (Fig. 7.2).

This study demonstrated that a huge difference in outcome can be expected according to the intensity and follow-up of the training programme and very little effect can be expected after training without close follow-up. It is worth noting that the significantly less effective group in this study had seven visits with a skilled PT and that adherence to the home training programme was high. Nevertheless, the effect was only 17%. More intensive training has also been shown to be more effective in two other RCTs (Glavind et al., 1996; Goode et al., 2003) and in one non-randomized study (Wilson et al., 1987) and is now the conclusion of several systematic reviews (Immamura et al., 2010; Hay-Smith et al., 2011; Moore et al., 2013). There is a dose–response issue in all sorts of training regimens (Haskel, 1994; Bouchard, 2001). Therefore, one reason for disappointing effects shown in some clinical practices or research studies may be due to insufficient training stimulus and low dosage. If low-dosage programmes are chosen as one arm in a RCT comparing PFMT with other methods, PFMT is bound to be less effective (Herbert and Bø, 2005).

**PFMT with biofeedback**

Biofeedback has been defined as ‘a group of experimental procedures where an external sensor is used to give an indication on bodily processes, usually in the purpose of changing the measured quality’ (Schwartz and Beatty, 1977). Biofeedback equipment has been developed within the area of psychology, mainly to measure sweating, heart rate and blood pressure during different forms of stress. Kegel (1948) always based his training protocol on thorough instruction of correct contraction using vaginal palpation and clinical observation. He combined PFMT with use of vaginal squeeze pressure measurement as biofeedback during exercise. Today, a variety of biofeedback apparatus is commonly used in clinical practice to assist with PFMT.

In urology or urogynaecology textbooks the term ‘biofeedback’ is often used to classify a method different from PFMT. However, biofeedback is not a treatment on its own.
It is an adjunct to training, measuring the response from a single PFM contraction. In the area of PFMT both vaginal and anal surface EMG, and urethral and vaginal squeeze pressure measurements, have been used to make patients more aware of muscle function, and to enhance and motivate patients’ efforts during training (Hay-Smith et al., 2009; Herderschee et al., 2011). However, one should be aware that erroneous attempts at PFM contractions (e.g. by straining) may be registered by manometers and dynamometers, and contractions of muscles other than the PFM may affect surface EMG activity. Therefore biofeedback cannot be used to register a correct contraction.

Since Kegel first presented his results, several RCTs have shown that PFMT without biofeedback is more effective than no treatment for SUI (Dumoulin and Hay-Smith, 2010). In a Cochrane review, Herderschee et al. (2011) found 24 RCTs or quasi-randomized trials comparing PFMT with and without biofeedback. They concluded that use of biofeedback may provide benefit in addition to

| Table 7.7 PEDro quality score of RCTs in systematic review of PFMT to treat stress urinary incontinence |
| Study | E | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | Total score |
|-------|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Henall et al., 1989 | + | + | - | ? | - | - | + | - | ? | + | + | 3 |
| Glavind et al., 1996 | + | + | + | + | - | - | + | - | + | + | + | 6 |
| Wong et al., 1997 | - | + | ? | + | - | - | - | ? | ? | - | - | 2 |
| Bø et al., 1999 | + | + | + | + | - | - | + | + | + | + | + | 8 |
| Mørkved et al., 2002 | + | + | + | + | - | - | + | + | + | + | + | 8 |
| Aksac et al., 2003 | + | + | + | + | - | - | - | + | + | + | + | 7 |
| Dumoulin et al., 2004 | + | + | ? | + | - | + | + | + | + | + | + | 8 |
| Zanetti et al., 2007 | + | + | + | + | - | - | - | + | + | + | + | 7 |

+, criterion is clearly satisfied; −, criterion is not satisfied; ?, not clear if the criterion was satisfied. Total score is determined by counting the number of criteria that are satisfied, except that ‘eligibility criteria specified’ score is not used to generate the total score. Total scores are out of 10.

**Figure 7.2** Pad-test results showed that only the ‘intensive’ pelvic floor muscle training group had a statistically significant reduction in urinary leakage.

*From Bø, Hagen et al., 1990, with permission.*
PFMT. In women with SUI or mixed incontinence, all but two RCTs have failed to show any additional effect of adding biofeedback to the training protocol for SUI (Shepherd et al., 1983; Castleden et al., 1984; Taylor and Henderson, 1986; Ferguson et al., 1990; Berghmans et al., 1996; Glavind et al., 1996; Sherman et al., 1997; Laycock et al., 2001; Pages et al., 2001; Wong et al., 2001; Aukee et al., 2002; Morkved et al., 2002; Aksac et al., 2003). Berghmans et al. (1996) demonstrated quicker progress in the biofeedback group. In the study of Glavind et al. (1996) a positive effect was demonstrated. However, this study was confounded by a difference in training frequency, and the effect might be due to a double training dosage, the use of biofeedback, or both. The results support the studies concluding that there is a dose–response issue in PFMT.

Very few of the studies comparing PFMT with and without biofeedback have used the exact same training dosage in the two groups (Herderschee et al., 2011). For example, Pages et al. (2001) compared 60 minutes of group training 5 days a week with 15 minutes of individual biofeedback training 5 days a week, and found that the individualized biofeedback training protocol was more effective assessed by the women’s report and measurement of PFM strength. When the two groups under comparison receive different dosage of training in addition to biofeedback, it is impossible to conclude what is causing a possible effect. In addition, other factors flaw the results of studies comparing PFMT with and without biofeedback. As PFMT is effective without biofeedback, a large sample size may be needed to show any beneficial effect of adding biofeedback to an effective training protocol. In most published studies comparing PFMT with PFMT combined with biofeedback, the sample sizes are small, and type II error may have been the reason for negative findings (Herderschee et al., 2011). However, in the two largest RCTs published, no additional effect was demonstrated from adding biofeedback.

Many women may not like to undress, go to a private room and insert a vaginal or rectal device to exercise (Prashar et al., 2000). On the other hand, some women find it motivating to use biofeedback to control and enhance the strength of the contractions when training. Any factor that may stimulate high adherence and intensive training should be recommended to enhance the effect of a training programme. Therefore, when available, biofeedback should be given as an option for home training, and the PT should use any sensitive, reliable and valid tool to measure the contraction force at office follow-up.

**PFMT with vaginal weighted cones**

Vaginal cones are weights that are put into the vagina above the levator plate (Herbison and Dean, 2009) (see Chapter 6, Fig. 6.12). The cones were developed by Plevnic (Hay-Smith et al., 2001) in 1985. The theory behind their use in strength training is that the PFM are contracted reflexively or voluntarily when the cone is perceived to slip out. The weight of the cone is supposed to give a training stimulus and make women contract harder with progressive weight. In a Cochrane review, combining studies including 17 RCT or quasi-randomized studies with 1484 women with both SUI and mixed incontinence (six trials published as abstracts only) it was concluded that training with vaginal cones is more effective than no treatment (Herbison and Dean, 2009).

Several RCTs have been found comparing PFMT with and without vaginal cones for SUI (Pieber et al., 1994; Cammu and van Nylen, 1998; Bø et al., 1999; Arvonen et al., 2001; Laycock et al., 2001). Bø et al. (1999) found that PFMT was significantly more effective than training with cones both to improve muscle strength and reduce urinary leakage. In three other studies there were no differences between PFMT with and without cones (Pieber et al., 1994; Cammu and van Nylen, 1998; Laycock et al., 2001). Cammu and van Nylen (1998) reported very low compliance and therefore did not recommend use of cones. Also in the study of Bø et al. (1999), women in the cone group had great motivational problems. Laycock et al. (2001) had a total drop-out rate in their study of 33%.

The use of cones can be questioned from an exercise science perspective. Holding the cone for the recommended time of 15–20 minutes may result in decreased blood supply, decreased oxygen consumption, muscle fatigue and pain, and recruit contraction of other muscles instead of the PFMs. In addition, many women report that they dislike using cones (Cammu and van Nylen, 1998; Bø et al., 1999). On the other hand, the cones may add benefit to the training protocol if used in a different way: the subjects can be asked to contract around the cone and simultaneously try to pull it out in lying or standing position, repeating this 8–12 times in three series per day, or they can use the cones during progressively graded activities of daily living. In this way, general strength training principles are followed, and progression can be added to the training protocol. Arvonen et al. (2001) used ‘vaginal balls’ and followed general strength training principles. They found that training with the balls was significantly more effective in reducing urinary leakage than regular PFMT.

**PFMT or electrical stimulation for SUI?**

Rationale and evidence for electrical stimulation for SUI are covered below (see p. 178). Here, studies comparing PFMT and electrical stimulation, and studies combining PFMT and electrical stimulation will be cited.

Hennalla et al. (1989a), Hofbauer et al. (1990) and Bø et al. (1999) found that PFMT was significantly better than electrical stimulation to treat SUI. Laycock and Jerwood (1996) and Hahn et al. (1991) found no difference, and Smith (1996) found that electrical stimulation was signifi-
Is bladder training equally effective as PFMT for SUI?

The rationale behind bladder training and evidence for bladder training in overactive bladder are discussed in the section above (p. 153). One study showed that bladder training had similar effects on SUI and urge incontinence in women (Fantl et al., 1991), and another RCT that bladder training had similar effects as PFMT in women with SUI and urgency and mixed incontinence (Elser et al., 1999). Contradictory to this, Sherburn et al. (2011) found that PFMT was significantly more effective than bladder training in elderly women with SUI. To date, there is no clear-cut understanding of how bladder training works, and it is difficult to understand how it can treat SUI if it does not include specific PFM contractions.

Is surgery more effective than PFMT for SUI?

Only one published study has compared surgery with PFMT as first-line treatment for SUI. In the RCT of Klarskov et al. (1986) the patients had different surgeries according to their problems. The PFMT programme was described as group training with five or more sessions with a PT, and it is not clear whether the participants had vaginal palpation to make sure they were able to contract the PFM correctly. At 4 months the PFMT group was less likely to report cure compared to women who had surgery. However, there was no statistical difference in the proportions reporting cure/improvement. At 12 months 10/24 women in the PFMT group reported satisfaction with the initial therapy versus 19/26 in the surgery group. Adverse effects were reported only in the surgery group, including new urge incontinence, retropubic or pelvic pain or dyspareunia.

Adverse effects of PFMT

Few, if any, adverse effects have been found after PFMT (Moore et al., 2013). Lagro-Jansson et al. (1992) found that one woman reported pain with exercise and three had an uncomfortable feeling during the exercises. Aukse et al. (2002) reported no side-effects in the training group, but found that two women interrupted the use of home biofeedback apparatus because they found the vaginal probe uncomfortable. These women were both postmenopausal. In other studies no side-effects have been found (Bø et al., 1999).

Long-term effect of PFMT for SUI

Several studies have reported long-term effect of PFMT (Bø and Herbert, 2013; Moore et al., 2013). However, usually women in the non-treatment or less effective intervention groups have gone on to retrieve treatment after cessation of the study period. Therefore, follow-up data are usually reported for either all women or for only the group with the best effect. As for surgery (Black and Downs, 1996; Dmochowski et al., 2013a), there are only a few long-term studies including clinical examination (Bø and Herbert, 2013). Klarskov et al. (1991) assessed only some of the women originally participating in the study. Lagro-Jansson et al. (1998) evaluated 88 of 110 women with SUI and urge or mixed incontinence 5 years after cessation of training and found that 67% remained satisfied with the condition. Only seven of 110 had been treated with surgery. Moreover, satisfaction was closely related to compliance to training and type of incontinence, with women with mixed incontinence being more likely to lose the effect. Women with SUI had the best long-term effect, but only 39% of them were exercising daily or ‘when needed’.

In a 5-year follow-up, Bø and Talseth (1996) examined only the intensive exercise group and found that urinary
leakage was significantly increased after cessation of organized training. Three of 23 had been treated with surgery. Two of these women who had not been cured after the initial training were satisfied with their surgery, and had no leakage on pad-test. The third woman had been cured after initial PFMT. However, after 1 year she stopped training because of personal problems connected to the death of her husband. Her incontinence problems returned and she had surgery 2 years before the 5-year follow-up. She was not satisfied with the outcome after surgery and had visible leakage on cough test and 17 g of leakage on the pad-test. Of the women, 56% had a positive closure pressure during cough and 70% had no visible leakage during cough at 5-year follow-up; 70% of the patients were still satisfied with the results and did not want other treatment options.

Cammu et al. (2000) used a postal questionnaire and medical files to evaluate the long-term effect on 52 women who had participated in an individual course of PFMT for urodynamic SUI. Eighty-seven per cent were suitable for analysis – 33% had had surgery after 10 years. However, only 8% had undergone surgery in the group that had originally had success after training, whereas 62% had undergone surgery in the group initially dissatisfied with training. Successful results were maintained after 10 years in two-thirds of the patients originally classified as successful.

Bø et al. (2005) reported current status of lower urinary tract symptoms from questionnaire data 15 years after cessation of organized training. They found that the short-term significant effect of intensive training was no longer present: 50% from both groups had interval surgery for SUI; however, more women in the less intensive training group had surgery within the first 5 years after ending the training programme. There were no differences in reported frequency or amount of leakage between women who had or had not had surgery, and women who had surgery reported significantly more severe leakage and to be more bothered by urinary incontinence during daily activities than those who had not.

The general recommendations for maintaining muscle strength are one session per week of moderate-to-hard intensity exercises (Garber et al., 2011). The intensity of the contraction seems to be more important than frequency of training. So far, no studies have evaluated how many contractions subjects have to perform to maintain PFM strength after cessation of organized training. In a study by Bø and Talseth (1996) PFM strength was maintained 5 years after cessation of organized training with 70% exercising more than once a week. However, number and intensity of exercises varied considerably between successful women (Bø, 1995). One series of 8–12 contractions could easily be instructed in aerobic dance classes or recommended as part of women’s general strength training programmes. On the other hand, we do not know how a voluntary precontraction before an increase in abdominal pressure will maintain or increase muscle strength. In the study of Cammu et al. (2000) the long-term effect of PFMT appeared to be attributed to the precontraction before sudden increases in intra-abdominal pressure, and not so much to regular strength training. Muscle strength was not measured in their study. Although not taught in the original programme, several women in the study of Bø et al. (2005) also had performed precontractions of the PFM before and during a rise in abdominal pressure during the long-term follow-up period.

Other programmes

Today there is a lot of interest in PFMT in combination with so-called ‘core training’ (stabilizing training for the lower spine including mTra and multifidus muscles). Yoga, Pilates, Feldenkrais and Mensendick classes are examples of exercise programmes that may include training of the PFM. All these programmes except yoga (which is much longer established) were developed in the 1920s and 1930s, and, as far as this author has ascertained, none originally included PFMT.

We refer to the systematic review of Bø and Herbert (2013) for an overview and discussion of the evidence for alternative methods to treat SUI, and to the article on when and how new therapies should become clinical practice from the same authors (Bø and Herbert, 2009).

In untrained individuals all stimulus for regular training have the potential for improving function, and a focus on and incorporation of PFMT in any fitness programme for women should therefore be welcomed. One should be aware, however, that many women may not be able to perform correct contractions without proper individual instruction. Lack of effect of such general programmes may therefore also be due to incorrect contractions.

Motivation

Several researchers have looked into factors affecting outcome of PFMT on urinary incontinence (Moore et al., 2013). No single factor has been shown to predict outcome, and it has been concluded that many factors traditionally supposed to affect outcomes such as age and severity of incontinence may be less crucial than previously thought. Factors that appear to be most associated with a positive outcome are thorough teaching of correct contraction, motivation, adherence with the intervention, and intensity of the programme.

Some women may find the exercises hard to conduct on a regular basis (Aleijnse, 2002). However, when analysing results of RCTs, adherence to the exercise programme is generally high, and drop-out rate is low (Moore et al., 2013). In a few studies low adherence and high drop-out rates have been reported (Ramsey and Thou, 1990; Laycock et al., 2001). Knowledge about behavioural sciences such as pedagogy and health psychology, and ability to explain
and motivate patients may be a crucial factor to enhance adherence and minimize drop-outs from training. In some studies such strategies have been followed, and high adherence has been achieved (Alewijnse, 2002; Chiarelli and Cockburn, 2002). In other studies, specific strategies have not been reported, but emphasis has been put on creating a positive, enjoyable and supportive training environment. Group training after thorough individual instruction may be a good concept if led by a skilled and motivating person (Bø et al., 1990a; Bø et al., 1999) (Figs 7.4 and 7.5).

PFMT concepts with no drop-outs (Berghmans et al., 1996) and adherence over 90% (Bø et al., 1999) are possible. In a study of Alewijnse (2002) most women followed advice to train 4–6 times a week 1 year after cessation of the training programme. The following factors predicted 50% adherence:

• positive intention to adhere;
• high short-term adherence levels;
• positive self-efficacy expectations;
• frequent weekly episodes of leakage before and after initial therapy.

Patients do not comply with treatment for a wide variety of reasons: long-lasting and time-consuming treatments, requirement of lifestyle changes, poor client–patient interaction, cultural and health beliefs, poor social support, inconvenience, lack of time, motivational problems and travel time to clinics have been listed (Paddison, 2002).

Sugaya et al. (2003) used a computerized pocket-size device giving a sound three times a day to remind the person to perform PFMT. To stop the sound the person needed to push a button, and by pushing the button for each contraction, adherence was registered: 46 women were randomly assigned to either instruction to contract the PFM following a pamphlet, or with the same pamphlet together with the sound device and instruction on how to use the device. The results showed a significant improvement in adherence.

Figure 7.4 When the patients are able to contract the pelvic floor muscles correctly it can be fun and motivating to conduct the strength training in a class. Group training classes for pelvic floor muscle training were developed by Bø in 1986 and the results of the first randomized controlled trial using group training for stress urinary incontinence were presented in the journal *Neurourology and Urodynamics* in 1990.

Figure 7.5 In between the pelvic floor muscle strength training other exercises are performed to music. The original class emphasizes strength training of the abdominal (including transversus abdominis), back and thigh muscles in addition to body awareness and relaxation (breathing and stretching) exercises. The class is 60 minutes with 45 minutes of exercising and 15 minutes for information, conversation and motivation for home training.
daily incontinence episodes and pad-test only in the device group: 48% were satisfied in the device group compared to 15% in the control group. It was reported that patients in the device group felt obliged to perform PFMT when the chime sounded.

**CONCLUSION**

RCTs with high methodological quality, systematic reviews and several Cochrane reviews have concluded that there is level A, grade 1 evidence that PFMT is more effective than no treatment, sham or placebo treatment for SUI. PFMT is recommended as first-line treatment for SUI. There is no evidence to suggest that adding use of biofeedback, electrical stimulation or vaginal cones brings any additional effect over PFMT alone.

**CLINICAL RECOMMENDATIONS**

- Teach the patient about the PFM and lower urinary tract function using diagrams, drawings and models.
- Explain a correct PFM contraction. Allow the patient to practise before checking ability to contract.
- Assess PFM contraction.
- If the woman is able to contract, set up an individual training programme to be conducted at home. Aim for close to maximum contraction, building up to three sets of 8–12 contractions per day. Ask the patient to suggest where and when exercises should be performed. Supply the patient with an exercise diary or biofeedback with computerized adherence registration. If available, discuss whether use of biofeedback motivates the patient to exercise.
- If the woman is unable to contract try manual techniques such as touch, tapping, massage and fast stretch or electrical stimulation. Be aware that most patients learn to contract if they are given some time by themselves at home to practise.
- Follow-up with weekly or more often supervised training. Supervised training can be conducted individually or in groups.
- Follow development in PFM function and strength closely, using responsive, reliable and valid assessment tools.
- In addition to a strength training regimen ask the patient to precontract and hold the contraction before and during coughing, laughing, sneezing and lifting (conscious precontraction, the Knack).
- Suggested assessment of urinary leakage and quality of life (QoL) before and after treatment:
  - ICIQ short form (Avery et al., 2004);
  - 3-day leakage episodes (Lose et al., 1998);
  - leakage index (Bø, 1994);
  - pad-test (48-hour, 24-hour, 1-hour, short tests with standardized bladder volume) (Lose et al., 1998);
  - general and disease-specific QoL questionnaires (SF-37, ICIQ UI-SF, Kings College, B-FLUTS) (Corcos et al., 2002).

**REFERENCES**


Bernstein, L., 1997. The pelvic floor muscles [thesis]. University of Copenhagen, Hvidovre Hospital, Department of Urology.

Female pelvic dysfunctions and evidence-based physical therapy


Bø, K., Herbert, R.H., 2013. There is not yet strong evidence that exercise regimens other than pelvic floor muscle training can reduce stress urinary incontinence in women: a systematic review. J. Physiother. 59, 159–168.


Evidence-Based Physical Therapy for the Pelvic Floor


INTRODUCTION

When a nerve is stimulated, signals travel both toward the periphery and toward the central nervous system. Electrical stimulation may elicit responses to these signals, which may come from the central nervous system or the tissues innervated by the nerve, or the central nervous system may be modified to reinterpret some signals (Fall and Lindstrom, 1994; Chancellor and Leng, 2002).

In respect of lower urinary tract dysfunctions, electrical stimulation is applied particularly to the pelvic floor muscles, bladder and sacral nerve roots. Electrical stimulation of the pelvic floor aims at stimulating motor fibres of the pudendal nerve, which may elicit a direct contraction of the pelvic floor muscles or the striated peri-urethral musculature, supporting the intrinsic part of the urethral sphincter closing mechanism (Fall and Lindstrom, 1991; Scheepens, 2003). As such, electrical stimulation might contribute to the compensation of a weak intrinsic sphincter, but it is questionable whether or not electrical stimulation in such cases would be the first choice treatment option or would have any additional value to a functional training (Berghmans et al., 1998; Hay-Smith et al., 2009).

In patients with detrusor overactivity or symptoms of urgency and urge urinary incontinence electrical stimulation can elicit direct contractions of the pelvic floor muscles, which stimulate afferent fibres of the pudendal nerve going to the sacral spinal cord that reflexively decrease the feeling/sensation of urgency and inhibit parasympathetic activity at the level of the sacral micturition centre in the sacral cord, in order to reduce involuntary detrusor contractions and reflexively activate the striated peri-urethral musculature. Electrical stimulation may be used as stand-alone therapy or in combination with pelvic floor muscle training (PFMT) (Moore et al., 2013). Electrical stimulation can be divided into two major forms, neurostimulation and neuromodulation. Neurostimulation of the pelvic floor aims at stimulating motor efferent fibres of the pudendal nerve, which may elicit a direct response from the effector organ, for instance a contraction of the pelvic floor muscles (Eriksen, 1989; Fall and Lindstrom, 1991; Scheepens, 2003). The object of neuromodulation is to remodel neuronal reflex loops, for instance the detrusor inhibition reflex, by stimulatingafferent nerve fibres of the pudendal nerve that influence these reflex loops. Thus, neuromodulation may elicit an indirect response from the effector organ, for instance detrusor muscle inhibition (Vodušek et al., 1986; Fall and Lindstrom, 1994; Weil et al., 2000; Berghmans et al., 2002).

Today it is still very difficult to clarify the potential value and benefits of electrical stimulation in the treatment of urinary incontinence, the most prevalent form of lower urinary tract dysfunctions (Moore et al., 2013). There are several reasons for this.

First, the nomenclature used to describe electrical stimulation has been inconsistent. Stimulation has sometimes been described on the basis of the type of current being used (e.g. faradic stimulation, interferential therapy), but is also described on the basis of the structures being targeted (e.g. neuromuscular electrical stimulation), the current intensity (e.g. low-intensity stimulation, or maximal stimulation), and the proposed mechanism of action (e.g. neuromodulation). In the absence of a clear unequivocal classification of electrical stimulation, the present author will make no attempt to classify the interventions that are considered.

Second, although it has been suggested that electrical stimulation as an intervention for urinary incontinence is using the natural neural pathways and micturition reflexes (Fall, 1998; Yamanishi and Yasuda, 1998) and the understanding of both neuroanatomy and neurophysiology of the central and peripheral nervous systems is increasing, there is still lack of a well-substantiated biological rationale supporting the use of electrical stimulation (Moore et al., 2013).
Third, the lack of a clear biological rationale seems to hamper reasoned choices of electrical stimulation parameters. Parameters, used in previous electrical stimulation studies, i.e. current source, pulse width and duration, current intensity (range), amplitudes, stimulus frequency, pulse shape, time and total number of sessions and rest/work ratio, and electrode placements vary according to type of urinary incontinence and type of electrical stimulation (Moore et al., 2013). Berghmans (Berghmans et al., 2002) reported that usually frequencies of 5–20Hz are used for urge urinary incontinence, 20–50Hz for stress urinary incontinence (SUI) and for mixed urinary incontinence (MUI) (around) 20Hz or high/low alternately (Hay-Smith et al., 2009). Pulse durations of 200 (Hay-Smith et al., 2009), 300 (Yamanishi et al., 2010), 400–600μsec (Everaert et al., 1999) and 1000μsec (Moore et al., 1999) for SUI have been reported, for detrusor overactivity 200–500μsec, for MUI depending on the dominant factor of UI (Smith, 2009; Berghmans et al., 2002). Pulse shape is generally rectangular, and biphasic pulses are preferred (Hay-Smith et al., 2009).

Although a wide range of parameters has been claimed to be successful, the most optimal set of parameters for each type of urinary incontinence has not been determined (Hay-Smith et al., 2009). Additional confusion is created by the relatively rapid developments in the area of electrical stimulation. Even for the same health problem, a wide variety of stimulation devices and protocols have been used (Moore et al., 2013) (see Table 7.8 below).

<table>
<thead>
<tr>
<th>Table 7.8 RCTs on electrical stimulation to treat stress urinary incontinence</th>
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<tbody>
<tr>
<td>Author</td>
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<tr>
<td>Design</td>
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<tr>
<td>Sample size and age (years)</td>
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<tr>
<td>Diagnosis</td>
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<tr>
<td>Training protocol</td>
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<td>Drop-out</td>
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<td>Adherence</td>
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<td>Results</td>
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<td>Author</td>
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<tr>
<td>Design</td>
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<td>Sample size and age (years)</td>
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<td>Diagnosis</td>
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<td>Training protocol</td>
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<td>Drop-out</td>
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<td>Adherence</td>
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<td>Results</td>
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(Continued)
### Table 7.8 RCTs on electrical stimulation to treat stress urinary incontinence—cont’d

<table>
<thead>
<tr>
<th>Author</th>
<th>Olah et al., 1990</th>
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<tbody>
<tr>
<td><strong>Design</strong></td>
<td>2-arm RCT</td>
</tr>
<tr>
<td><strong>Sample size and age (years)</strong></td>
<td>69 women; mean age 43.2 ± 8.9 (VC), 47.9 ± 13.0 (ES)</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>Continence-frequency chart 1 week pretreatment, pelvic floor strength with VC, 1 hour pad-test</td>
</tr>
<tr>
<td><strong>Training protocol</strong></td>
<td>VC group 1x/week for 4 weeks, active PFMT with VC 2x/day 15 min, increasing weight after 2 successful occasions; ES-IFT 3x/week for 4 weeks; 0–100 Hz, 4 vacuum electrodes, 2 abdominal, 2 thighs, I max, 15 min</td>
</tr>
<tr>
<td><strong>Drop-out</strong></td>
<td>15/69 (22%)</td>
</tr>
<tr>
<td><strong>Adherence</strong></td>
<td>Not reported</td>
</tr>
<tr>
<td><strong>Results</strong></td>
<td>Weekly leakages (g) mean ± SD: VC from 22.0 ± 31.4 to 8.2 ± 14.5 to 3.9 ± 9.4 (after 6 mth); IFT 19.3 ± 22.6 to 7.7 ± 11.7 to 5.3 ± 9.2 (after 6 mth)</td>
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<tr>
<td></td>
<td>UI (g) mean ± SD: VC 27.7 ± 38.8 to 14.0 ± 36.7 to 2.8 ± 8.3; IFT from 32.2 ± 49.1 to 10.5 ± 17.3 to 9.7 ± 28.4 (after 6 mth)</td>
</tr>
<tr>
<td></td>
<td>No difference between groups</td>
</tr>
<tr>
<td></td>
<td>Cured/improved: VC 4/15 of 24, 10/7 of 24 after 6 mth; IFT 4/23 of 30, 12/15 of 30</td>
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<thead>
<tr>
<th>Author</th>
<th>Hofbauer et al., 1990</th>
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<tr>
<td><strong>Design</strong></td>
<td>4 arm RCT</td>
</tr>
<tr>
<td><strong>Sample size and age (years)</strong></td>
<td>43 women; mean age 57.5 ± 12</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>Cystoscopy, cystometry, UPP, micturition diary</td>
</tr>
<tr>
<td><strong>Training protocol</strong></td>
<td>ES constant 3x/week 10 min for 6 weeks perineal and lumbar electrodes, faradic, I variable until contraction; PFMT + abd/add 20 min 2x/week + home exercises; sham ES</td>
</tr>
<tr>
<td><strong>Drop-out</strong></td>
<td>Not reported</td>
</tr>
<tr>
<td><strong>Adherence</strong></td>
<td>Not reported</td>
</tr>
<tr>
<td><strong>Results</strong></td>
<td>Cured/improved/unchanged: ES + PFMT 3/4/4; PFMT 6/1/4; ES 1/2/8; sham ES 0/0/10</td>
</tr>
<tr>
<td></td>
<td>MUCP, FUL, pressure transmission no significant changes pre- and post-treatment</td>
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<thead>
<tr>
<th>Author</th>
<th>Blowman et al., 1991</th>
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<tr>
<td><strong>Design</strong></td>
<td>Double blind 2-arm RCT</td>
</tr>
<tr>
<td><strong>Sample size and age (years)</strong></td>
<td>14 women; age range 33–68</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>Urodynamics, filling cystometry, coughing-induced leakage while standing</td>
</tr>
<tr>
<td><strong>Training protocol</strong></td>
<td>PFMT + visual feedback with perineometer 4x/day; home (sham) surface ES 60 min/day; perineal and buttocks, ES 10 Hz, 4 s hold/relax, pulse width 80 μs, 2 weeks 35 Hz, 15 min/day; ES no contraction, minimal sensation, 4 weeks</td>
</tr>
<tr>
<td><strong>Drop-out</strong></td>
<td>1/14 (7%)</td>
</tr>
<tr>
<td><strong>Adherence</strong></td>
<td>Not reported</td>
</tr>
<tr>
<td><strong>Results</strong></td>
<td>No significant decrease median (range) IEF/week in sham ES from 12.5 (1–31) to 6 (0–21), significant decrease ES from 5 (0–14) to 0 (0–1)</td>
</tr>
<tr>
<td></td>
<td>Max. perineometer in sham ES median (range) pre-/post-treatment from 3.5 (1–5) to 5 (3–13), ES 1 (0–8) to 5 (2–16); no side-effect of (sham) ES reported; IEF 0 in 6/7 ES, 1/6 in sham ES; questionnaire after 6 mth ES no ES, 4 sham ES further treatment needed</td>
</tr>
</tbody>
</table>
Table 7.8 RCTs on electrical stimulation to treat stress urinary incontinence—cont’d

<table>
<thead>
<tr>
<th>Author</th>
<th>Design</th>
<th>Sample size and age (years)</th>
<th>Diagnosis</th>
<th>Training protocol</th>
<th>Drop-out</th>
<th>Adherence</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hahn et al., 1991</td>
<td>2-arm RCT PFMT ES If not cured after 6mth other arm offered</td>
<td>20 women; mean age 47.2 (range 24–64)</td>
<td>Urodynamics, cystometry, pad-test, cystourethroscopy</td>
<td>PFMT fast Pmax 5 s hold and relax and slow-twitch P submax 2 s hold and relax various positions, 5–10x, 6–8x/day, endurance 30–40 s; IFT vaginal probe, alternating pulses 10/20/50 Hz, home device (Contelle) 6–8 h/night</td>
<td>2 IFT after unsuccessful PFMT/13</td>
<td>Not reported</td>
<td>Pad-test: 5/20 cured 1 treatment course (1 PFMT, 4 IFT); PFMT, IFT significant improvement, in-between NS; 13 second course significant improvement; subjective improvement 2 cured/11 improved Pad-test after 4 years: 4/14 further improvement, 8/14 unchanged, 2/14 deterioration; subjective improvement: 1/14 improved, 8/14 unchanged, 5 deteriorated</td>
</tr>
<tr>
<td>Laycock and Jerwood, 1993 (I)</td>
<td>2-arm RCT PFMT ES</td>
<td>46 women, age range 28–59</td>
<td>Urodynamically proven GSI; digital palpation (grading Oxford scale)</td>
<td>Mean10 ES-IFT sessions, bipolar, perineal and symphysis pubis, 30 min, 1 max, 1/10–40/40 Hz 10 min each; 6 weeks PFMT 5 MVCs every hour, from 2nd visit VC 10 min, 2x/day</td>
<td>ES: no drop-outs; PFMT 6/23 (26%)</td>
<td>After therapy in ES group 1 subject (7%) every day home maintenance PFMT, 6 (40%) nearly every day, 8 (53) 1x/week</td>
<td>Pad-test; significant decrease (p &lt;0.003) both groups; PFM strength ES significant improvement (p=0.0035), PFMT n.s.; micturition diary IEF significant decrease in both groups; subjective assessment IEF both groups equally effective; review questionnaire after 2 years &gt;30% ES maintained improvement</td>
</tr>
<tr>
<td>Laycock and Jerwood, 1993 (II)</td>
<td>2-arm RCT PFMT ES</td>
<td>30 women, age range 16–66</td>
<td>See Laycock I</td>
<td>IFT: see Laycock I; sham IFT: no current, rest similar IFT</td>
<td>IFT no drop-outs; sham IFT 4/15 (27%)</td>
<td>After therapy in ES group 2 subjects (15.4%) every day home maintenance PFMT, 5 (38.5%) nearly every day, 4 (30.8%) 1x/week, 2 (15.4%) &lt;1x/week</td>
<td>Pad-test: IFT mean 56.8% decrease weight pre/post treatment, sham IFT 21.4%; in between significant difference; perineometer: PFMC significant increase strength only in IFT; micturition chart: IEF reduction only in IFT, severity reduction idem; review questionnaire after mean 16 mth 20% IFT maintained improvement</td>
</tr>
<tr>
<td>Sand et al., 1995</td>
<td>2-arm RCT PFMT ES, sham ES. Multicentre</td>
<td>52 women age mean ± SD 53.2 ± 11.4 years</td>
<td>Urodynamic-proven GSI, UCP &gt;20 cm H₂O, and LPP &gt;60 cm H₂O at max. cyst. capacity</td>
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(Continued)
### Table 7.8 RCTs on electrical stimulation to treat stress urinary incontinence—cont’d

<table>
<thead>
<tr>
<th>Training protocol</th>
<th>Vaginal electrode, ES pulse duration 0.3 ms, I max, first 2 weeks 5/10 s, later 5/5 s hold/relax; sham ES 1 mA max, 15–30 min 2x/day 12 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drop-out</td>
<td>8/52 (15%)</td>
</tr>
<tr>
<td>Adherence</td>
<td>61% used ES &gt;50 out of planned 70 hours (80%) vs 89% sham ES</td>
</tr>
<tr>
<td>Results</td>
<td>ES vs sham ES after 12 weeks IEF/24 h, IEF/week, UI during pad-test, PFMT strength on perineometry significantly better in ES; no irreversible adverse events, vaginal irritation/infection/urinary tract infection/pain 14%/11%/3%/9% ES and 12%/12%/12%/6% sham ES</td>
</tr>
<tr>
<td>Author</td>
<td>Smith, 1996</td>
</tr>
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</table>

| Design            | 2-arm RCT                                                                                                                      |
| Sample size and age (years) | Subgroup GSI (type II) 18 women, age range 26–72 |
| Diagnosis         | Cystoscopy only when indicated, complex video urodynamic study, i.e. uroflow, UPP, cystometry, Vasalva LPP                        |
| Training protocol | ES: 5 s contractions (range 3–15), duty circle 1:2, treatment time 15–60 min 2x/day for 4 mth, I 5–80 mA; PFMT: 60 contr./day, fast- and slow-twitch |
| Drop-out          | None                                                                                                                           |
| Adherence         | 80%                                                                                                                           |
| Results           | IEF, pads >50% improved, objective improvement 44% PFMT, 1/4/5 cured/improved/unchanged; 66% ES, 2/4/3; in between NS             |
| Author            | Brubaker et al., 1997                                                                                                            |

| Design            | 2-arm RCT                                                                                                                      |
| Sample size and age (years) | 148 women, subgroup GSI 60 women, age mean 57 (SD 12) |
| Diagnosis         | Urodynamics, micturition diary                                                                                                 |
| Training protocol | ES: transvaginal, 20 Hz, 2/4 s work/rest, pulse width 0.1 μs, bipolar square wave, I 0–100 mA: sham ES same parameters, no I, both groups 8 weeks treatment |
| Drop-out          | 18%                                                                                                                           |
| Adherence         | ES vs sham ES mean compliance 87% vs 81% at 4 & 8 treatment weeks                                                            |
| Results           | ES vs sham ES, 6 weeks 24-h frequency NS; 6 weeks, no. of accidents/24 h (average) NS; adequate subjective improvement p=0.027; QoL NS; no analysis diaries because of incomplete data |
| Author            | Luber and Wolde-Tsadik, 1997                                                                                                   |

| Design            | 2-arm RCT double blind:                                                                                                         |
| Sample size and age (years) | 45 women GSI, mean age 53.8 |
| Diagnosis         | Urodynamics, micturition diary, questionnaire, cotton-tip test: hypermobility urethra                                           |
| Training protocol | ES: 2x 15 min sessions/day for 12 weeks, home device, pulse width 2 ms, 2/4 s work/rest, freq. 50 Hz, I 10–100 mA; sham ES same parameters, I no sensation |
| Drop-out          | 1/45 (2.2%)                                                                                                                     |
| Adherence         | Measured by internal memory home device                                                                                         |
| Results           | Difference NS between groups (ES 20 women, sham ES 24 women in subjective cure/improvement, objective cure (diaries, incontinence questionnaire, urodynamics); no adverse events |
| Author            | Knight et al., 1998                                                                                                              |

| Design            | 3-arm RCT                                                                                                                      |
| Sample size and age (years) | 70 women GSI age range 24–68 |
| Clinic ES+PFMT/BF | Home ES+PFMT/BF | PFMT/BF |

Author Smith, 1996

Design 2-arm RCT

ES PFMT

Sample size and age (years)

Subgroup GSI (type II) 18 women, age range 26–72

Diagnosis Cystoscopy only when indicated, complex video urodynamic study, i.e. uroflow, UPP, cystometry, Vasalva LPP

Training protocol ES: 5 s contractions (range 3–15), duty circle 1:2, treatment time 15–60 min 2x/day for 4 mth, I 5–80 mA; PFMT: 60 contr./day, fast- and slow-twitch

Drop-out None

Adherence 80%

Results IEF, pads >50% improved, objective improvement 44% PFMT, 1/4/5 cured/improved/unchanged; 66% ES, 2/4/3; in between NS

Author Brubaker et al., 1997

Design 2-arm RCT

ES Sham ES

Sample size and age (years)

148 women, subgroup GSI 60 women, age mean 57 (SD 12)

Diagnosis Urodynamics, micturition diary

Training protocol ES: transvaginal, 20 Hz, 2/4 s work/rest, pulse width 0.1 μs, bipolar square wave, I 0–100 mA: sham ES same parameters, I no sensation

Drop-out 18%

Adherence ES vs sham ES mean compliance 87% vs 81% at 4 & 8 treatment weeks

Results ES vs sham ES, 6 weeks 24-h frequency NS; 6 weeks, no. of accidents/24 h (average) NS; adequate subjective improvement p=0.027; QoL NS; no analysis diaries because of incomplete data

Author Luber and Wolde-Tsadik, 1997

Design 2-arm RCT double blind:

ES Sham ES

Sample size and age (years)

45 women GSI, mean age 53.8

Diagnosis Urodynamics, micturition diary, questionnaire, cotton-tip test: hypermobility urethra

Training protocol ES: 2x 15 min sessions/day for 12 weeks, home device, pulse width 2 ms, 2/4 s work/rest, freq. 50 Hz, I 10–100 mA; sham ES same parameters, I no sensation

Drop-out 1/45 (2.2%)

Adherence Measured by internal memory home device

Results Difference NS between groups (ES 20 women, sham ES 24 women in subjective cure/improvement, objective cure (diaries, incontinence questionnaire, urodynamics); no adverse events

Author Knight et al., 1998

Design 3-arm RCT

Clinic ES+PFMT/BF Home ES+PFMT/BF PFMT/BF

Sample size and age (years)

70 women GSI age range 24–68
## Table 7.8 RCTs on electrical stimulation to treat stress urinary incontinence—cont’d

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Urodynamics, micturition diary, pad-test, perineometry</th>
</tr>
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<tbody>
<tr>
<td>Training protocol</td>
<td>Baseline treatment: home PFMT after instruction PT, max. 10 10/4 s hold/relax, repetitions recorded, max. 10 fast-twitch contractions, 6x/day. Baseline treatment nightly low I home ES, vaginal probe, trains of 10 Hz, 35 Hz occasionally, pulse width 200 μs, duty circle 5/5 s. Baseline treatment + 16 30-min clinic ES, I max, 35 Hz, pulse width 250 ms, together with voluntary contraction</td>
</tr>
<tr>
<td>Drop-out</td>
<td>13/70 (18.6%); 24% in home ES (NS), ITT-analysis of all</td>
</tr>
<tr>
<td>Adherence</td>
<td>Median percentage compliance home ES (72.5%); PFMT/BF (90%); difference between groups NS</td>
</tr>
<tr>
<td>Results</td>
<td>Pad-test after 6 mth: significant reduction urine loss in all 3 groups, clinic ES best, after 12&gt; reduction; objective improvement/cured after 6 mth clinic ES (n=20) vs home ES (n=19) vs controls (n=18) 80%/52.8%/72.3%</td>
</tr>
<tr>
<td></td>
<td>Micturition diaries data incomplete, not analysed; PFM strength significant increase in all groups, biggest in clinic ES (NS)</td>
</tr>
<tr>
<td>Author</td>
<td>Bø et al., 1999</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Design</th>
<th>4-arm RCT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample size and age (years)</td>
<td>122 women GSI, mean (range) age 49.5 (24–70)</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Urodynamics, uroflowmetry, cystometry, pad-test with standard bladder volume</td>
</tr>
<tr>
<td>Training protocol</td>
<td>PFMT: 8–12 VPFMC 3x/day at home % 1x/week office; ES: vaginal intermittent stimulation, 50 Hz 30 min/day; VC: 20 min/day</td>
</tr>
<tr>
<td>Drop-out</td>
<td>15/122 (12%) primary analysis and ITT analysis of all</td>
</tr>
<tr>
<td>Adherence</td>
<td>Mean (SE) adherence PFMT 93% (1.5%), ES 75% (2.8%), VC 78% (4.4%); PFMT vs ES or VC significantly better, ES vs VC NS</td>
</tr>
<tr>
<td>Results</td>
<td>Significant improvement pre/post treatment all treatment groups: PFMT vs no treatment significant difference (p &lt;0.01) PFMT: 44% cured; no treatment 6.7%; change in PFM strength significantly greater in PFMT (p = 0.03), not in ES or VC; ITT analysis same results; PFMT vs no treatment significant change in pad-test after 6 mth, IEF (p &lt;0.01), Social Activity Index (p &lt;0.01) and Leakage Index (p &lt;0.01). No urodynamic parameters changed in any group pre/post treatment</td>
</tr>
<tr>
<td>Author</td>
<td>Jeyaseelan et al., 2000</td>
</tr>
</tbody>
</table>

| Design                             | 2-arm RCT |
| Sample size and age (years)        | 27 women GSI, age not reported |
| Diagnosis                          | Urodynamics, 7 day micturition diary, 20-minute pad-test |
| Training protocol                  | ES consisted of background low frequency (target slow-twitch fibres) and intermediate frequency with an initial doublet (target fast-twitch fibres), vaginal probe, 1 hr/day, 8 weeks. Sham ES: 1 250 μs/min for 1 hour, I no effect |
| Drop-out                           | 3/27 (11%) |
| Adherence                          | ES: 71–98%; sham ES: 64–100% |
| Results                            | Perineometry: PFM strength between and within groups no significant changes pre/post treatment (p=0.86); digital assessment: significant within changes pre/post treatment; endurance PFM by perineometry: ES 73% ± 116% improved, sham ES reduction −6% ± 24%, difference between groups NS. Pad-test, micturition diary, IIQ: no significant changes between groups. UDI: ES>reduction than sham ES (p=0.03) |
| Author                             | Goode et al., 2003 |

(Continued)
Table 7.8  RCTs on electrical stimulation to treat stress urinary incontinence—cont’d

| Design | 3-arm RCT  
| ES + PFMT/BF  
| PFMT/BF  
| Controls (self-administered PFMT) |
| Sample size and age (years) | 200 women, age range 40–78 |
| Diagnosis | Urodynamics, cystometry, micturition diary, QoL questionnaires |
| Training protocol | PFMT: 1x/2 weeks for 8 weeks, anorectal BF for awareness PFM, hold/relax 20 min, verbal and written instructions for 3x/day PFMT at home, duration hold/relax max 10 s each. ES: vaginal probe, biphasic, 20 Hz, pulse width 1 ms, hold/relax 1:1, I max up to 100 mA 15 min/2 days. Controls: written instructions, booklet |
| Drop-out | 18.2% in PFMT, 11.9% in ES, 37.3% in controls, ITT analysis |
| Adherence | Not reported |
| Results | Mean reduction 68.6% PFMT/BF, 71.9% ES + PFMT/BF, 52.5% controls condition. In comparison with controls both interventions significantly more effective, but not significantly different from each other (p = 0.60). ES + PFMT/BF significantly better patient self-perception of outcome (p < 0.001) and satisfaction with progress (p = 0.02) |
| Author | Castro et al., 2008 |

| Design | 4-arm RCT  
| ES  
| Vaginal cones (VC)  
| PFMT  
| No treatment (controls) |
| Sample size and age (years) | 118 women urodynamically proven (predominant) SUI, mean age 54.2, range 41–69 |
| Diagnosis | Urodynamically proven SUI |
| Training protocol | PFMT: 10x 5 s contractions, 5 s rest, then 20x 2/2, 20x 1/1, 5x 10/10, followed by 5x strong contractions with stimulated cough/1 min rest; general warming-up and stretching exercises at end  
| ES: transvaginal 20 min, 3x/week; 50 Hz, biphasic, pulse width 500 μs, I 0–100 mA, duty cycle 5/10 s  
| VC: 1 session with PT 3 days/week supervised physical therapy; 45 min holding heaviest weight. |
| Drop-out | 17 (14%) of which 9 (7.6%) lack of success (2 PFMT, 1 ES, 4 VC, 2 controls); 8 (7.4%) other reasons (1 PFMT, 2 ES, 2 VC, 3 controls) |
| Adherence | Mean compliance PFMT 92%, ES 91%, VC 93% after 6 months treatment |
| Results | Cure objective (pad-test): 12 (46%) PFMT, 13 (48%) ES, 11 (46%) VC, 2 (8%) controls. Active treatment significantly better than controls (p = 0.003). Difference between active groups NS  
| QoL: active treatment significantly better than controls (p = 0.002). Increase QoL: PFMT 28.4%, 32.4% ES, 30.3% VC, −3.6% controls. Difference between active groups NS |
| Author | Eyjólfsdóttir et al., 2009 |

| Design | 2-arm RCT  
| PFMT + ES  
| PFMT |
| Sample size and age (years) | 24 women, range 27–73 years |
| Diagnosis | SUI |
| Training protocol | PFMT both groups: 15 min/twice a day for 9 weeks  
| ES: intermittent ES |
| Drop-out | Not reported |
| Adherence | Not reported |
It has been suggested that electrical stimulation restores continence by:

- strengthening the structural support of the urethra and the bladder neck (Plevnic et al., 1991);
- securing the resting and active closure of the proximal urethra (Erlandson and Fall, 1977);
- strengthening the pelvic floor muscles (Sand et al., 1995);
- inhibiting reflex bladder contractions (Fall and Lindström, 1994; Berghmans et al., 2002);
- modifying the vascularity of the urethral and bladder neck tissues (Fall and Lindström, 1991, 1994; Plevnic et al., 1991).

In the context of conservative or non-surgical, non-medical therapy electrical stimulation can be applied using surface electrodes (Brubaker, 2000; Goldberg and Sand, 2000; Govier et al., 2001; Jabs and Stanton, 2001).

Surface electrodes include: transcutaneous electrical stimulation (Brubaker, 2000; Jabs and Stanton, 2001; Berghmans et al., 2002) or transcutaneous electrical nerve stimulation (TENS), via suprapubic, sacral or penile/clitoral attachment of electrodes (Yamanishi et al., 2000), vaginal/anal plug electrodes (Moore et al., 1999), plantar/thigh and similar stimulation (Walsh et al., 1999) and other surface placement of electrodes such as for interferential or maximum electrical stimulation; percutaneous electrical stimulation (Govier et al., 2001; Amarenco, 2003), e.g. posterior tibial nerve stimulation, percutaneous nerve evaluation and electroacupuncture.

There are two main types of electrical stimulation:

1. Long-term or chronic electrical stimulation is delivered below the sensory threshold aiming at detrusor inhibition by afferent pudendal nerve stimulation. The electrically evoked activity is suggested to result in reflex activation of hypogastric efferents and central inhibition of pelvic efferent mechanisms sensitive to low-frequency stimulation (Fall and Lindström, 1994). The device is used 6–12 hours a day for several months (Eriksen, 1989).

2. Maximal electrical stimulation uses a high-intensity stimulus (just below the pain threshold). It aims to improve urethral closure. Fall (Fall and Lindström, 1991) suggested a direct and reflexogenic contraction of striated periurethral musculature. Also detrusor inhibition by afferent pudendal nerve stimulation has been suggested (Berghmans et al., 2002). Maximal electrical stimulation is applied with short duration (15–30 minutes), is used several times a week (1–2 times daily, also using portable devices at home) (Yamanishi et al., 1997; Yamanishi and Yasuda, 1998; Yamanishi et al., 2000).

In addition to clinic-based mains-powered electrical stimulation, portable electrical stimulation devices for self care by patients themselves at home have been developed (Berghmans et al., 2002). In the literature, authors suggest that intermittent, short-term stimulation (maximal electrical stimulation) by means of a portable, home-use device should usually be employed.

Electrical stimulation has been used for patients with stress urinary incontinence, symptoms of urgency, frequency and/or urge urinary incontinence, nocturia, detrusor overactivity and mixed urinary incontinence (Moore et al., 2013).

In the rest of this chapter we will address the question about the most appropriate electrical stimulation protocol for female patients with SUI, whether electrical stimulation is better than no treatment, placebo or control treatment, whether electrical stimulation is better than any other single treatment, and whether or not (additional) electrical stimulation to other (additional) treatments adds any benefit. Finally, we will address the results of electrical stimulation on PFM strength and eventual adverse events reported in the included studies. Information about male patients with SUI can be found in Chapter 8.

### METHODS

The following qualitative summary of the evidence regarding electrical stimulation in adult patients with SUI is based on RCTs included in four systematic reviews (Berghmans et al., 1998; Berghmans et al., 2000; Hay-Smith et al., 2001; Moore et al., 2013), with addition of trials performed after publication of the reviews and/or located through additional electronic searching on

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**Table 7.8 RCTs on electrical stimulation to treat stress urinary incontinence—cont’d**

| Results | UI episodes frequency and quantity questionnaire and VAS scale: subjective cure/improvement in 70% of all women, no report on rates cured/improved or group differences. Oxford scale, vaginal palpation, EMG: both groups significant increase in PFM strength (PFMT p = 0.007; PFMT + ES p = 0.005, difference between groups NS) |

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FUL, functional urethral length; GSI, genuine stress incontinence; IEF, incontinence episodes frequency; IFT, interferential therapy; LPP, leak point pressure; MUCP, maximal urethral closing pressure; MVC, maximal voluntary contraction; NS, not statistically significant; PFMT, pelvic floor muscle training; UCP, urethral closing pressure; UPP, urethral pressure profile; VC, voluntary contraction. For other abbreviations, see text.
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PubMed from 1998 till 2013 and the Cochrane Library. Additionally, we searched the literature used in the most recent (Fifth) International Consultation on Incontinence (Moore et al., 2013). Published abstracts were excluded.

**EVIDENCE FOR ELECTRICAL STIMULATION TO TREAT SUI SYMPTOMS**

Table 7.8 provides details of results of all included studies (n = 17; one study consisted of two separate RCTs [Laycock and Jerwood, 1993]). The PEDro rating scale was used to classify the methodological quality of the included studies (Table 7.9). The studies had low to high methodological quality.

It appeared that there was considerable variation in electrical stimulation (ES) protocols with no consistent pattern emerging.

Interferential therapy was used in four trials (Henalla et al., 1989; Olah et al., 1990; Laycock and Jerwood, 1993; Alves et al., 2011). Few trials clearly stated whether direct or alternating currents were being used.

The most commonly used descriptors were frequency and pulse duration. Eight trials used a single frequency, ranging from 20 Hz (Brubaker et al., 1997; Goode et al., 2003) to 50 Hz (Hahn et al., 1991; Smith, 1996; Bø et al., 1999; Luber and Wolde-Tsadik, 1997; Castro et al., 2008; Alves et al., 2011). Two trials included stimulation at both 10 Hz and 35 Hz (Blowman et al., 1991; Knight et al., 1998), although the protocols were different, one at combined low and intermediate frequency (Jeyaseelan et al., 2000). Other protocols included stimulation at 12.5 Hz and 50 Hz (Sand et al., 1995), 10–50 Hz (Shepherd et al., 1984), 0–100 Hz (Henalla et al., 1989; Olah et al., 1990), and finally a 30-minute treatment including 10 minutes at 1 Hz, 10 minutes 10–40 Hz and 10 minutes at 40 Hz (Laycock and Jerwood, 1993). Pulse durations ranged from 0.08 ms (Blowman et al., 1991) up to 100 ms (Brubaker et al., 1997; Alves et al., 2011). Ten trials also detailed the duty cycle used during stimulation. The ratios ranged from 1:3 (Bø et al., 1999), and 1:2 (Brubaker et al., 1997; Luber and Wolde-Tsadik, 1997; Castro et al., 2008; Alves et al., 2011) to 1:1 (Blowman et al., 1991; Knight et al., 1998; Goode et al., 2003) and two trials alternated between a ratio of 1:1 and 1:2 (Sand et al., 1995; Smith, 1996).

Eight trials asked women to use the maximum tolerable intensity of stimulation (Olah et al., 1990; Laycock and Jerwood, 1993; Sand et al., 1995; Brubaker et al., 1997; Bø et al., 1999; Goode et al., 2003; Castro et al., 2008; Alves et al., 2011), and one trial increased output until there was a noticeable muscle contraction (Hofbauer et al., 1990). The trial compared ‘low intensity’ and ‘maximal intensity’ protocols. The trials by Hofbauer et al. (1990), Knight et al. (1998) and Goode et al. (2003) also asked women to add a voluntary PFM contraction to the stimulated contraction, although in the trial of Knight et al. (1998) this was only for the maximal stimulation group.

Current was most commonly delivered via a single vaginal electrode (Hahn et al., 1991; Sand et al., 1995; Smith, 1996; Brubaker et al., 1997; Luber and Wolde-Tsadik, 1997; Knight et al., 1998; Bø et al., 1999; Goode et al., 2003; Castro et al., 2008; Alves et al., 2011). One trial used both vaginal and buttock electrodes (Shepherd et al., 1995). In three trials external electrodes were used: abdomen and inside thighs (Olah et al., 1990), perineal body and symphysis pubis (Laycock and Jerwood, 1993), perineal and buttock (Blowman et al., 1991), and in two studies the electrode placement was not clearly described (Henalla et al., 1989; Hofbauer et al., 1990).

The length and number of treatments was also highly variable. The longest treatment periods included daily treatment at home for 6 months (Hahn et al., 1991; Knight et al., 1998; Bø et al., 1999) and 20 minutes clinic-based treatment three times a week for 6 months (Castro et al., 2008). Medium length treatment periods were based on once-daily treatment at home for 8 weeks every other day (Goode et al., 2003) and twice-daily treatment at home for 8 (Brubaker et al., 1997) to 12 weeks (Sand et al., 1995; Luber and Wolde-Tsadik, 1997). The shortest treatment periods were all for clinic-based stimulation, ranging from 1 (Henalla et al., 1989; Laycock and Jerwood, 1993), 12 (Olah et al., 1990) to 16 (Knight et al., 1998) and 18 sessions in total (Hofbauer et al., 1990).

Comparing two protocols with different intensity of ES, Knight et al. (1998) found a trend, across a range of outcomes including self-report of cure or improvement, pad-test and PFM strength measurement, measured by vaginal squeeze pressure, for women who received clinic-based maximal stimulation to benefit more than women in the low intensity stimulation group, although most differences were not significant.

Also comparing two protocols but now with low and medium frequency current, Alves et al. (2011) found that the two ES protocols applied were equally effective in the treatment of SUI, based on a 1 hour pad-test as an objective outcome measure of UI, a VAS scale to evaluate subjective severity of UI and perineal pressure performed with a perineometer to test PFM maximum voluntary contraction.

**Is ES better than no treatment, control or placebo treatment?**

Henalla et al. (1989) has compared ES with no treatment in women with SUI. Eight of the 25 women receiving ES were ‘objectively’ cured or improved (negative pad-test or more than 50% reduction in pad-test) at 3 months, versus none of the 25 women in the no treatment group. One trial has compared ES with control intervention (women were offered use of the Continence Guard [Coloplast AS],...
used infrequently by 14 out of 30 controls) in women with SUI (Bø et al., 1999). Bø et al found that ES was better than control intervention for change in leakage episodes over 3 days, using Social Activity Index and Leakage Index. However, only one of these measures (change in leakage episodes over 3 days) remained significant (p = 0.047) with intention to treat analysis. PFM activity was significantly improved in the ES group after treatment, but the change in activity was not significant when compared with controls. There was no difference in the primary outcome
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measure, i.e., pad-test with standardized bladder volume. Two of 30 controls were cured (<2g leakage) on the pad-test compared to 7/25 in the ES group. One of 30 women in the control group reported the condition was ‘unproblematic’ after treatment versus 3/25 in the ES group, but 28/30 and 19/25 wanted further treatment, respectively.

Six trials compared electrical stimulation with placebo electrical stimulation in women with urodynamic stress incontinence (Hofbauer et al., 1990; Blowman et al., 1991; Laycock and Jerwood, 1993; Sand et al., 1995; Luber and Wolde-Tsadik, 1997; Jeyaseelan et al., 2000). Blowman et al. (1991) compared ES/PFMT versus placebo ES/PFMT in women with urodynamic stress incontinence and for the purposes of analysis this trial was considered to be a comparison of ES with placebo ES. Hofbauer et al. (1990) provided minimal detail of participants, methods and stimulation parameters. Laycock and Jerwood (1993) used clinic-based, short-term (10 treatments) maximal stimulation with an interferential current applied with external surface electrodes. The ES treatment regimen of Jeyaseelan et al. (2000) consisted of a new stimulation pattern, i.e., background low frequency (to target the slow-twitch fibres) and intermediate frequency with an initial doublet (to target the fast-twitch fibres) applied with a vaginal probe. Three trials were based on daily home stimulation for six (Blowman et al., 1991), eight (Jeyaseelan et al., 2000) or 12 weeks (Sand et al., 1995; Luber and Wolde-Tsadik, 1997).

The two most comparable trials in terms of stimulation parameters reported contrasting findings. Sand et al. (1995) found that the ES group has significantly greater changes in the number of leakage episodes in 24 hours, number of pads used, amount of leakage on pad-test and PFM activity (PFM strength measurement, measured by vaginal squeeze pressure) than the placebo stimulation group. In addition, the ES group had significantly improved subjective measures (e.g. visual analogue measure of severity) than the placebo group. Neither group demonstrated significant change in the quality of life measure (SF 36). In contrast, Luber and Wolde-Tsadik (1997) did not find any statistically significant differences between ES and placebo ES groups for rates of self-reported cure or improvement, objective cure (negative stress test during urodynamics), number of incontinence episodes in 24 hours, or Valsalva leak point pressure.

The other trials generally favoured ES over placebo ES. Laycock and Jerwood (1993) generally found significantly greater improvements in the ES group (pad-test, PFM activity, self-reported severity), although the decrease in incontinence episodes was not significantly different between the groups post treatment. Blowman et al. (1991) found a significant decrease in the number of leakage episodes in the ES group only. Hofbauer et al. (1990) reported that 3/11 women in the ES group were cured/improved (not defined) versus 0/11 in the placebo ES group.

Jeyaseelan et al. (2000) did not find statistically significant differences between the two study groups when PFM strength was measured by a device measuring vaginal squeeze pressure, but in contrast, when strength was assessed using digital assessment a statistical significant difference was found. When endurance was assessed an improvement in favour of ES was found over time in the ES group, but not in the sham ES group. The authors suggested that between-group differences may not be significant as a result of the high degree of variance combined with a small sample size. No changes were reported using a pad-test or diaries, but a significant change in favour of the ES group using the UDI-score (Jeyaseelan et al., 2000).

Is ES better than any other single treatment?

Henalla et al. (1989) compared ES (interferential) with vaginal oestrogens (Premarin). Eight of 25 women in the stimulation group reported they were cured or improved versus 3/24 in the oestrogen therapy group. There was a significant reduction in leakage on pad-test in the stimulation group but not the oestrogen group. In contrast, the maximum urethral closure pressure was significantly increased in the oestrogen group but not the stimulation group. Long-term follow-up (9 months) found that subjectively one of the eight women in the ES group who had reported cure/improvement post treatment had recurrent symptoms, as did all three women in the oestrogen group once oestrogen therapy ceased.

Comparing ES with PFMT, using a pad-test as mentioned before, only Bø et al. (1999) found a statistically significant difference in favour of PFMT. It was not clear if the cure data reported by Hofbauer et al. (1990) were derived from a symptom scale or voiding diary; these data were therefore excluded. Only Bø and colleagues measured leakage episodes and quality of life (Social Activity Index) in SUI women. There was no statistically significant difference between the groups for either outcome. At 9 months post treatment, Henalla and co-workers found three of 17 PFMT women and one of eight in the electrical stimulation group reported recurrent symptoms.

In both the trials of Olah et al. (1990) and of Bø et al. (1999) there was no statistically significant difference between vaginal cones (VC) and ES groups for self-reported cure, self-reported cure/improvement or leakage episodes in 24 hours. Bø and colleagues did not find any statistically significant difference between the groups in quality of life (Social Activity Index). Olah and co-workers had to exclude some women from their trial prior to randomization because they could not use cones in the vagina (e.g. wedging of cones).

One study (Castro et al., 2008) in a four-arm RCT compared women with urodynamically proven SUI (USUI) with ES, PFMT, VC and a control group with no treatment. Only data of those women (101 out of 118) who completed the study were analysed for final results. Based on a pad-test with standardized bladder volume, 48% of ES, 46% of PFMT, 46% of VC and only 8% of controls were...
cured, defined as <2 g in pad weight. All active groups were effective but superior to no treatment with no significant difference between the active groups (Castro et al., 2008). The increase of quality of life was 32.4% in the ES, 28.4% in the PFMT and 30.3% in the VC groups, while there was a decrease in the no treatment group of 3.6%. There was no significant difference in quality of life between groups.

**Is (additional) ES better than other (additional) treatments?**

For comparisons of ES with biofeedback-assisted PFMT versus biofeedback-assisted PFMT alone versus a control condition, reporting was limited to a single trial. In the study of Goode et al. (2003), intention-to-treat analysis showed that incontinence was reduced by a mean of 68.6% with biofeedback-assisted PFMT, 71.9% with ES with biofeedback-assisted PFMT, and 52.5% with the control condition. In comparison with the control group both interventions were significantly more effective, but they were not significantly different from each other (p = 0.60). The ES with biofeedback-assisted PFMT had significantly better patient self-perception of outcome (p < 0.001) and satisfaction with progress (p = 0.02).

Two trials compared ES in combination with PFMT versus PFMT alone in women with stress incontinence (Hofbauer et al., 1990; Luber and Wolde-Tsadik, 1997). As both arms in these trials received the same PFMT the trials are essentially investigating the effect of electrical stimulation. Hofbauer gave minimal detail of participants, methods and stimulation parameters. In a three-arm RCT, Knight et al. (1998) compared PFMT versus PFMT with home-based low-intensity ES versus PFMT with clinic-based maximal-intensity stimulation. Ten of 21 women in the PFMT group, 9/25 women in the low-intensity stimulation group, and 16/24 in the maximum-intensity stimulation group reported cure or great improvement. All three groups had significant improvements in pad-test after treatment, with no significant differences in the percentage reduction between the groups. Similarly all three groups had improvements in vaginal squeeze pressure, but there were no significant differences in improvement.

Overall, Knight et al did not find any clear benefits of electrical stimulation in addition to PFMT. This finding is similar to that of Hofbauer et al. (1990) which found no significant differences between the groups receiving combined electrical stimulation/PFMT and PFMT alone.

**Muscle strength**

Several studies reported on PFM strength as an outcome measure (Shepherd et al., 1984; Blowman et al., 1991; Laycock and Jerwood, 1993; Sand et al., 1995; Knight et al., 1998; Bo et al., 1999; Jeyaseelan et al., 2000; Alves et al., 2011). In all but trial I in the study of Laycock and Jerwood (1993) a (kind of) device, measuring PFM strength by vaginal squeeze pressure, was used, with contrasting results between the studies. Laycock and Jerwood did use digital assessment in that trial.

Shepherd et al. (1984) did not find any difference of PFM strength between groups, although no statistical tests were performed to confirm this.

An improvement of PFM strength in both groups (PFMT+ES versus PFMT+sham ES), with more improvement in the PFMT+ES was reported in the study of Blowman et al. (1991). However, no statistical tests were performed to test statistical significance.

When digitally tested, Laycock and Jerwood found a pre-/post-treatment statistically significant improvement (p = 0.0035) only in the ES group (PFMT vs ES [interferential therapy]). In this trial they did not report the in-between results. In the second trial they used PFMT strength measurement, measured by vaginal squeeze pressure to measure PFM strength at pelvic floor muscle maximal contraction and found a significant increase only in the ES group.

Sand et al. (1995) performed PFM strength measurements using a device measuring vaginal squeeze pressure in 35 patients and 17 controls who used identical sham devices before and after a 15-week treatment period. The active group had a significant improvement in vaginal muscle strength when compared to the controls. In the active group mean (+ SE) change of vaginal muscle strength (mmHg) before and after treatment was 4.6 ± 1.4, and in the control group 1.1 ± 1.5 (p = 0.02).

Knight et al found a significant increase of PFM strength in all groups, the biggest in the ES group in a clinical setting. However, there was no significant difference between groups. In contrast with the ES group, Bo and colleagues (1999) reported significant improvement of PFM strength only in the PFMT group (compared with no treatment). As indicated before, Jeyaseelan et al. (2000) did not detect any statistically significant differences between ES and sham ES when PFM strength was measured using a device measuring vaginal squeeze pressure. However, if strength was assessed using digital assessment a statistical significant difference in favour of ES was found.

The difference between included studies with respect to outcome of pelvic floor muscle strength, using a device measuring PFM strength by vaginal squeeze pressure, can be explained by the huge variation in measurement protocols, devices used and assessment differences. For instance, in the studies of Shepherd et al. (1984) and Blowman et al. (1991) no statistical tests were performed. Knight et al. (1998) and Laycock and Jerwood (1993) did not blind the outcome measurement assessors, while Sand et al. (1995), Bo et al. (1999) and Jeyaseelan et al. (2000) did.

**Adverse events**

Four trials (Hahn et al., 1991; Sand et al., 1995; Smith, 1996; Bo et al., 1999) reported side-effects related to ES, including vaginal irritation or infection, urinary tract
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infection or pain and/or vaginal bleeding. Sand et al reported that all adverse events were reversible. Besides the ES group, the VC group also reported adverse events in the trial by Bø and colleagues (1999).

CONCLUSION

- There is a marked lack of consistency in the electrical stimulation protocols; this implies a lack of understanding of the physiological principles of rehabilitating urinary incontinence through electrical stimulation used in clinical practice to treat women with stress urinary incontinence.
- There is insufficient evidence to judge whether electrical stimulation is better than no or placebo treatment for women with stress urinary incontinence.
- Although conclusive evidence is lacking, PFMT seems to be better than electrical stimulation in women with stress urinary incontinence.
- There is insufficient evidence to determine whether electrical stimulation is better than vaginal oestrogens or vaginal cones in women with stress urinary incontinence.
- At present it seems that there is no extra benefit in adding electrical stimulation to PFMT.
- There is need for more basic research to explore the working mechanism of electrical stimulation in women with stress urinary incontinence, and to determine the best electrical stimulation protocol(s) and outcome measures for this kind of patient.

CLINICAL RECOMMENDATIONS

- Up to now there is no convincing evidence from RCTs that electrical stimulation is a useful treatment in women with stress urinary incontinence. So far, it is impossible to recommend the most optimal electrical stimulation regimen and protocol.
- A protocol based on the hypothesis that electrical stimulation might help patients who are unaware how to contract the PFM and are not capable of doing so voluntarily to regain awareness of the PFM, should be considered to be tested in a high-quality RCT.

REFERENCES


7.2 Female overactive bladder

Pelvic floor muscle training for overactive bladder

Kari Bø

INTRODUCTION

In clinical practice, many patients with overactive bladder (OAB) symptoms are treated with pelvic floor muscle training (PFMT) with and without biofeedback, electrical stimulation, bladder training, or medication, and often many of the interventions are combined. When different methods are combined it is not possible to analyse the cause–effect of the different interventions. In most systematic reviews on efficacy of PFMT to prevent and treat urinary incontinence, studies including patients with symptoms or urodynamic diagnosis of stress urinary incontinence (SUI), urge urinary incontinence (UUI) and mixed urinary incontinence (MUI) are combined. This makes it impossible to understand the real effect of the different interventions on each condition.

Although there are new theories suggesting PFM dysfunction as a common cause of the two main diagnoses (SUI and UUI) (Artibani, 1997; Mattiasson, 1997), the mechanisms behind the PFM dysfunction in each of these diagnoses are not yet thoroughly understood, and pathophysiological factors may be very different (rupture of the pelvic floor and connective tissue during childbirth for SUI, caffeine-induced urethral incontinence in an elderly woman). Optimally, the physical therapy intervention should relate to the underlying pathophysiological condition. PFMT may have different cure and improvement rates for SUI and UUI, and the combination of heterogeneous patient groups in systematic reviews and meta-analyses may disseminate the real cure rate for each of the diagnoses. In addition, and most important, an optimal PFMT protocol may be different for the two conditions due to a different theoretical rationale. In this chapter we will therefore cover studies including only patients with symptoms/diagnosis of OAB and studies where PFMT with or without biofeedback or vaginal cones was the only intervention.

RATIONALE FOR EFFECT OF PFMT FOR OAB

The rationale behind the use of PFMT to treat symptoms of OAB is based on observations from electrical stimulation and urodynamic assessment during PFM contraction. Godec et al. (1975) studied 40 patients with cystometrograms, taken during and after 3 minutes of 20 Hz functional electrical stimulation (FES). The results showed that, during FES, hyperactivity of the bladder was diminished or completely abolished in 31 of 40 patients. One minute after stimulation cessation, the inhibition was still present. Mean bladder capacity also increased significantly, from $151 \pm 126$ ml to $206 \pm 131$ ml ($p < 0.05$).

De Groat (1997) noted that during the storage of urine, distension of the bladder produces low-level vesicalafferent firing. This stimulates the sympathetic outflow to the bladder outlet (base and urethra), and the pudendal outflow to the external urethral sphincter. He stated that these responses occur by spinal reflex pathways, representing ‘guarding reflexes’ that promote continence. Sympathetic firing also inhibits the detrusor muscle and bladder ganglia. Morrison (1993) claimed that the excitatory loop through Barrington’s micturition centre is switched on at bladder pressures between 5 and 25 mmHg, whereas the inhibitory loop through the raphe nucleus is active predominantly above 25 mmHg. The inhibition is at the automatic level, with the person not being conscious of the increasing tone in the PFM and urethral wall striated muscles.

Shafik and Shafik (2003) investigated the effect of a voluntary PFM contraction on detrusor and urethral pressures in 28 patients with OAB (mean age 48.8 years±10.2 years, 18 men and 10 women) and 17 healthy volunteers (mean age 42.6 years±9.8 years, 12 men and 5 women). They found that during PFM contraction the urethral pressure significantly increased and vesical pressure significantly decreased in both patients and healthy subjects. The change during PFM contraction was significantly larger in the healthy volunteers. The authors concluded that PFM contractions led to a decline of detrusor pressure, an increase of urethral pressures and suppression of the micturition reflex, and that the results encourage PFM contractions in treatment of OAB.

Clinical experience tells us that patients can successfully inhibit urgency, detrusor contraction and urinary leakage by walking, bending forwards, crossing their legs, using hip adductor muscles with or without conscious co-contraction of the PFM, or by conscious contraction of the PFM alone. After inhibition of the urgency to void and detrusor contraction, the patients may gain time to reach the toilet and thereby prevent leakage. The reciprocal inhibition reflex runs via cerebral control, recruiting ventral horn motor neurons for voluntary PFM contraction and inhibiting the parasympathetic excitatory pathway for the micturition reflex via Onuf’s ganglion. This mechanism has been exploited as part of bladder training regimens (Burgio et al., 1998). There may therefore be two main
hypotheses for the mechanism of PFMT to treat urgency incontinence:

- intentional contraction of the PFM during urgency, and holding of the contraction till the urge to void disappears;
- strength training of the PFM with long-lasting changes in muscle morphology, which may stabilize neurogenic activity.

None of the studies in this field (neither uncontrolled studies nor RCTs) have evaluated whether changes in the inhibitory mechanisms really occur after PFMT. In addition, research in this area is relatively new, and there does not seem to be any consensus on the optimal exercise protocol to prevent or treat OAB (Bø and Berghmans, 2000). The theoretical basis of how PFMT may work in the treatment of OAB therefore remains unclear (Berghmans et al., 2000).

**METHODS**

This systematic review is based on three former systematic reviews (Berghmans et al., 2000; Bø and Berghmans, 2000; Greer et al., 2012), Cochrane reviews (Dumoulin and Hay-Smith, 2010; Hay-Smith et al., 2011; Herderschee et al., 2011; Rai et al., 2012) and the literature found in the International Consensus on Incontinence (ICI) meeting (Moore et al., 2013). In addition we have conducted an electronic search on PubMed limited to the past 10 years. Only fully published randomized controlled trials (RCTs) including female patients with OAB symptoms (frequency, urgency and UUI) alone were included. Methodological quality is classified according to the PEDro rating scale, which has been found to have high reliability (Maher et al., 2003).

**EVIDENCE FOR PFMT TO TREAT OAB SYMPTOMS**

Four RCTs using PFMT alone to treat symptoms of OAB were found (Nygaard et al., 1996; Berghmans et al., 2002; Millard, 2004; Wang et al., 2004). The results of the studies are presented in Table 7.10, and methodological quality in Table 7.11. The studies had moderate to high methodological quality.

Nygaard et al found (1996) significant improvement in many variables in the subgroup of women with detrusor instability. There was no difference in outcome between the two randomized groups, and no comparison with non-treated controls.

Berghmans et al. (2002) did not demonstrate any significant effect of their exercise protocol compared to an untreated control group. Wang et al. (2004) found that the significant subjective improvement/cure rate of OAB was the

**Table 7.10 RCTs of PFMT to treat overactive bladder symptoms**

<table>
<thead>
<tr>
<th>Author</th>
<th>Nygaard et al., 1996</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Design</strong></td>
<td>2-arm RCT: with and without audiotape</td>
</tr>
<tr>
<td><strong>Sample size and age (years)</strong></td>
<td>71 women, mean age 53 years (SD 13); 17 with detrusor instability</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>History and urodynamics, cystometry</td>
</tr>
<tr>
<td><strong>Training protocol</strong></td>
<td>Two 5-minute daily sessions for 3 months of PFMT with or without audiotape. Telephoned at 2, 6 and 10 weeks. Clinic visit with PFM assessment by vaginal palpation at 4, 8 and 12 weeks</td>
</tr>
<tr>
<td><strong>Drop-out</strong></td>
<td>16 (22.5%), 3 with detrusor instability (17.6%)</td>
</tr>
<tr>
<td><strong>Adherence</strong></td>
<td>Not reported</td>
</tr>
<tr>
<td><strong>Results</strong></td>
<td>No difference between PFMT with or without audiotape. Significant improvement in incontinence episodes per day, number of voids per night, urge score, number of pads used per day and muscle strength in women with detrusor instability</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Author</th>
<th>Berghmans et al., 2002</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Design</strong></td>
<td>4-arm RCT LUTE, ES, ES+LUTE, no treatment</td>
</tr>
<tr>
<td><strong>Sample size and age (years)</strong></td>
<td>68 women, mean age 55.2 (SD 14.4)</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>Ambulatory urodynamics + micturition diary (DAI score ≥0.5 included)</td>
</tr>
<tr>
<td><strong>Training protocol</strong></td>
<td>9 treatments once a week + daily home training programme</td>
</tr>
<tr>
<td><strong>Drop-out</strong></td>
<td>LUTE: bladder retraining, selective contraction of the PFM to inhibit detrusor contraction, 20s hold, toilet behaviour</td>
</tr>
<tr>
<td><strong>Adherence</strong></td>
<td>10/68 (15%)</td>
</tr>
<tr>
<td><strong>Results</strong></td>
<td>ITT analysis of all</td>
</tr>
<tr>
<td></td>
<td>Significant decrease in DAI score (0.22, p &gt;0.001), but no difference compared with no treatment</td>
</tr>
</tbody>
</table>

(Continued)
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same between the electrical stimulation group and in the biofeedback-assisted PFMT group, but lower in the PFMT home training group. Millard (2004) did not show any additional benefit for a simple PFMT protocol (two-page written instruction, no assessment of ability to contract, and no follow-up or supervised training). The effect of PFMT on OAB is therefore questionable.

**Quality of the intervention: dose–response issues**

Quality of the interventions is difficult to judge because there are no direct recommendations on how PFMT should be conducted to inhibit urgency and detrusor contraction. The published studies have all used different exercise protocols. Berghmans et al. (2002) and Millard (2004) included intentional contraction of the PFM to inhibit detrusor contractions in addition to a strength training programme. However, we have no information about how many conducted the exercises in Millard’s study, and Berghmans et al. (2002) also included bladder training in their protocol. The protocol from Berghmans et al did not show any effect when compared with untreated controls, but if there had been an effect it would not be possible to tell whether this was due to the exercises or the bladder training. In Millard’s study (2004) a very weak exercise protocol was conducted. There was no control of ability to contract the PFM, patients were left alone to exercise and there was no report on adherence to the exercise protocol. The exercise period varied between 9 and 12 weeks in duration in the four RCTs in this area. This may be too short to treat a complex condition such as OAB.

**CONCLUSION**

There are few RCTs in this area and the results are difficult to interpret. In general the studies have moderate to high

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### Table 7.10 RCTs of PFMT to treat overactive bladder symptoms—cont’d

<table>
<thead>
<tr>
<th>Author</th>
<th>Design</th>
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<tbody>
<tr>
<td></td>
<td>2-arm RCT international multicentre, 54 sites: tolterodine, tolterodine+PFMT</td>
</tr>
<tr>
<td>Sample size and age (years)</td>
<td>480 women (75%) and men, mean age 53.4 (SD 17.4)</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Symptoms of OAB ≥6 months: frequency ≥8×/day, urgency and urge incontinence ≥1/24 h</td>
</tr>
<tr>
<td>Training protocol</td>
<td>12 weeks</td>
</tr>
<tr>
<td>Drop-out</td>
<td>Written instruction on PFMT 10s hold×15 twice a day; 20 contractions once a day</td>
</tr>
<tr>
<td>Adherence</td>
<td>ITT analysis of all</td>
</tr>
<tr>
<td>Results</td>
<td>Both groups had significant reduction in incontinence episodes, numbers of micturitions, urgency episodes, improvement in perception of bladder symptoms</td>
</tr>
<tr>
<td></td>
<td>No significant difference between groups</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Author</th>
<th>Design</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>3-arm RCT PFMT, PFMT with biofeedback, ES</td>
</tr>
<tr>
<td>Sample size and age (years)</td>
<td>120 women, mean age 52.7 (SD 13.7)</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Symptoms of OAB &gt;6 months, frequency ≥8×/day, urge incontinence ≥once/day</td>
</tr>
<tr>
<td>Training protocol</td>
<td>12 weeks</td>
</tr>
<tr>
<td>Drop-out</td>
<td>Home exercise: based on individual PFM strength 3×/day</td>
</tr>
<tr>
<td>Adherence</td>
<td>Same home training+in-clinic biofeedback twice a week</td>
</tr>
<tr>
<td>Results</td>
<td>PFMT: 83%</td>
</tr>
<tr>
<td></td>
<td>PFMT+biofeedback: 75%</td>
</tr>
<tr>
<td></td>
<td>ES: 79%</td>
</tr>
<tr>
<td></td>
<td>Home exercise PFMT: 14.5 days</td>
</tr>
<tr>
<td></td>
<td>PFMT+biofeedback: 8.5 days</td>
</tr>
<tr>
<td></td>
<td>Improvement/cured: PFMT 38%, PFMT/biofeedback 50%</td>
</tr>
<tr>
<td></td>
<td>PFM strength: no significant differences between exercise groups, but between both exercise groups and ES. No change in urodynamic parameters. Significant change in several QoL measures for different groups</td>
</tr>
</tbody>
</table>

DAI score, detrusor activity index formed from results of extramural ambulatory cystometry and micturition diary; LUTE, lower urinary tract exercise. For other abbreviations, see text.
methodological quality, but the exercise protocols may not have been optimal. Because the pathophysiological background for OAB is not clear, it is difficult to plan an optimal training protocol. Based on the theoretical knowledge and symptoms of bladder overactivity it seems reasonable to put more emphasis on the inhibition mechanisms of the PFM contraction, and teaching and follow-up of patients trying to contract the PFM when there is an urge to void. There is a need for more basic research to understand the role of a voluntary PFM contraction in inhibition of the micturition reflex and future RCTs with high interventional and methodological quality are recommended.

### Table 7.11 PEDro quality score of RCTs in systematic review of PFMT to treat overactive bladder symptoms

<table>
<thead>
<tr>
<th>Study</th>
<th>E</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>Total score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nygaard et al., 1996</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>?</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>5</td>
</tr>
<tr>
<td>Berghmans et al., 2002</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>8</td>
</tr>
<tr>
<td>Millard, 2004</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>8</td>
</tr>
<tr>
<td>Wang et al., 2004</td>
<td>?</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>5</td>
</tr>
</tbody>
</table>

+, criterion is clearly satisfied; −, criterion is not satisfied; ?, not clear if the criterion was satisfied. Total score is determined by counting the number of criteria that are satisfied, except that ‘eligibility criteria specified’ score is not used to generate the total score. Total scores are out of 10.

### CLINICAL RECOMMENDATIONS

- To date there is no convincing evidence from RCTs to support the use of PFMT in the treatment of OAB. There are no training protocols to recommend.
- Clinical experience and basic research show that it may be possible to learn to inhibit detrusor contraction by intentionally contracting the PFM and holding the contraction to stop the urge to void. A protocol based on patients’ experiences needs to be tested in a high-quality RCT.

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Evidence-Based Physical Therapy for the Pelvic Floor


Electrical stimulation for overactive bladder

Bary Berghmans

INTRODUCTION

Clinical experience has shown that overactive bladder function with associated urge urinary incontinence (UUI) is not amenable to surgical correction (Millard and Oldenburg, 1983; Ulmsten, 1999). Based on poor studies that did not compare sacral neuromodulation with other treatment methods, a recent Cochrane review concluded that sacral neuromodulation can be of benefit in selected patients with overactive bladder symptoms in which other methods of treatment have failed (Herbison and Arnold, 2009; Dmochowski et al., 2013). Therefore, it is important first to find another satisfactory treatment modality for patients with this problem. Pharmaceutical agents in general lead to disappointing results, with success rates of 60–70% in all adults and 25–75% in adults >65 years (Andersson et al., 2013) for the most effective single agents. But all these agents show side-effects to a greater or lesser extent in most of the patients, limiting their usefulness (Andersson et al., 2013). They are frequently not continued indefinitely (Andersson et al., 2013) and lead to poor tolerability in about 15% of them (Sussman and Garely, 2002). The short duration of most clinical trials and the lack of long-term follow-up give little information about the short- and long-term efficacy and acceptability of drugs (Hay-Smith et al., 2002; Madhuvarata et al., 2012).

Although combination therapy is claimed to be more successful, the use of drugs produces many side effects, inevitably leading to non-compliance and the recurrence of incontinence (Millard and Oldenburg, 1983; Resnick, 1998; Hay-Smith et al., 2002). Besides bladder (re)training and PFMT with or without biofeedback, electrical stimulation (ES) is one of the physical therapy treatment modalities that is used for the management of women with bladder overactivity (OAB).

The theoretical basis of how ES for the treatment of OAB actually works still remains unclear. Is it the change in pelvic floor muscle activity during nervous excitation that automatically should inhibit or better prevent detrusor overactivity (Messelink, 1999; Lewis and Cheng, 2007)? Is it a learning process that should make the patient aware of contracting the PFM during urgency in order to inhibit involuntary detrusor contraction (reciprocal inhibition) (Messelink, 1999) or a cortical inhibition (Pannek et al., 2010)? Is it that increase in strength of the PFM could provide more inhibition of the overactivity of the bladder (Messelink, 1999)? The different physical therapy treatment modalities are therefore still based on hypotheses for the underlying pathologies causing bladder overactivity. However, clinical experience has shown that different physical therapy treatment modalities generally will provide some progress in most individuals with OAB. Improved bladder control can occur even in the cognitively
impaired individual (Engel et al., 1990; McCormick et al., 1990; Schnelle et al., 1990; Colling et al., 1992).

**Rationale for Electrical Stimulation for OAB**

The literature concerning electrical stimulation in the management of OAB and urge urinary incontinence is very difficult to interpret, due to the lack of a well-substantiated biological rationale underpinning the use of electrical stimulation. The mechanisms of action may vary depending on the cause(s) of OAB and the structure(s) being targeted by ES, e.g. pelvic floor muscle or detrusor muscle, peripheral or central nervous system. Eriksen (Eriksen, 1989; Eriksen and Eik-Nes, 1989) and Fall (2000) claimed that electrical stimulation theoretically stimulates the detrusor inhibition reflex (DIR) and pacifies the micturition reflex, resulting in a decrease of overactive bladder dysfunction. Schmidt (1988) hypothesized that the electrical stimulus was thought to activate the pudendal nerve, contracting the pelvic floor musculature and external urinary sphincter.

Elabbady et al. (1994) and Weil (2000) suggested that electrical stimulation of pelvic floor muscles induces a reflex contraction of the striated para-urethral and periurethral muscles, accompanied by a simultaneous reflex inhibition of the detrusor muscle. This reciprocal response depends on a preserved reflex arc through the sacral micturition reflex centre. In order to obtain a therapeutic effect of pelvic floor stimulation in women with OAB, peripheral innervation of the pelvic floor muscles must at least partially be intact (Eriksen, 1989).

This means that, when increasing stimulation is applied on the nerve, improved contraction of the muscles is obtained, resulting in more efficient detrusor inhibition (Schmidt, 1988; Elabbady et al., 1994; Hoebeke et al., 2001).

However, according to Weil (2000), detrusor inhibition is not the result of activating somatosensory efferents of the pudendal nerve (Schultz-Lampel, 1997). Schultz-Lampel (1997) holds the β-fibres of the sacral nerve afferents responsible for the electrically induced inhibition of detrusor contraction. Pudendal afferent β-fibres from the urinary sphincter and/or pelvic floor induce electrical inhibition of detrusor contractions (Schultz-Lampel, 1997). As these fibres are large in diameter, these nerve cells can be depolarized with minimal amounts of energy. Therefore, electrical stimulation should not be applied through muscle contraction, nor should excess energy be applied to produce depolarization of the smaller nerve fibres, like B and unmyelinated C-fibres, which result in a painful sensation (Weil, 2000).

Electrical stimulation therapy alone, both external or internal, is suggested to inhibit the parasympathetic motor neurons to the bladder and to enable an effective reduction or inhibition of detrusor activity by stimulation of (large diameter) afferents of the pudendal nerve (Eriksen, 1989; Eriksen and Eik-Nes, 1989; Elabbady et al., 1994; Fall and Lindström, 1994; Fall, 2000; Weil, 2000; Hoebeke et al., 2001).

**Evidence for Electrical Stimulation to Treat OAB (Symptoms)**

At present few studies are performed regarding the efficacy of ES for OAB (Moore et al., 2013). Systematic reviews revealed only weak evidence on the efficacy of ES alone or in combination with pelvic floor muscle training (PFMT) for women with UUI (Berghmans al, 2000; Moore et al., 2013).

However, these findings did not prove the ineffectiveness of ES as a treatment modality for bladder overactivity as a whole. It was our assumption that the lack of efficacy is most likely caused by methodological flaws like heterogeneity of study groups and suboptimal research designs.

Electrical stimulation for OAB is provided by clinic-based mains-powered machines or portable battery-powered stimulators (Fig. 7.6). Also in this area, ES offers a seemingly infinite combination of current types, waveforms, frequencies, intensities, electrode placements, electrical stimulation probes, etc. (Fig. 7.7). Without that clear biological rationale, mentioned above, it is difficult to make reasoned choices of ES parameters. Hence, as in ES studies for stress urinary incontinence, we see a wide variety of stimulation devices and protocols being used for OAB.

This section reviews the evidence in women comparing non-surgical ES with no treatment, placebo ES and comparisons of different ES protocols. It also includes trials comparing non-surgical ES with any other single intervention (e.g. magnetic stimulation, PFMT, weighted vaginal cones, surgery, medication, etc.) and trials comparing ES with any other combined intervention versus that other combined intervention alone.
Methods

Four systematic reviews (Berghmans et al., 1998, 2000; Hay-Smith et al., 2001; Moore et al., 2013) have been published that include trials relevant to this chapter. The following qualitative summary of the evidence regarding electrical stimulation is based on the trials included in all of the previous systematic reviews with addition of trials performed after publication of the reviews and/or located through additional searching. This search was conducted in the same fashion as described above in the section of this chapter on ES in women with SUI.

To be included here a trial needed to (a) be a RCT, (b) include women with OAB or UUI symptoms, and (c) compare different electrical stimulation protocols or investigate the effect of electrical stimulation versus no treatment, placebo treatment, any other single treatment, with any other combined intervention versus that other combined intervention. Published abstracts and reports of trials in progress were excluded.

Quality of data

The two trials by Yamanishi and co-workers (Yamanishi et al., 2000a; Yamanishi et al., 2000b), the trial by Soomro et al. (2001) and Walsh et al. (2001) included both men and women with OAB and urinary incontinence. It is possible that the effects of stimulation might be different between genders (due to difference in electrode placement for example). So, although some of these studies included a large number of women with OAB and/or UUI symptoms and reported significant objective and/or subjective results in favour of ES in comparison to no or placebo treatment, for reasons of heterogeneity of inclusion criteria, i.e., differences in gender, we decided not to use these studies for the analysis of results where they did not perform subgroup analysis or did not differentiate the effects of treatment in women versus men. Only the study of Yamanishi et al. (2000b) could partly be used, where the authors did report results from subgroups according to gender. Table 7.12 provides details of results of all included studies (n = 12).

Again, the PEDro rating scale was used to classify the methodological quality of the included studies (Table 7.13). The studies had low (n = 2), moderate (n = 1) to high (n = 9) methodological quality.

Table 7.12

<table>
<thead>
<tr>
<th>Author</th>
<th>Arruda et al., 2008</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Design</strong></td>
<td>3-arm RCT</td>
</tr>
<tr>
<td></td>
<td>ES</td>
</tr>
<tr>
<td></td>
<td>Oxybutynin</td>
</tr>
<tr>
<td></td>
<td>PFMT</td>
</tr>
<tr>
<td><strong>Sample size and age (years)</strong></td>
<td>64 women, age range 35–80</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>DO subjective response, voiding diary, urodynamics. UUI dominant in all MUI; SUI factor in 28.6% ES, 31.8 oxybutynin, 28.6% PFMT</td>
</tr>
<tr>
<td><strong>Training protocol</strong></td>
<td>12 weeks; 2x/week 20 min clinic ES: vaginal electrode, 10 Hz, pulse with 1.0 ms, intermittent biphasic, 1 max (range 10–100 mA). Oxybutynin: immediate release 5 mg 2x/day. PFMT: 2x/week 45 min clinic sessions: in supine, sitting and orthostatic positions 40 fast (2 and 5s) and 20 10 s sustained contractions, 10 s relaxation, same home regimen</td>
</tr>
<tr>
<td><strong>Drop-out</strong></td>
<td>13/77 women: 4 unable to comply with ES, 4 oxybutynin, 5 PFMT, 1 no show at post-treatment urodynamics</td>
</tr>
<tr>
<td><strong>Adherence</strong></td>
<td>1 no show at post-treatment urodynamics</td>
</tr>
<tr>
<td>Results</td>
<td>Subjective symptomatic improvement: ES 52%, oxybutynin 77%, PFMT 76%. Urgency resolved: ES 52%, oxybutynin 64%, PFMT 57%. Urodynamic evaluation normal in 57% ES, oxybutynin 36%, PFMT 52%. Maximum detrusor involuntary contraction pressure decreased in all groups (p &lt; 0.05). All treatments equally effective. Oxybutynin: high percentage of dry mouth. Side-effects: only reported for oxybutynin: 72.7% dry mouth, micturition difficulty 9.1%. Follow-up 1 year: ES 36.4%; oxybutynin 58.8%, PFMT 56.2% persistent improvement</td>
</tr>
<tr>
<td>Author</td>
<td>Berghmans et al., 2002</td>
</tr>
<tr>
<td>Design</td>
<td>4-arm RCT</td>
</tr>
<tr>
<td>LUTE</td>
<td></td>
</tr>
<tr>
<td>ES</td>
<td></td>
</tr>
<tr>
<td>ES+LUTE</td>
<td></td>
</tr>
<tr>
<td>No treatment</td>
<td></td>
</tr>
<tr>
<td>Sample size and age (years)</td>
<td>68 women, mean (SD) age 55.2 (14.4)</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Ambulatory urodynamics + micturition diary (DAI-score ≥ 0.5 included)</td>
</tr>
<tr>
<td>Training protocol</td>
<td>9 treatments once a week daily home programme</td>
</tr>
<tr>
<td>LUTE: bladder retraining; selective contraction of the PFM to inhibit detrusor contraction; 20 seconds hold; toilet behaviour</td>
<td></td>
</tr>
<tr>
<td>Drop-out</td>
<td>10/68 (15%)</td>
</tr>
<tr>
<td>Adherence</td>
<td>92% (reported for all groups together)</td>
</tr>
<tr>
<td>Results</td>
<td>Dunnett's t-test: ES compared to no treatment significant difference in decrease in DAI-score (0.23, p &gt; 0.039), other treatment groups no difference compared with no treatment</td>
</tr>
<tr>
<td>Author</td>
<td>Bower et al., 1998</td>
</tr>
<tr>
<td>Design</td>
<td>3-arm RCT</td>
</tr>
<tr>
<td>Low frequency (LF) ES</td>
<td></td>
</tr>
<tr>
<td>High frequency (HF) ES</td>
<td></td>
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<tr>
<td>Sham ES</td>
<td></td>
</tr>
<tr>
<td>Sample size and age (years)</td>
<td>80 women; 49 OAB, 31 sensory urge; mean (SD) age 56.5 (16.9)</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Urodynamics (cystometry)</td>
</tr>
<tr>
<td>Training protocol</td>
<td>LF ES: transcutaneous 10Hz, pulse width 200 μs, sacral placement, I max; HF ES: 150 Hz, 200 μs, suprapubic placement, I max; sham ES (placement at random); 1 session during filling cystometry</td>
</tr>
<tr>
<td>Drop-out</td>
<td>None</td>
</tr>
<tr>
<td>Adherence</td>
<td>Not applicable</td>
</tr>
<tr>
<td>Results</td>
<td>OAB: sham ES no sign change first desire to void (p = 0.69), max cystometric capacity and detrusor pressure idem; both active ES groups reduction max detrusor pressure, significant increase first desire to void, no change max cystometric capacity; no change in detrusor pressure at first desire to void; 44% in both active ES groups stable. Sensory urgency: significant increase first desire to void only in 150 Hz active ES; max cystometric capacity increase only in sham ES!</td>
</tr>
<tr>
<td>Author</td>
<td>Brubaker et al., 1997</td>
</tr>
<tr>
<td>Design</td>
<td>2-arm RCT</td>
</tr>
<tr>
<td>ES</td>
<td></td>
</tr>
<tr>
<td>Sham ES</td>
<td></td>
</tr>
<tr>
<td>Sample size and age (years)</td>
<td>148 women, subgroup OAB 28 women, age mean 57 (SD 12)</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Urodynamics, micturition diary</td>
</tr>
<tr>
<td>Training protocol</td>
<td>ES: transvaginal 20 Hz, 2/4s work/rest, pulse width 0.1 ms, bipolar square wave, I0–100 mA</td>
</tr>
<tr>
<td>Sham ES: same parameters, no I, both groups 8 weeks treatment</td>
<td></td>
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</tbody>
</table>

Table 7.12: Randomized controlled trials on electrical stimulation to treat OAB and/or urgency urinary incontinence symptoms—cont’d
### Table 7.12 Randomized controlled trials on electrical stimulation to treat OAB and/or urgency urinary incontinence symptoms—cont’d

| Drop-out | 18% |
| Adherence | ES vs sham ES mean compliance 87% vs 81% at 4 and 8 treatment weeks |
| Results | ES vs sham ES 54% (n = 33) OAB pretreatment reduced to 27% (n = 16) post treatment (p = 0.0004) vs sham ES 47% (n = 28) to 42% (p = 0.22) |
| 24 hr frequency NS; 6 weeks no. of accidents/24 h (average) NS; adequate subjective improvement p = 0.027; QoL NS difference; no analysis diaries because of incomplete data |

### Design
2-arm RCT

#### Sample size and age (years)
72 women with urgency/UUI (predominant)

#### Diagnosis
History-taking, physical examination; optional urodynamics, cystoscopy

#### Training protocol
ES: 10 20-min sessions 1–2x/week, vaginally and/or transanally, 5–10 Hz, I max tolerable

#### Design
2-arm RCT

#### Sample size and age (years)
35 women with UUI (OAB symptoms predominantly); age, median (range) ES (n = 18) 57.5 (36–78); trospium hydrochloride (n = 17) 60.0 (37–78)

#### Diagnosis
History-taking, physical examination; urodynamics

#### Training protocol
ES: 18 20-min sessions 3x/week, vaginal probe, 5 Hz, biphasic, symmetrical rectangular pulse, I max tolerable total duration 100μs

#### Design
2-arm RCT

#### Sample size and age (years)
72 women with urgency/UUI (predominant)

#### Diagnosis
History-taking, physical examination; optional urodynamics, cystoscopy

#### Training protocol
ES: 10 20-min sessions 1–2x/week, vaginally and/or transanally, 5–10 Hz, I max tolerable

#### Drop-out
15%:
ES 6/37
Tolterodine 5/35

#### Adherence
Tolterodine: 2/32 stopped at 6 weeks, 8/31 at 6 months, 1/32 ES switched to tolterodine

#### Results
Micturitions/24 hr no significant difference between groups; mean voided volume idem

Within groups: both groups significant decrease in micturitions/24 hr and increase in mean voided volume

Subjective: at 6 months ES 73%, tolterodine 71% lesser degree of bother from bladder symptoms

Tolterodine 6% higher bother, ES equal

Cured/improved no significant difference between groups

### Author
Franzen et al., 2010

### Design
2-arm RCT

#### Sample size and age (years)
35 women with UUI (OAB symptoms predominantly); age, median (range) ES (n = 18) 57.5 (36–78); trospium hydrochloride (n = 17) 60.0 (37–78)

#### Diagnosis
History-taking, physical examination; urodynamics

#### Training protocol
ES: 18 20-min sessions 3x/week, vaginal probe, 5 Hz, biphasic, symmetrical rectangular pulse, I max tolerable total duration 100μs

Trospium hydrochloride (Spasmex 30 mg tablet), 6 weeks dose 45 mg/day

#### Drop-out
11.4%:
ES: 2/18

### Adherence
Compliance measured in trospium hydrochloride group but results not given

### Results
Urodynamic parameters: no significant differences between groups during all controls (after 6 weeks [end treatment], 10 and 18 weeks). Both groups equal progress

Voiding diary: no significant differences between groups during all controls (after 6 weeks [end treatment], 10 and 18 weeks), ES better progress

Subjective: VAS urgency severity. IIQ-7 significant decrease in both groups. No significant difference between groups

Treatment satisfaction: 87.6% ES and 93.3% trospium hydrochloride

Side-effects significantly less in ES (5/18, 27.7%) than in trospium hydrochloride (8/17, 47%)

### Author
Ozdedeli et al., 2010
<table>
<thead>
<tr>
<th>Author</th>
<th>Smith, 1996</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Design</strong></td>
<td>2-arm RCT</td>
</tr>
<tr>
<td></td>
<td>ES</td>
</tr>
<tr>
<td><strong>Sample size and age</strong></td>
<td>Propanthelinebromide</td>
</tr>
<tr>
<td>(years)</td>
<td>Subgroup detrusor instability, 38 women, age range 44–73</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>Cystoscopy only when indicated, complex video urodynamic study, i.e. uroflow, UPP, cystometrography, Vasalva LPP</td>
</tr>
<tr>
<td><strong>Training protocol</strong></td>
<td>Study group (SG): ES 5 s contractions (range 3–15), duty circle 1:2, treatment time 15–60 min 2x/day for 4 mth, I 5-max 25 mA</td>
</tr>
<tr>
<td></td>
<td>Control group (CG): Propantheline bromide 7.5–45 mg 2–3x/day; written/verbal instructions timed voiding and bladder retraining</td>
</tr>
<tr>
<td><strong>Drop-out</strong></td>
<td>None</td>
</tr>
<tr>
<td><strong>Adherence</strong></td>
<td>&gt;80%</td>
</tr>
<tr>
<td><strong>Results</strong></td>
<td>CG: IEF 50% improved; SG ES: IEF 72% improved, including 4 pts cured, &gt; bladder capacity trend both groups; no improvement urodynamic variables; in between NS</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Author</th>
<th>Soomro et al., 2001b</th>
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</thead>
<tbody>
<tr>
<td><strong>Design</strong></td>
<td>2-arm RCT</td>
</tr>
<tr>
<td></td>
<td>ES</td>
</tr>
<tr>
<td><strong>Sample size and age</strong></td>
<td>Oxybutynin</td>
</tr>
<tr>
<td>(years)</td>
<td>43 patients: 30 women, 13 men; mean (SD) age 50 (15)</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>OAB symptoms, SF-36 QoL, Bristol urinary symptom questionnaire; clinical assessment urodynamics, uroanalysis, urine cytology</td>
</tr>
<tr>
<td><strong>Training protocol</strong></td>
<td>ES: transcutaneous, 2 self-adhesive pads bilateral perianal region (S2/3 dermatome), I variable tickling sensation, 20 Hz, 200 μs, continuous, 6 hours daily; oxybutynin 2.5 mg orally 2x/day, titrated to 5 mg, orally 3x/day by day 7</td>
</tr>
<tr>
<td></td>
<td>Control group (CG): No current</td>
</tr>
<tr>
<td><strong>Drop-out</strong></td>
<td>Not reported</td>
</tr>
<tr>
<td><strong>Adherence</strong></td>
<td>Not reported</td>
</tr>
<tr>
<td><strong>Results</strong></td>
<td>Overall no differences between groups in symptoms, urodynamic data or SF-36 QoL, side effects</td>
</tr>
</tbody>
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<table>
<thead>
<tr>
<th>Author</th>
<th>Walsh et al., 2001b</th>
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<tbody>
<tr>
<td><strong>Design</strong></td>
<td>2-arm RCT</td>
</tr>
<tr>
<td></td>
<td>ES</td>
</tr>
<tr>
<td></td>
<td>Sham ES</td>
</tr>
<tr>
<td><strong>Sample size and age</strong></td>
<td>146 patients: 111 women, 35 men with urgency incontinence; mean (range) age 47 (17–79)</td>
</tr>
<tr>
<td>(years)</td>
<td>Clinical assessment: history and examination, uroanalysis, pelvic ultrasonography, cystourethroscopy, urodynamics</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>ES n = 74: DV/ DH/ SU 28/18/28; sham ES n = 72: 27/17/28</td>
</tr>
<tr>
<td><strong>Training protocol</strong></td>
<td>Both groups: transcutaneous neurostimulator, bilateral S3 dermatomes; ES antidromic S3 neurostimulation, 10 Hz, 200 μs, continuous mode, I max</td>
</tr>
<tr>
<td></td>
<td>Sham ES: no current</td>
</tr>
<tr>
<td><strong>Drop-out</strong></td>
<td>None</td>
</tr>
<tr>
<td><strong>Adherence</strong></td>
<td>Not applicable</td>
</tr>
<tr>
<td><strong>Results</strong></td>
<td>ES: pre/post stimulation significantly &gt; mean volumes bladder capacity at first desire to void (+57.3), strong desire to void (+68.4), urge (+55.2) and max capacity (+59.5) (p=0.0002); sham ES no changes</td>
</tr>
</tbody>
</table>

*(Continued)*
Table 7.12  Randomized controlled trials on electrical stimulation to treat OAB and/or urgency urinary incontinence symptoms—cont’d

<table>
<thead>
<tr>
<th>Author</th>
<th>Wang et al., 2004</th>
</tr>
</thead>
<tbody>
<tr>
<td>Design</td>
<td>3-arm RCT</td>
</tr>
<tr>
<td></td>
<td>PFMT</td>
</tr>
<tr>
<td></td>
<td>PFMT + BF</td>
</tr>
<tr>
<td></td>
<td>ES</td>
</tr>
<tr>
<td>Sample size and age (years)</td>
<td>120 women; mean age 52.7 (SD 13.7)</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Symptoms of OAB &gt; 6 mth, frequency ≥ 8 times/day, urge incontinence ≥ once/day</td>
</tr>
<tr>
<td>Training protocol</td>
<td>12 weeks</td>
</tr>
<tr>
<td></td>
<td>Home exercise based on individual PFM strength 3 times/day</td>
</tr>
<tr>
<td></td>
<td>Same home training in addition clinic BF twice a week</td>
</tr>
<tr>
<td>Drop-out</td>
<td>17/120 (14%)</td>
</tr>
<tr>
<td>Adherence</td>
<td>PFMT: 83%</td>
</tr>
<tr>
<td></td>
<td>PFMT + BF: 75%</td>
</tr>
<tr>
<td></td>
<td>ES: 79%</td>
</tr>
<tr>
<td>Results</td>
<td>Home exercise: PFMT 14.5 days; PFMT + BF 8.5 days</td>
</tr>
<tr>
<td></td>
<td>Urge incontinence resolved 30%; modified 6%; unchanged 64%</td>
</tr>
<tr>
<td></td>
<td>PFMT + BF: resolved 38%; modified 12%; unchanged 50%</td>
</tr>
<tr>
<td></td>
<td>ES: resolved 40%; modified 11.5%; unchanged 48.5%</td>
</tr>
<tr>
<td></td>
<td>Improvement/cured: PFMT 38%; PFMT + BF 50%; ES 51.5%</td>
</tr>
<tr>
<td></td>
<td>PFM strength: no significant differences between exercise groups, but between both exercise groups and ES in favour of exercise groups. No change in urodynamic parameters. Significant change in several QoL measures for different groups. Between ES and PFMT + BF no significant differences in improvement/reduction rate; between ES and PFMT yes</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Author</th>
<th>Yamanishi, Yasuda et al., 2000a</th>
</tr>
</thead>
<tbody>
<tr>
<td>Design</td>
<td>2-arm RCT</td>
</tr>
<tr>
<td></td>
<td>ES</td>
</tr>
<tr>
<td></td>
<td>Sham ES</td>
</tr>
<tr>
<td>Sample size and age (years)</td>
<td>68 patients; 39 women, 29 men; mean (SD) age 70 (11.2)</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Uroanalysis, urine cytologic examination, clinical assessment, neurologic, anatomical, urodynamics (cystometrogram, cystometry)</td>
</tr>
<tr>
<td>Training protocol</td>
<td>ES: alternating 10 Hz pulses, 1 ms pulse duration, I max tolerable, in women vaginal plug, 15 min 2x/day for 4 weeks</td>
</tr>
<tr>
<td>Drop-out</td>
<td>12%</td>
</tr>
<tr>
<td>Adherence</td>
<td>Not reported</td>
</tr>
<tr>
<td>Results</td>
<td>N IEF significance &lt; in ES, not in sham ES, significant intergroup difference in favour of ES, favouring ES significant intergroup change in nocturia (p = 0.03), same for QoL (p = 0.045), significantly &gt; maximum cystometric capacity and first desire to void in ES vs sham ES; trend favouring ES daily frequency of pad changes (p = 0.06)</td>
</tr>
<tr>
<td></td>
<td>Subgroup analysis of self report of cure/improvement according to sex: in women significant difference in favour of ES (p = 0.0091) in no. of cured/improved</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Author</th>
<th>Yamanishi, Sakakibara et al., 2000b</th>
</tr>
</thead>
<tbody>
<tr>
<td>Design</td>
<td>2-arm RCT</td>
</tr>
<tr>
<td></td>
<td>ES</td>
</tr>
<tr>
<td>Sample size and age (years)</td>
<td>32 patients: 17 women, 15 men; mean (SD) age 62.3 (16.6)</td>
</tr>
<tr>
<td></td>
<td>Magnetic stimulation (MS)</td>
</tr>
</tbody>
</table>
Quality of the intervention: dose–response issues

Some ES protocols were poorly reported, lacking detail of stimulation parameters, devices and methods of delivery. However, on the basis of the details that have been reported it appeared that there was considerable variation in ES protocols. Also the ES dosage (type of current, frequency, duration and intensity) varies significantly between studies (Hay-Smith et al., 2001; Moore et al., 2013). Looking into the studies on OAB patients included in the Cochrane systematic review reveals that length of the intervention varies between 4 months of daily stimulation (Smith, 1996) and a single episode of stimulation (Bower et al., 1998), intensity varies between 5 mA and maximal tolerable intensity, and length of each session between 20 minutes and several hours. Frequency of stimulation is once or twice every day in all RCTs except in studies by Smith (1996), Franzen et al. (2010) and Ozdedeli et al. (2010) (Moore et al., 2013).

Despite the many clinical series that have been reported, the common issues of patient selection, dose–response issues and electrical parameters still remain unsolved.

Patient selection criteria should most likely have to include neurophysiological sacral arc testing and assessment of detrusor muscle status, because some forms of muscle dysfunction respond less to neural inhibitory effects (Brubaker, 2000). Also, there is still no consensus how much stimulation is required for an optimal effect (Moore et al., 2013). Currently, most RCTs stimulate patients to use such an intensity of current that a maximally tolerable motor response of the pelvic floor is achieved (Moore et al., 2013). But it is still unknown whether or not a contraction of the pelvic floor is really necessary to achieve detrusor inhibition or whether just excitation of the pudendal afferents is sufficiently effective for this kind of inhibition.

Electrical parameters

Current

Although it appeared that all the ES trials reviewed here used alternating current, only six trials specifically stated this to be: biphasic (Berghmans et al., 2002; Ozdedeli et al., 2010); bipolar (Brubaker et al., 1997); biphasic pulsed current (Smith, 1996; Wang et al., 2004; Arruda et al., 2008).

Pulse shape

Five trials and the trials of Yamanishi and co-workers (Yamanishi et al., 2000a; Yamanishi et al., 2000b) were the only ones to detail the pulse shape: rectangular (Berghmans et al., 2002; Ozdedeli et al., 2010); square (Brubaker et al., 1997; Yamanishi et al., 2000a; Yamanishi et al., 2000b); symmetric (Wang et al., 2004); asymmetric (Smith, 1996); balanced with 2-second ramp up and 1-second ramp down.

Frequency

Twelve trials gave details of the frequencies used and these ranged from 5 Hz (Ozdedeli et al., 2010), 5–10 Hz (Franzen et al., 2010), 10 Hz (Bower et al., 1998; Yamanishi et al., 2000a; Yamanishi et al., 2000b; Walsh et al., 2001; Wang et al., 2004; Arruda et al., 2008) to 20 Hz (Brubaker et al., 1997; Soomro et al., 2001), a combination of 12.5 and 50 Hz (Smith, 1996), 150 Hz (Bower et al., 1998), and a random frequency of 4–10 Hz (Berghmans et al., 2002).

Pulse duration

Pulse durations were also reported in 11 trials, and these were 0.1 ms (Brubaker et al., 1997; Ozdedeli et al., 2010), 0.2 ms (Bower et al., 1998; Soomro et al., 2001; Walsh et al., 2001; Berghmans et al., 2002), 0.3 ms (Smith, 1996), 0.4 ms (Wang et al., 2004), and 1 ms (Yamanishi et al., 2000a; Yamanishi et al., 2000b; Arruda et al., 2008).

Table 7.12 Randomized controlled trials on electrical stimulation to treat OAB and/or urgency urinary incontinence symptoms—cont’d

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Training protocol</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urodynamics (cystometrogram, cystometry)</td>
<td>ES: home- and clinic-bound device; alternating 10 Hz pulses, 1 ms pulse duration, 1 max tolerable, in women vaginal plug, 15 min 2x/day for 4 weeks</td>
<td>No significant intergroup differences between groups for max cystometric capacity and bladder capacity at first desire to void; OAB cured in 3/15 (20%) in MS, 0/17 in ES; &gt;50 ml increase max cystometric capacity in 13/15 pts in MS, 6/17 in ES; no adverse events in either group</td>
</tr>
</tbody>
</table>

CG, control group; IEF, incontinence episodes frequency; LPP, leak point pressure; LUTE, lower urinary tract exercises; N, number; NS, not statistically significant; SG, stimulation group; UPP, urethral pressure profile. For other abbreviations, see text.

*Partly included in results for subgroup analysis according to gender.

*Not included in analysis of results because of inclusion of both women and men.
Duty circle
Two trials used a duty cycle ratio of 1:2 (Smith, 1996; Brubaker et al., 1997), in one trial this was 2:1 (Wang et al., 2004).

Intensity of stimulation
Intensity of stimulation progressed from 5 to 25 mA in the trial by Smith (1996). Ten trials used the maximum tolerable intensity (Brubaker et al., 1997; Bower et al., 1998; Yamanishi et al., 2000a; Yamanishi et al., 2000b; Walsh et al., 2001; Berghmans et al., 2002; Wang et al., 2004; Arruda et al., 2008; Ozdedeli et al., 2010; Franzen et al., 2010). In the trial of Soomro et al. (2001) patients were asked to control the amplitude of intensity to produce a tickling sensation.

Mode of delivery of current
Current was most commonly delivered by a vaginal electrode (Smith, 1996; Brubaker et al., 1997; Yamanishi et al., 2000a; Yamanishi et al., 2000b; Berghmans et al., 2002; Wang et al., 2004; Arruda et al., 2008; Ozdedeli et al., 2010; Franzen et al., 2010) and over S3 sacral dermatomes (Walsh et al., 2001), although one trial used external surface stimulation.

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### Table 7.13 PEDro quality score of RCTs in systematic review of electrical stimulation to treat OAB and/or urgency urinary incontinence symptoms

<table>
<thead>
<tr>
<th>Study</th>
<th>E</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>Total score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arruda et al., 2008</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>?</td>
<td>+</td>
<td>−</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Berghmans et al., 2002</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>8</td>
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<tr>
<td>Bower et al., 1998</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>?</td>
<td>+</td>
<td>+</td>
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<td>+</td>
<td>8</td>
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<tr>
<td>Brubaker et al., 1997</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>+</td>
<td>−</td>
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<td>Franzen et al., 2010</td>
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<td>+</td>
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<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>8</td>
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<tr>
<td>Ozdedeli et al., 2010</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>+</td>
<td>−</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>7</td>
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<tr>
<td>Smith, 1996</td>
<td>+</td>
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<td>−</td>
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<td>+</td>
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<td>+</td>
<td>+</td>
<td>4</td>
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<tr>
<td>Soomro et al., 2001***</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
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<td>Walsh et al., 2001***</td>
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<td>7</td>
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<tr>
<td>Wang et al., 2004</td>
<td>?</td>
<td>+</td>
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<td>−</td>
<td>−</td>
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<td>+</td>
<td>−</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>5</td>
</tr>
<tr>
<td>Yamanishi et al., 2000a**</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<tr>
<td>Yamanishi et al., 2000b***</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>?</td>
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<td>−</td>
<td>−</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>6</td>
</tr>
</tbody>
</table>

+, criterion is clearly satisfied; −, criterion is not satisfied; ?, not clear if the criterion was satisfied. Total score is determined by counting the number of criteria that are satisfied, except that ‘eligibility criteria specified’ score is not used to generate the total score. Total scores are out of 10.
Length and number of treatments

The length and number of treatments was also highly variable. The longest treatment period was 4 months of daily stimulation (Smith, 1996). Medium-length treatment periods were based on twice-daily stimulation for 4 (Yamanishi et al., 2000a), 8 (Brubaker et al., 1997), 9 (Berghmans et al., 2002), or 12 weeks (Wang et al., 2004; Arruda et al., 2008). In the crossover trial of Soomro et al. (2001), after randomization patients received 6 weeks of electrical stimulation for 6 hours daily or oxybutynin. After a washout period of 2 weeks they started in the second arm of treatment for another 6 weeks. The shortest treatment period consisted of a single episode of stimulation after the voiding phase of cystometry before filling was repeated (Bower et al., 1998).

Is ES better than no treatment, control or placebo treatment?

In a four-arm RCT in 83 women with detrusor overactivity, Berghmans et al. (2002) investigated the effect of no treatment, ES alone, a combination of PFMT and bladder training alone (which in this study was defined as lower urinary tract exercises), and ES in combination with lower urinary tract exercises. An important fact in this study was that women in the ES group received not only weekly clinic-based ES, but also a twice-daily ES programme with a home device, that also measured patient’s compliance of use of ES. The main outcome measures were change in the Detrusor Overactivity Index (DAI) (Berghmans et al., 2002), the Incontinence Impact Questionnaire (Berghmans et al., 2001) and the adapted Dutch Incontinence Quality of Life questionnaire (DI-QOL). The no treatment group showed no significant change at all pre to post treatment. In comparison with no treatment, there was a significant improvement in the ES alone group for the DAI (Berghmans et al., 2002). The ES alone group turned out to have statistically significant lower self-professed impact of incontinence on daily life activities (Berghmans et al., 2001). Using the DI-QOL ES alone improved self-professed incontinence control in daily life activities.

Yamanishi et al. (2000b) investigated maximum intensity stimulation delivered daily for 4 weeks in 29 men and 39 women with detrusor overactivity. There was significantly more improvement in a number of outcomes in the ES group compared with the placebo ES group post treatment (i.e. nocturia, number of leakage episodes, number of pad changes, quality of life score (using a questionnaire chart recording ‘0 = delighted’, ‘1 = mostly satisfied’, ‘2 = dissatisfied’ and ‘3 = mostly dissatisfied or unhappy’), urodynamic evidence of improvement in detrusor overactivity, self-report of cure or improvement). For a single outcome, self-report of cure/improvement, subgroup analysis on the basis of sex was reported. Women in the active ES group were much more likely to report cure/improvement than women in the placebo ES group.

Bower et al. (1998) used a single stimulation episode given after the voiding phase of cystometry and before bladder filling was repeated. The results were reported separately for women with detrusor overactivity and those with urgency. For women with detrusor overactivity both stimulation groups (10 Hz sacral electrodes, and 150 Hz symphysis pubis electrodes) showed significant improvements in urodynamic measures when compared with the placebo stimulation group (i.e. reduction in maximum detrusor pressure, increase in first desire to void, proportion of women with a stable bladder). However, there were no significant differences between stimulation and placebo groups for change in maximum cystometric capacity or detrusor pressure at first desire to void. Fewer measures were reported for women with urgency. The only significant findings were a significant increase in first desire to void in the 150 Hz group, and a significant increase in the maximum cystometric capacity in the placebo ES group.

One further trial (Brubaker et al., 1997) that compared ES with placebo ES in a group of women with urodynamic stress incontinence, detrusor overactivity or both, conducted a subgroup analysis on the basis of diagnosis and found that women with pre-treatment detrusor overactivity who received active stimulation were significantly less likely to have urodynamic evidence of detrusor overactivity post treatment.

Due to availability of only a single study in women comparing ES with no treatment and the variation in stimulation protocols comparing electrical stimulation with placebo stimulation, it is difficult to interpret the findings of trials. However, for women with detrusor overactivity there is an absolute trend in favour of active stimulation over no treatment or placebo stimulation.

Is ES better than any other single treatment?

In a three-arm RCT in 103 women with OAB Wang et al. (2004) did compare the effects of ES with PFMT and with biofeedback-assisted PFMT (BAPFMT). Assessment was performed pre and post treatment using the King’s Health Questionnaire for subjective cure/improvement, and urinary symptoms like urgency, diurnal frequency, urgency incontinence, dysuria and nocturia for more objective outcomes. As secondary outcomes, PFM strengthening and urodynamic data were used. More study details can be found in Table 7.10.
Wang et al. (2004) did not find any statistically significant difference between the groups for self-reported cure or cure/improvement. PFMT women had statistically significantly fewer leakage episodes per day. Although there were no statistically significant differences in the general health perception, incontinence impact, role limitation, physical limitation, social limitation, sleep/energy and personal relationships, domains of the quality of life measure (King’s Health Questionnaire), the ES group had statistically significantly better scores post treatment for emotions and severity measures, compared to the exercise regimens and in total score compared to PFMT only. Some women using ES reported discomfort during treatment.

The trial of Smith (1996) compared electrical stimulation and medication (propantheline bromide) in women with detrusor overactivity with or without urodynamic stress incontinence. He did not find any statistically significant differences in outcome (self-reported improvement and urodynamic parameters) between the two groups.

Arruda et al. (2008) compared in a three-arm study intermittent electrical stimulation with medication (oxybutynin immediate release 5 mg) in women with DO and MUI (UUI dominant on urodynamics) and with pelvic floor muscle training. No significant differences between groups were found in outcome of effects (based on self-reported cure/improvement and urodynamics, equally effective directly after treatment and after 12 months follow-up). No side-effects were reported for electrical stimulation, only for oxybutynin.

Franzen et al. (2010) showed no significant differences comparing electrical stimulation with tolterodine SR 4 mg orally once per day for 6 months in women with (predominant) urge and UUI symptoms. Both therapies were effective without any statistical difference in cured or improved patients between groups.

In a trial comparing ES with trospium hydrochloride 45 mg daily in women with (predominant) UUI no statistically significant differences were found between groups using both objective (urodynamic parameters, voiding diary) and subjective (Vas urgency severity, IIQ-7, treatment satisfaction) outcome measures. Side-effects were statistically significantly higher in the medication group.

With only a few single trials comparing electrical stimulation with PFMT, BAPFMT, or medication there is insufficient evidence to determine if electrical stimulation is better than PFMT, BAPFMT, propantheline bromide, anticholinergic or antimuscarinic therapy in women with detrusor overactivity. From the studies comparing ES with medication, it seems that ES is equally as effective as medication. However, one has to be very cautious about such statements because different kinds of ES (protocols) were compared to different kinds of medication.

In summary, because of sparse availability of trials, there is insufficient evidence to determine if electrical stimulation is better than PFMT, BAPFMT or medication for women with detrusor overactivity.

Is (additional) ES better than other (additional) treatments?

In this section no studies were found, so no conclusion can be drawn as to whether or not there is any benefit of adding ES to another treatment modality in women with OAB.

CONCLUSION

- ES protocols and designs in studies for women with OAB and/or UUI symptoms are largely inconsistent. One reason for this is insufficient understanding of the physiological rationale of the working mechanism and basic principles of electrical stimulation used in clinical practice to treat these women.
- There is some evidence to judge that an intensive programme of clinic-based and home electrical stimulation is better than no or placebo treatment for women with OAB and/or UUI symptoms. Unfortunately, some of the relevant studies in this area included both women and men, making interpretation of results in women only very difficult.
- There is insufficient evidence to determine whether electrical stimulation is better than PFMT, BAPFMT or medication in women with OAB and/or UUI symptoms.
- At present there are no studies that have investigated extra benefit of adding ES to other treatment (modalities).
- There is need for more basic research to find out the working mechanism of ES in women with OAB and/or UUI symptoms, and to determine the best ES protocol(s) for this kind of patient.

CLINICAL RECOMMENDATIONS

- If available, ES should be applied both in clinical practice and at the patient’s home – maybe as the treatment of first choice in this diagnostic group. So far, it is impossible to recommend the most optimal ES regimen and protocol. But if ES is applied, do use an intensive (parameters, number of sessions, duration of therapy) ES regimen with both clinic-based and home devices. A protocol that has proven to be effective (Fall and Madersbacher, 1994; Berghmans et al., 2002) consisted of the following parameters:
  - stochastic frequency: 4–10 Hz; freq. mod. 0.1 s
  - intensity: I max
  - pulse duration: 200–500 μs
  - biphasic, duty circle 13 s 5/8
  - shape of current: rectangular
REFERENCES


Evidence-Based Physical Therapy for the Pelvic Floor

7.3 Urinary incontinence related to the peripartum period

Siv Mørkved, Kari Bø

INTRODUCTION

Current exercise guidelines recommend all pregnant women to be physically active on preferably all weekdays throughout pregnancy and to conduct both cardiovascular and strength training exercise (Artal and O'Toole, 2003; Wolfe and Davies, 2003; RCOG, 2006). The prescription for exercise is more detailed for the cardiovascular component of training than the strength training component. This may, to some extent, be explained by the fact that there are fewer published clinical trials on strength training programmes for pregnancy and birth outcomes than endurance training (Kramer, 2005; Melzer et al., 2010).
Pregnancy and childbirth are known risk factors for weakening and causing injury to the perineum and pelvic floor. Stretch and rupture of peripheral nerves, connective tissue and muscles may cause urinary and faecal incontinence, pelvic organ prolapse, sensory and emptying abnormalities of the lower urinary tract, defecation dysfunction, sexual dysfunction and chronic pain syndromes (Bump and Norton, 1998). About 50% of women lose some of the supporting function of the pelvic floor due to childbirth (Swift, 2000), and recent research using ultrasound and MRI report prevalence of major injuries to the pelvic floor muscles of 20–26% following vaginal delivery (DeLancey et al., 2003, 2008; Dietz and Lanzarone, 2005). Hence, vaginal childbirth can be considered equivalent to a major sports injury, but has not been given the same attention concerning prevention or treatment.

Urinary incontinence is the most prevalent symptom of pelvic floor dysfunction; prevalence rates varying between 32 and 64% (Milsom et al., 2009). Stress urinary incontinence is defined as ‘complaint of involuntary loss of urine during effort or physical exertion (e.g. sporting activities), or on sneezing and coughing’ (Haylen et al., 2010) and is the most common form of urinary incontinence (UI) in all age groups. Prevalence rates between 4.5% (swimming) and 80% (trampoline jumping) have been found in young elite athletes (Bo, 2004). In the general female population urinary incontinence causes withdrawal from exercise and fitness activities and is a barrier to regular participation in physical activities (Bo, 2004). Surprisingly, strength training of the pelvic floor muscles is not mentioned at all in the Guidelines of the American College of Obstetricians and Gynecologists (Artal and O’Toole, 2003) and only briefly mentioned in the British and Canadian guidelines. Furthermore, there are no or few references to evidence from clinical controlled trials in the existing guidelines (Wolfe and Davies, 2003; RCOG, 2006).

Two important questions are: (1) whether UI and other pelvic floor disorders can be prevented by training the pelvic floor muscles before problems arise (primary prevention), or (2) whether women at risk at an early stage can be identified with a view to secondary prevention using pelvic floor muscle training (PFMT). Reviews on PFMT in prevention of UI report inconsistent results and there seems to be some doubt about the effect (Brostrom and Lose, 2008; Hay-Smith et al., 2008). This may be due to use of different inclusion criteria of studies and different criteria to classify studies as either prevention or treatment interventions. Some authors do not separate between antenatal or postpartum interventions (Brostrom and Lose, 2008) and there seems to be little attention towards dose–response issues in the training protocols. The aims of the present systematic review were to answer the following questions:

1. Is there evidence that pregnant women should be advised to do PFMT to prevent or treat UI?
2. Is there evidence that postpartum women should be advised to do PFMT to prevent or treat UI?
3. What is the most optimal training dosage for effective antenatal and postpartum PFMT in prevention and treatment of UI?
4. What is the long-term effect of PFMT during pregnancy and after childbirth?

**RESEARCH METHODS**

PubMed (search date June 12, 2012), the Cochrane Central Register of Controlled Trials (CENTRAL in the Cochrane Library, Wiley, Issue 6 of 12, June 2012), Embase (through OvidSP, 1980 to 2012 week 24) and Physiotherapy Evidence Database (PEDro 12 June 2012) were searched to identify studies. Keywords used in different combinations in the search were: pregnancy, pelvic floor muscle, exercise, training, incontinence, after delivery, postpartum, childbirth, effect, prevention. Inclusion criteria were quasi-experimental and randomized controlled trials written in English or Scandinavian languages. Both meeting abstracts and full publications were included. In addition to database searches, reference lists of selected papers were searched and manual search was undertaken of meeting abstract books published by the World Confederation of Physical Therapy (1993–2011), International Continence Society and International Urogynecology Association (1990–2011).

Scoring of methodological quality was done according to the PEDro rating scale giving one point for each of the following factors for internal validity: random allocation, concealed allocation, baseline comparability, blinded assessment, blinded subjects, blinded therapists, adequate follow up (≥85%), intention-to-treat (ITT) analysis, between-group comparison, report of point estimates and variability (Maher et al., 2003). The two authors independently scored the studies. Any disagreement was resolved with consensus.

**RESULTS**

The database searches resulted in 117 references after deduplication. In addition to the studies included in the Cochrane systematic review (Hay-Smith et al., 2008), eight new RCTs (Dinc et al., 2009; Elliott et al., 2009; Mason et al., 2010; Bo and Haakstad, 2011; Dias et al., 2011; Ko et al., 2011; Kim et al., 2012; Stafne et al., 2012) and one quasi-experimental study (Sansawang and Serisathien, 2012) were found. Eight were short-term original studies and one (Elliott et al., 2009) was a 7-year follow-up study.

**PFM exercises during pregnancy to prevent UI, including both women with and without UI** (Table 7.14)

Ten RCTs (Sampselle et al., 1998; Hughes et al., 2001; Reilly et al., 2002; Mørkved et al., 2003; Mason et al., 2010;
<table>
<thead>
<tr>
<th>Author</th>
<th>Sampselle et al., 1998</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Table 7.14</strong></td>
<td><strong>Studies assessing the effect of pelvic floor muscle exercises during pregnancy to prevent urinary incontinence, including both women with and without urinary incontinence at inclusion</strong></td>
</tr>
<tr>
<td><strong>Author</strong></td>
<td>Hughes et al., 2001 (abstract)</td>
</tr>
<tr>
<td><strong>Author</strong></td>
<td>Reilly et al., 2002</td>
</tr>
<tr>
<td><strong>Design</strong></td>
<td>2 arm RCT</td>
</tr>
<tr>
<td><strong>Design</strong></td>
<td>2-arm RCT</td>
</tr>
<tr>
<td><strong>Design</strong></td>
<td>2 arm RCT</td>
</tr>
<tr>
<td><strong>Study population</strong></td>
<td>n = 72 primigravid women recruited at 20 weeks of pregnancy. Some women had existing UI. Groups comparable at baseline. Single centre, USA</td>
</tr>
<tr>
<td><strong>Study population</strong></td>
<td>n = 1169 pregnant nulliparous women recruited at 20 weeks of pregnancy. Some women had existing UI. Single centre, UK</td>
</tr>
<tr>
<td><strong>Study population</strong></td>
<td>n = 268 primigravid, continent women with increased bladder neck mobility recruited at 20 weeks of pregnancy. Single centre, UK</td>
</tr>
<tr>
<td><strong>Training protocol</strong></td>
<td>C: Routine care</td>
</tr>
<tr>
<td><strong>Training protocol</strong></td>
<td>I: Tailored PFMT programme beginning with muscle identification, progressing to strengthening. 30 contractions per day at max or near max intensity from 20 weeks of pregnancy. Correct VPFMC checked</td>
</tr>
<tr>
<td><strong>Drop-out/Adherence</strong></td>
<td>Losses to follow-up: 36</td>
</tr>
<tr>
<td><strong>Outcomes</strong></td>
<td>Change in mean UI symptom score: 35 wk pregnancy: C 0.20; I −0.02; p = 0.07</td>
</tr>
<tr>
<td><strong>Outcomes</strong></td>
<td>SUI Bristol Female Urinary Tract Symptoms Questionnaire: 36 wk pregnancy: C 66%; I 61%. OR (95% CI): 0.78 (0.59–1.04)</td>
</tr>
<tr>
<td><strong>Outcomes</strong></td>
<td>6 mth post partum: C 38%; I 36%. OR (95% CI): 0.90 (0.64–1.28)</td>
</tr>
<tr>
<td><strong>Adverse events not stated. Self-reported adherence. Partial ITT analysis</strong></td>
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<td><strong>Adverse events not stated</strong></td>
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<td><strong>Adverse events not stated</strong></td>
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</tr>
<tr>
<td><strong>ITT analysis: ~38% in the intervention group were doing PFMT twice or more per week</strong></td>
<td></td>
</tr>
</tbody>
</table>
### Table 7.14 Studies assessing the effect of pelvic floor muscle exercises during pregnancy to prevent urinary incontinence, including both women with and without urinary incontinence at inclusion—cont’d

<table>
<thead>
<tr>
<th>Outcomes</th>
<th>Self-reported UI at 3 mth post partum:</th>
<th>C 36/110 (32.7%); I 23/120 (19.2%); RR (95% CI) 0.59 (0.37–0.92); p = 0.023</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>QoL: Higher score in the exercise group (p = 0.004)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pad-test: NS</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Bladder neck mobility: NS difference</td>
<td></td>
</tr>
<tr>
<td></td>
<td>PFM strength: NS difference</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Self-reported UI at 8 yr follow-up:</td>
<td></td>
</tr>
<tr>
<td></td>
<td>C 38.8%; I 35.4%; p = 0.75</td>
<td></td>
</tr>
</tbody>
</table>

### Design

2-arm RCT

Control (C): (n = 153) customary information from general practitioner/midwife

Intervention (I): (n = 148) 12 weeks of intensive PFMT

Control (C): (n = 94)

Intervention (I): (n = 94)

### Study population

n = 301 primigravid women recruited at 20 weeks of pregnancy. Some women had existing UI

Three outpatient physical clinics in Norway

188/301 (62%) returned the questionnaire

### Training protocol

C: Customary information from general practitioner/midwife. Not discouraged from PFMT. Correct PFM contraction checked at enrolment

I: 12 weeks of intensive PFMT (in a group) led by PT, with additional home exercises 10 max contractions (each held for 6 s) and to the last 4 were 3–4 fast contractions added, repeated twice daily, between 20 and 36 weeks of pregnancy. Correct VPFMC checked at enrolment

Control group received information about the results of the trial and the training programme, about 1 year after delivery

### Drop-out/Adherence

Losses to follow-up 12/301 (5 I and 7 C)

Adherence to PFMT: 81% adherence to PFMT in I group. Adverse events not stated. ITT analysis: 45% adherence to PFMT in both groups

### Outcomes

Self-reported UI at 36 weeks of pregnancy:

C 74/153 (48%); I 48/148 (32%); RR (95% CI) 0.67 (0.50–0.89); p = 0.007

UI at 3 mth post partum:

C 49/153 (32%); I 29/148 (19.6%); RR (95% CI) 0.61 (0.40–0.90); p = 0.018

PFM strength: significant difference in favour of the intervention group

UI at 6 years follow up:

C 17%; I 23%; p = 0.276

### Author

Mørkved et al., 2003

Mørkved et al., 2007

(abstract): 6-year follow-up

### Design

2 arm RCT

Control (C): (n = 34 after drop outs). No PFMT

Intervention (I): (n = 38 after drop-outs) PFMT

### Study population

75 pregnant nulliparous continent women recruited at 20 weeks of pregnancy.

Single setting, Mexico

### Training protocol

C: Requested not to perform PFMT during pregnancy or post partum

I: Individual PFMT with PT, 10 VPFMC each held for 8 s each followed by 3 fast 1-s contractions; 6 s rest. Clinic appointments weekly for 8 weeks, then weekly phone calls up to 20 weeks. Biofeedback and training diary. Correct VPFMC checked

### Drop-out/Adherence

Losses to follow up 3/75 (4%)

Adherence to PFMT: 84% attended 7 or 8 physical therapy appointments. ITT analyses

(Continued)
Table 7.14  Studies assessing the effect of pelvic floor muscle exercises during pregnancy to prevent urinary incontinence, including both women with and without urinary incontinence at inclusion—cont’d

| Outcomes | UI:
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>28 wk pregnancy: C 17%; I 0%; p&lt;0.05</td>
<td></td>
</tr>
<tr>
<td>35 wk pregnancy: C 47%; I 0%; p&lt;0.05</td>
<td></td>
</tr>
<tr>
<td>6 wk post partum: C 47%; I 15%; p&lt;0.05</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Author</th>
<th>Mason et al., 2010</th>
</tr>
</thead>
<tbody>
<tr>
<td>Design</td>
<td>2 arm RCT</td>
</tr>
<tr>
<td>Study population</td>
<td>n=311 nulliparous pregnant women with no symptoms of SUI at 11–14 weeks of pregnancy. Two hospitals in England</td>
</tr>
<tr>
<td>Training protocol</td>
<td>C: No treatment</td>
</tr>
<tr>
<td>I: 45 min physical therapy class once per month for 4 months. Additional home exercises 8–12 max contractions (each held for 6 s) and to the last 4 were added 3–4 fast contractions, repeated twice daily, between 20 and 36 weeks of pregnancy. Correct VPFMCP checked at enrolment in most women</td>
<td></td>
</tr>
<tr>
<td>Drop-out/Adherence</td>
<td>Losses to follow up: 8%</td>
</tr>
<tr>
<td>Some significant differences between responders and non-responders. 90 women (31.4%) completed all sets of questionnaires. 91/141 (49.1%) in the intervention group attended a PFMT class</td>
<td></td>
</tr>
<tr>
<td>Outcomes</td>
<td>Significantly more PFMT in the intervention group compared to the control group</td>
</tr>
<tr>
<td>Self-reported UI:</td>
<td></td>
</tr>
<tr>
<td>36 wk pregnancy: C 51/96 (53%); I 24/60 (40%); OR (95%CI) 1.7 (0.884–3.269); p=0.138</td>
<td></td>
</tr>
<tr>
<td>3 mth post partum: C 33/80 (41.3%); I 23/68 (33.8%); OR (95%CI) 1.374 (0.702–2.688); p=0.397</td>
<td></td>
</tr>
<tr>
<td>Difference in symptoms and episodes of UI between groups NS</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Author</th>
<th>Bø and Haakstad, 2011</th>
</tr>
</thead>
<tbody>
<tr>
<td>Design</td>
<td>2-arm RCT</td>
</tr>
<tr>
<td>Study population</td>
<td>n=105 nulliparous women recruited within 24 weeks of pregnancy. Some women had existing UI. Single centre, Norway</td>
</tr>
<tr>
<td>Training protocol</td>
<td>C: Usual care</td>
</tr>
<tr>
<td>I: 12–16 weeks of aerobic exercise classes twice per week during pregnancy, including intensive PFMT (in a group) led by aerobic instructor. Additional home exercises 10 max contractions (each held for 6 s) and to the last 4 were added 3–4 fast contractions x 3, per day. Correct VPFMCP was not checked at enrolment</td>
<td></td>
</tr>
<tr>
<td>Drop-out/Adherence</td>
<td>Losses to follow up: 21/105 (10 I and 11 C)</td>
</tr>
<tr>
<td>Adherence to training sessions: 40%. Adverse events not stated. Not ITT analysis</td>
<td></td>
</tr>
<tr>
<td>Outcomes</td>
<td>Self-reported UI:</td>
</tr>
<tr>
<td>36–38 wk pregnancy: C 7/53; I 9/52</td>
<td></td>
</tr>
<tr>
<td>3 months post partum: C 6/53; I 5/52; NS</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Author</th>
<th>Dias et al., 2011</th>
</tr>
</thead>
<tbody>
<tr>
<td>Design</td>
<td>3-arm RCT</td>
</tr>
<tr>
<td>Study population</td>
<td>n=87 primigravidae; women recruited at 18 weeks of pregnancy. Some women had existing UI. Single centre, Brazil</td>
</tr>
<tr>
<td>Training protocol</td>
<td>C: no exercising; S: exercising under supervision of a PT monthly + daily home exercises</td>
</tr>
<tr>
<td>O: unsupervised daily home exercises</td>
<td></td>
</tr>
<tr>
<td>Drop-out/Adherence</td>
<td>Losses to follow up: Unspecified</td>
</tr>
<tr>
<td>Outcomes</td>
<td>Self-reported UI at 38 weeks of pregnancy: C 96%; S 6.9%; O 6.9%</td>
</tr>
</tbody>
</table>
Female pelvic floor dysfunctions and evidence-based physical therapy

Chapter

Table 7.14  Studies assessing the effect of pelvic floor muscle exercises during pregnancy to prevent urinary incontinence, including both women with and without urinary incontinence at inclusion—cont’d

<table>
<thead>
<tr>
<th>Author</th>
<th>Ko et al., 2011</th>
</tr>
</thead>
<tbody>
<tr>
<td>Design</td>
<td>2-arm RCT</td>
</tr>
<tr>
<td>Control (C): (n = 150) routine care</td>
<td>Intervention (I): (n = 150) 20 weeks of intensive PFMT</td>
</tr>
<tr>
<td>Study population</td>
<td>n = 300 nulliparous women recruited at 16–24 weeks of pregnancy. Some women had existing UI. Single centre, Taiwan</td>
</tr>
<tr>
<td>Training protocol</td>
<td>C: Routine antenatal care. I: Individual PFMT with PT once per week between 20 and 36 weeks of pregnancy, with additional home exercises 3 sets of 8 contractions (each held for 6 s) repeated twice daily. Instructed to contract the PFM when coughing or sneezing</td>
</tr>
<tr>
<td>Drop-out/Adherence</td>
<td>Losses to follow up: 0%</td>
</tr>
<tr>
<td>Adherence PFMT:</td>
<td>−87% practised PFMT at least 75% of the time. Adverse events not stated</td>
</tr>
<tr>
<td>Outcomes</td>
<td>ITT analysis</td>
</tr>
<tr>
<td>Self-reported UI:</td>
<td>36 wk pregnancy: C 76/150 (51%); I 52/150 (34%); p = &lt;0.01</td>
</tr>
<tr>
<td>3 days post partum: C 62/150 (41%); I 46/150 (30%); p = 0.06</td>
<td></td>
</tr>
<tr>
<td>6 wk post partum: C 53/150 (35%); I 38/150 (25%); p = 0.06</td>
<td></td>
</tr>
<tr>
<td>6 mth post partum: C 42/150 (27%); I 25/150 (16%); p = 0.04</td>
<td></td>
</tr>
<tr>
<td>Significant improvement in Intervention group in scores on the Incontinence Impact Questionnaire and Urogenital Distress Inventory, in late pregnancy and up to 6 mth post partum</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Author</th>
<th>Stafne et al., 2012</th>
</tr>
</thead>
<tbody>
<tr>
<td>Design</td>
<td>2-arm RCT</td>
</tr>
<tr>
<td>Control (C): (n = 426) customary information from general practitioner/midwife</td>
<td>Intervention (I): (n = 429): 12 weeks of intensive PFMT</td>
</tr>
<tr>
<td>Study population</td>
<td>n = 855 pregnant women recruited at 20 weeks of pregnancy. Some women had existing UI. Two hospitals in Norway</td>
</tr>
<tr>
<td>Training protocol</td>
<td>C: customary information from general practitioner/midwife and written information. Not discouraged from PFMT</td>
</tr>
<tr>
<td>I: 12 weeks of exercise class including led by PT, with additional home exercises 3 x 10 max contractions (each held for 6 s and to the last 4 were added 3–4 fast contractions) at least 3 times per week between 20 and 36 weeks of pregnancy. Correct VPFMC checked at enrolment</td>
<td></td>
</tr>
<tr>
<td>Drop-out/Adherence</td>
<td>Losses to follow-up: 93/855 (32 intervention and 61 controls)</td>
</tr>
<tr>
<td>Adherence to PFMT:</td>
<td>−67% adherence to PFMT in I group; −40% adherence to PFMT in C group. No adverse events. ITT analysis</td>
</tr>
<tr>
<td>Outcomes</td>
<td>Self-reported UI at 34–38 weeks pregnancy:</td>
</tr>
<tr>
<td>Any UI: C 192/365 (53%); I 166/397 (42%); p = 0.004</td>
<td></td>
</tr>
<tr>
<td>UI once per week or more: C 68/365 (19%); I 44/397 (11%); p = 0.004</td>
<td></td>
</tr>
</tbody>
</table>

ITT, intention-to-treat analysis; NS, not statistically significant; OR, odds ratio; RR, relative risk; SD, standard deviation; VPFMC, voluntary pelvic floor muscle contraction. For other abbreviations, see text.

Bø and Haakstad, 2011; Dias et al., 2011; Ko et al., 2011; Stafne et al., 2012; Gorbea Chavez et al., 2004) and two long-term follow-up studies (Mørkved et al., 2007; Agur et al., 2008) were identified. In all studies women were recruited at before 22 weeks of pregnancy. All the trials except the RCT by Stafne et al. (2012) included primigravid/nulliparous women. Three trials were primary prevention trials including only continent women (Reilly et al., 2002; Gorbea Chavez et al., 2004; Mason et al., 2010), one trial included only women at risk of developing UI (with increased bladder neck mobility) and no previous UI (Reilly et al., 2002). Seven studies included women who had not been selected on the basis of incontinence or risk factors (Sampselle et al., 1998; Hughes et al., 2001; Mørkved et al., 2003; Bø and Haakstad, 2011; Dias et al., 2011; Ko et al., 2011; Stafne et al., 2012). However, in two of these trials (Mørkved et al., 2003; Stafne et al., 2012) results from the subgroup of women who were continent at inclusion were reported (primary prevention). PEDro scores varied between 7 and 8 out of 10 in the trials published as articles (see Table 7.18 below). The abstracts were difficult to score due to limited information.
Training protocol

The exercise period started between 20 and 22 weeks of pregnancy in six studies (Sampselle et al., 1998; Stafne et al., 2012), between 11 and 14 weeks in one (Mason et al., 2010) and between 16 and 24 weeks in three trials (Bø and Haakstad, 2011; Dias et al., 2011; Ko et al., 2011). However, the length of the training period, the follow up by health professionals, the training intensity and frequency varied.

The training protocol in all the studies, except for one (Hughes et al., 2001), addressed both regular home training and follow-up (monthly and weekly) by a physical therapist (PT), few (up to 30 contractions per day) and strong (near maximal) contractions. Hughes et al. (2001) used a protocol consisting of only one individual session and one group session in addition to regular home training.

In all studies except for two (Gorbea Chavez et al., 2004; Dias et al., 2011), the control groups were not discouraged from doing PFMT on their own, but received standard care including advice about PFMT. In one trial (Mørkved et al., 2003) the control group was given the same individual instructions in correct PFM contraction (including vaginal palpation and feedback) as the training group. Adherence to the PFMT protocol was reported in most trials (Sampselle et al., 1998; Reilly et al., 2002; Mørkved et al., 2003; Gorbea Chavez et al., 2004; Mason et al., 2010; Bø and Haakstad, 2011; Ko et al., 2011; Stafne et al., 2012), however different classification systems of adherence were used. No specific questionnaires/instruments to report adherence were used. Some studies used exercise diaries (Reilly et al., 2002; Mørkved et al., 2003; Stafne et al., 2012).

Outcomes

Clinically relevant and statistically significant effects of the interventions were documented in seven trials (Sampselle et al., 1998; Reilly et al., 2002; Mørkved et al., 2003; Gorbea Chavez et al., 2004; Dias et al., 2011; Ko et al., 2011; Stafne et al., 2012), showing a significant reduction in symptoms, episodes of UI or a lower percentage of women with UI in late pregnancy or during the first 3 months after delivery. A specific preventive effect of PFMT was shown in the studies by Reilly et al. (2002), Gorbea Chavez et al. (2004) and in the subgroup of women with no previous UI at inclusion in the trials from Mørkved et al. (2003) and Stafne et al. (2012). No adverse effects of the interventions were reported. Sampselle et al. (1998) found that the short-term effect was not present at one-year follow-up. Eight years follow-up data from Reilly et al’s (2002) trial showed no significant difference in UI between the original intervention and control groups (Agur et al., 2008). Mørkved et al. (2007) reported that the percentage of continent women in the training group was similar at 3 months and 6 years follow-up, while the percentage of continent women in the control group had increased in the period, and the statistically significant difference between groups was no longer present.

**Pelvic floor muscle exercises during pregnancy to treat UI, including only women with UI (Table 7.15)**

Two RCTs (Woldringh et al., 2007; Dinc et al., 2009) and one quasi-experimental study were found (Sangsawang and Serisathien, 2012). Incontinent parous or nulliparous women were included. PEDro scores were 5 and 7 out of 10 (see Table 7.18 below).

Training protocol

The training protocols and follow-up varied. In the trial by Woldringh et al. (2007) the programme consisted of three individual sessions during pregnancy weeks 23–30 and one 6 weeks after delivery, while the control group received routine care including instruction on PFMT. The drop-out rate was about 50% and the adherence to regular PFMT among the women that stayed in the training group was 77%. Dinc et al. (2009) addressed both regular home training and follow-up between 20 and 36 weeks of pregnancy, and few (up to 30 contractions per day) and close to maximal contractions, while the study by Sangsawang and Serisathien (2012) used a 6-week training programme.

Outcomes

Woldringh et al. (2007) found no difference in UI between the intervention and control groups during pregnancy and at the follow-up at 6 and 12 months post partum. Conversely, Dinc et al. (2009) and Sangsawang and Serisathien (2012) demonstrated a significant difference in UI after the intervention period in favour of the training group, both in late pregnancy and 6–8 weeks post partum.

**Pelvic floor muscle exercises after delivery to prevent UI, including women with and without UI (Table 7.16)**

Five short-term studies were found (Sleep and Grant, 1987; Mørkved and Bø, 1997; Meyer et al., 2001; Chiarelli and Cockburn, 2002; Ewings et al., 2005), and in addition long-term results from two studies (Mørkved and Bø, 2000; Chiarelli et al., 2004) have been reported. Two of the short-term studies were RCTs (Sleep and Grant, 1987; Chiarelli and Cockburn, 2002), one a nested RCT (Ewings et al., 2005), one a quasi-randomized study (Meyer et al., 2001) and one a matched controlled study (Mørkved and Bø, 1997). PEDro scores varied between 4 and 8 out of 10 (see Table 7.16). The studies included both primi- and multiparous women. Chiarelli and Cockburn (2002)
### Table 7.15 Studies assessing the effect of pelvic floor muscle exercises during pregnancy to treat urinary incontinence, including only women with urinary incontinence at inclusion

<table>
<thead>
<tr>
<th>Author</th>
<th>Woldringh et al., 2007</th>
</tr>
</thead>
<tbody>
<tr>
<td>Design</td>
<td>2-arm RCT</td>
</tr>
<tr>
<td>Control (C): (n = 152) routine care</td>
<td></td>
</tr>
<tr>
<td>Intervention (I): (n = 112) 4 sessions of individual instruction in PFMT</td>
<td></td>
</tr>
<tr>
<td>Study population</td>
<td>n = 264 women with UI at 22 weeks of pregnancy. Multicentre, The Netherlands</td>
</tr>
<tr>
<td>Training protocol</td>
<td>C: routine care; nearly 2/3 received some instruction on PFMT</td>
</tr>
<tr>
<td></td>
<td>I: 3 sessions of individual therapy during week 23–30 of pregnancy and one 6 weeks after delivery, combined with written information</td>
</tr>
<tr>
<td>Losses to follow-up</td>
<td>% losses to follow up (C/I): 35 wk 17/14; 8 wk post partum 25/18; 6 mth post partum 30/29; 12 mth post partum: 42/35</td>
</tr>
<tr>
<td>Adherence</td>
<td>Adherence to PFMT: −54% in I group participated during the whole study period, and 77% of these women reported regular PFMT at 35 weeks of pregnancy; −50% in C group participated during the whole study period, and 40% of these women reported regular PFMT at 35 weeks of pregnancy. Adverse events not stated. ITT analysis</td>
</tr>
<tr>
<td>outcomes</td>
<td>Self-reported severity of any UI:</td>
</tr>
<tr>
<td></td>
<td>35 wk pregnancy: C 93%; I 88%; p = 0.33</td>
</tr>
<tr>
<td></td>
<td>8 wk post partum: C 68%; I 62%; p = 0.44</td>
</tr>
<tr>
<td></td>
<td>6 mth post partum: C 60%; I 56%; p = 0.63</td>
</tr>
<tr>
<td></td>
<td>12 mth post partum: C 63%; I 58%; p = 0.61</td>
</tr>
<tr>
<td></td>
<td>12 mth post partum: negative correlation between training intensity and severity of UI</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Author</th>
<th>Dinc et al., 2009</th>
</tr>
</thead>
<tbody>
<tr>
<td>Design</td>
<td>2-arm RCT</td>
</tr>
<tr>
<td>Control (C): (n = 46) PFMT</td>
<td></td>
</tr>
<tr>
<td>Intervention (I): (n = 46) PFMT</td>
<td></td>
</tr>
<tr>
<td>Study population</td>
<td>n = 92 pregnant women recruited at 20–34 weeks of pregnancy. All women had existing UI. Primi- and multiparous. Single centre, Turkey</td>
</tr>
<tr>
<td>Training protocol</td>
<td>C: No treatment</td>
</tr>
<tr>
<td></td>
<td>I: 3–16 weeks of intensive PFMT, with thorough instruction and additional home exercises between 20 and 36 weeks of pregnancy. 3 sets of 10–15 contractions 2–3 times per day. Both fast and slow (3–10 s) contractions</td>
</tr>
<tr>
<td>Losses to follow-up</td>
<td>Losses to follow-up: 24/92 (6 in both groups) after first evaluation; second, 12 lost to follow-up (5 I and 7 C)</td>
</tr>
<tr>
<td>Adherence</td>
<td>Adherence to PFMT: Not reported. Not ITT analysis</td>
</tr>
<tr>
<td>outcomes</td>
<td>Self-reported UI: at 36–38 wk pregnancy: C 25/35 (71.4%); I 16/37 (43.2%)</td>
</tr>
<tr>
<td></td>
<td>at 6–8 wk post partum: C 13/33 (38.4%); I 6/35 (17.1%)</td>
</tr>
<tr>
<td></td>
<td>Significant difference in episodes of UI, urgency, number of voids and amount of urine in pad-test in favour of I group both at 36–38 weeks pregnancy and at 6–8 weeks post partum</td>
</tr>
<tr>
<td></td>
<td>PFM strength: significant difference (p = 0.00) in favour of I both at 36–38 weeks pregnancy and at 6–8 weeks post partum</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Author</th>
<th>Sangsawang and Serisathien, 2012</th>
</tr>
</thead>
<tbody>
<tr>
<td>Design</td>
<td>Quasi-experimental design, pre- and post-test</td>
</tr>
<tr>
<td>Control (C): (n = 35) PFMT</td>
<td></td>
</tr>
<tr>
<td>Intervention (I): (n = 35) PFMT</td>
<td></td>
</tr>
<tr>
<td>Study population</td>
<td>n = 70 with SUI at gestational age of 20–30 weeks</td>
</tr>
<tr>
<td>Training protocol</td>
<td>C: No treatment</td>
</tr>
<tr>
<td></td>
<td>I: 6 weeks PFMT</td>
</tr>
<tr>
<td>Losses to follow up</td>
<td>Losses to follow-up: 4 in I group</td>
</tr>
<tr>
<td>Adherence</td>
<td>Adherence to PFMT: Not reported. Not ITT analysis</td>
</tr>
<tr>
<td>outcomes</td>
<td>Severity of SUI after I: Significantly lower frequency and amount of urine leakage and score of perceived SUI severity in I group</td>
</tr>
</tbody>
</table>

ITT, intention-to-treat analysis; NS, not statistically significant; OR, odds ratio; VPFMC, voluntary pelvic floor muscle contraction; RR, relative risk; SD, standard deviation. For other abbreviations, see text.
### Table 7.16: Studies assessing the effect of pelvic floor muscle exercises after delivery to prevent urinary incontinence, including both women with and without urinary incontinence at inclusion

<table>
<thead>
<tr>
<th>Author</th>
<th>Sleep and Grant, 1987</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Design</strong></td>
<td>2-arm RCT</td>
</tr>
<tr>
<td>Control (C):</td>
<td>(n = 900) current standard care</td>
</tr>
<tr>
<td>Intervention (I):</td>
<td>(n = 900) current standard care + individual sessions PFMT</td>
</tr>
<tr>
<td><strong>Study population</strong></td>
<td>n = 1800 postpartum women recruited within 24 hours of vaginal delivery. Some women had existing UI. Single centre, England</td>
</tr>
<tr>
<td><strong>Training protocol</strong></td>
<td>C: current standard antenatal and postnatal care. Recommended to do PFM contractions as often as remembered and mid-stream urine stop. 4-week health diary</td>
</tr>
<tr>
<td></td>
<td>I: as above plus one individual session daily while in hospital with midwifery coordinator. 4-week health diary including section recommending a specific PFMT task each week</td>
</tr>
<tr>
<td><strong>Losses to follow-up/Adherence</strong></td>
<td>Losses to follow-up at 3 months: 84/900 in C group and 107/900 in I group</td>
</tr>
<tr>
<td><strong>Outcomes</strong></td>
<td>Adherence to PFMT: 3 months post partum 58% in I group and 42% in C group. Adverse events not stated. Not ITT analysis</td>
</tr>
<tr>
<td></td>
<td>Self-reported UI 3 mth post partum: C 175/793 (22%); I 180/816 (22%); RR (95% CI) 1 (0.83, 1.20)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Author</th>
<th>Markved and Bø, 1997</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Design</strong></td>
<td>Prospective matched controlled:</td>
</tr>
<tr>
<td>Control (C):</td>
<td>(n = 99) customary written postpartum instructions from the hospital</td>
</tr>
<tr>
<td>Intervention (I):</td>
<td>(n = 99) 8 weeks PFMT</td>
</tr>
<tr>
<td>Control (C):</td>
<td>(n = 81)</td>
</tr>
<tr>
<td>Intervention (I):</td>
<td>(n = 81)</td>
</tr>
<tr>
<td><strong>Study population</strong></td>
<td>n = 198 women 8 weeks post partum. Some women had existing UI. The criteria for matching: age (± 2 years), parity (1, 2, 3, 4 ≥ deliveries) and type of delivery. Single centre, Norway</td>
</tr>
<tr>
<td></td>
<td>n = 180 women one year post partum. All women who had participated in a matched controlled trial were contacted by telephone one year after delivery</td>
</tr>
<tr>
<td><strong>Training protocol</strong></td>
<td>C: customary written postpartum instructions from the hospital. Not discouraged from performing PFMT on their own. Correct PFM contraction checked at enrolment</td>
</tr>
<tr>
<td></td>
<td>I: 8 weeks of intensive PFMT (in a group) led by PT, with additional home exercises 10 max contractions (each held for 6 s) and to the last 4 were added 3–4 fast contractions, repeated twice daily, between 8 and 16 weeks post partum. Correct VPFMC checked at enrolment</td>
</tr>
<tr>
<td><strong>Losses to follow-up/Adherence</strong></td>
<td>Losses to follow-up in the I group: 7 women</td>
</tr>
<tr>
<td><strong>Outcomes</strong></td>
<td>Adherence to PFMT: −100% in I group; −65% in C group. Adverse events not stated</td>
</tr>
<tr>
<td></td>
<td>All longitudinal changes were conducted using a constant sample, including the 81 matched pairs that attended all tests. −53% in I group and 24% in C group reported that they were doing PFMT between 16th week and one year post partum</td>
</tr>
<tr>
<td></td>
<td>Self-reported UI at 16 weeks post partum: C 28/99 (28.3%); I 14/99 (14.1%); p = 0.015</td>
</tr>
<tr>
<td></td>
<td>Standardized pad-test: C 13/99 (13.1%); I 3/99 (3.0%); p = 0.009</td>
</tr>
<tr>
<td></td>
<td>PFM strength: significant difference in favour of I</td>
</tr>
<tr>
<td></td>
<td>Self-reported UI at 12 mth post partum: C 31/81 (38%); I 14/81 (17%); p = 0.003</td>
</tr>
<tr>
<td></td>
<td>Standardized pad-test: C 14/81 (13%); I 5/81 (3%); p &lt; 0.03</td>
</tr>
<tr>
<td></td>
<td>PFM strength: significant difference in favour of I</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Author</th>
<th>Meyer et al., 2001</th>
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</thead>
<tbody>
<tr>
<td><strong>Design</strong></td>
<td>Allocated to 2 groups:</td>
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<tr>
<td>Control (C):</td>
<td>(n = 56) no education</td>
</tr>
<tr>
<td>Intervention (I):</td>
<td>(n = 51) 12 sessions PFMT over 6 weeks with PT</td>
</tr>
<tr>
<td><strong>Study population</strong></td>
<td>n = 107 primiparous women recruited 12–39 weeks of pregnancy: 9/56 controls and 16/51 in the intervention group had self-reported SUI. Single centre, Switzerland</td>
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</table>
Table 7.16  Studies assessing the effect of pelvic floor muscle exercises during pregnancy to prevent urinary incontinence, including both women with and without urinary incontinence at inclusion—cont’d

<table>
<thead>
<tr>
<th>Training protocol</th>
<th>C (n = 56): no pelvic floor re-education offered from 2 to 10 mth post partum</th>
<th>I (n = 51): begun at 2 mth post partum. 12 sessions over 6 weeks with PT. PFMT followed by 20 min of BF and 15 min of ES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Losses to follow-up/Adherence</td>
<td>Losses to follow-up: 0%</td>
<td>Adherence not reported. Adverse events not stated. Not ITT analysis</td>
</tr>
<tr>
<td>Outcomes [Numbers and percentage (%)]</td>
<td>Self-reported SUI 10 mth post partum: C 8/56 (32%); I 6/51 (12%); RR (95% CI) 0.82 (0.31, 2.21)</td>
<td>Subjects cured: C 1/51 (2%), p = 1.0; I 10/56 (19%), p = 0.02</td>
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<tr>
<td></td>
<td>PFM strength: NS</td>
<td>Bladder neck position and mobility: NS</td>
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<td></td>
<td>Urodynamic parameters: NS</td>
<td></td>
</tr>
<tr>
<td>Author</td>
<td>Chiarelli and Cockburn, 2002</td>
<td>Chiarelli et al., 2004</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Design</th>
<th>2-arm RCT</th>
<th>2-arm RCT</th>
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<tbody>
<tr>
<td>Control (C): (n = 350) usual care</td>
<td>Control (C): (n = 294) usual care</td>
<td></td>
</tr>
<tr>
<td>Intervention (I): (n = 370) continence promotion</td>
<td>Intervention (I): (n = 275) continence promotion</td>
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<tr>
<td>Study population</td>
<td>n = 720 postnatal women following forceps or ventouse delivery, or delivered a baby ≥4000g. Some women had existing UI. Recruited at postnatal ward. Multicentre (3), Australia</td>
<td></td>
</tr>
<tr>
<td>Training protocol</td>
<td>C: usual care</td>
<td>I: continence promotion: one contact with physiotherapist on postnatal ward and another at 8 weeks post partum (correct PFM contraction checked at second visit). Intervention included individually tailored PFMT, use of transversus abdominus contraction, the 'Knack', techniques to minimize perineal descent, post partum wound management. Written and verbal information. Adherence strategies</td>
</tr>
<tr>
<td>Losses to follow-up/Adherence</td>
<td>Losses to follow-up: 6% in each group</td>
<td>Adherence to PFMT: C 57.6%; I 83.9%</td>
</tr>
<tr>
<td></td>
<td>Adherence not reported. Adverse events not stated. ITT analysis</td>
<td>Adverse events not stated. ITT analysis</td>
</tr>
<tr>
<td>Outcomes [Numbers and percentage (%)]</td>
<td>Self-reported UI 3 mth post partum: C 126/328 (38.4%); I 108/348 (31.0%) (95% CI: 0.22%–14.6%), p = 0.044</td>
<td>OR of incontinence for the women in the I group compared with C group: 0.65 (0.46–0.91), p = 0.01</td>
</tr>
<tr>
<td></td>
<td>Self-reported UI 12 mth post partum: difference between groups NS</td>
<td>Self-reported UI 12 mth post partum: difference between groups NS</td>
</tr>
<tr>
<td>Practice of PFMT at 12 mth promotes continence at this time</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Author</td>
<td>Ewings et al., 2005</td>
<td>Ewings et al., 2005</td>
</tr>
</tbody>
</table>

VPFMC, voluntary pelvic floor muscle contraction; NS, not statistically significant; CI, confidence interval; OR, odds ratio; RR, risk ratio; SD, standard differentiation. For other abbreviations, see text.
included only women with forceps or ventouse delivery or birth of a baby weighing 4000 g or more.

Training protocol

In three studies the training period started while the women were still at the hospital (Sleep and Grant 1987; Chiarelli and Cockburn, 2002; Ewings et al., 2005), while the training started 8 weeks after delivery in the other studies. Length of the training period, follow-up by health professionals, training intensity and frequency varied. Sleep and Grant (1987) gave one individual session of PFMT while in hospital in addition to standard care and recommended the women in the intervention group to do a specific PFMT task each week at home for 4 weeks. The 8-week training protocol in the study by Mørkved and Bø (1997) addressed individual instructions in PFM contractions, regular home training (two sets of 10 near-maximal contractions per day) and close weekly follow-up in groups. Meyer et al. (2001) added biofeedback and electrical stimulation to the 6-week PFMT programme, while the intervention group in the RCT by Chiarelli and Cockburn (2002) received individually tailored PFMT including two individual contacts with a PT and thorough information. The Health Beliefs Model was used as a framework to underpin the development of a successfully implemented postnatal continence programme. In addition, social marketing strategies were implemented in the development of materials used within the programme (Chiarelli and Cockburn, 2002). Adherence to the PFMT protocol was reported in four studies (Sleep and Grant, 1987; Mørkved and Bø, 1997; Chiarelli and Cockburn, 2002; Ewings et al., 2005), however different classification systems of adherence were used. Some studies used exercise diaries (Sleep and Grant, 1987; Mørkved and Bø, 1997; Chiarelli and Cockburn, 2002).

Most studies compared PFMT with current standard care, allowing self-managed PFMT but not introducing supervised intervention. In one study (Mørkved and Bø, 1997) the control group were given the same individual instructions in correct PFM contraction (including vaginal palpation and feedback) as the training group.

Outcomes

Three studies (Mørkved and Bø, 1997; Meyer et al., 2001; Chiarelli and Cockburn, 2002) reported clinically relevant and statistically significant effects of the interventions, with a significant reduction in symptoms or frequency of UI after the intervention period. Two trials reported no significant results of the intervention (Sleep and Grant, 1987; Ewings et al., 2005). No adverse effects of the interventions were reported. Mørkved and Bø (2000) found that the effect of PFMT was still present one year after cessation of the training programme, while Chiarelli and co-workers demonstrated short-term effects, but no difference in UI between groups at one- and six-year follow-up (Chiarelli and Cockburn, 2002; Chiarelli et al., 2004). However, Chiarelli et al. (2004) reported that continued adherence to PFMT at 12 months was predictive of UI at that time, with less UI among women training the PFM.

The effect of pelvic floor muscle exercises after delivery to treat UI, including only women with UI (Table 7.17)

Four RCTs were found (Wilson and Herbison, 1998; Glazener et al., 2001; Dumoulin et al., 2004; Kim et al., 2012), and two follow-up studies (Glazener et al., 2005; Elliott et al., 2009). PEDro scores were between 4 and 8 out of 10 (see Table 7.18). All the women included were continent, and they were recruited from 3 months (Wilson and Herbison, 1998; Glazener et al., 2001) or more (Dumoulin et al., 2004) after delivery. Both primi- and multiparous women were included.

Training protocol

The interventions followed different training protocols. All the trials included individual instructions in PFMT. Wilson and Herbison (1998) and Glazener et al. (2001) advised the women to perform 80–100 contractions per day and introduced 3–4 follow-up sessions in the period up to 9 months after delivery. Dumoulin et al. (2004) addressed close follow-up (weekly) by a PT and used a training protocol including a lower number of high intensity contractions. In the 8-weekly physical therapy appointments they included biofeedback and electrical stimulation in the training programme. Only Dumoulin et al. (2004) introduced an intervention in the control group (massage), while the two other trials compared PFMT with current standard care, allowing self-managed PFMT but no control intervention. Adherence to the PFMT protocol was reported in two trials (Wilson and Herbison, 1998; Glazener et al., 2001), but none of them used exercise diaries.

Outcomes

All trials (Wilson and Herbison, 1998; Kim et al., 2012; Mørkved and Bø, 2000; Glazener et al., 2001; Dumoulin et al., 2004) reported clinically relevant and statistically significant short-term effects of PFMT, with a significant reduction in symptoms or frequency of UI. No adverse effects of the interventions were reported. Glazener et al. (2001) found no difference in UI between groups at 6-year follow-up, while Elliott et al. (2009) reported that in the PFMT groups over 50% of the women were still continent according to pad-testing after 7 years. Incontinence-specific signs, symptoms and quality of life remained better than before treatment although not as good as immediately after cessation of the supervised training.
### Table 7.17 Studies assessing the effect of pelvic floor muscle exercises after delivery to treat urinary incontinence, including only women with urinary incontinence at inclusion

<table>
<thead>
<tr>
<th>Author</th>
<th>Wilson and Herbison, 1998</th>
</tr>
</thead>
</table>
| **Design** | 2-arm RCT  
Control (C): (n=117) standard postnatal PFM exercises  
Intervention (I): (n=113) 12 weeks of intensive PFMT |
| **Study population** | n=230 women with UI 3 mth post partum. Single centre, New Zealand |
| **Training protocol** |  
C: standard postnatal PFM exercises  
I: instructions by PTs (80–100 fast/slow contractions daily) 3, 4, 6 and 9 mth post partum.  
Use of perineometer to teach awareness of VPFMC. Three groups: (a) 39 women performed only PFMT; (b) 36 women only trained with vaginal cones 15 mins per day; (c) 38 women used both (a) and (b) |
| **Losses to follow-up/Adherence** | Losses to follow-up 12 mth outcome assessment: 36.9%. C 91/117; I 54/113  
Adherence to PFMT: last month 89%; every day 48% |
| **Outcomes** | 12 mth postnatally mean number of VPFMC 86 in I group and 35 in C group  
Self-reported UI at 12 mth post partum: C 69/91 (76%); I 27/54 (50%); p=0.003  
Pad-test: difference NS; perineometry: difference NS |

| Author | Glazener et al., 2001  
Glazener et al., 2005 |
|--------|----------------------|
| **Design** | 2-arm RCT  
Control (C): (n=6) no visit  
Intervention (I): (n=371) advice+visits  
3 centres: Aberdeen, Birmingham, Dunedin  
6-year follow-up  
Control (C): n=253  
Intervention (I): n=263 |
| **Study population** | n=747 women with UI 3 mth postnatally  
Multi-centre trial: New Zealand, UK  
n=516 |
| **Training protocol** | C: no visit  
I: assessment of UI, with advice on PFMT (80–100 fast/slow contractions daily) followed up 5, 7 and 9 months after delivery supplemented by bladder training if appropriate at 7 and 9 mths |
| **Losses to follow-up/Adherence** | Lost to follow-up at 12 mth: 31%. C 35%; I 25%  
Adherence to PFMT: in the 11th postnatal mth, 78% in I group (mean 20 VPFMC) and 48% in C group (mean 5 VPFMC) had done some PFMT. ITT analysis  
Lost to follow-up: 30%. Adherence (performing any PFMT): C 50%; I 50% |
| **Outcomes** | Self-reported UI at 12 mth post partum:  
Any UI: C 169/245 (69%); I 167/279 (59.9%); p=0.037, severe UI: C 78/245 (31.8%); I 55/279 (19.7%) (p=0.002)  
Severe UI at 6 years follow up: C 99/253 (39%); I 100/263 (38%); p=0.867 |

| Author | Dumoulin et al., 2004  
Elliott et al., 2009 (abstract) |
|--------|------------------|
| **Design** | 3-arm RCT:  
(1) Control (C): n=20  
(2) PFM rehabilitation: n=21  
(3) PFM rehabilitation+training of deep abdominal muscles: n=23 |
| **Study population** | n=64 parous women under 45 years, still presenting symptoms of SUI at least once per week 3 months or more after their last delivery. Recruited during annual gynaecological visit at an obstetric clinic, Canada |

(Continued)
This review of randomized and quasi-experimental studies in the field of PFMT during pregnancy and after delivery highlights the very large heterogeneity in the populations studied, use of inclusion and exclusion criteria, ways of including participants, use of outcome measures and content of the PFMT interventions. The 2008 Cochrane review (Hay-Smith et al., 2008) concluded that women without prior UI who were randomized to intensive antenatal PFMT were 56% less likely to report UI in late pregnancy and about 30% up to 6 months post partum. Postnatal women with persistent UI 3 months after delivery were 20% less likely than those not receiving PFMT to report UI 12 months after delivery. Hay-Smith et al. (2008) stated that it is unclear if the population-based approach is effective and that there was not enough evidence about the long-term effects. Brostrøm and Lose (2008) concluded from a narrative review that published studies on PFMT in general are small, underpowered and of uneven quality, and the available evidence suggests a lack of long-term efficacy of peripartum PFMT. Here we focus on the methodological quality of the studies, dose–response issues in exercise trials and challenges in long-term assessment of PFMT during pregnancy and after childbirth.

### Methodological quality

Using the PEDro rating scale, 10 is the top score. However, in exercise trials 7–8 out of 10 reflects high quality, accepting that the two criteria related to blinding of the therapist and patient are almost impossible to meet in this kind of intervention. In this review, 7 of 18 studies received a PEDro score of 7 or 8, as detailed in Table 7.18.

In addition to the PEDro criteria, sample size is a crucial factor in RCTs. Small sample size may cause type II error, meaning that a possible effect is not revealed because of low power. On the other side, it is also well known that a large sample size may overestimate results in clinical trials as

### Table 7.17

Studies assessing the effect of pelvic floor muscle exercises after delivery to treat urinary incontinence, including only women with urinary incontinence at inclusion—cont’d

| Training protocol | (1) 8 weekly sessions of massage  
| (2) PFM rehabilitation: weekly sessions supervised by physiotherapist for 8 weeks; 15 min ES + 25 min PFMT with BF + home training 5 days per week  
| (3) PFM rehabilitation as group 2 + 30 mins of deep abdominal muscle training  
| Losses to follow-up/Adherence | Losses to follow up: 3%. Adherence rate not stated. Adverse events not stated. ITT analysis.  
| Performing any PFMT: 54%  
| Outcomes | Self-reported UI after the intervention period:  
| Objective cure (less than 2 g urine on pad-test): (1) Control: 0/19; (2) PFM rehabilitation: 14/20; (3) PFM rehabilitation + training of deep abdominal muscles: 17/23. Significant difference in favour of the intervention groups (p = 0.001). Difference between the two intervention groups NS  
| Incontinence Impact Questionnaire: Significant difference in favour of the intervention groups  
| PFM strength: difference NS  
| Objective cure (less than 2 g urine on pad-test) performed by 26 out of 35 women: 14/26 = 53%  
| Incontinence Impact Questionnaire: significantly better than at baseline  
| Author | Kim et al., 2012  
| Design | 2-arm RCT:  
| Control intervention (C): n = 10  
| Intervention (I): n = 10  
| Study population | Post-partum women with UI (n = 20)  
| Single centre, Korea  
| Training protocol | C: Unsupervised PFMT  
| I: Supervised PFMT  
| Losses to follow-up/Adherence | Losses to follow up: 2/20. Adherence: not reported. Adverse events not stated. No ITT analysis  
| Outcomes | Significant difference in favour of the supervised PFMT group after the intervention period on:  
| Bristol Female Lower Urinary Tract Symptoms; vaginal squeeze pressure  

ITT, intention to treat analysis; NS, no statistically significant difference; OR, odds ratio; VPFMC, voluntary pelvic floor muscle contraction; RR, relative risk; SD, standard deviation. For other abbreviations, see text.
Table 7.18  PEDro quality score of RCTs in systematic review of studies assessing the effect of pelvic floor muscle exercises during pregnancy (to prevent/treat urinary incontinence)

<table>
<thead>
<tr>
<th>Study</th>
<th>E</th>
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<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>Total score</th>
</tr>
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<tr>
<td>Sleep and Grant, 1987</td>
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<td>+</td>
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<td>Mørkved and Bø, 1997</td>
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<td>Kim et al., 2012</td>
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<td>4</td>
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</tbody>
</table>

+, criterion is clearly satisfied; –, criterion is not satisfied; ?, not clear if the criterion was satisfied. Total score is determined by counting the number of criteria that are satisfied, except that ‘eligibility criteria specified’ score is not used to generate the total score. Total scores are out of 10.

*Studies published as only abstracts are not included.
Evidence-Based Physical Therapy for the Pelvic Floor

small and clinically irrelevant effect sizes may reach statistical significance. We disagree with Brostrøm and Lose (2008) that most antenatal and postpartum PFMT trials are small, as most of them have several hundred participants. However, there are two big trials in this area with 1169 and 1800 participants (Sleep and Grant, 1987; Hughes et al., 2001) that are of great concern when judging the effect of antenatal and postpartum PFMT. These two trials have applied very weak interventions, meaning very few visits with either a PT or a midwife. Herbert and Bo (2005) have shown how one trial with huge numbers clearly dilutes the effect of smaller high-quality studies when pooling them in a meta-analysis. The training dosage in the two above-mentioned studies was minimal and had extremely little potential for bringing significant effects. In addition, the training period in one of the studies was only 4 weeks (Sleep and Grant, 1987).

Quality of the intervention: dose–response issues

There is a strong dose–response relationship in exercise training. Type of exercise and frequency, intensity and duration of the training, as well as adherence to the exercise protocol will decide the effect size (Bo et al., 1990; Imamura et al., 2010). In the area of PFMT the six trials with no or little effect have either used inadequate training dosages (Sleep and Grant, 1987; Hughes et al., 2001), left the participants alone to train (Sleep and Grant, 1987; Hughes et al., 2001; Chiarelli and Cockburn, 2002) or have huge drop-outs and/or low adherence to the training protocol (Sleep and Grant, 1987; Ewing et al., 2005; Woldringh et al., 2007; Mason et al., 2010; Bo and Haakstad 2011). If the patients are not following the training protocol, we cannot evaluate the effect of PFMT. Conclusion can only be drawn on the feasibility of the programme, which is another research question. None of the studies used specific questionnaires or instruments to assess adherence. Questions about home exercise were either asked in general questionnaires or in a personal interview and some studies used exercise diaries. Registration of adherence to the supervised training sessions was done by those providing the supervision. Self-report by the participants may overestimate actual adherence, and we recommend that future studies improve the methods used to register adherence.

Several RCTs in the PFMT literature support the early finding by Bo et al. (1990) that there is a very large difference in the effect size between programmes with more or less intensive training and follow-up (Imamura et al., 2010). The term ‘intensive training’ comes from the RCT of Bo et al. (1990), but the interpretation of this term can be questioned. The general recommendations for effective strength training to increase muscle cross-sectional area and strength are three sets of 8–12 close-to-maximum contractions 3–4 times a week (Haskell, 1994). Intensity in the exercise science literature on strength training is defined as the percentage of 1 repetition maximum (1RM), meaning how close the contraction is to the maximal contraction (Fleck and Kraemer, 2004). Bo et al. (1990) emphasized close-to-maximum contractions and strength measurements were done throughout the training period. The same protocol has been used in several peripartum studies, and all of these trials show clinically relevant and statistically significant effect (Mørkved and Bo, 1997; Reilly et al., 2002; Mørkved et al., 2003; Dumoulin et al., 2004; Gorbea Chavez et al., 2004; Dinc et al., 2009; Ko et al., 2011; Kim et al. 2012; Sangsawang and Serisathien, 2012; Stafne et al., 2012). In a recent assessor blinded RCT of PFMT to reduce pelvic organ prolapse, Brækken et al. (2010) found that this protocol significantly increased PFM strength and muscle thickness, reduced muscle length and area of the levator hiatus, in addition to lifting the position of the bladder neck and rectal ampulla. Hence, PFMT is changing muscle morphology, working in the same way as strength training of general skeletal muscles.

Training volume is the total workload of training (Fleck and Kraemer, 2004). In the PFMT literature, exercise programmes with only one supervised individual or group training session per week is named intensive. Some physicians suggest that follow-up once a week does not translate into clinical reality (Brostrøm and Lose, 2008). However, it is common to offer physical therapy at least 2–3 times a week for other conditions such as neck and low back pain, injured athletes are given supervised training at least once a day, and in rehabilitation centres patients are exercising several hours per day. There are no pharmaceutical companies that would allow treatment or research with their drugs with an ineffective dosage. Nor would anyone suggest that surgeons should do suboptimal surgery. In the long run, there is no money to be saved on low or suboptimal training dosages in physical therapy because treating a large number of patients with ineffective interventions can be very costly. Furthermore, by recommending low dosage or unsupervised training, the patients with no or little effect believe they have tried PFMT and may not be motivated for conducting a new period of more optimal dosage and supervised training before opting for other treatment options. Evidence-based practice means to use protocols from high-quality RCTs showing worthwhile effect sizes (Herbert and Bo, 2005; Bo and Herbert, 2009).

Another specific problem in studies evaluating the effect of antenatal and postpartum PFMT is that in most countries it is established practice to advise all women to do PFMT. Hence most of the PFMT studies have compared PFMT with ‘usual care’. ‘Usual care’ can vary between thorough individual instruction with clinical assessment and motivation for training to providing women with written information only. In some studies the control group has done substantial PFMT (Woldringh et al., 2007; Gorbea Chavez et al., 2004) compared the effect of PFMT with a group specifically asked not to train the PFM, and the difference between groups was highly significant with no women reporting UI in the PFMT group compared to 47% in the control group. To date there are no studies comparing the effect of ‘usual
care’ with no exercise. For some women being able to perform strong contractions and being highly motivated for training, such initiatives may be enough, and there will be difficulties showing differences between the intervention and the control group. However, studies have shown that few women exercise regularly with a recommended dosage during pregnancy and after childbirth without supervision (Bø et al., 2007a; Bø, Owe, 2007).

Physical therapists, nurses and physicians conducted the PFMT in all the clinical trials included in the present review, and to date there has been no comparison of effects of interventions given by different professionals. Given the widespread prevalence of UI in the female population and the evidence for PFMT, we suggest that PFMT should be part of general strength training programmes for women. This would imply that proper teaching of PFM function and dysfunction and how to teach PFMT correctly should be part of the curricula in exercise science, fitness and sports studies.

**Long-term effects**

Another general critique of the effect of PFMT is a possible lack of long-term benefit, especially in the peripartum studies (Brostrom and Lose, 2008). However, the effect of any training programme will diminish with time if not continued. In general, strength gains decline at a slower rate than that at which strength increases due to training. There are few studies investigating the minimal level of exercise necessary to maintain the training effect. A 5–10% loss of muscle strength per week has been shown after training cessation (Fleck and Kraemer, 2004). Greater losses have been shown in the elderly (65–75-year-olds) compared to younger (20–30-year-olds), and for both groups the majority of strength loss was from week 12–31 after cessation of training. The rate of strength loss may depend on length of the training period prior to detraining, type of strength test used and the specific muscle groups examined. Fleck and Kraemer (2004) concluded that research has not yet indicated the exact resistance, volume and frequency of strength training or the type of programme needed to maintain the training gains. However, studies indicate that to maintain strength gains or slow strength loss, the intensity should be maintained, but the volume and frequency of training can be reduced. One to two days a week seems to be an effective maintenance frequency for individuals already engaged in a resistance training programme (Kraemer and Ratamess, 2004).

So far, no studies have evaluated how many contractions subjects have to perform to maintain PFM strength after cessation of organized training. However, a long-term effect cannot be expected if the women stop exercising. In addition, long-term effect, meaning for more than one year, in pregnant and postpartum women is almost impossible to evaluate, as many women would be pregnant again during the follow-up period. This is likely to negatively interfere with the short-term effect. Furthermore, in most trials the control groups are given information or supervised training after cessation of the RCT. This was shown in the study by Mørkved et al. (2007), where the control group received the training programme after the results of the RCT were published. In the following period up to 6 years the adherence to the PFMT programme was similar in the original control group and the training group. The continence rate in the training group was nearly the same at 3 months and 6 years follow-up, while the number of incontinent women in the control group had decreased in the period. However, in another study, Mørkved and Bø (1997, 2000) showed that the initial effect of postpartum PFMT was maintained one year after delivery. Hence, the demand for long-term follow-up studies of PFMT in general can be questioned, and longer follow-up periods than one year after birth, in our opinion, are not warranted.

**CONCLUSION**

Based on studies with relevant sample size, high adherence to a strength training protocol and close follow-up, pelvic floor muscle training both during pregnancy and after delivery can prevent and treat urinary incontinence. The most optimal dosage for effective PFMT is still not known. However, a training protocol following general strength training principles, emphasizing close to maximum contractions and at least an 8-week training period can be recommended. Evidence-based practice of PFMT during pregnancy and after delivery implies using protocols from high-quality RCTs showing clinically relevant and statistically significant results. Given the detrimental negative effect of a non-functioning pelvic floor on women’s participation in sport and physical activity, there is a need to update the exercise in pregnancy guidelines. New guidelines for exercise during pregnancy and after childbirth should include detailed recommendations for effective PFMT, and we provide an outline in Box 7.1.

**ACKNOWLEDGMENT**

The authors thank Ingrid Ingeborg Riphagen, Unit for Applied Clinical Research, Department of Cancer Research and Molecular Medicine, Norwegian University of Science and Technology, for her contribution to the work being reported, by conducting the data searches.

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Box 7.1 How to tell if you are contracting the pelvic floor muscles correctly

- Sit on the arm of a chair or the edge of a table. Lift the pelvic floor up from the surface you are sitting on by pulling up and contracting around the urethra, vagina and rectum. Squeeze so hard that you feel a slight trembling in your vagina. When you squeeze hard enough, you can feel the lower part of the stomach being pulled in slightly at the same time. Release the contraction without pressing downward. Try to feel the difference between relaxing and tightening the pelvic floor.

- Try to stop the flow when you are urinating. If these muscles are weak, it may be difficult to stop the flow when it is strongest. You can then test yourself towards the end of urination, which is much easier. This is only a test to see whether you are using the muscles correctly. Do not use urination for training, as this can interfere with the ability to empty your bladder completely.

- If you are not sure about whether you are doing it correctly, contact your doctor and ask for a referral to a physical therapist with special training in women’s health.

Training programme

Lift up and inward around your urethra, vagina and rectum. Squeeze as hard as you can during each contraction and try to hold it for 6–8 seconds before you gently relax. Relax and breathe with a slow, regular and gentle rhythm out and in both during and between the muscle contractions. Do 8–12 repetitions in three sets. If this seems too difficult, start with fewer repetitions. Choose one or more of these starting positions:

1. Sit with your legs apart and your back straight. Lift upwards and inwards around the openings in the pelvic floor.

2. Stand with your legs apart, and check that the buttock muscles are relaxed while you squeeze the pelvic floor muscles.

3. Kneel on all fours with your knees out to the side and feet together. Lift the pelvic floor upwards and inwards.

REFERENCES


Chapter

Female pelvic floor dysfunctions and evidence-based physical therapy
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7.4 Pelvic organ prolapse

Clinical assessment of pelvic organ prolapse

Matthew D Barber

BACKGROUND

Pelvic organ prolapse (POP) is the downward descent of the female pelvic organs (vagina, uterus, bladder and/or rectum) into or through the vagina. POP is a common clinical condition, encountered on a daily basis by PTs specializing in the care of women with pelvic floor disorders. Loss of vaginal or uterine support in women presenting for routine gynaecology care is seen in up to 43–76% of women (Swift, 2000; Ellerkmann et al., 2001). Many women with pelvic organ prolapse are asymptomatic and do not require treatment. This is particularly true for women with prolapse that is mild and does not extend beyond the hymen. However, approximately 3–6% of women have descent of the uterus or vagina beyond the hymen and approximately 3% of women report symptomatic vaginal bulging (Nygaard et al., 2008). Women who develop symptoms may present with a single symptom such as vaginal bulging or pelvic pressure or they may present with multiple complaints. Ellerkmann et al. (2001) found that in 237 women evaluated for POP 73% reported urinary incontinence, 86% reported urinary urgency and/or frequency, 34–62% reported voiding dysfunction and 31% complained of faecal incontinence. Some are the result of the prolapsing vagina itself and some are caused by coexisting or associated dysfunction of the bladder, lower gastrointestinal tract or pelvic floor. In either circumstance, the evaluation of a patient with vaginal prolapse requires a comprehensive review of the full spectrum of pelvic floor symptoms, an assessment of how these symptoms affect their quality of life and a pelvic examination to evaluate pelvic organ descent.

HISTORY

As women with POP can present with multiple pelvic floor symptoms it is important that the clinical assessment should include a comprehensive history including an assessment for vaginal bulge symptoms and associated lower urinary and gastrointestinal symptoms and symptoms of sexual dysfunction (Box 7.2). The symptom that most strongly correlates with advanced POP is the presence of a vaginal bulge that can be seen or felt (Swift et al., 2003; Bradley and Nygaard, 2005; Tan et al., 2005). Additionally, the hymen seems to be an important cut-off point for symptom development. Women with prolapse beyond the hymen have more pelvic floor symptoms and are much more likely to report a vaginal bulge than women with prolapse at or above the hymen (Swift et al., 2003; Tan et al., 2005). In fact, the presence of vaginal bulge symptoms is a highly specific symptom for predicting the presence of prolapse beyond the hymen on a straining examination (specificity 99–100%) (Bradley and Nygaard, 2005; Barber et al., 2006). Less specific symptoms such as pressure and heaviness have a much weaker relation to loss of vaginal support (Samuelsson et al., 1999; Ellerkmann et al., 2001; Burrows et al., 2004). Lower urinary tract complaints are frequent in women with POP. In some circumstances, loss of vaginal support directly affects bladder or urethral function, resulting in symptoms. In other cases, the relation between prolapse and lower urinary tract dysfunction is less clear. The anterior vaginal wall supports the bladder and urethra. Loss of this support results in urethral...
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Hypermobility and cystocele formation, which is thought to contribute to development of stress urinary incontinence (DeLancey, 1994). Therefore, the fact that pelvic organ prolapse and stress urinary incontinence sometimes coexist is not surprising, particularly when prolapse is mild. In contrast, women with POP that extends beyond the hymen are less likely to complain of stress incontinence and more likely to have obstructed voiding symptoms such as urinary hesitancy, intermittent flow, weak or prolonged stream, feeling of incomplete emptying, the need to manually reduce (splinting) the prolapse to initiate or complete urination and, in rare cases, urinary retention (Samuelsson et al., 1999; Ellerkmann et al., 2001; Burrows et al., 2004; Barber, 2005). The mechanism for this appears to be mechanical obstruction resulting from urethral kinking that occurs with progressively worsening anterior vaginal prolapse. As many as 30% of women with Stage 3 or 4 POP have elevated post void residuals (PVR > 100 ML) (Coates et al., 1997). Symptoms of POP, particularly bulging and pressure, are generally worse after long periods of standing or exercise and often improved when the patient is lying supine. Complaints of vaginal bleeding, discharge or infection may result from vaginal ulcerations sometimes seen with advanced prolapse. Unexplained vaginal bleeding should prompt a work-up for other causes of bleeding, particularly in postmenopausal women, where an evaluation of the endometrium via biopsy or ultrasound is typically warranted.

POP, as with other pelvic floor disorders, rarely results in severe morbidity or mortality; rather it causes symptoms that can impact a woman’s daily activities and negatively affect her quality of life. As such, it is essential to understand not only the symptoms a particular patient experiences but the degree of bother caused by these symptoms and how it impacts her quality of life. This understanding is essential for determining when and what treatment to offer. Current management options for women with symptomatic POP include observation, pessary use and surgery. Women with even advanced POP may have minimal symptoms and report little or no bother as a result of their POP. In these cases, observation or ‘watchful waiting’ is perfectly appropriate. Women with advanced POP who choose observation should be examined periodically to identify the development of new symptoms or conditions that might prompt treatment.

**QUESTIONNAIRES**

Pelvic floor symptoms can be assessed in a number of ways. Obviously, taking a thorough clinical history is an important method of assessing the patient’s symptoms and their effect on the patient’s life. However, in situations where a standardized, reproducible assessment is desired, clinical histories can be problematic, as they typically take on a different form for each clinician and patient encounter. The most valid way of measuring the presence, severity and impact of pelvic floor symptoms on a patient’s activities and well-being is through the use of psychometrically robust self-administered questionnaires (Barber, 2005).

Several valid, reliable and responsive questionnaires are available and have been widely used in women with POP, including symptom questionnaires, measures of health-related quality of life and sexual function questionnaires (Box 7.3).

**PHYSICAL EXAMINATION**

Patients presenting with symptoms suggesting POP should undergo a pelvic examination. The pelvic examination should be performed with the patient resting and straining while supine and standing in order to define the extent of the prolapse and determine the segments of the vagina involved (anterior, posterior or apical) (Bump et al., 1996).
It is important that a clinician reproduces the maximum extent of prolapse that the patient exhibits in their daily life. The extent of prolapse of the anterior vaginal wall can be evaluated by placing a Sim’s speculum or the posterior blade of a bivalve speculum in the vagina to retract the posterior vaginal wall. The patient is asked to strain and the extent of anterior vaginal prolapse is noted. The blade is then placed to retract the anterior vaginal wall and the patient strains to reveal any posterior prolapse. A rectovaginal examination can be useful to identify the presence of a rectocele and determine the integrity of the perineal body. A bivalve speculum is inserted and the cervix or, in women who have had a hysterectomy, the vaginal cuff is identified to evaluate apical vaginal support. While the patient strains, the speculum is slowly withdrawn and the descent of the vaginal apex is noted. In women with prolapse that protrudes beyond the hymen for a long duration, the vagina and/or cervix can become hypertrophied and develop erosions. A bimanual examination is performed to rule out coexistent gynaecologic pathology and pelvic muscle strength is assessed as described in the previous sections.

Although numerous prolapse grading systems have been described, the most widely used and accepted is the Pelvic Organ Prolapse Quantification system (POP-Q). The POP-Q system was introduced in 1996 jointly by the Society of Gynecologic Surgeons, the American Urogynecologic Society and the International Continence Society as the accepted method for describing pelvic support and comparing examinations over time and after interventions (Bump et al., 1996). This system has since been similarly adopted by the National Institutes of Health, the International Urogynecologic Association (IUGA) and the World Health Organization’s International Consultation on Incontinence (Weber et al., 2001; Haylen et al., 2010).

This prolapse grading system has been shown to have good inter- and intra-examiner reproducibility in multiple studies and, while other prolapse grading systems are still used by some, POP-Q has become the most commonly used system in the peer-review literature (Hall et al., 1996; Kobak et al., 1996; Muir et al., 2003).

The POP-Q examination systematically defines the degree of prolapse during a pelvic exam by measuring anterior, posterior and apical segments of the vaginal wall in centimetres relative to a fixed anatomical structure, the vaginal hymen, with the patient performing maximal strain. This descriptive system contains a series of site-specific measurements of the woman’s pelvic organ support. It can be easily learned and taught by means of a video tutorial (Steele et al., 1998). Prolapse in each segment is evaluated and measured relative to the hymen (not introitus), which is a fixed anatomic landmark that can be identified consistently and precisely. The anatomic position of the six defined points for measurement should be centimetres above or proximal to the hymen (negative number) or centimetres below or distal to the hymen (positive number), with the plane of the hymen being defined as zero. For example, a cervix that protrudes 3 cm distal to the hymen should be described as +3 cm. Six points (two on the anterior vaginal wall, two in the superior vagina and two on the posterior vaginal wall) are located with reference to the plane of the hymen (Fig. 7.8). In addition, three other measurements are made in centimetres: the length of the table.
perineal body (from posterior forchette to mid-anus), the length of the genital hiatus (from external urethral meatus to posterior forchette) and the total vaginal length (tvl) (measured from the hymen to the apex/posterior fornix, non-straining). In addition to these site-specific measurements, the POP-Q system provides a highly reliable and reproducible staging system (Table 7.19) to describe the overall extent of pelvic organ descent. Stages are assigned according to the most severe portion of the prolapse when the full extent of protrusion has been demonstrated.

In addition to its widespread adoption and proven re-producbility, another advantage of the POP-Q system is its relative precision (nine site-specific measurements in 1 cm increments) which has allowed an improved understanding of the relationship between the anatomic characteristics of pelvic organ prolapse and the development of specific pelvic floor symptoms (Barber, 2007). When evaluating pelvic organ support in a study, investigators should perform a standardized evaluation including the POP-Q before and after the intervention. Details of this evaluation should be reported, including the position in which the examination was performed, the fullness of the bladder, the type of vaginal specula, retractors and measuring devices used, and the method used to ensure that the maximal extent of prolapse is seen. It is critical that the examiner sees and describes the maximum protrusion noted by the individual during her daily activities. Disadvantages of the system include its relative complexity and the exclusion of some anatomic findings that some investigators believe to be essential for complete patient description, such as vaginal calibre, status of paravaginal support, pelvic floor descent and urethral mobility.

**Table 7.19 Staging system for pelvic organ prolapse, based on the POP-Q system**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Definition</th>
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<tbody>
<tr>
<td>0</td>
<td>No prolapse is demonstrated. Points Aa, Ap, Ba and Bp are all at −3 and the absolute value of either point C or D is ≤[tvl −2]cm</td>
</tr>
<tr>
<td>I</td>
<td>Criteria for stage 0 are not met, but the most distal portion of the prolapse is &gt;1 cm above the hymen (its absolute value is ≤−1 cm)</td>
</tr>
<tr>
<td>II</td>
<td>The most distal portion of the prolapse is ≤1 cm proximal to or distal to the hymen (its absolute value is ≤+1 but ≥ −1 cm)</td>
</tr>
<tr>
<td>III</td>
<td>The most distal portion of the prolapse is &gt;1 cm below the plane of the hymen but protrudes no further than 2 cm less than tvl (its absolute value is &gt;+1 but &lt;[tvl −2] cm)</td>
</tr>
<tr>
<td>IV</td>
<td>Essentially, complete eversion of the total length of the vagina is demonstrated. The distal portion of the prolapse protrudes to at least [tvl −2] cm (its absolute value is ≥[TVL−2] cm)</td>
</tr>
</tbody>
</table>

For description of measurement points see Figure 7.8 and Bump et al., 1996.

**REFERENCES**


**CONCLUSION**

The clinical evaluation of a patient with POP requires a comprehensive review of the full spectrum of pelvic floor symptoms, an assessment of how these symptoms affect their quality of life and a pelvic examination to evaluate pelvic organ descent. Ancillary testing will largely depend upon the symptoms that the patient presents with but may include cystoscopy, urodynamics, dynamic MRI and/or pelvic floor ultrasound. A number of valid, reliable questionnaires exist to measure the presence and severity of pelvic floor symptoms and their impact on a patient’s activities and the well-being of women with POP, which complement the information gained from a clinical history. Pelvic examination using the POP-Q system to quantitatively assess the degree of anterior, posterior and apical pelvic organ support is an essential component of the clinical evaluation of women with POP. The POP-Q system is a valid and reliable system that provides a comprehensive description of pelvic organ support and overall stage of prolapse that is easy to learn and clinically useful as both baseline assessment and outcome measure after treatment. The system has also facilitated clinical and translational research in multiple areas related to female pelvic floor disorders.
Use of pessaries to prevent and treat pelvic organ prolapse

Patricia Neumann

INTRODUCTION

A pessary is an intravaginal device designed to provide support to the vaginal walls in women with pelvic organ prolapse (POP). Pessaries, made of various materials from pomegranates to wool, have been used since ancient times, but today they are available in silicone, which has the advantages of not absorbing secretions and odours and of being hypoallergenic (Atnip, 2009). Pessaries are manufactured in a range of shapes and sizes (Fig. 7.9). A pessary may be used in the management of POP with the aim of preventing prolapse progression, in reducing symptom severity and for women who are unsuitable or unwilling to undergo surgery (Oliver et al., 2011).

POP is a major concern for women as some 50% of women have some degree of POP, with a serious impact on quality of life in more severe cases (Swift, 2000).
Women face an 11% lifetime risk of requiring surgery for POP (Olsen et al., 1997) and also the prospect of surgical failure, which may be as high as 58%, particularly in younger women (Whiteside et al., 2004). This presents a dilemma for women when making management decisions.

**Economic considerations**

Although there are minimal data on the costs of POP surgery (Moore et al., 2009), the direct costs of POP surgery in the United States were estimated to be in excess of 1 billion US dollars in 1997 (Subak et al., 2001), which represents a substantial cost to the healthcare system, particularly when translated into current values. Pessaries are widely advocated as a safe and inexpensive way to manage POP (Atnip, 2009; Oliver et al., 2011) but the possible economic benefits of pessary management over surgery have not been researched (Bugge et al., 2013).

**Scope of practice**

Pessaries, pelvic floor muscle training (PFMT) and lifestyle advice are recommended by the Fourth International Consultation on Incontinence as conservative management options for women with POP (Hay-Smith et al., 2009). PFMT now has level 1 evidence from a number of high-quality studies underpinning its recommendation. PFMT is widely provided by physical therapists (PTs) in primary care. Pessaries are generally prescribed by gynaecologists (Cundiff et al., 2000; ACOG, 2007; Gorti et al., 2009).

Reports that nurses have successfully prescribed and fitted pessaries in nurse-led pessary clinics (McIntosh, 2005; Hanson et al., 2006; Maito et al., 2006), raised the question of whether PTs could also be trained to prescribe and fit pessaries in primary care as part of the medical team. Pessary fitting has not been within the scope of practice of pelvic floor or continence-trained PTs in Australia, or elsewhere to the author’s knowledge, although other vaginal devices, such as those for stress urinary incontinence, are commonly prescribed and fitted. Expanding PTs’ scope of practice to include pessary use could have economic advantages and be more convenient for women, but minimal standards and demonstrated competencies need to be established first to ensure safe practice.

**Clinical practice guidelines**

As no clinical practice guidelines were found on pessary fitting, an Australian expert working party consisting of a urogynaecologist, gynaecologists, pelvic floor PTs and continence nurses collaborated with the International Centre for Allied Health Evidence to produce the ‘Guidelines for the use of support pessaries in the management of pelvic organ prolapse’ and the accompanying treatment algorithm. The guidelines are also available from the Australian National Health & Medical Research Council through their guideline portal (Clinical Practice Guideline, 2012). They are suitable for use by medical and suitably qualified allied health professionals.
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Training

Workshops were subsequently held to train theoretical and practical skills, based on the guidelines, for a multi-disciplinary group of doctors, nurses and pelvic floor PTs. Only limited numbers of the PTs have since started to fit pessaries in their clinical practice (Neumann et al., submitted). A number of barriers to change of practice were identified and this is the subject of ongoing study. There are cogent arguments, both economic and practical, for pessaries to be fitted in primary care by PTs who already provide prolapse management with PFMT and lifestyle advice. PTs have demonstrated that they can be trained to perform a Pelvic Organ Prolapse Quantification (POP-Q) assessment, a necessary precursor to pessary fitting. There was substantial agreement with POP-Q assessment between a gynaecologist and a PT (weighted kappa 0.64) and a PT on two occasions (weighted kappa 0.71) (Stark et al., 2010). Research into optimal training in pessary fitting for PTs and how to achieve acceptance of this role within the wider medical community is an exciting challenge for the future.

EVIDENCE FOR THE USE OF PESSARIES TO MANAGE POP

Despite the widespread use of pessaries, there is no level 1 evidence for their effectiveness when compared with alternative management options such as surgery or PFMT, or with placebo (Bugge et al., 2013). There is also a lack of high-level evidence for many aspects of pessary use, such as the best type of pessary to use for different types of prolapse, their indications for use and optimal pattern of care (Bugge et al., 2013).

Although there is a persuasive biological rationale for using a pessary together with PFMT to manage POP, i.e. to elevate the pelvic organs above the levator plate and then to train the muscles, this has been the subject of only one randomized controlled trial to date and results are not yet available (Hagen et al., 2011).

Effect of pessaries on pelvic floor symptoms

The only randomized controlled trial to date compared management with two different types of pessary, a ring and Gellhorn, in a cross-over trial (Cundiff et al., 2007). One hundred and thirty-four women were randomly assigned to be fitted first with either a ring or Gellhorn pessary and each was worn for 3 months. Approximately 60% of the women who completed the study were effectively fitted with a pessary and experienced both statistically and clinically significant symptom relief measured by the Pelvic Floor Distress Inventory and Pelvic Floor Impact Questionnaire, i.e. across the domains of bladder, prolapse and bowel. There was no statistically significant difference in scores on either measure between groups after 3 months of pessary use. Although this study concluded that both types of pessary were equally effective in relieving symptoms, the high rate of attrition (40%) suggests that the results should be interpreted with caution.

There are ethical considerations, which make random assignment to pessary or surgical treatment in a controlled trial difficult. One prospective non-randomized trial compared treatment with a pessary or surgery in 554 women with symptomatic POP (Abdoool et al., 2011). One hundred and ninety-five women were treated with surgery and 359 women were fitted with a pessary. One year later, 63% of those fitted with a pessary continued to use it. Ring pessaries were fitted most frequently (83%), followed by a Gellhorn (14%), a cube (0.03%) and donut (0.02%). One year later, when compared with pre-treatment, there was a statistically significant improvement in most parameters of PFM function (prolapse, urinary, faecal urgency and sexual symptoms) and quality of life in both pessary users and those treated surgically. Difficulty with bowel emptying improved only in the surgically treated group. When the two groups were compared at 1 year, there was no statistically significant difference in symptoms of prolapse, bladder, bowel and sexual function or quality of life between groups, providing evidence of the equivalence of both treatments over the first year of treatment in terms of patient reported outcomes. Importantly, the costs of treatment may have differed between pessary and surgical management but these outcomes were not evaluated. Concomitant training of PFM and of appropriately coordinated defecation in pessary users could improve the outcomes achieved with pessaries and deserves further study.

Effect of pessaries on quality of life

There are also a number of level 3 studies using validated symptom questionnaires suggesting that pessaries are effective in improving not only prolapse symptoms but also satisfaction (Clemens et al., 2004), sexual function (Fernando et al., 2006; Kuhn et al., 2009), quality of life (Lamers et al., 2011; Manchana and Bunyavejchevin, 2012) and goal attainment in domains such as general health (Komesu et al., 2007).

Complications

A 5-year prospective study found that if women were successfully wearing a pessary after 4 weeks, then they were likely to still be using a pessary 5 years later, without any concomitant increase in complications (Lone et al., 2011).

Although pessaries are widely considered to be a safe form of treatment, relatively minor complications such as vaginal bleeding, erosion and infection can occur in as many as 56% of women using pessaries long term.
There are also some reports of serious complications, such as cervical incarceration and impacted or embedded pessaries, which are more likely when pessaries are neglected and not changed regularly (Arias \textit{et al.,} 2008; Sarma \textit{et al.,} 2009). Based on expert opinion, women are advised to remove and re-insert the pessary themselves at regular intervals (i.e. ‘self-care’) to reduce the likelihood of complications (Bash, 2000; Atnip, 2009).

\textbf{Therapeutic effect}

There is level 3 evidence to suggest that pessaries may have a therapeutic effect. Nineteen women who had used a pessary for at least a year removed the pessary 48 hours before objective testing using the POP-Q system, i.e. on full Valsalva. An improvement in objective measures of prolapse was noted in 4 (21\%) subjects and there was no deterioration in stage of prolapse in any of the others (Handa and Jones, 2002). Further level 3 evidence suggests possible morphological changes associated with pessary use. Three months of Gellhorn pessary use significantly reduced the dimensions of the genital hiatus on straining compared with baseline measures (Jones \textit{et al.,} 2008). These two studies reported on older cohorts of women with mean ages of 75 and 64 years respectively.

\textbf{RATIONALE FOR THE ROLE OF PTS IN PESSARY FITTING}

Such findings suggest a possible rationale for fitting pessaries in younger women to prevent prolapse progression or in younger women with levator avulsion injuries who are at high risk of developing a prolapse (Dietz and Simpson, 2008), particularly if they have chronic lung disease, constipation or work in environments where heavy or repetitive lifting is required (Mouritsen \textit{et al.,} 2007). These are populations typically seen by pelvic floor PTs, who would be well placed to provide a holistic programme of PFM rehabilitation, lifestyle advice and pessary care in the primary care setting in collaboration with the woman’s general practitioner. Pessaries potentially provide a management option that enables women to engage in physical activity to maintain ideal weight and physical fitness, when, anecdotally, women with prolapse are afraid to exercise because they may exacerbate their symptoms. The potential for such a holistic physical therapy approach to delay the progression of prolapse or reduce the need for surgery merits further study, particularly in view of our ageing populations and the pressure on restricted health resources.

\textbf{CLINICAL RECOMMENDATIONS}

- Clinical practice guidelines, based on low levels of evidence, are available for healthcare professionals, including PTs, trained in pessary fitting.
- Pessaries may be used as first-line therapy for women with pelvic organ prolapse.
- Most women, offered a pessary and preferring it to surgery, can be successfully fitted and obtain symptom relief.
- If possible, women should be taught ‘self-care’ to prevent complications.
- Regular follow-up and examination by a medical practitioner is essential to avoid rare but potentially serious complications.

\textbf{REFERENCES}


Evidence-Based Physical Therapy for the Pelvic Floor

INTRODUCTION

The prevalence of symptomatic pelvic organ prolapse (POP) is reported to be 3–28% (Tjøgestad et al., 2005; Nygaard et al., 2008; Lawrence et al., 2008; Milsom et al., 2009; Slieter-ten Hove et al., 2009). Mechanical symptoms such as vaginal bulging and perception of pelvic heaviness are the most prevalent and specific symptoms of POP (Mouritsen, 2005; Srikrishna et al., 2010), and these symptoms may greatly impair quality of life and result in restriction of participation in, for example, physical activity (Srikrishna et al., 2008).

Pelvic floor muscle training in prevention and treatment of pelvic organ prolapse

Kari Bø, Helena Frawley

Evidence-Based Physical Therapy for the Pelvic Floor

INTRODUCTION

The prevalence of symptomatic pelvic organ prolapse (POP) is reported to be 3–28% (Tjøgestad et al., 2005; Nygaard et al., 2008; Lawrence et al., 2008; Milsom et al., 2009; Slieter-ten Hove et al., 2009). Mechanical symptoms such as vaginal bulging and perception of pelvic heaviness are the most prevalent and specific symptoms of POP (Mouritsen, 2005; Srikrishna et al., 2010), and these symptoms may greatly impair quality of life and result in restriction of participation in, for example, physical activity (Srikrishna et al., 2008).
It is estimated that approximately 50% of all women lose some of the supportive mechanisms of the pelvic floor due to childbirth, leading to different degrees of POP (Thakar and Stanton, 2002). In the UK, POP accounts for 20% of women on waiting lists for major gynaecological surgery (Thakar and Stanton, 2002). The prevalence of surgery for POP is considerable: up to 19% of women in Australia (Smith et al., 2010) and 20% in The Netherlands (de Boer et al., 2011). Prolapse recurs in up to 70% of women after surgery (Iglesia et al., 2010), and about one-third of operated women undergo at least one further surgical procedure for prolapse (Olsen et al., 1997). Potential risk factors for POP have been listed as constipation, pelvic surgery, genetic factors, familial transmission, Caucasian ethnicity, pregnancy and vaginal delivery (especially instrumental vaginal delivery), generalized connective tissue disorders (Ehlers–Danlos disease and Marfan’s syndrome), chronic anemia, chronic obstructive pulmonary disorders, low educational level/low income and hard work/exercise (Milsom et al., 2009). In a one-to-one age- and parity-matched case–control study, Brækken et al. (2009) compared 49 women with POP-Q stage ≥ II with 49 controls stage 0 and I and found no difference in postmenopausal status, current smoking, current low intensity exercise, type of birth, birth weight, presence of striae, diastasis recti abdominis and joint hypermobility. However body mass index (BMI), socioeconomic status, heavy occupational work, anal sphincter lacerations, PFM strength and endurance were independently related to POP. The combination of weak PFM and low vaginal resting pressure gave the highest odds ratio for POP (Brækken et al., 2009). The high prevalence and its increase with age highlight the need for preventative measures that could reduce both the incidence and the impact of POP.

Prolapse may be asymptomatic until the descending organ reaches the introitus, and therefore POP may not be recognized until an advanced condition is present (Handa et al., 2004; Milsom et al., 2009). In some women the prolapse advances rapidly, while others remain stable for many years. Most clinicians have considered that POP does not seem to regress. However, Handa et al. (2004) found that spontaneous regression is common, especially for minor prolapse. In addition, in a 5-year follow-up of 160 women with symptomatic POP and 120 women without symptomatic POP, Miedel et al. (2011) found 47% had an unchanged POP-Q stage, 40% showed regression and only 13% showed progression. Thirty per cent had no change in ‘feeling of a vaginal bulge’ and 2% of control women developed symptomatic POP. The authors concluded that only a small proportion of women with symptomatic POP worsen within 5 years.

Treatment of POP can be conservative (lifestyle interventions and/or pelvic floor muscle training [PFMT]), mechanical (use of a pessary) or surgical (Hagen and Stark, 2011). A survey of UK women’s health physical therapists (PTs) showed that many women attending physical therapy practice presented with a mixture of pelvic floor dysfunctions such as stress urinary incontinence (SUI) and prolapse, and that 92% of the PTs assessed and treated women with POP (Hagen et al., 2004). The most commonly used treatment was PFMT with and without biofeedback. A recent Cochrane review on PFMT found four RCTs for PFMT to treat POP and two RCTs on PFMT supplementing surgery versus surgery alone (Hagen and Stark, 2011). They concluded that there was some evidence indicating a positive effect of PFMT for prolapse symptoms and severity of POP, and that there is need for a large study of PFMT supplementing surgery. Furthermore, RCTs involving lifestyle change and prevention of POP are warranted.

**Rationale for PFMT in Prevention and Treatment of POP**

There are two main hypotheses of mechanisms of how PFMT may be effective in prevention and treatment of SUI (Bø, 2004), and the same theories may apply for a possible effect of PFMT to prevent and treat POP. The two hypotheses are: Hypothesis 1: Women learn to consciously contract before and during increases in abdominal pressure (also termed ‘bracing’ or ‘performing the Knack’), and continue to perform such contractions as a behaviour modification to prevent descent of the pelvic floor; and Hypothesis 2: Women are taught to perform regular strength training in order to build up ‘stiffness’ and structural support of the pelvic floor over time (Bø, 2004).

**Conscious contraction (bracing or ‘performing the Knack’) to prevent and treat POP**

Research on basic and functional anatomy supports conscious contraction of the PFM as an effective manoeuvre to stabilize the pelvic floor (Miller et al., 2001; Peschers et al., 2001). However, to date there are no studies on how much strength or what neuromotor control strategies are necessary to prevent descent during cough and other physical exertions, nor how to prevent gradual descent due to activities of daily living or over time. In an RCT comparing PFMT plus lifestyle advice plus the Knack, to lifestyle advice only plus the Knack, Brækken et al. (2010a) found no effect of teaching participants to do the Knack on vaginal resting pressure, PFM strength and endurance, while the PFMT group significantly increased the PFM strength by 13 cmH₂O, an effect size of 1.2, and the effect size in change of muscle endurance in favour of the PFMT group was 0.9. There was no assessment of adherence to the Knack protocol so it is still unknown whether the women actually performed this manoeuvre during the 6 months training period. An interesting but difficult research question to test...
is whether women at risk for POP can prevent development of prolapse by performing the Knack during increases in intra-abdominal pressure. Since it is possible to learn to hold a hand over one’s mouth before and during coughing, it is perhaps possible to learn to precontract the PFM before and during simple and single tasks such as coughing, lifting and isolated exercises such as performing abdominal exercises. However, it is unlikely that multiple task activities and repetitive movements such as running, playing tennis, aerobics and dance activities can be conducted simultaneously with intentional co-contractions of the PFM.

**Strength training**

The theoretical rationale for intensive strength training (exercise) of the PFM to treat POP is that strength training may build up the structural support of the pelvic floor by elevating the levator plate to a permanently higher location inside the pelvis and by enhancing hypertrophy and stiffness of the PFM and connective tissue (Fig. 7.10). This would facilitate a more effective automatic motor unit firing, thus preventing descent during increases in abdominal pressure. The training may also lift the pelvic floor and thereby the protruding organs in a cranial direction. The levator hiatus may narrow and the pelvic organs may be held in place during increase in intra-abdominal pressure (Bø, 2004). Brækken et al. (2010a) showed that the PFMT group had a statistically significant increased thickness of the PFM, elevation of the bladder neck and rectal ampulla and decrease of the levator hiatus area and length of the PFM compared to the control group.

The aim of this section is to provide an updated systematic review of RCTs on PFMT to prevent and treat POP.

### EVIDENCE FOR PFMT IN THE PREVENTION AND TREATMENT OF POP

#### Research methods

The basis for this review included searches on Cochrane, PubMed, PEDro and the abstract books from International Continence Society and International Urogynecology Annual Meetings from 2000 to 2012 for randomized controlled trials (RCTs) on PFMT to prevent or treat POP. Methodological quality of the studies is classified according to the PEDro scoring system (Maher et al., 2003).

#### Results

No RCTs or studies using other designs have been found to evaluate the effect of PFMT on POP in primary prevention, i.e. to stop prolapse from developing (Moore et al., 2013). Table 7.20 shows the eight RCTs assessing PFMT to treat POP or POP symptoms. One study compared PFMT with no treatment (Ghroubi et al., 2008) while Piya-Anant et al. (2003) compared a combination of PFMT and advice to drink water and eat vegetables to reduce constipation and straining on stool and compared this with no treatment. Typically most RCTs compared PFMT plus lifestyle intervention, against lifestyle interventions alone. Lifestyle intervention included use of precontraction of the PFM before and during increase in intra-abdominal pressure (the Knack) and advice to avoid pushing down during defecation (Brækken et al., 2010a, 2010b) or general lifestyle advice (Hagen et al., 2009; Hagen et al., 2011; Frawley et al., 2012). None has compared the effect of these lifestyle interventions with untreated controls and there is no report of adherence to these protocols. Hence, the effect of lifestyle interventions on POP is still unknown. Brækken et al. (2010a) did not find any effect of advice to use the Knack on muscle morphology.

The RCTs are all in favour of PFMT to be effective in treating POP, demonstrating statistically significant improvement in symptoms (Ghroubi et al., 2008; Hagen et al., 2009; Brækken et al., 2010b; Stupp et al., 2011; Hagen et al., 2011; Frawley et al., 2012; Kashyap et al., 2013) and/or prolapse stage (Piya-Anant et al., 2003; Hagen et al., 2009; Brækken et al., 2010b; Stupp et al., 2011;
| Table 7.20  RCTs on PFMT to treat pelvic organ prolapse |
|-----------------|-------------------|----------------|-----------------|
| **Author**      | Piya-Anant et al., 2003 |
| **Design**      | RCT                |
| **Study population** | 654 women >60 years in Thailand. Anterior vaginal wall POP |
| **Intervention** | PFMT: 2 years of 30 contractions per day + eat more fruit and vegetables and drink 2L of water per day |
| **Adherence/Drop-out** | Control: no intervention, same follow-up |
| **Outcome measures** | Adherence: not reported |
| **Results**     | No report of how many drank water and ate more vegetables |
| **Design**      | Ghroubi et al., 2008 |
| **Study population** | 47 women from Tunisia, mean age 53.4 (SD 11), stages I and II anterior vaginal wall POP |
| **Intervention** | PFMT: 2 times per week for 5 weeks with individual PFMT + advice on healthy living by PT; home training 20 contractions per day for 7 weeks |
| **Adherence/Drop-out** | Control: no treatment |
| **Outcome measures** | Drop-out: 0 |
| **Results**     | Adherence: not reported |
| **Design**      | Hagen et al., 2009 |
| **Study population** | 47 women, mean age 56 years (SD 9) with symptomatic stages I and II POP in UK, all kinds of POP |
| **Intervention** | PFMT for 16 weeks, 5 visits with PT. Home exercise: 6 sets of max 10 contractions per day, use of diary + lifestyle advice sheet |
| **Adherence/Drop-out** | Control: no treatment |
| **Outcome measures** | Drop-out not reported. POP-Q data missing for 27/47 |
| **Results**     | 91% attended at least 3 PT sessions, 65% attended 5 visits. 61% rated as good/moderate compliers |
| **Design**      | Brækken et al., 2010a, 2010b |
| **Study population** | 109 women, mean age 48.8 years (SD 11.8), mean BMI 25.6 (SD 4.5), mean parity 2.4 (0.7) with POP-Q stages I, II and III. All kinds of POP |
| **Intervention** | PFMT: information on not to strain on toilet + ‘the Knack’; 3 sets of 8–12 contractions per day, diary; weekly visits with PT for 3 months, every second week for 3 months |
| **Adherence/Drop-out** | Control: information on not to strain on toilet; ‘the Knack’ |

(Continued)
Adherence/Drop-out

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<td>Hagen et al., 2011</td>
<td>447 women, mean age 56.8 years (SD 11.5). Symptomatic POP-Q stage I, II, III</td>
<td>PFMT (n = 224): 5 appointments with PT over 16 weeks + home exercise (see Hagen, 2009) and lifestyle advice</td>
<td>One drop-out in each group. 79% adhered to ≥80% of exercise sessions</td>
<td>POP-Q stage: 11 (19%) in the PFMT vs 4 (8%) controls improved one stage (p = 0.04)</td>
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<td>Control (n = 222): lifestyle advice</td>
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<td>Elevation of the bladder neck: ↑ 2.3 mm vs ↓ 0.6 mm; diff. 3.0 mm (95% CI: 1.5–4.4), p &lt; 0.001</td>
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<td>ICIQ UI-SF: Effect size 0.66 in favour of PFMT, diff. 2.63 (95% CI: 0.95–4.30), p &lt; 0.01</td>
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<td>Group 1 (n = 21): PFMT 7 visits with PT. 14 weeks training period. Use of quick pull of a vaginal cone and stretch reflex followed by active PFM contraction. Use of Knack during different tasks. Home exercise: 3 sets of 8–12 maximum voluntary contractions held for 6–10 seconds. PTs called patients every fortnight. Global stretching and lifestyle: weight loss, fluid intake, constipation, avoidance of heavy lifting</td>
<td>Drop-out at 6 months: PFMT – 16%; Control – 14%.</td>
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<td>Group 2 (n = 16): Control group taught how to perform PFM contractions with no protocol. Same lifestyle and global stretching as PFMT group</td>
<td>Adherence: 80% attended 4 or 5 PT sessions</td>
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Quality of the intervention: dose–response issues

There was some variation in the content of the interventions, but overall it appears that the interventions seem to be of better quality in PFMT for POP compared to interventions for SUI. The study from Ghroubi et al. (2008) was in the French language and only provided the abstract in English. Two RCTs have been published only as abstracts to date and contain limited information regarding the intervention (Hagen et al., 2011; Frawley et al., 2012). All studies used PFMT and all research groups included assessment to determine correctness of PFM contractions. The training period varied between 14 weeks and 2 years, and number of visits with the PT varied between 4 and 18 times. Pelvic floor muscle training was taught individually in all trials and was combined with a home training programme. Drop-outs were low and adherence high in all studies. The highest number of visits with the PT was in the study of Brækken et al. (2010a, 2010b), which included 18 visits over 6 months. This study had high adherence and only two drop-outs and showed the best overall results in change in POP stage and symptom reduction. Interestingly, an RCT from India compared 6 months’ training using a self-instruction manual only with the instruction manual plus one-to-one PFMT for the patients as well, and

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<td><strong>Outcome measures</strong></td>
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<td><strong>Results</strong></td>
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<td><strong>Adherence/Drop-out</strong></td>
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<td><strong>Outcome measures</strong></td>
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<td><strong>Results</strong></td>
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</table>

For abbreviations, see text.
showed the same overall beneficial results in symptoms and POP-Q stage (Kashyap et al., 2013). Because of the use of different outcome measures in POP symptoms it is difficult to compare results based on dose–response issues. No studies compared different training dosages.

**Hypopressive technique**

‘The hypopressive technique’ is a technique taught by physical therapists in France, Italy, Spain and Brazil. The technique was developed by Caufriez (1997) and involves a combination of a breathing technique and contraction of the abdominal muscles which has shown to pull the prolapse upwards/inwards. Resende et al. (2012) assessed 36 nulliparous PTs with vaginal surface EMG during PFM contraction, hypopressive technique and a combination of the two. They found that PFM contraction was more effective than the hypopressive technique to increase sEMG activation of the PFM and that there were no additional effects from adding the hypopressive technique. The hypopressive technique was significantly more effective than PFM contraction in activation of the transverse abdominal muscle.

In an RCT, Stupp et al. (2011) found that both PFMT and PFMT plus the hypopressive technique were significantly more effective than lifestyle advice in increasing muscle strength (Oxford grading) and muscle activation (sEMG), but there was no additional effect of adding the hypopressive technique to PFMT. Ultrasound assessment of the cross-sectional area (CSA) of the levator ani muscle showed increased CSA in the PFMT and the PFMT plus the hypopressive technique compared to the lifestyle group, but there was no additional effect of the hypopressive technique on CSA (Bernardes et al., 2012).

**Should PFMT be an adjunct to prolapse surgery?**

Surgery for POP is common, with a lifetime risk of undergoing a single operation for either prolapse or incontinence by age 80 ranging from 11.1% (Olsen et al., 1997) to 20% (Smith et al., 2013).
2010). As POP surgery is so prevalent and results not always satisfactory, a few studies have investigated the effect of adding PFMT to surgery, to test additional treatment benefits. Jarvis et al. (2004) studied the effect of PFMT and bladder/bowel training on women undergoing surgery for POP/UI, with an RCT of 60 women. The number of women undergoing POP-only surgery was not specified. Thirty women were randomized to each of the treatment and control groups. The intervention consisted of PFMT, functional bracing of PFM prior to rises in abdominal pressure, bladder/bowel training and advice to reduce straining during voiding and defecation. Significant improvements in bladder quality of life and urinary symptom-specific scores were found in the treatment group. Subjects in the treatment group also demonstrated an increase in digital palpation score and maximum vaginal squeeze pressure compared with subjects in the control group, who showed a decrease in squeeze pressure.

In an assessor-blinded RCT comparing the effect of POP surgery with and without a structured physical therapy programme, Frawley et al. (2010) did not find a significant effect of PFMT at 1 year follow-up after surgery. The physical therapy intervention comprised a PFMT strength training protocol, supplemented by bladder and bowel advice. This was provided over eight sessions: one preoperative and seven postoperative sessions; day 3 postoperatively, week 6, 7, 8, 10 and 12, and a final appointment at 9 months postoperatively. Further trials are currently under way, which will hopefully provide a more definitive answer on this intervention (Barber et al., 2009).

CONCLUSION

There is now level 1, grade A evidence that PFMT is effective in treatment of POP. There are no studies in primary prevention or lifestyle intervention, and there is a need for further RCTs to investigate the effect of PFMT in combination with POP surgery on POP symptoms and stage of prolapse. Morphological changes have been found after PFMT, which may indicate a possible preventive effect. There are no long-term studies on the effectiveness of PFMT for POP, but such studies would be difficult to perform as women are frequently offered other treatments, e.g. surgery, after cessation of the PFMT intervention. One can assume that PFMT must be continued to maintain short-term results (Bø and Aschehoug, 2007).

CLINICAL RECOMMENDATIONS

- Watchful waiting is recommended for POP stage I to III if symptom bother is absent or low, as POP stage and symptoms can both regress.
- There are convincing results from eight RCTs that PFMT is effective in reducing symptoms of POP and stage of POP in women who have not had previous POP surgery, therefore PFMT should be first-line treatment for women presenting with symptoms of POP.
- Given the high incidence of de novo (new compartment) or recurrent POP after POP surgery and complications after POP surgery, PFMT may be considered as an adjunct to surgery for POP, as the treatment does no harm and may improve results.
- PFMT for POP patients requires proper teaching, assessment and feedback of correct contraction (vaginal palpation).
- PFMT must be supervised in addition to a home training programme.

REFERENCES


Evidence-Based Physical Therapy for the Pelvic Floor


7.5 Female sexual dysfunction

**Assessment**

Alessandra Graziottin

**INTRODUCTION**

Women’s sexuality has only recently emerged as a central concern after years of neglect in the medical world. The current challenge is to blend together the biological, psychosexual and context-related components of women’s sexual response in a comprehensive and meaningful scenario (Basson et al., 2000, 2004; Banner et al., 2006; Dennerstein et al., 2006, 2007; Graziottin, 2006a, 2006b, 2006c; Graziotti et al., 2006; Whipple and Graziotti, 2006; Graziottin, 2007; Graziotti and Rovei, 2007; Bertolasi et al., 2008; Handa et al., 2008; Frasson et al., 2009; Graziotti, 2009; Knoepp et al., 2010; Graziotti, 2011; Graziotti and Serafini, 2011; Faubion et al., 2012; Graziotti and Serafini, 2012; Clayton and Groth, 2013; Fashokun et al., 2013; Graziotti et al., 2013; Salonia et al., 2013; Graziotti, 2014a, 2014b; Graziotti and Gambini, 2014; Lukasiewicz and Graziotti, 2014). In this perspective, the role of pelvic floor function and dysfunctions is of the highest importance (Alvarez and Rockwell, 2002; Bourcier et al., 2004; Graziotti, 2001a, 2005, 2007; Knoepp et al., 2010; Graziotti and Murina, 2011; Faubion et al., 2012; Fashokun et al., 2013).

Levator ani’s tone, strength and performance is a major contributor to vaginal receptivity, vaginal responsiveness, coital competence and pleasure (for both partners), and for the orgasmic muscular response (Graziotti, 2007; Knoepp et al., 2010; Faubion et al., 2012; Fashokun et al., 2013). Indirectly, pelvic floor disorders (PFD) may impair genital arousal and, through a negative feedback, may affect the potential for physical and emotional satisfaction, and for sexual desire and mental arousal, thus potentially affecting the whole of a woman’s sexual response, particularly when coital pain is a disruptive factor (Fig. 7.11) (Graziotti, 2000, 2001a, 2004; Graziotti and Murina, 2011; Salonia et al., 2013; Lukasiewicz and Graziotti, 2014).

Hyperactivity of the pelvic floor is causally associated with sexual pain disorders, namely dyspareunia and vaginismus (Glazer et al., 1995; Graziotti, 2005; Graziotti et al., 2004a, Harlow et al., 2001; Harlow and Stewart, 2003; Lamont, 1978; McKay et al., 2001; Graziotti and Murina, 2011; Faubion et al., 2012; Fashokun et al., 2013) and overexertion of the pelvic floor muscles (PFM) may lead to myalgia and ’Kegel’ dyspareunia (DeLancey et al., 1993).

The pelvic floor is central in understanding how physiological events such as vaginal deliveries may modulate levator ani’s sexual competence in a lifespan perspective (Bassler and Schuessler, 2004; Glazener, 1997). Pelvic floor disorders are a common denominator in urogenital, proctological and sexual comorbidities (Barlow et al., 1997; Cardozo et al., 1998; Lauman et al., 1999; Weiss, 2001; Graziotti, 2004; Peters et al., 2007; Faubion et al., 2012; Clayton and Groth, 2013; Fashokun et al., 2013). Iatrogenic problems, consequent to urogenital surgery, may in parallel affect and impair both a woman’s well-being and sexual response (Graziotti, 2001b, 2006c).

Increasing evidence stresses the role of genital inflammation as a key aetiological factor of pelvic pain, sexual pain disorders and associated comorbidities, on one hand, and of neuroinflammation, depression and sickness behaviour on the other (Graziotti, 2009; Graziotti et al., 2013).

**Figure 7.11** Circular model describing female sexual function and the interfering role of sexual pain disorders. This model contributes to the understanding of frequent overlapping of sexual symptoms reported in clinical practice (comorbidity) because different dimensions of sexual response are correlated from a pathophysiological point of view. Potential negative or positive feedback mechanisms operate in sexual function: dyspareunia and/or vaginismus has a direct inhibiting effect on genital arousal/lubrication and vaginal receptivity and may have an indirect inhibiting effect on orgasm, satisfaction and libido, with close interplay between biological and psychosexual factors. Pelvic floor disorders of the hyperactive type causally related to sexual pain disorders may rapidly affect the sexual response. The model also includes mood as a key modulator of desire and central arousal. Mood is affected by pain through direct and indirect mechanisms. Neuroinflammation is the most powerful biological pain-associated factor that causes mood impairment up to a frank depression. It is also currently recognized as a key causative factor in the so-called ‘sickness behaviour’. *(Modified from Graziotti 2000, with permission.)*
Evidence-Based Physical Therapy for the Pelvic Floor

Last, but not least, new insights into the role of the hyperactivity of the pelvic floor in adolescence and, possibly, infancy, as predictors of vulnerability to further sexual pain disorders (vaginismus and dyspareunia) and to vulvar vestibulitis/provoked vestibulodynia/vulvodynia open a new preventive window for female sexual dysfunctions (FSD) (Harlow et al., 2001; Chiocchia and Graziottin, 2004; Graziottin, 2005; Peters et al., 2007; Graziottin and Murina, 2011). Appropriate management of early hyperactivity of the pelvic floor could hopefully prevent the urogenital and sexual comorbidities that affect so many young lives (Peters et al., 2007; Salonia et al., 2013).

In this book, dedicated to physical therapy for the pelvic floor, FSD is reviewed paying special attention to the genital components of women's sexual response in physiological and pathological conditions. However, the role of the biological and medical factors should always be considered in the appropriate psychosexual and sociocultural context.

THE COMPLEXITY OF WOMEN’S SEXUALITY

Women’s sexuality is multifactorial, rooted in biological, psychosexual and context-related factors (Dennerstein et al., 1999; Basson et al., 2000, 2004; Binik et al., 2002; Levin, 2002; Graziottin, 2004; Leiblum and Rosen, 2000; Klausmann, 2002; Plaut et al., 2004; Segraves and Balon, 2003a; Banner et al., 2006; Dennerstein et al., 2006, 2007; Graziottin, 2006a, 2006b, 2006c; Graziottin et al., 2006; Whipple and Graziottin, 2006; Graziottin, 2007; Graziottin and Rovei, 2007; Bertolasi et al., 2008; Handa et al., 2008; Frasson et al., 2009; Graziottin, 2009; Knoepp et al., 2010; Graziottin, 2011; Graziottin and Serafini, 2011; Faubion et al., 2012; Graziottin and Serafini, 2012; Clayton and Groth, 2013; Fashokun et al., 2013; Graziottin et al., 2013; Salonia et al., 2013; Graziottin, 2014a, 2014b; Graziottin and Gambini, 2014; Lukasiewicz and Graziottin, 2014), correlated to couple dynamics and family and sociocultural issues (Fig. 7.12). It is multisystemic: in men and women, a physiologic response requires the integrity of the hormonal, vascular, nervous, muscular, connective and immune systems; this fact has been too often overlooked in women until recently (Goldstein and Berman, 1998; Graziottin, 2000; Meston and Frolich, 2000; Bachmann et al., 2002; Levin, 2002; Graziottin and Brozzo, 2004; O’Connell et al., 2004; Handa et al., 2008; Knoepp et al., 2010; Graziottin, 2011; Graziottin and Serafini, 2011; Faubion et al., 2012; Graziottin and Serafini, 2012; Fashokun et al., 2013; Graziottin et al., 2013; Salonia et al., 2013; Graziottin, 2014a, 2014b; Graziottin and Gambini, 2014; Lukasiewicz and Graziottin, 2014).

Three major dimensions – female sexual identity, sexual function and sexual relationship – interact to give women’s sexual health its full meaning or its problematic profile (Graziottin, 2000, 2004; Graziottin and Basson, 2004; Banner et al., 2006; Dennerstein et al., 2006, 2007; Graziottin, 2014b). Women’s sexuality is discontinuous throughout the life cycle and is dependent on biological (reproductive events) as well as personal, current contextual and relationship variables (Basson et al., 2000, 2004; Fashokun et al., 2013; Graziottin et al., 2013; Graziottin, 2014a, 2014b; Lukasiewicz and Graziottin, 2014).

FSD is age-related, progressive and highly prevalent, affecting up to 20–43% of premenopausal women (Lauman et al., 1999), and 48% of older women who are still sexually active in the late postmenopause (Dennerstein et al., 2003, 2007; Graziottin and Koochaki, 2003; Graziottin et al., 2009a).

FSD may occur along a continuum from dissatisfaction (with potential integrity of the physiological response but emotional/affective frustration) to dysfunction (with or without pathological modifications), to severe pathology (Basson et al., 2000, 2004). Pelvic floor disorders are among the most important and yet neglected medical contributors to FSD (Graziottin, 2001a, 2005; Peters et al., 2007; Faubion et al., 2012; Fashokun et al., 2013). However, sexual dissatisfaction, disinterest and even dysfunction may be appropriate for an ‘antisexual’ context (e.g. a partner affected by male sexual disorder or abusive) and they should not be labelled per se as ‘diseases’ or dysfunctions requiring medical treatment (Bancroft et al., 2003).

FSD may occur with or without significant personal (and interpersonal) distress (Bancroft et al., 2003; Graziottin and Koochaki, 2003; Banner et al., 2006; Dennerstein et al., 2006, 2007; Graziottin, 2006a, 2006b, 2006c; Graziottin et al., 2006; Whipple and Graziottin, 2006; Graziottin, 2007; Graziottin and Rovei, 2007; Knoepp et al., 2010; Graziottin et al., 2013). Sociocultural factors may further modulate the perception, expression and complaining

![Figure 7.12 Leading aetiologies of female sexual dysfunctions.](www.fsdeducation.eu)
modality (i.e. the ‘wording’) of a sexual disorder. The meaning of sexual intimacy is a strong modulator of the sexual response and of the quality of satisfaction a woman experiences besides the simple adequacy of the physical response (Kaplan, 1979; Klausmann, 2002; Levine, 2003; Basson, 2003; Plaut et al., 2004). The quality of feelings for the partner and the partner’s health and sexual problems may further contribute to FSD (Dennerstein et al., 2003; Graziottin and Althof, 2011; Martin-Morales et al., 2011).

Sexual problems reported by women are not discrete and often co-occur, comorbidity being one of the leading characteristics of FSD (Basson et al., 2000, 2004; Peters et al., 2007; Graziottin et al., 2013; Salonia et al., 2013).

Comorbidity between FSD and medical conditions (e.g. urological, gynaecological, proctological, metabolic, cardiovascular and neurological) is increasingly recognized (Graziottin, 2000, 2004; Peters et al., 2007; Knoepp et al., 2010; Graziottin et al., 2013; Salonia et al., 2013). For example, latent classes analysis of sexual dysfunctions by risk factors in women indicate that urinary tract symptoms have a RR = 4.02 (2.75–5.89) of being associated with arousal disorders and a RR = 7.61 (4.06–14.26) of being associated with sexual pain disorders according to the epidemiological survey of Laumann et al. (1999), credited as being the best survey carried out so far. The attention dedicated to pelvic-floor-related comorbidities – both between FSD and between FSD and medical conditions – in this contribution reflects the clinical relevance of this association, especially in the urogynaecological (Peters et al., 2007; Salonia et al., 2013) and proctological domain (Handa et al., 2008; Faubion et al., 2012).

**CLASSIFICATION OF FSD**

Over the past decades, the classification of FSD has undergone intense scrutiny and revisions that mirror the new understanding of its complex aetiology. Until a decade ago, the classification of FSD, which constitutes the frame of reference for an appropriate diagnosis, was focused almost entirely on its psychological and relational components. Indeed, FSD was included in the broader manual of ‘psychiatric’ disorders (American Psychiatric Association, 1987, 2000, 2013). The first and second consensus conferences on FSD (Basson et al., 2000, 2004) set out to define FSD with special attention to bringing together the current level of evidence with definitions to fit women’s wording and experiences. The latest classification of the consensus conference on FSD is shown in **Box 7.4**.

**Clinical history**

For a more accurate definition of the sexual symptoms, healthcare providers should also briefly investigate the so-called ‘descriptors’ of the disorders, as defined by the International Consensus Conferences held in 1998 and 2003 (Basson et al., 2000, 2004, and well described in the different chapters of the *Standard Practice in Sexual Medicine Book*, 2006 (Banner et al., 2006; Dennerstein et al., 2006; Giraldi and Graziottin, 2006; Graziottin et al., 2006; Whipple and Graziottin, 2006). They include the following.

**The aetiology of the disorder**

The aetiology of the disorder is further detailed in predisposing, precipitating and maintaining factors (Box 7.5) (Graziottin, 2003a, 2003b; Graziottin and Brotto, 2004; Graziottin and Leiblum, 2005). Each category includes biological, psychosexual and contextual causes (Banner et al., 2006; Dennerstein et al., 2006; Giraldi and Graziotti, 2006; Graziottin, 2006c; Graziottin et al., 2006; Whipple and Graziottin, 2006; Clayton and Groth, 2013; Salonia et al., 2013).

**Biological descriptors** include hormonal dysfunctions, PFDs, cardiovascular problems, neurological conditions (particularly pain-related) (Binik et al., 2002; Binik, 2005; Gruenwald et al., 2007), metabolic disorders (diabetes mellitus [Pontioli et al., 2013]) and affective disorders (depression and anxiety [Graziottin et al., 2013]). All the medical conditions that may directly or indirectly affect sexuality through their multisystemic impact and/or the consequences of pharmacological, surgical and/or radiotherapy treatment should be considered in the differential diagnosis of potential contributors to the reported FSD (Graziottin, 2006b; Lukasiewicz and Graziottin, 2014). Loss of sexual hormones, consequent to natural or iatrogenic menopause, is a major contributor to FSD (Dennerstein et al., 2003; Graziottin, 2010). It can be addressed with appropriate hormonal replacement therapy (Graziottin, 2000, 2004; Bachmann et al., 2002; Graziottin and Basson, 2004; Graziottin, 2010). Current medication use and substance abuse should be actively investigated (Segraves and Balon, 2003a; Graziottin and Serafini, 2011).

**Psychosexual descriptors** refer to emotional/affective psychic factors such as negative upbringing/losses/truma (physical, sexual, emotional) (Basson, 2003; Rellini and Meston, 2004), psychological factors associated with chronic pelvic pain (Graziottin, 2011), body image issues (Graziottin et al., 2006), binge eating disorders affecting self-esteem and self-confidence, attachment dynamics (secure, avoidant, anxious) (Clulow, 2001) that may also modulate the level of trust in the relationship, the intensity of the commitment and the confidence in loving and attitude towards affective and erotic intimacy.

**Contextual descriptors** include past and current significant relationships (Leiblum and Rosen, 2000; Basson, 2003), cultural/religious restrictions (Basson et al., 2000, 2004), current interpersonal difficulties (Klausmann, 2002; Liu, 2003), partner’s general health issues and/or
Box 7.4 Classification of female sexual disorders

**Women’s sexual interest/desire disorder**
- Absent or diminished feelings of sexual interest or desire, absent sexual thoughts or fantasies and a lack of responsive desire. Motivations (here defined as reasons/incentives), for attempting to become sexually aroused are scarce or absent. The lack of interest is considered to be more than that due to a normative lessening with the life cycle and duration of a relationship.

**Sexual aversion disorder**
- Extreme anxiety and/or disgust at the anticipation of/or attempt to have any sexual activity.

**Subjective sexual arousal disorder**
- Absence of or markedly diminished cognitive sexual arousal and sexual pleasure from any type of sexual stimulation. Vaginal lubrication or other signs of physical response still occur.

**Genital sexual arousal disorder**
- Complaints of absent or impaired genital sexual arousal. Self-report may include minimal vulvar swelling or vaginal lubrication from any type of sexual stimulation and reduced sexual sensations from caressing genitalia. Subjective sexual excitement still occurs from non-genital sexual stimuli.

**Combined genital and subjective arousal disorder**
- Absence of or markedly diminished subjective sexual excitement and awareness of sexual pleasure from any type of sexual stimulation as well as complaints of absent or impaired genital sexual arousal (vulvar swelling, lubrication).

**Persistent sexual arousal disorder**
- Spontaneous, intrusive and unwanted genital arousal (e.g. tingling, throbbing, pulsating) in the absence of sexual interest and desire. Any awareness of subjective arousal is typically but not invariably unpleasant. The arousal is unrelieved by one or more orgasms and the feelings of arousal persist for hours or days.

**Women’s orgasmic disorder**
- Despite the self-report of high sexual arousal/excitement, there is either lack of orgasm, markedly diminished intensity of orgasmic sensations or marked delay of orgasm from any kind of stimulation.

**Dyspareunia**
- Persistent or recurrent pain with attempted or complete vaginal entry and/or penile vaginal intercourse.

**Vaginismus**
- The persistent or recurrent difficulties of the woman to allow vaginal entry of a penis, a finger, and/or any object, despite the woman’s expressed wish to do so. There is often (phobic) avoidance and anticipation/fear/experience of pain, along with variable involuntary pelvic muscle contraction. Structural or other physical abnormalities must be ruled out/addressed.

From Basson et al., 2004.

Box 7.5 Factors contributing to female sexual dysfunction

**Predisposing factors**

**Biological**
- Anaemia
- Endocrine disorders (hypoandrogenism, hypoestrogenism, hyperprolactinaemia, hypothyroidism)
- Menstrual cycle disorders/premenstrual syndrome
- Recurrent vulvovaginitis and/or cystitis
- Pelvic floor disorders: lifelong or acquired
- Drug treatments affecting hormones or menstrual cycle
- Contraceptive methods inappropriate for the woman and couple in that period of life
- Chronic diseases (diabetes mellitus, cardiovascular, neurological or psychiatric disease, etc.)
- Disorders associated with premature ovarian failure (POF): genetic, autoimmune
- Benign diseases (e.g. endometriosis) predisposing to iatrogenic menopause and dyspareunia
- Iatrogenic menopause: bilateral oophorectomy, chemotherapy, radiotherapy
- Persistent residual conditions (e.g. dyspareunia/chronic pain associated with endometriosis)

**Psychosexual**
- Inadequate/delayed psychosexual development
- Binge eating disorders and restrictive eating disorders
- Previous negative sexual experiences: sexual coercion, violence, or abuse
- Body image issues/concerns
- Affective disorders (dysthymia, depression, anxiety)
- Inadequate coping strategies
- Inadequate sexual education (attitudes towards contraception and sexually transmitted diseases)
- Dissatisfaction with social/professional role(s)
- Borderline personality traits
- Gender dysphoria
## Box 7.5  Factors contributing to female sexual dysfunction—cont’d

<table>
<thead>
<tr>
<th>Contextual</th>
<th>Maintaining factors</th>
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<tbody>
<tr>
<td>• Ethnic/religious/cultural messages, expectations, and constraints regarding sexuality</td>
<td>• Diagnostic omissions: unaddressed predisposing/precipitating biological aetiologies, anaemia first</td>
</tr>
<tr>
<td>• Social ambivalences towards female sexuality, when separated from reproduction or marriage</td>
<td>• Untreated or inadequately treated comorbidities</td>
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<tr>
<td>• Negative social attitudes towards female contraception</td>
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<td>• Low socioeconomic status/reduced access to medical care and facilities</td>
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<td>• Support network</td>
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<td><strong>Precipitating factors</strong></td>
<td><strong>Biological</strong></td>
</tr>
<tr>
<td><strong>Biological</strong></td>
<td>• Diagnostic omissions: unaddressed predisposing/precipitating biological aetiologies, anaemia first</td>
</tr>
<tr>
<td>• Negative reproductive events (unwanted pregnancies, abortion, traumatic delivery with damage of the pelvic floor, child’s problems, infertility)</td>
<td>• Untreated or inadequately treated comorbidities</td>
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<tr>
<td>• Postpartum depression</td>
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<tr>
<td>• Vulvovaginitis/sexually transmitted diseases</td>
<td>• Sexual: sexual aversion disorders, arousal difficulties with vaginal dryness, anorgasmia, introital and/or deep dyspareunia, post-coital cystitis</td>
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<tr>
<td>• Sexual pain disorders</td>
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<tr>
<td>• Age at menopause</td>
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<tr>
<td>• premature ovarian failure (POF) – menopause before age 40</td>
<td>• metabolic: diabetes mellitus</td>
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<tr>
<td>• premature menopause – menopause between age 40 and 45</td>
<td>• psychiatric: depression, anxiety, phobias</td>
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<tr>
<td>• Biological vs iatrogenic menopause (especially for premature menopause)</td>
<td>• Neuroinflammation and pain-related symptoms and disorders</td>
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<tr>
<td>• Iatrogenic menopause</td>
<td>• Pharmacological treatments</td>
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<tr>
<td>• androgen (besides oestrogen) loss</td>
<td>• Substance abuse</td>
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<tr>
<td>• associated disorder/disease</td>
<td>• Multisystemic changes associated with chronic disease or secondary to menopause</td>
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<tr>
<td>• Extent and severity of menopausal symptoms and impact on well-being</td>
<td></td>
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<tr>
<td>• Current medical disorders</td>
<td>• vascular</td>
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<tr>
<td>• Current pharmacological treatment</td>
<td>• muscular</td>
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<tr>
<td>• Substance abuse (mainly alcohol and opiates)</td>
<td>• neurological</td>
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<tr>
<td><strong>Psychosexual</strong></td>
<td>• immunological</td>
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<tr>
<td>• Loss of loving feelings toward partner</td>
<td>• Contraindications to hormone replacement therapy (HRT)</td>
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<tr>
<td>• Unpleasant/humiliating sexual encounters or experiences</td>
<td>• Inadequacy of hormone replacement therapy in ameliorating menopause-associated biological symptoms</td>
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<tr>
<td>• Affective disorders (depression, anxiety)</td>
<td>• Low or loss of sexual self-confidence</td>
</tr>
<tr>
<td>• Relationship of fertility loss to fulfilment of life goals</td>
<td>• Performance anxiety</td>
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<tr>
<td><strong>Contextual/relational</strong></td>
<td>• Distress (personal, emotional, occupational, sexual)</td>
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<tr>
<td>• Relationship discord</td>
<td>• Diminished affection for or attraction to partner</td>
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<td>• Life-stage stressors (e.g. child’s diseases, divorce, separation, partner infidelity)</td>
<td>• Unaddressed affective disorders (depression and/or anxiety)</td>
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<tr>
<td>• Partner’s sexual problems (premature ejaculation and/or erectile deficits)</td>
<td>• Negative perception of menopause-associated changes</td>
</tr>
<tr>
<td>• Loss or death of close friends or family members</td>
<td>• Body image concerns and increased body changes (wrinkles, body shape/weight, muscle tone; invalidating outcomes of disease, surgery or traumas)</td>
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<tr>
<td>• Lack of access to medical/psychosocial treatment and facilities</td>
<td>• Omission of menopause and female sexual dysfunction from provider’s diagnostic and therapeutic approach</td>
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<tr>
<td>• Economic difficulties</td>
<td>• Lack of access to adequate care</td>
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<td></td>
<td>• Partner’s general health or unaddressed sexual problems (premature ejaculaion and/or erectile deficit)</td>
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<td></td>
<td>• Ongoing interpersonal conflict (with partner or others)</td>
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<tr>
<td></td>
<td>• Environmental constraints (lack of privacy, lack of time)</td>
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</tbody>
</table>

Modified from Graziottin, 2007.
sexual dysfunctions (Dennerstein et al., 1999, 2003; Graziottin and Althof, 2011; Martin-Morales et al., 2011), inadequate stimulation and unsatisfactory sexual and emotional contexts (Levine, 2003; Graziottin and Leiblum, 2005).

**Generalized or situational?**

Is the disorder generalized (with every partner and in every situation) or situational, specifically precipitated by partner-related or contextual factors, which should be specified (Basson et al., 2000, 2004; Graziottin and Althof, 2011; Martin-Morales et al., 2011)? Situational problems usually rule out medical factors that tend to affect the sexual response with a more generalized effect (Graziottin, 2004).

**Lifelong or acquired?**

Has the disorder been lifelong (from the very first sexual experience) or is it acquired after months or years of satisfying sexual intercourse? Asking the woman what in her opinion is causing the current FSD may offer useful insights into the aetiology of the disorder, particularly when it is acquired (Plaut et al., 2004; Banner et al., 2006; Dennerstein et al., 2006; Giraldi and Graziottin, 2006; Graziottin, 2006c; Whipple and Graziottin, 2006; Graziottin et al., 2009b; Buster, 2013; Clayton and Groth, 2013).

**Level of distress**

The level of distress indicates a mild, moderate, or severe impact of the FSD on personal life (Bancroft et al., 2003; Graziottin and Koochaki, 2003; Banner et al., 2006; Knoepf et al., 2010; American Psychiatric Association, 2013). Sexual distress should be distinguished from non-sexual distress and from depression. The degree of reported distress may have implications for the woman’s motivation for therapy and for prognosis.

An interdisciplinary team is the most valuable resource for a patient-centred approach, both for diagnostic accuracy and tailored treatment. Key professional figures include a medical sexologist, gynaecologist, urologist, psychiatrist, endocrinologist, physiologist, anaesthetist, neurologist, proctologist, dermatologist, psychotherapist (individual and couple) and physical therapist. Physical therapists are emerging as a key resource in addressing PFDs, which are finally receiving the attention they deserve as key biological factors in the aetiology of FSD.

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### WOMEN’S SEXUAL DESIRE/INTEREST DISORDER

Hypoactive sexual desire disorder (HSDD) is the sexual dysfunction most frequently reported by women (Dennerstein et al., 2003; Graziottin and Koochaki, 2003). The complaint of low desire becomes a sexual disorder when it causes severe personal distress to the woman. Population data indicate a prevalence of low desire in 32% of women between 18 and 59 years of age (Laumann et al., 1999). A European survey of 2467 women, in France, UK, Germany and Italy (Graziottin and Koochaki, 2003) indicated that the percentage of women with low sexual desire was 19% in the age cohort from 20 to 49 years, 32% in the same age cohort in women who had experienced surgical menopause, 46% in postmenopausal women aged 50 to 70 years with natural menopause, and 48% in the same age cohort, after surgical menopause.

The percentage of women distressed by their loss of desire and having a HSDD was 27% in premenopausal women and 28% after surgical menopause, in the age cohort 20–49 years, respectively; 11% in women with natural menopause; and 14% in those with surgical menopause aged 50–70 years (Graziottin and Koochaki, 2003). The likelihood of HSDD increases with age, while the distress associated with the loss of desire is inversely correlated with age.

Surgical menopause secondary to bilateral oophorectomy has a specific damaging effect due to the loss of ovarian oestrogens and androgens (Graziottin, 2010). Ovaries contribute to more than 50% of total body androgens in the fertile age. A European survey on 1356 women indicated that women with surgical menopause had an odds ratio (OR) of 1.4 (CI=1.1, 1.9; p=0.02) of having low desire. Surgically menopausal women were more likely to have HSDD than premenopausal or naturally menopausal women (OR=2.1; CI=1.4, 3.4; p=0.001). Sexual desire scores and sexual arousal, orgasm and sexual pleasure were highly correlated (p <0.001). Women with HSDD were more likely to be dissatisfied with their sex life and their partner relationship than women with normal desire (p <0.001) (Dennerstein et al., 2007; Derogatis et al., 2009; Graziotti et al., 2009a). A well-tailored Hormonal Replacement Therapy programme may address the hormonal contributor of postmenopausal female sexual dysfunctions (Al-Azzawi et al., 2010).

The leading biological aetiology of HSDD includes not only hormonal factors (low testosterone, low oestrogens, high prolactin, or low thyroid hormones), but also anaemia, depression (Graziottin et al., 2013) and/or comorbidity with major diseases (Gruenwald et al., 2007; Clayton and Groth, 2013; Pinto et al., 2013; Salonia et al., 2013; Lukasiwiecz and Graziottin, 2014) (see Box 7.5). Premature iatrogenic menopause is the most frequent cause of a biologically determined generalized loss of desire; the younger the woman, the higher the distress this loss causes to her (Graziottin and Basson, 2004; Graziottin, 2010). Key questions to address women’s desire disorders are summarized in Box 7.6. Unaddressed pain associated with sexual pain disorders, and causally related, among others, to hyperactivity of the pelvic floor up to a frank myalgia, is a frequently overlooked predisposing, precipitating and maintaining factor of acquired loss of desire.
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(Graziottin, 2000; Graziottin, 2004; Graziottin and Brotto, 2004; Handa et al., 2008; Faubion et al., 2012). Figure 7.13 summarizes the diagnostic flowchart in HSDD as a model useful in the clinical practice.

What the clinician should look for

If a possible biological aetiology is suggested by the clinical history, the clinician should assess (Plaut et al., 2004; Graziottin et al., 2009b; Clayton and Groth, 2013) the following:

- **Hormonal profile:**
  - total and free testosterone, dihydroepiandrosterone sulphate (DHEAS), prolactin, 17β-oestradiol, sex hormone binding globulin (SHBG), with a plasma sample on the third or fourth day from the beginning of the menses in fertile women;
  - follicle stimulating hormone (FSH) and all of the above, in perimenopausal women;
  - thyroid stimulating hormone (TSH) when individually indicated.
- **The pelvic floor:** in all its components, with an accurate gynaecological, sexological and/or physiatric examination, particularly when comorbidity with arousal, orgasm and/or sexual pain disorders is reported.
- **Psychosexual factors and affective state:** depression first, with referral to a psychiatrist, sex therapist or couples therapist for a comprehensive diagnosis if indicated (Leiblum and Rosen, 2000; Graziottin and Leiblum, 2005; Clayton and Groth, 2013).

AROUSAL DISORDERS

Central arousal disorders (‘I do not feel mentally excited’) are comorbid with loss of sexual desire; they can be separated from it only with difficulty (Giraldi and Graziottin, 2006). A current trend of classification tends to consider together HSDD and central arousal disorders (Giraldi et al., 2013). Genital arousal disorders, with their key subjective symptom, vaginal dryness, are increasingly reported with age. In epidemiological surveys 19–20% of women complain of arousal disorders (Lauman et al., 1999). This figure may increase to 39–45% in postmenopausal sexually active patients (Dennerstein et al., 2003). Persistent genital arousal disorder (PGAD) is a rare but disabling sexual condition, with different aetiologies and unclear treatment (Facelle et al., 2013).

Mental arousal may be triggered through different pathways: biologically by androgens and oestrogens, psychologically by motivational forces such as intimacy needs (i.e. the affective needs of love, tenderness, attention, bonding and commitment) (Laan and Everaerd, 1995). With successful genital arousal, most women produce increased quantities of vaginal transudate. The neurotransmitter

Box 7.6 Sexual history for hypoactive sexual desire disorders and associated sexual comorbidities

**General well-being**

- How do you feel (physically and mentally)?
- Are you currently sexually active?
- If not, is that a concern for you? If yes, how’s your sex life?

**Sexual function**

- Have you always suffered from low sexual desire (lifelong) or has it faded recently (acquired)?
- Do you suffer from other sexual symptoms?
- For example, do you experience vaginal dryness?
- Do you have difficulty in getting aroused or lubricated?
- Do you have difficulty reaching orgasm?
- Do you feel pain during or after intercourse?
- Do you suffer from cystitis 24–72 hours after intercourse and/or other urinary symptoms?
- Is there any lifestyle-related factor that may affect your sexual desire (e.g. body weight, alcohol or drug abuse, little sleep, fatigue, professional distress)?
- What, in your opinion, is causing or worsening your sexual disorder? Is it a psychological problem, a past or current negative event (e.g. sexual harassment or abuse), something related to your physical health or your relationship, or something else?

**Sexual relationship**

- Do you have a stable relationship?
- How’s your relationship? Are you satisfied with it?
- How is your partner’s health (general and sexual)? Does he suffer from premature ejaculation or erectile deficit?
- Do you feel that your current sexual problem is more dependent on a physical or couple (loving/intimacy) problem?
- Is your sexual problem present in every context and/or with different partners (generalized), or do you complain of it in specific situations or with a specific partner (situational)?
- What made you aware of it and willing to look for help (e.g. intolerable personal frustration, fear of losing the partner, partner’s complaints, new hope for effective treatment, more self-confidence in reporting)?
- Are you personally interested in improving your sex life?

Modified from Graziottin, 2007.
vasoactive intestinal peptide (VIP) stimulates this neurogenic transudate production. Oestrogens are believed to be powerful 'permitting factors' for VIP (Levin, 2002; Graziottin and Gambini, 2014). The neurotransmitter nitric oxide (NO) stimulates the neurogenic congestion of the clitoral and vestibular bulb corpora cavernosa (Levin, 2002). Reduction in vaginal lubrication is one of the most common complaints of postmenopausal women. When the plasma oestradiol concentration is below 50 pg/ml (the normal range in fertile women being 100–200 pg/ml) vaginal dryness is increasingly reported. Physiological studies indicate that after menopause the vaginal pH increases from 3.5–4.5 to 6.0–7.39 owing to decreased glycogen production and metabolism to lactic acid, with dramatic modification of the vaginal ecosystem, and an average reduction of vaginal secretions of 50%.

Leading biological aetiologies of arousal disorders include loss of sexual hormones, primarily oestrogen, and PFDs.

Hyperactivity of the pelvic floor may reduce the introital opening, causing dyspareunia (Graziottin and Murina, 2011). (Unwanted) pain is indeed the strongest reflex inhibitor of genital arousal: genital arousal disorders, and the consequent vaginal dryness, are often comorbid with dyspareunia (Graziottin, 2001a, 2004) and with recurrent cystitis/postcoital cystitis/bladder pain disorders (Peters et al., 2007; Salonia et al., 2013). Psychosexual and relational factors may also concur in the aetiology of introital coital pain (‘introital dyspareunia’) (Box 7.7).

A hypoactive or damaged pelvic floor (after traumatic deliveries, with macrosomic children or vacuum extraction) (Baessler and Schuessler, 2004) may contribute to genital arousal disorder because it reduces the pleasurable sensations the woman (and partner) feel during intercourse (Graziottin et al., 2004a; van Delft et al., 2014).

**What the clinician should look for**

When a patient complains of an arousal disorder, the clinician should check (Plaut et al., 2004; Banner et al., 2006; Dennerstein et al., 2006, 2007; Graziottin, 2006a, 2006b, 2006c; Graziottin et al., 2006; Whipple and Graziottin,
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Box 7.7 Aetiology of dyspareunia

Many causes may overlap or be associated with coital pain with complex pathophysiological interplay. The relative weight of each cause in the individual woman may change with chronicity of pain, progressive involvement of other pelvic organs (pelvic comorbidity), systemic comorbid diseases such as fibromyalgia or chronic fatigue syndrome, and level of associated neuroinflammation, depression and sickness behaviour.

Biological

Superficial/introital and/or mid-vaginal dyspareunia
- Infectious: vulvitis, vaginitis, cystitis
- Inflammatory and pain-related, with mast cells’ upregulation and vulvar vestibulitis/provoked vestibulodynia
- Hormonal: vulvovaginal atrophy
- Anatomical: fibrous hymen, vaginal agenesis
- Muscular: primary or secondary hyperactivity of levator ani muscle
- Iatrogenic: poor outcome of genital surgery, including episiotomy/episiorraphy, pelvic radiotherapy
- Neurological: inclusive of neuropathic pain
- Connective and immunological: lichen sclerosus and Sjögren’s syndrome
- Vascular

Deep dyspareunia
- Endometriosis
- Pelvic inflammatory disease (PID)
- Chronic pelvic pain and referred pain
- Outcome of pelvic or endovaginal radiotherapy
- Abdominal nerve entrapment syndrome
- Pelvic varicocele

Psychosexual
- Comorbidity with desire and/or arousal disorders, or vaginismus
- Past sexual harassment and/or abuse
- Affective disorders: depression and anxiety
- Catastrophism as leading psychological coping modality

Context or couple related
- Lack of emotional intimacy
- Inadequate foreplay
- Conflicts: verbally, physically or sexually abusive partner
- Poor anatomical compatibility (penis size and/or infantile female genitalia)
- Sexual dissatisfaction and consequent inadequate arousal

Modified from Graziottin, 2007.

2006; Al-Azzawi et al., 2010; Graziottin and Murina, 2011; Buster, 2013; Pontiroli et al., 2013) the following:
- hormonal profile (see above), more so in hypoestrogenic conditions such as long-lasting secondary amenorrhoea, puerperium, menopause (especially iatrogenic);
- general and pelvic health, focusing on pelvic floor trophism: vaginal, clitoral, vulvar, connective and muscular (looking for both hypertonic and hypotonic pelvic floor dysfunctions) (Graziottin, 2001a, 2004; Faubion et al., 2012; Fashokun et al., 2013);
- vaginal pH with a simple stick because vaginal acidity correlates well with oestrogen tissue levels (Graziottin, 2004);
- biological factors, such as vulvar vestibulitis or poor outcome of perineal/genital surgery causing introital and/or pelvic pain (see dyspareunia (Graziottin and Murina, 2011);
- vascular factors that may impair the genital arousal response (smoking, hypercholesterolaemia, atherosclerosis, hypertension, diabetes mellitus) (Goldstein and Berman, 1998);
- relational issues, inhibition and/or erotic illiteracy if a poor quality of mental arousal, poor or absent foreplay are reported; if this is so refer the willing couple to the sexual or couple therapist (Leiblum and Rosen, 2000).

ORGASMIC DISORDERS

Anatomic factors can modulate women’s orgasmic potential (Jannini et al., 2012; Oakley et al., 2014). Many physiologic factors still remain underinvestigated (Levin, 2014). Orgasmic disorder has been reported in an average of 24% of women during their fertile years in the epidemiological study of Lauman et al. (1999). During hormonal contraception reduction of the androgenic levels may contribute to orgasmic disorders (Smith et al., 2014) but the finding is controversial in the medical literature.

After the menopause, 39% of women complain of orgasmic difficulties, with 20% complaining that their clitoris ‘is dead’. Topical sexual hormones may partially reverse the complaint (Fernandes et al., 2014). Orgasm is a sensorimotor reflex that may be triggered by a number of physical and mental stimuli.

Genital orgasm requires:
- integrity of the pudendal sensory nerve fibres (S2, S3, S4) and corticomedullary fibres;
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- cavernosal structures that when engorged and adequately stimulated convey pleasant sensory stimuli to the medullary centre and the brain;
- adequate motor response of the PFMs.

A short medullary reflex may trigger a muscular response characterized by involuntary contraction (three to eight times, in single or repetitive sequences) of the levator ani. The medullary reflex may be eased or blocked, respectively, by corticomedullary fibres that convey both excitatory stimuli when central arousal is maximal and inhibitory ones when arousal is poor. Performance anxiety may activate adrenergic input, which disrupts the arousal response. Inhibitory fibres are mostly serotonergic: this explains the inhibitory effects of selective serotonin reuptake inhibitors (SSRIs) on orgasm in both men and women (Seagraves and Balon, 2003; Levin, 2014). Fear of leaking during intercourse may inhibit coital intimacy and/or orgasm (Barlow et al., 1997; Cardozo et al., 1998): leakage during coital thrusting is usually associated with stress incontinence, while leakage at orgasm is associated with urge incontinence.

Significant age-associated changes in the content of smooth muscle and connective tissue in the clitoral cavernosa contributing to age-associated clitoral sexual dysfunction causing hypo-anorgasmia, have been demonstrated from the first to the sixth decade of life and beyond by computer-assisted histomorphometric image analysis (Tarcan et al., 1999).

What the clinician should look for

Using the information emerging from the clinical history as a starting point, the physician should assess (Whipple and Graziotti, 2006; Buster, 2013):

- hormonal balance;
- signs and symptoms of vulvar dystrophy and, specifically, of clitoral and vaginal involution (Graziotti, 2004);
- traumatic consequences of female genital mutilation (infibulation);
- signs and symptoms of urge, stress or mixed incontinence, with either a hypotonic or hypertonic pelvic floor (Barlow et al., 1997; Cardozo et al., 1998);
- iatrogenic influences when potentially orgasm inhibiting drugs are prescribed.

**Sexual Pain Disorders**

Various degrees of dyspareunia are reported by 15% of coitally active women, and 22.5–33% of postmenopausal women. Vaginismus occurs in 0.5–1% of premenopausal women. However, mild hyperactivity of the pelvic floor, that could coincide with grade I or II of vaginismus according to Lamont (1978), may permit intercourse, causing coital pain (Graziotti, 2003b, 2005; Graziotti and Murina, 2011).

Vaginal receptiveness is a prerequisite for intercourse, and requires anatomical and functional tissue integrity, both in resting and aroused states. Normal trophism, both mucosal and cutaneous, adequate hormonal impregnation, lack of inflammation, particularly at the introitus, normal tonicity of the perivaginal muscles, vascular, connective and neurological integrity, and normal immune response are all considered necessary to guarantee vaginal ‘habitability’. Vaginal receptiveness may be modulated by psychosexual, mental and interpersonal factors, all of which may result in poor arousal with vaginal dryness (Plaut et al., 2004; Graziotti, 2006a; Graziotti and Murina, 2011).

Fear of penetration, and a general muscular arousal secondary to anxiety, may cause a defensive contraction of the perivaginal muscles leading to vaginismus (Reissing et al., 2003, 2004; van der Velde et al., 2001). This disorder may be the clinical correlate of a primary neurodystonia of the pelvic floor, as recently proven with needle electromyography (Graziotti et al., 2004a; Bertolasi et al., 2008). It may be so severe as to prevent penetration completely. Vaginismus is the leading cause of un Consummated marriages in women. The defensive pelvic floor contraction may also be secondary to genital pain of whatever cause (Graziotti, 2006a, 2006b; Graziotti and Murina, 2011).

Dyspareunia is the common symptom of a variety of coital pain-causing disorders (see Box 7.7). Vulvar vestibulitis/provoked vestibulodynia is its leading cause in premenopausal women (Friedrich, 1987; Glazer et al., 1995; Graziotti, 2001a; Graziotti and Brotto, 2004; Graziotti et al., 2004b; Heddini et al., 2012). The diagnostic triad is:

1. Severe pain upon vestibular touch or attempted vaginal entry.
2. Exquisite tenderness to cotton-swab palpation of the introital area (mostly at 5 and 7, when looking at the introitus as a clock-face).
3. Dyspareunia (Friedrich, 1987).

From the pathophysiological point of view, vulvar vestibulitis/provoked vestibulodynia involves the upregulation of:

- the immunological system (i.e. of introital mast cells with hyperproduction of both inflammatory molecules and nerve growth factors [NGF]) (Bohm-Starke et al., 1999, 2001a, 2001b; Bornstein et al., 2002, 2004; Graziotti and Murina, 2011);
- the pain system, with proliferation of local pain fibres induced by the NGF (Bornstein et al., 2002, 2004), which may contribute to neuropathic pain (Graziotti and Brotto, 2004) and neuroinflammation, is a key
contributor to comorbid depression (Graziottin et al., 2013);

- hyperactivity of the levator ani, which can be antecedent to vulvar vestibulitis (Graziottin et al., 2004; Graziottin, 2005), or secondary to the introital pain (Graziottin and Murina, 2011).

In either case, addressing the muscle component is a key part of treatment (Glazer et al., 1995; Bergeron et al., 2001; McKay et al., 2001; Graziottin and Murina, 2011). Hyperactivity of the pelvic floor may be triggered by non-genital, non-sexual causes, such as urological factors (urge incontinence, when tightening the pelvic floor may be secondary to the aim of reinforcing the ability to control the bladder (Salonia et al., 2013), or anorectal problems (anismus, haemorrhoids, rhagades) (Faubion et al., 2012; Fashokun et al., 2013). Comorbidity with other sexual dysfunctions – loss of libido, arousal disorders, orgasmic difficulties, and/or sexual pain-related disorders – is frequently reported with persisting/chronic dyspareunia.

What the clinician should look for

The diagnostic work-up should focus on (Graziottin, 2006a; Graziottin and Murina, 2011):

- physical examination to define the ‘pain map’ (Graziottin, 2001a; Graziottin and Basson, 2004; Graziottin and Murina, 2011) (any site in the vulva, mid-vagina and deep vagina where pain can be elicited) because location of the pain and its characteristics are the strongest predictors of type of organicity, and including pelvic floor trophism (vaginal pH), muscular tone, strength and performance (Alvarez and Rockwell, 2002; Bourcier et al., 2004), signs of inflammation (primarily vulvar vestibulitis) (Friedrich, 1987; Graziottin and Brotto, 2004; Graziottin and Murina, 2011), poor outcomes of pelvic (Graziottin, 2001b) or perineal surgery (primarily episiotomy/episiorraphy), postpartum sexual pain, which is still a very neglected area, deserving the highest medical attention (Glazener, 1997; Graziottin, 2006c), associated urogenital (Peters et al., 2007; Salonia et al., 2013) and rectal pain syndromes, myogenic or neurogenic pain (Bohm-Starke, 2001a, 2001b; Bornstein et al., 2002, 2004) and vascular problems (Goldstein and Berman, 1998; Pontiroli et al., 2013);

- psychosexual factors, poor arousal and coexisting vaginismus (Leiblum, 2000; Pukall et al., 2005; Frasson et al., 2009);

- relationship issues (Reissing et al., 2003; Smith et al., 2013);

- hormonal profile, if clinically indicated, when dyspareunia is associated with vaginal dryness (Al-Azzawi et al., 2010).

Pain is rarely purely psychogenic, and dyspareunia is no exception. Like all pain syndromes, it usually has one or more biological aetiological factors. Hyperactive PFDs are a constant feature. However, psychosexual and relationship factors, generally lifelong or acquired low libido because of the persisting pain, and lifelong or acquired arousal disorders due to the inhibitory effect of pain, should be addressed in parallel to provide comprehensive, integrated and effective treatment.

ETHICAL, LEGAL AND COUNSELLING RELATED CONSIDERATIONS

The topic of sexuality requires special attention to confidentiality and informed consent depending on the profession of the clinician and any local laws that place limits on confidentiality, such as in the reporting of sexual abuse. Although the discussion of sexual matters is often an appropriate part of medical evaluation and treatment, it is also important not to sexualize the clinical setting when it is not necessary. Patients may be confused or embarrassed by comments about their attractiveness, disclosure of intimate personal information by the clinician, or by sex-related questions that are neither clinically relevant nor justifiable. The modesty of the patient should be respected in touching, disrobing and draping procedures (Plaut et al., 2004). Key aspects of appropriate counselling attitudes are summarized in Box 7.8.

Box 7.8 Talking with patients about sexual issues

- Ask pointed questions and request clarification that will result in sufficiently specific data about the patient’s symptoms

- Be sensitive to the optimal time to ask the most emotionally charged questions

- Look for and respond to non-verbal cues that may signal discomfort or concern

- Be sensitive to the impact of emotionally charged words (e.g. rape, abortion)

- If you are not sure of the patient’s sexual orientation, use gender-neutral language in referring to his or her partner

- Explain and justify your questions and procedures

- Teach and reassure as you examine

- Intervene to the extent that you are qualified and comfortable; refer to qualified medical or mental health specialists as necessary

From Plaut et al., 2004, with permission.
CONCLUSION

To address the complexity of FSD requires a balanced clinical perspective between biological and psychosexual/relational factors. Apart from counselling the FSD complaint in a competent way where the issue is openly raised by the patient, physicians and physical therapists can contribute to improving the quality of (sexual) life of their patients by routinely asking them during the clinical history taking: ‘How’s your sex life?’ so offering an opening for current or future disclosure. The wish is that the new attention to women’s right for a better sexual life will significantly help increase the physician’s confidence in asking and listening to complaints of FSD and his or her ‘clinical impact factor’ (i.e. his or her ability to appropriately diagnose and effectively treat FSD).

In the tailoring of treatment, the physical therapist has a crucial role, especially in sexual pain disorders, either lifelong or acquired, and in acquired desire, arousal or orgasmic disorders secondary to coital pain. The enthusiasm that many physical therapists have when they can effectively treat or co-treat FSD for which a woman has been doctor-shopping for years are mirrored by the woman’s satisfaction in finally feeling listened to, respected in the truth of her coital pain or other sexual complaints, and re-empowered in her body confidence, when she is taught how to command and appropriately relax her key muscles for sex and love.

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dysfunction in the United States: prevalence and predictors. JAMA 281 (6), 537–542.
There is no effective therapy without accurate and comprehensive diagnosis. This is even more true for female sexual dysfunction (FSD), which usually has a multifactorial aetiology. It is a prevalent problem, afflicting approximately 40% of women (Allahdadi et al., 2009). Biological, psychosexual and context-related factors (Basson et al., 2000, 2004), further characterized as predisposing, precipitating and maintaining (Graziottin and Brotto, 2004; Graziottin, 2005; Graziottin, 2011) may interact to give the FSD that the woman is complaining about its specific individual characteristics. FSD is defined by the World Health Organization as ‘the various ways in which a woman is unable to participate in a sexual relationship as she would wish’ (NIH Consensus Conference, 1993; American Psychiatric Association, 2013). FSD is more complex and difficult to categorize due to a woman’s perception about sex when compared to males. FSD is a multifaceted disorder, comprising anatomical, psychological, physiological, as well as social-interpersonal components. With several existing FSD definitions, the most descriptive encompasses FSD as the persistent/recurring decrease in sexual desire or arousal, the difficulty/inability to achieve an orgasm, and/or the feeling of pain during sexual intercourse (Salonia et al., 2004; American Psychiatric Association, 2013).

The accurate diagnosis of FSD is currently a challenge for researchers and clinicians. The temptation of searching for the aetiology, preliminary to finding the optimal treatment, is usually inappropriate, and continuously frustrated by the complexity of female sexuality (Allahdadi et al., 2009). The physicians should master the basis of anatomy and physiology of sexual function to get the instruments to diagnose and treat women’s sexual dysfunction (FSD) (Graziottin, 2007a, 2007b). Sexual function includes desire/interest, central and peripheral arousal with genital congestion and vaginal lubrication, orgasm, resolution and satisfaction. Recent debate supported a common reading of sexual desire/interest and central arousal in women.

The delay in the medical approach to FSD and the persistent psychological perspective make it difficult to have evidence-based medical treatments of FSD except in the domain of sexual hormones. As a result of diagnostic delays, inadequacies, and gender biases, no treatment for FSD is currently approved with this specific indication with the exception of a clitoral device indicated for female arousal disorders (Wilson et al., 2001). From the clinical point of view, an integrated diagnostic and treatment approach is therefore necessary to tailor treatment according to the individual and couple’s needs at the best of our current scientific and clinical knowledge (Basson et al., 2000, 2004; Graziottin, 2001a, 2004a, 2004b; Plaut et al., 2004; Clayton and Hamilton, 2010; Clayton and Groth, 2013).

The available evidence for treatment of FSD will be reviewed. Special focus will be given to the role of the physical therapist in addressing the muscle and pelvic floor-related contributors to FSD.

**INTRODUCTION**

**DIAGNOSTIC KEY POINTS**

Key points in the FSD diagnosis, preliminary to a well tailored treatment, should be:

- **Accurate listening** to the complaint’s wording, to verbal and non-verbal messages, with:
  - definition of the nature of the disorders;
  - is it lifelong or acquired?
  - is it generalized or situational?
  - is it organic, psychogenic, contextual or, as in most cases, mixed, with definition of key predisposing, precipitating and maintaining factors?
  - how severe is the distress it causes?
  - are there sexual and/or medical (e.g. urogenital, proctological) associated comorbidity – comorbidities may be other types of FSD, but also other medical conditions, such as urological, gynaecological, proctological, metabolic, cardiovascular and neurological diseases – for example, urinary tract symptoms have a relative risk (RR) of: 4.02 (2.75–5.89) of being associated with arousal disorders; 7.61 (4.06–14.26) of being associated with sexual pain disorders (Laumann et al., 1999);
  - partner’s related issues;
  - the personal motivation the woman has (or does not have) for treatment of FSD, which includes the meaning of the symptom for the woman.

- **Accurate examination** of the woman, and particularly of the external genitalia, vagina and pelvic floor (Graziottin, 2004a, 2004b, 2011; Graziottin et al., 2001a, 2001b, 2001c) – careful physical examination should be performed because the biological aetiology of FSD is better diagnosed when attention is paid to vulvovaginal trophism with pH recording; hypo- or hypertonic pelvic floor conditions, with tender and trigger point evaluation; diagnosis of inflammation and infection, with culture examinations when indicated; and the pain map accurate description (Graziottin et al., 2001c; Graziottin and Murina, 2011) because location of pain and its onset
characteristics are the strongest predictors of its biological aetiology (Meana et al., 1997; Graziottin and Murina, 2011; Clayton and Groth, 2013).

This is mandatory when genital arousal disorders, sexual pain disorders (vaginismus and dyspareunia) and orgasm disorders are complained of. It may be useful even when sexual desire disorders and/or subjective sexual arousal disorders (‘I do not feel mentally excited’) are the leading complaints to diagnose biologically rooted comorbidities with other FSD. Comorbidity should be accurately recorded with attention to which sexual disorder came first. A competent physical examination is required, focused on detecting all the clinical signs, and an attention to the frequent comorbidities (medical and sexual) that vulvar pain can be associated with. There are medical comorbidities, as vulvodynia may be associated with bladder symptoms (post-coital cystitis, painful bladder syndrome), endometriosis, irritable bowel syndrome, fibromyalgia, headache. And sexual comorbidities, with coital pain (dyspareunia) being the leading symptom, with its cohort of secondary loss of desire, vaginal dryness, orgasmic difficulties and sexual dissatisfaction (Graziottin and Murina, 2011).

On the positive side, the cascade of positive feedback when a treatment is effective may cause a significant improvement in all domains of sexual response as several studies have proved (Shifren et al., 2000; Laan et al., 2001; Alexander Leventhal et al., 2004; Simunic et al., 2003; Graziottin and Basson, 2004; Graziottin et al., 2009a).

In stable couples, current feelings for the partner (i.e. quality of the relationship, and the quality of the partner’s sexuality [inclusive of general and sexual health]) should be investigated as well (Dennerstein et al., 1999, 2003, 2007; Klausmann, 2002; American Psychiatric Association, 2013).

The woman’s general health should be examined, with special focus on conditions that may directly or indirectly impair the woman’s mental and/or genital response (Basson et al., 2000, 2004; Graziottin, 2000, 2003a, 2004a, 2004b; Graziottin, 2007b; Clayton and Groth, 2013; Pontiroli et al., 2013).

**PRINCIPLES OF FSD THERAPY**

A growing body of evidence implicates hormonal factors in the genesis of FSD (Sarrel, 1998; Shifren et al., 2000; Laan et al., 2001; Alexander Leventhal et al., 2004; Simunic et al., 2003; Graziottin and Basson, 2004; Clayton and Groth, 2013). Key endocrine hormones—oestrogen, progesterone and testosterone—are involved in the sexual response (Bancroft, 2005). And deficiencies in the levels of androgens potentially are involved in FSD (Bancroft, 2002). Low levels of testosterone are associated with a decline in libido, arousal, genital sensation and orgasm (Davis, 2000). Indeed, during a woman’s entire reproductive life span, sex hormones exert both organizational and activational effects on sexual behaviour. The action of hormones is mediated by non-genomic and genomic pathways (Graziottin and Gambini, 2014). Current evidence indicates that there is a specific place in the treatment of FSD for pharmacological hormones, for the most part in postmenopausal women (Sarrel, 1998; Shifren et al., 2000; Laan et al., 2001; Simunic et al., 2003; Alexander Leventhal et al., 2004; Graziottin and Basson, 2004; Graziottin, 2010). Sexual hormones may be delivered by various routes: oral, transdermal, nasal, vaginal, through subcutaneous implants or intrauterine devices. The most important difference between the oral route and those that bypass the first hepatic pass is that the oral treatment induces an increase of sex hormone-binding globulin (SHBG) by as much as 133%, thus significantly reducing free testosterone (Vehkavara et al., 2000). Levels of SHBG seem to be unaffected by hormones delivered via transdermal, nasal and vaginal routes.

Depending on the aetiological diagnosis of the leading disorder, the therapy should consider one or more of the following leading options.

**Libido disorder**

Libido and subjective sexual arousal disorder (‘I do not feel mentally excited’), often diagnosed in comorbidity, either lifelong or, more frequently, acquired, may benefit from the following treatments.

**Medical treatment**

**Hormones**

**Androgen**

The major androgens in women include testosterone (T) and dihydrotestosterone (DHT), dehydroepiandrosterone sulphate (DHEAS), dehydroepiandrosterone (DHEA) and androstenedione (A) (Bachmann et al., 2002). Testosterone is the most potent androgen. Plasma testosterone levels range from 0.2 to 0.7 ng/ml (0.6–2.5 mmol/l), with significant fluctuations related to the phase of the menstrual cycle. Testosterone is converted to DHT, but can also be aromatized to estradiol (E2) in target tissues; DHT is the principal ligand to androgen receptors in women as well. Androgens peak in the early 20s, then decline steadily (Burger et al., 2000). Androgens play a primary role in female physiopathology: The age-related reduction in the production of ovarian and adrenal androgens may significantly affect women’s health. The decline of circulating androgens results from a combination of two events: reduced ovarian production and age-related decline in adrenal androgen synthesis. The relative androgen deficiency in pre- and postmenopausal women may induce impairment of sexual function, libido, well-being, energy and may contribute to reduced cognitive functions (Pluchino et al., 2013a).

**Testosterone in premenopausal women**: evidence concerning the role of hormones, particularly testosterone, in...
premenopausal women is limited. Very few studies have been done in premenopausal subjects. Goldstat et al. (2003) focused their controlled study on a small group of premenopausal women; subjects with lifelong hypoactive sexual desire disorder with testosterone levels in the lower one-third or less of the normal range may significantly benefit from testosterone cream when compared to placebo.

**Testosterone in postmenopausal women:** menopause can be natural or iatrogenic. Iatrogenic menopause may result from surgery, chemotherapy or radiation therapy. The most common surgical cause of menopause is bilateral oophorectomy, which leads to a sudden 50% fall in circulating testosterone levels (Bachmann et al., 2002). Plasma testosterone values at or below the lowest quartile of the normal range for women in their reproductive years also suggest a diagnosis of androgen insufficiency syndrome.

A recent, systematic review of all available data from randomized and placebo-controlled trials of treatment for FSD in postmenopausal women concluded that use of many frequently used treatments is not supported by adequate evidence (Madelska and Cummings, 2003). In their review of randomized, controlled trials involving the use of testosterone in oestrogen-replete women, Alexander Leventhal et al. (2004) found general support for the positive effect of testosterone on different dimensions of women’s sexuality. One limit of this analysis is that some of the reviewed studies involved supraphysiological doses. In a study by Shifren et al. (2000), the total testosterone was raised above the normal range, but the free and bioavailable testosterone remained within the normal range. Sherwin (2002) and more recently Alexander Leventhal et al. (2004) in their reviews of randomized, controlled trials, found that adding androgens to the standard oestrogen replacement had added sexual benefit in different domains, sexual desire first. It has been further demonstrated that surgically menopausal women receiving testosterone experience significant increases in total satisfying sexual activity versus women receiving placebo, significant improvement in all domains of sexual function and decreases in personal distress, with a favourable safety profile (Kingsberg, 2008; Graziottin et al., 2009a).

**Ostrogens and progestogens**

In naturally postmenopausal women, progesterone or progestogens protect the endometrium. The positive effect of oestrogens on the well-being and sexuality of postmenopausal women may be variably modulated according to the type of progestogens added in the hormonal replacement therapy (Graziottin and Leiblum, 2005; Simon, 2010). Progesterone, the physiological hormone, may have a mildly inhibiting effect on sexual desire. Progestogens, synthetic molecules with progestinic action, have a wide spectrum of actions from strongly antiandrogenic to neutral to androgenic, according to:

- their structure (whether they are derived from 17-OH-progesterone, 19-nortestosterone or 17-alphaaspiironolactone) and their consequent varying pattern of interaction with different hormonal receptors (Schindler, 1999; Stanczyk, 2002; Graziottin and Leiblum, 2005) – progestogens may interact with progestinic, oestrogenic, androgenic, glucocorticoid, and mineralocorticoid receptors, so the consequent metabolic and sexual profile differs;
- their variable binding affinity to SHBG, which modulates the quantity of free testosterone available for its biological action;
- the variable inhibition of the type 2,5-aldareductase, which activates testosterone (T) into DHT.

To assimilate progestogens in a unique category focusing on a generalized ‘class effect’ is wrong and may lead to inappropriate conclusions (Graziottin and Leiblum, 2005). The progestogen with the most favourable effect on sexual function in hormonal replacement therapy is nor-ethisterone, with a positive impact on desire, arousal, orgasm and satisfaction in natural postmenopausal women with an intact uterus. Controlled head-to-head studies are necessary to evaluate the correlation between the pharmacological profile and the clinical effect.

**Tibolone**

Tibolone is a 19-nortestosterone derivate with mild oestrogenic, progestinic and androgenic activity. It lowers SHBG, thus increasing free E2, testosterone and DHEA-S levels. It is not available in the United States, but is widely used in Europe. In randomized studies comparing it with placebo, tibolone (2.5 mg/day) alleviated vaginal dryness and dyspareunia, increasing libido, arousal and sexual satisfaction in postmenopausal women with natural or surgical menopause (Laan et al., 2001; Madelska and Cummings, 2002). On the contrary the current evidence does not suggest an important effect of tibolone on sexual function (Nasti et al., 2013). More studies are required to really understand the role of this hormone on female sexuality.

**DHEA-S**

Studies conducted in elderly women have shown a positive effect of DHEA-S on mental well-being and on motivational aspects of sexuality with a mild relief of climacteric symptoms (Stomati et al., 2000; Labrie et al., 2001). It has been demonstrated recently that delta-5 androgen therapies seem to enhance the sexual response in experimental animal models and in clinical trials (Pluchino et al., 2013b).

**Hyproprolactinaemic drugs**

Prolactin is the most powerful inhibiting hormone where sexual desire is considered, with increasing inhibiting effect with increasing plasma levels. Hyproprolactinaemic drugs are useful to improve sexual desire when the prolactin level is supraphysiological.

**Antidepressants**

Affective disorders, namely depression and anxiety, when associated with sexual desire disorders, should be
addressed with a mixed approach, both pharmacological and psychodynamic (Alexander Leventhal and Kotz, 2004; American Psychiatric Association, 2013). There is consistent evidence to suggest that antidepressant medication adversely affects one or more of the three phases of sexual response (desire, arousal and orgasm). Antidepressants with strong serotonergic properties have the highest rate of sexual side-effects (La Torre et al., 2013). Among antidepressants, bupropion seems to have the most positive effect on sexual desire (Seagraves and Balon, 2003; Clayton et al., 2004). Comorbidity between low testosterone and depression should be considered and appropriately treated.

**Pelvic floor rehabilitation**

A few physicians and medical sexologists recommend careful physical examination of the woman complaining of low desire on the wrong assumption that the disorder is either ‘all psychogenic and/or couple-dependent’ or at best ‘hormone-dependent’. Low desire can result from negative feedback from disappointing arousal, coital pain, coital anorgasmia, dissatisfaction. Indeed, low desire may be concomitant to sexual aversion disorders associated with vaginismus (with a variable hyperactivity of the pelvic floor) (Graziottin et al., 2004a; Graziottin et al., 2009b) or secondary to sexual pain disorders such as dyspareunia associated with vulvar vestibulitis (Graziottin et al., 2001b; Graziottin and Murina, 2011), in which defensive contraction of levator ani is common (Glazer et al., 1995; Bergeron et al., 2001; McKay et al., 2001; Graziottin et al., 2004b, 2004c).

**Antalgic treatment**

When loss of desire is acquired and secondary to persistent chronic coital pain, antalgic treatment aimed at reducing or eliminating pain (especially if neuropathic) is preliminary to effective normalization of sexual desire (Vincenti and Graziottin, 2004).

**Psychosexual treatment**

*Individual psychosexual or behavioural therapy*

Individual psychosexual or behavioural therapy is the approach of choice if the FSD aetiology includes sexual inhibitions, poor erotic skills, poor body image, low self-confidence or previous abuse (Leiblum and Rosen, 2000; Graziottin, 2003b; Rellini and Meston, 2004; American Psychiatric Association, 2013).

*Couple therapy*

Couple therapy is used when symbiotic dynamics with poor differentiation according to Schnarch (2000) or conflicts and/or destructive dynamics are reported.

**Box 7.9 Referral resources**

- Medical sexologist or gynaecologist trained in sexual medicine: FSD requires appropriate medical diagnosis and treatment
- Urologist or andrologist: when the partner has erectile or ejaculatory dysfunction that requires medical intervention
- Family physician trained in sexual medicine: for sexual dysfunctions in either partner
- Oncologist: when hormonal treatment is considered for patients who have had cancer
- Psychiatrist: when depression and anxiety are associated with FSD
- Sex therapist: to carry out the psychosexual therapy
- Couple therapist: when relationship issues are a primary contributor to the sexual dysfunction
- Individual psychotherapist: when personal psychodynamic issues are inhibiting sexual function
- Physical therapist: when hyper- or hypotonicity of pelvic floor is contributory

Modified from Plaut et al., 2004, with permission.

**Referral**

The multisystemic and multifactorial aetiology of FSD require a professional multidisciplinary team. Appropriate referral is a key part of successful treatment (see Box 7.9) (Plaut et al., 2004; Clayton and Hamilton, 2010; Buster, 2013). For example, referral of the partner to the uroandrologist should be recommended when male disorders (premature ejaculation, erectile deficit, libido disorders) emerge as critical co-factors in the aetiology of FSD (i.e. if the partner appears to be the ‘symptom inducer’ and the woman is the ‘symptom carrier’ [Kaplan, 1979; Plaut et al., 2004; Graziottin and Althof, 2011]).

Acquired libido disorder should be treated on the basis of the leading aetiologic factor, especially if it is comorbid with other lifelong or acquired FSD, such as pain disorder, arousal disorder or orgasm disorder (Graziottin et al., 2001b), or biological factors such as iatrogenic menopause (Graziottin and Basson, 2004).

**Arousal disorders**

Subjective sexual arousal disorders, either lifelong or acquired, usually in comorbidity with sexual desire disorders, should be treated as mentioned above. Postmenopausal mixed genital and subjective arousal disorders may benefit from systemic hormonal replacement therapy, especially androgens (see above) (Traish et al., 2002; Alexander Leventhal et al., 2004). However, to date, no drug treatment approved by the US Food and Drug Administration (FDA) and the European Medicines Agency (EMA) is
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available to treat women with HSDD/FSIAD. As a result, there is an unmet need for a drug treatment for HSDD/FSIAD (Poels et al., 2014).

Isolated acquired genital arousal disorders may benefit from the following.

Medical treatment

Topical oestrogens
A number of studies suggest that topical vaginal oestrogens may significantly reduce vaginal dryness, increase genital arousal and reduce dyspareunia (Riouxf et al., 2000; Simunic et al., 2003; Dessole et al., 2004; Graziotiit and Serafini, 2011; Griebling et al., 2012). A multicentre, double-blind, randomized, placebo-controlled study (n = 1612 postmenopausal women with urogenital and sexual complaints) indicates that 25 μg of oestradiol applied vaginally twice a week for a year may significantly improve six vaginal symptoms and signs: vaginal dryness (p < 0.0001), itching/burning (p < 0.0001), recurrent vaginitis (p < 0.0001), petechiae (p < 0.0002), dyspareunia (p < 0.0001) and vaginal atrophy (p < 0.0001), and five bladder symptoms and signs: dysuria (p < 0.003), frequency/nocturia (p < 0.001), urinary tract infection (p < 0.034), urinary incontinence, urge mostly (p < 0.002), and urinary atrophy (p < 0.001) (Simunic et al., 2003). Furthermore, cystometry performed at baseline and after 12 months indicates that the maximal cystometric capacity increases from 200 ml to 290 ml (p < 0.023); the bladder volume at first urgency increases from 140 ml to 180 ml (p < 0.048); and bladder volume at strong urgency increases from 130 ml to 170 ml (p < 0.045). The comorbidity between urogenital and sexual symptoms in postmenopausal women may therefore be effectively addressed with a topical vaginal treatment that is easy to use and safe both for the endometrium and the breast (Cody et al., 2012).

Topical testosterone
Testosterone propionate powder 1% or 2% in white petroleum jelly applied in minimal daily quantity to the clitoris and the vulvar region may improve genital arousal in the external genitalia (Notelevitz, 2002). Improvement in sexuality score was greatest with combined oestrogen-androgen therapy (Raghunandan et al., 2010).

Vasoactive drugs
Evidence on the effectiveness of vasoactive drugs (sildenafil, vardenafil, tadalaflag) in addressing genital arousal disorders in women is negative (Leddy et al., 2012), or at best controversial, with one exception (Berman et al., 2003). The frequent comorbidity with desire disorders, the frequent couple issues, the difficulty in diagnosing a ‘pure’ genital arousal disorder and the lack of a personal motivation for a pharmacological treatment of genital arousal disorder may explain the substantial lack of efficacy in comparison to men’s genital arousal disorders (i.e. erectile deficit of vascular aetiology).

Clitoral vacuum device
Clitoral vacuum device is the only FDA-approved treatment for genital arousal disorders with a vascular and/or neurogenic aetiology (Wilson et al., 2001). It may be useful in women treated for invasive carcinoma of the cervix who have undergone surgery and pelvic radiotherapy.

Pelvic floor rehabilitation
Genital arousal disorders may be secondary to coital pain: unwanted pain is the strongest reflex inhibitor of vaginal congestion and lubrication. Diagnosing and treating the muscular component of coital pain (both in vaginismus and dyspareunia) is a key part of the medical treatment (Glazer et al., 1995; Bergeron et al., 2001; McKay et al., 2001; Graziotiit, 2004c) and is preliminary to resuming a normal vasocongestive response (Graziotiit and Brotto, 2004). It has been recently demonstrated that arousal function and orgasm are related to better pelvic floor muscle function (Lowenstein et al., 2010). Hypoactivity of the muscles (low tone), more frequent after vaginal delivery, leads to poor sexual function and lack of pleasure during coitus and orgasm. In contrast, hyperactivity (high tone) may be pathophysiologically linked to the sexual pain disorders called dyspareunia (namely coital pain) and vaginismus (Graziotiit and Giraldi, 2006).

Psychosexual treatment
Psychological interventions are effective treatment options for sexual dysfunction. However, evidence varies considerably across single disorders. Good evidence exists to date for female hypoactive sexual desire disorder (HSDD) and female orgasmic disorder (Fruhauf et al., 2013). Indications for psychosexual treatment of subjective sexual arousal disorders overlap with those for desire disorders. Co-treatment may therefore effectively address comorbidity. However, treatment of the potential parallel biological aetiology of the genital arousal disorder is mandatory if cure for the reported FSD is to be achieved (Plaut et al., 2004). Couple psychotherapy should be proposed when relational dynamics are contributing to maintenance of the sexual problem (Leiblum and Rosen, 2000; Clulow, 2001; Buster, 2013).

Orgasm disorders
Orgasm disorders have a prevalent psychogenic aetiology in young women (Mah and Binik, 2004). Biological factors – age, menopause-related loss of sexual hormones, pelvic floor disorders, iatrogenic issues (such as antidepressant serotonergic drugs inhibiting orgasm), and comorbidities (mainly with stress and urge incontinence) – become increasingly important with increasing age (Graziotiit, 2004a; De Rogatis et al., 2009b; Clayton and Groth, 2013). According to the aetiologic diagnosis, the main therapeutic options include the following.
Medical treatment

Systemic and/or topical hormonal replacement therapy
Systemic and/or topical hormonal replacement therapy is discussed above. Testosterone has a special role in the treatment of orgasmic disorders associated with loss of sexual hormones, especially after bilateral oophorectomy (Shifren et al., 2000; Sherwin, 2002; Alexander Leventhal et al., 2004). It behaves as ‘initiator’ in the brain and as ‘modulator’ in the cavernosal bodies, where it works as ‘permitting factor’ for nitric oxide (NO), in women as well as in men (Graziottin, 2004c; Raghunandan et al., 2010).

Change of pharmacological treatment inhibiting orgasm (e.g. antidepressants such as selective serotonin reuptake inhibitor [SSRI] or tricyclics) should be considered when feasible from the medical point of view if orgasm inhibition is reported as a side-effect (see above, La Torre et al., 2013). Bupropion seems to be a better choice (Clayton et al., 2004; Segraves and Balon, 2003b).

Pelvic floor rehabilitation
Pelvic floor rehabilitation is of the highest importance for hypotonic conditions of the pelvic floor, as pioneered by Kegel (1952), after delivery (Glazener, 1997; Baessler and Schuessler, 2004; Graziottin and Gambini 2014); even more so when incontinence is a strong inhibiting orgasmic factor. Fear of leaking during thrusting in stress incontinence and at orgasm in urge incontinence is often under-reported and yet is a powerful disruptor of orgasm potential. Orgasm inhibition may also be secondary to coital pain (Graziottin et al., 2001b; Graziottin and Murina, 2011). Again, accurate diagnosis of comorbidity and appropriate cotreatment with relaxation of the pelvic floor in this latter case is key.

Psychosexual treatment

Individual psychosexual or behavioural therapy
Lifelong ‘isolated’ orgasmic disorders may benefit from a behavioural educational treatment, encouraging self-knowledge and eroticism with the experience of higher arousal sensations, use of vibrators or of a clitoral device up to orgasm (Meston et al., 2004). More often, however, the orgasmic disorder is associated with poor arousal with or without performance anxiety. These conditions should therefore be treated together (Leiblum and Rosen, 2000; American Psychiatric Association, 2013).

Couple therapy
Lifelong orgasm difficulties may need couple therapy when sexual inhibitions, poor erotic skills and/or low self-confidence are shared by the couple (Meston et al., 2004; American Psychiatric Association, 2013). Appropriate behavioural and pharmacological treatment of premature ejaculation should be proposed to the partner when it causes inadequate coital stimulation and increasing erotic dissatisfaction in the female partner (Graziottin and Althof, 2011).

If all of the sexual response is impaired, with significant comorbidity with desire and arousal disorders, accurate treatment of predisposing, precipitating and maintenance factors, biological, psychosexual and/or contextual, should be proposed (Plaut et al., 2004; American Psychiatric Association, 2013).

Sexual pain disorders

Dyspareunia and vaginismus because of coital pain directly inhibit genital arousal and vaginal receptivity. Indirectly, they may affect orgasm potential, the physical and emotional satisfaction, causing loss of desire up to avoidance of sexual intimacy. Dyspareunia may have many biological aetologies: the leading cause of coital pain in premenopausal women is vulvar vestibulitis, whereas postmenopausally it is vaginal dryness. Dyspareunia may benefit from the following treatments. (See also Box 7.10.)

Medical treatment

Multimodal therapy
Vulvar vestibulitis should be treated with a combined treatment aimed at reducing:

- upregulation of mast cells, the main source of inflammatory mediators and recently considered as cellular sensors in inflammation and immunity (Beghdadi et al., 2011), as coordinators of peripheral inflammatory processes (Kinet, 2007; Abraham and St John, 2010), and as important players in the development and maintenance of neuroinflammation due to their capacity to directly or indirectly interact with glial cells (Milligan and Watkins, 2009; Nelissen et al., 2013), both by reducing the agonist stimuli (such as Candida infections, micro abrasions of the introital mucosa because of intercourse with a dry vagina and/or a contracted pelvic floor, chemicals, allergens, etc.) that cause degranulation leading to chronic tissue inflammation, and/or with antagonist modulation of its hyper-reactivity, with amitriptyline or aliamides gel (Graziottin and Brotto, 2004; Graziottin et al., 2004b; Graziottin and Murina, 2011), and palmytoiletanolamide, a food supplement (Graziottin et al., 2013);

- upregulation of the pain system secondary to proliferation of introital pain fibres (Bohm-Starke et al., 1999, 2001a, 2001b; Bornstein et al., 2002, 2004) induced by nerve growth factor produced by the upregulated mast cells. Mast cells’ granules contain many factors implicated in neurogenic inflammation like NGF, tumour necrosis factor (TNF), protease and cytokines (Frenzel and Hermine, 2013), and the lowered central pain threshold (Pukall et al., 2006) – a thorough understanding of
the pathophysiology of pain in its nociceptive and neuropathic component, is mandatory – antalgic treatment should be prescribed: locally, with electroanalgesia (Nappi et al., 2003) or, in severe cases, with the ganglion impar block; systemically with tricyclic antidepressant or gabapentin in the most severe cases (Graziottin and Broto, 2004; Vincenti and Graziottin, 2004). Preliminary data suggest a positive impact in reducing neuroinflammation with palmytoiletanolamide, a food supplement that reduces mast cells’ upregulation (Graziottin et al., 2013);

- upregulation of the muscular response, with hyperactivity of the pelvic floor (Graziottin et al., 2004a), which may precede vulvar vestibulitis when the predisposing factor is vaginismus (Abramov et al., 1994; Graziottin et al., 2001b; Graziottin and Murina, 2011) or be acquired in response to genital pain (Graziottin et al., 2004a, 2004b; Graziottin and Murina, 2011) – in controlled studies, electromyographic feedback (Glazer et al., 1995; Bergeron et al., 2001; McKay et al., 2001) has proven to significantly reduce pain of vulvar vestibulitis;

self-massage, pelvic floor stretching and physical therapy may also reduce the muscular component of coital pain (Graziottin, 2004a; Graziottin and Broto, 2004), but high-quality randomized controlled trials are needed to determine the true effect of such interventions; for hyperactivity of the pelvic floor, treatment with type A botulinum toxin has been proposed (Bertolasi, 2004, personal communication) – individually tailored combinations of this approach are useful for treating introital dyspareunia with different aetiologies from vulvar vestibulitis.

Deep dyspareunia, secondary to endometriosis, pelvic inflammatory disease (PID), chronic pelvic pain and other less frequent aetiologies, requires specialist treatment that goes beyond the scope of this chapter.

Topical hormones

Vaginal oestrogen treatment is mandatory when vaginal dryness is causing postmenopausal dyspareunia, either spontaneous or iatrogenic (Graziottin, 2001a, 2001b, 2004a; Simunic et al., 2003; Graziottin and Murina, 2011). Vulvar treatment with testosterone may be considered when vulvar dystrophy and/or lichen sclerosus contribute

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**Box 7.10 Treatment of the medical causes of dyspareunia**

**Inflammatory aetiology (upregulation of mast cells)**

Pharmacological modulation of mast cell hyper-reactivity

- Antidepressants: amitriptyline
- Aliamides – topical gel
- Reduction of agonist factors causing mast cell hyper-reactivity
- Recurrent *Candida* or *Gardnerella vaginitis*
- Microabrasions of the introital mucosa:
  - from intercourse with a dry vagina
  - from inappropriate lifestyles
- Allergens/chemical irritants
- Physical agents
- Neurogenic stimuli

**Muscular aetiology (upregulation of the muscular system)**

- Self-massage and levator ani stretching
- Physical therapy of the levator ani
- Electromyographic biofeedback
- Type A botulinum toxin

**Neurological aetiology (upregulation of the pain system)**

**Systemic analgesia**

- Gabapentin
- Pregabalin

**Local analgesia**

- Electroanalgesia
- Ganglion impar block
- Surgical therapy:
  - vestibulectomy

**Hormonal aetiology**

**Hormonal therapy**

- Local:
  - vaginal oestrogens
  - testosterone for the vulva
- Systemic:
  - hormonal replacement therapies

*Aliamides are a class of endogenous molecules with an anti-inflammatory activity. The most important is the palmytoiletanolamide, belonging to the class of fatty acid amides, chemically known as N-2-idrossietil)-esadecanamide. They work through the downregulation of the hyperactive mast cells. In Italy they are available in the form of vaginal gel and now as pills. They constitute an innovative approach to the vaginal and bladder chronic inflammation, secondary to mast cells’ upregulation and to neuroinflammation, associated to the upregulation of mast cells and microglia.
to introital dyspareunia. These data need more studies because of the controversial results presented in the scientific literature (Chi et al., 2011).

**Psychosexual treatment**

**Psychosexual and/or behavioural therapy**

Psychosexual and/or behavioural therapy is the leading treatment of lifelong vaginismus (Leiblum, 2000). It should be offered in parallel with progressive rehabilitation of the pelvic floor and pharmacological treatment to modulate the intense systemic arousal in the subset of intensely phobic patients (Plaut et al., 2004; Graziottin et al., 2009a). In this latter group, comorbidity with sexual aversion disorder should be investigated and treated first (Frasson et al., 2009).

Psychosexual and/or behavioural therapy contributes to the multimodal treatment of lifelong dyspareunia, which is reported in one-third of our patients (Graziottin et al., 2001b; De Rogatis et al., 2009b; Graziottin and Murina, 2011). Anxiety, fear of pain and sexual avoidance behaviours should be addressed as well. The shift from pain to pleasure is key from the sexual point of view. Sensitive and committed psychosexual support to the woman and the couple is mandatory.

### WHEN THE PHYSICAL THERAPIST COUNTS

Pelvic floor muscles are critically involved in the physiology and pathophysiology of women’s sexual response (Fashokun et al., 2013). The physical therapist should be part of the multidisciplinary team involved at the sexual medicine centre (Graziottin et al., 2009a). He or she should diagnose and address the following.

**Hyperactivity/hypertonus of the pelvic floor**

The physical therapist should diagnose and address:

- primary pelvic floor hyperactivity in children and adolescents, thus preventing one of the most neglected predisposing factors to dyspareunia and vulvar vestibulitis (Chiozza and Graziottin, 2004; Graziottin, 2005; Harlow et al., 2001);
- acquired hyperactivity with levator ani myalgia by overexertion (i.e. ‘Kegel dyspareunia’; DeLancey et al., 1993; Faubion et al., 2012);
- lifelong hyperactivity of the pelvic floor in vaginismus and lifelong or acquired hyperactivity in dyspareunia of any aetiology (Graziottin, 2003a; Graziottin et al., 2004a; Faubion et al., 2012);
- levator ani tender and/or trigger points with referred pain (Travell and Simons, 1983; Alvarez and Rockwell, 2002; Graziottin and Murina, 2011);
- levator ani hyperactivity associated with recurrent cystitis, urge incontinence and dyspareunia (Graziottin, 2004a; Whitmore et al., 2007; Salonia et al., 2013);
- systemic postural problems in chronic pelvic pain, dyspareunia and vaginismus (Faubion et al., 2012);
- chronic pelvic pain and chronic coital pain-associated myalgias and pertinent antalgic treatment (Bourcier et al., 2004; Graziottin, 2011).

**Hypotonicity/hypotonus of the pelvic floor**

The physical therapist should diagnose and address:

- pelvic floor damage after delivery;
- hypotonicity worsening after the menopause;
- pelvic floor hypotonus in comorbidity with urogenital and/or proctological disorders (Wesselmann et al., 1997; Bourcier et al., 2004).

Perineal pain is common after delivery and may impair normal sexual functioning. Dyspareunia following vaginal delivery is reported by 60% of women at 3 months, 30% at 6 months. Trauma to the perineum has been associated with dyspareunia during the first 3 months after birth. It is reported in four trials (2497 women) that the perineal massage during the last months of pregnancy may be a possible solution able to reduce the likelihood of perineal trauma and pain. Antenatal digital perineal massage was associated with an overall reduction in the incidence of trauma requiring suturing and women practising perineal massage were less likely to have an episiotomy (Beckmann and Stock, 2013):

- overall reduction in the incidence of trauma RR 0.91
- incidence of episiotomy RR 0.84
- reduction of pain at three months post-partum RR 0.45.

These data require a further validation in the short, mean and long period to better evaluate the impacts on the women’s sexuality.

The physical therapist may also help the patient to increase awareness of the role of the levator ani in sexual receptivity and vaginal sensitivity to increase the woman’s and her partner’s coital pleasure.

### CONCLUSION

The complexity of FSD requires a dedicated diagnostic and therapeutic team, sharing a common pathophysiological and psychodynamic cultural scenario with the aim of offering the most integrated understanding of the meaning of the symptoms and the most effective comprehensive treatment.
Pelvic floor muscles are critically involved in the physiology and pathophysiology of a woman's sexual response. Physical therapists may therefore greatly contribute to improving women's sexual health. They deserve appreciation and an increasing role in the multimodal treatment of FSD. There is, however, an urgent need for high-quality randomized controlled trials to evaluate the effect of different physical therapy interventions for FSD. A collaboration between physical therapists and sexologists/gynaecologists in future research projects in this important field is highly recommended.

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Female pelvic floor dysfunctions and evidence-based physical therapy

Chapter

7


8.1 Urinary incontinence and other lower urinary tract symptoms

INTRODUCTION

Prostate surgery is one of the major causes of urinary incontinence (UI) in the male population. Randomized controlled studies considering physical therapy for men with incontinence after prostatectomy have been published since the end of the 1990s (Mathewson-Chapman, 1997; Chang et al., 1998; Moore et al., 1999; Bales et al., 2000; Franke et al., 2000; Van Kampen et al., 2000; Porru et al., 2001; Sueppel et al., 2001; Floratos et al., 2002; Parekh et al., 2003; Wille et al., 2003). From 2004 to 2013, 22 new studies have focused on physical therapy and incontinence after prostatectomy (Ip, 2004; Yokoyama et al., 2004; Filocamo et al., 2005; Burgio et al., 2006; Manassero et al., 2007; Zhang et al., 2007; Liu et al., 2008; Moore et al., 2008; Overgard et al., 2008; Robinson et al., 2008; Mariotti et al., 2009; Centemero et al., 2010; Dobbelman et al., 2010; Marchiori et al., 2010; Ribeiro et al., 2010; Yamanishi et al., 2010; Glazener et al., 2011; Goode et al., 2011; Nilsson et al., 2012; Park et al., 2012; Tienforti et al., 2012; Geraerts et al., 2013). A Cochrane review and clinical guidelines for incontinence after prostatectomy have described the evidence for physical therapy for male incontinence (Campbell et al., 2012; Lucas et al., 2012).
Evidence-Based Physical Therapy for the Pelvic Floor

Besides urinary incontinence, men may suffer from other lower urinary tract symptoms. These symptoms in men include filling symptoms or irritative symptoms – frequency, urgency, urgency incontinence, nocturia – and voiding symptoms or obstructive symptoms – hesitancy, weak stream, straining, incomplete emptying, intermittency, terminal and post-voiding dribble (Dorey, 2001; Abrams et al., 2003). Although physical therapy should have the potential to alleviate lower urinary tract symptoms, the number of studies concerning these symptoms is scarce. The efficacy of physical therapy for terminal and post-void dribble is investigated in four randomized controlled studies (Paterson et al., 1997; Chang et al., 1998; Porru, 2001; Dorey et al., 2004).

Postprostatectomy incontinence

Urinary incontinence is a common consequence in many men undergoing prostate surgery (Diokno, 1998; Dorey, 2000; Peyromaure et al., 2002).

The prostate gland is part of the male sex gland and can be divided into three zones: the central zone (25%, situated just under the bladder), the transition zone (5% around the urethra) and the peripheral zone (70% around the other zones). The transition zone is the site of the development of benign prostatic hyperplasia. Prostate hyperplasia (Fig. 8.1) can be treated by transurethral or transvesical resection of the prostatic adenoma. Seventy-five per cent of all prostatic adenocarcinomas are situated in the peripheral zone. Localized prostate cancer can be treated by radical prostatectomy (RP) and this treatment is commonly thought to be the most effective (Baert et al., 1996) (Fig. 8.2). Radical prostatectomy can be performed via an open, laparoscopic or robot-assisted laparoscopic approach (Fig. 8.3).

Removal of the prostate can lead to leakage of urine. The occurrence of incontinence, especially in the early recovery period after surgery, is hard to accept for all patients. Patients express fear of odour, shame, increased self-consciousness and embarrassment and there is a trend that incontinent patients appear to benefit from support (Moore et al., 1999).

Pelvic floor muscle training (PFMT), biofeedback and electrical stimulation with a transcutaneous or a rectal electrode have been suggested to improve incontinence after prostate surgery (Hunter et al., 2004). The rationale for this treatment is that pelvic floor contraction may improve the strength of the external urethral sphincter during periods of increased abdominal pressure. PFMT results in hypertrophy of the striated muscles, increasing the external mechanical pressure on the urethra. Moreover, contraction of the pelvic floor leads to inhibition of detrusor contraction; therefore incontinence can be improved (Berghmans et al., 1998).

Incidence and pathophysiology

The incidence of incontinence after transurethral and open adenectomy is distinctly low and incontinence resolves in a few days or months. Initially incontinence rates around 9% are reported and about 1% at 12 months postoperatively (Lourenco et al., 2008; Milsom et al., 2009). Only Glazener et al. (2011) found higher numbers in a recent trial, with 17% of patients with incontinence at 6 weeks after transurethral prostatectomy (TURP) and still 10% of incontinent patients at 12 months. In general it is believed that incontinence is a troubling long-term problem in only a small proportion of patients after TURP (Van Kampen et al., 1997). The incidence of UI after RP varies widely. Immediately after catheter removal, continence rate is reported to be 10–41% after open RP (Van Kampen et al., 2000; Ficarra et al., 2009) and between 13.1% and 68.9% after robot-assisted laparoscopic prostatectomy (Tewari et al., 2003; Joseph et al., 2006; Menon et al., 2007; Ficarra et al., 2009). One year after RP, several reports from prestigious academic centres claimed that 95% of patients were continent (Walsh et al., 1994; Myers, 1995; Poon et al., 2000). However, other studies cast a rather more pessimistic light on the problem. They reported that 30–40% of the patients were wearing an incontinence pad one year or more after surgery (Basilis et al., 1995; Bishoff et al., 1998; Boccon-Gibod, 1997). Twelve months after surgery, 61–94% (open) (Coelho et al., 2010; Ficarra et al., 2012), 69–97% (robot) (Coelho et al., 2010; Ficarra et al., 2012) and 48–95% (laparoscopic) (Coelho et al., 2010) of patients have regained continence.

Figure 8.1 The prostate gland, hyperplasia, prostate cancer.
Several studies compared UI after open and robot RP. Different studies found that patients achieved continence much earlier after robot than after open RP (Ficarra et al., 2009), but other studies could not confirm this. Variation in reported frequency of incontinence depends on the definition of incontinence, the difference in outcome measures, various follow-up periods and the person (patient, physician, urologist or therapist) who assesses (Donnellan et al., 1997; Fowler et al., 1995; Moore et al., 1999).

Incontinence after adenectomy for prostate hyperplasia is mostly due to bladder dysfunction as bladder overactivity or poor compliance, more than sphincter injury. After RP, intrinsic sphincter deficiency is the primary cause of incontinence and ranges from 60 to 97% (Baert et al., 1996). An overlooked cause is detrusor overactivity. Outlet obstruction that results in overflow incontinence is rare (Foote et al., 1991; Baert et al., 1996; Gudziak et al., 1996; Haab et al., 1996; Grise and Thurman, 2001).

A small group of patients reported terminal and postmicturition dribble in the early postoperative period (Chang et al., 1998; Porru et al., 2001). This is due to urethral dysfunction because of decreased or absent post-void urethral milking resulting in residual unexpelled urine in the bulbous urethra (Wille et al., 2000).

Many risk factors were described that increase the possibility of UI after RP: previous transurethral resection, shortened functional urethral length, no preservation of the bladder neck, no preservation of the neurovascular bundles, higher age, less surgical expertise and more advanced clinical and pathological stage of the tumour (Aboseif et al., 1994; Eastham et al., 1996; Van Kampen et al., 1998).

**EVIDENCE FOR EFFECT OF PFMT IN PREVENTION AND TREATMENT OF URINARY INCONTINENCE**

We analysed literature on UI in men in order to generate clinical recommendations. Overall effectiveness of conservative management of postprostatectomy UI has
been widely investigated (Burgio et al., 1989; Meaglia et al., 1990; Ceresoli et al., 1995; Moul, 1998; Dorey, 2000). Symptoms of incontinence after prostatectomy tend to improve over time without intervention. The specific effectiveness of a physical therapeutic approach for incontinence after prostatectomy can only be evaluated in randomized controlled studies. Different types of intervention are described. PFMT involves any method of training the pelvic floor muscles including pelvic floor muscle exercises (PFME), biofeedback (BF) and electrical stimulation (ES). Biofeedback involves the use of a device to provide visual or auditory feedback. Electrical stimulation involves any type of stimulation by using a rectal probe or transcutaneous electrodes (Fig. 8.4). This method is used to facilitate awareness of contraction of the pelvic floor muscles or to inhibit detrusor contraction.

Although relaxation of the pelvic floor muscles is as important as contraction, up to now, no study has paid attention to that aspect of the therapy.

Moore et al. (2003), Hunter et al. (2004, 2007) and Campbell et al. (2012) have carried out Cochrane reviews concerning conservative management for postprostatectomy UI. Guidelines on UI were published by Lucas et al. (2012). There was a wide variation in outcome measures of incontinence. Assessment of incontinence was mostly based on the number of pads where 0 and 1 pad was defined as continent. Different pad-tests (20-, 40-, 60-minutes and 24- and 48-hour pad-test) were used to assess incontinence objectively (Moore et al., 2003; Hunter et al., 2004). Other assessments were voiding diaries for incontinent episodes, strength of the pelvic floor by digital test, visual analogue scale (VAS) and quality of life (QoL) questionnaires for incontinence (Herr, 1994; Laycock, 1994; Emberton et al., 1996). The American Urological Association Symptom Score (AUSS) and the International Prostate Symptom Score (IPSS) assess lower urinary tract symptoms (Barry et al., 1992).

Figure 8.3 Open (A), laparoscopic (B) and robot (C) radical prostatectomy.
Research methods

We identified 33 eligible trials considering physical therapy for men with incontinence after prostatectomy: two on physical therapy and incontinence after transurethral resection of the prostate, 30 on physical therapy and incontinence after radical prostatectomy and one on physical therapy and incontinence after transurethral resection and radical prostatectomy (Table 8.1). No abstracts were included. In only one study (Chang et al., 1998) patients of the control and the experimental group were not randomized. All other trials were randomized. The methodological quality of all identified studies concerning incontinence after prostatectomy based on PEDro ranged between 1 and 8 out of 10 (Table 8.2).

Results

The following hypotheses were tested for the role of PFMT in alleviating UI after adenectomy or radical prostatectomy:

**PFMT is better than no treatment or placebo (7 trials)**

In five trials (Chang et al., 1998; Van Kampen et al., 2000; Filocamo et al., 2005; Manassero et al., 2007) a significant difference in incontinence was found between the experimental and control group. In the first

Text continued on page 287

<table>
<thead>
<tr>
<th>Table 8.1 Randomized controlled studies of physical therapy for incontinence after prostatectomy</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Author</strong></td>
</tr>
</tbody>
</table>
| **Design** | 2-arm RCT  
Experimental group (E): preoperative PFMT+EMG BF  
Control group (C): preoperative information about PFMT |
| **Sample size and age (years)** | 100 men (E = 50, C = 50), mean age 59.3 in E and 60.9 in C |
| **Diagnosis** | Questionnaire UI, number of pads |
| **Training protocol** | E: one treatment 45 min preoperatively BF with surface electrodes + home exercises pre- and postoperatively  
C: information (written and brief verbal information) pre- and postoperatively + same home exercises as E  
Home: 10–15 contractions of 5–10 s, 4x/day |
| **Drop-out** | 3% |
| **Results** | No significant difference in incidence of incontinence (number of pads) between E and C group at 1–6 months after surgery (p between 0.271 and 0.648) |

| **Author** | Burgio et al., 2006 |
| **Design** | 2-arm RCT  
Experimental group (E): preoperative PFMT+EMG BF  
Control group (C): postoperative verbal information of surgeon |

(Continued)
### Table 8.1 Randomized controlled studies of physical therapy for incontinence after prostatectomy—cont’d

<table>
<thead>
<tr>
<th>Sample size and age (years)</th>
<th>125 men (E = 63, C = 62), mean age 60.7 in E and 61.1 in C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diagnosis</td>
<td>Number of leakage (diary), pad use, IIQ, QoL questionnaires</td>
</tr>
<tr>
<td>Training protocol</td>
<td>E: one treatment preoperatively BF with rectal probe + home exercises pre- and postoperatively</td>
</tr>
<tr>
<td></td>
<td>Home: 15 contractions of 2–10 s, 3x/day</td>
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<tr>
<td></td>
<td>C: brief verbal information to interrupt urine stream postoperatively once a day</td>
</tr>
<tr>
<td>Drop-out</td>
<td>10% after surgery; 18% after 6 months</td>
</tr>
<tr>
<td>Results</td>
<td>Significant difference in duration of incontinence (number of leakage) between E and C group at 6 months after surgery (p = 0.04); number of patients wearing pads (p &lt; 0.05)</td>
</tr>
<tr>
<td>Author</td>
<td>Centemero et al., 2010</td>
</tr>
</tbody>
</table>

#### Design
2-arm RCT
Experimental group (E): preoperative and postoperative PFME
Control group (C): postoperative PFME

#### Sample size and age (years)
118 men (E = 59, C = 59), median age 60.5 in E and 57.5 in C

#### Diagnosis
Self-reported continence, ICS male SF score, 24-h pad-test

#### Training protocol
E: 8 guided PFME preoperatively + 30 days home exercises preoperatively and 8 guided PFME during 1 month after catheter removal, at home; PFME postoperatively till continence, 30 min daily
C: same postoperative programme

#### Drop-out
0%

#### Results
Significant difference in incontinence between E and C group:
- at 1 and 3 months after surgery (p = 0.018/0.028) for self-reported continence
- at 1 and 3 months after surgery (p = 0.002/0.002) for ICS male SF score
- at 1 and 3 months after surgery (p = 0.040/0.033) for 24-h pad-test

#### Author
Chang et al., 1998

<table>
<thead>
<tr>
<th>Design</th>
<th>2-arm CT not randomized</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample size and age (years)</td>
<td>50 men after TURP (E = 25, C = 25) mean age 65 in E (range 51–74) and 66 in C (range 45–79)</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Digital evaluation for strength of pelvic floor (0–4), questionnaires, voiding diary, uroflow</td>
</tr>
<tr>
<td>Training protocol</td>
<td>E: PFMT 4 weeks</td>
</tr>
<tr>
<td></td>
<td>Home: 30 exercises 4x/day</td>
</tr>
<tr>
<td></td>
<td>C: no treatment</td>
</tr>
<tr>
<td>Drop-out results</td>
<td>Not reported</td>
</tr>
<tr>
<td>Results</td>
<td>Significant difference in strength of pelvic floor between E and C group only at week 4 (p &lt; 0.05)</td>
</tr>
<tr>
<td></td>
<td>Significant difference in length of between void interval between E and C group at week 1 till 4 (p &lt; 0.01)</td>
</tr>
<tr>
<td></td>
<td>Significant difference in incontinence between E and C group at week 3 and 4 (p &lt; 0.05)</td>
</tr>
<tr>
<td></td>
<td>Significant difference in terminal dribbling between E and C group at week 4 (p &lt; 0.05)</td>
</tr>
<tr>
<td></td>
<td>Significant difference in QoL between E and C group at week 4 (p &lt; 0.01)</td>
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<tr>
<td></td>
<td>No significant difference in uroflow between E and C group at week 4</td>
</tr>
</tbody>
</table>

#### Author
Dubbelman et al., 2010

<table>
<thead>
<tr>
<th>Design</th>
<th>2-arm RCT</th>
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<tbody>
<tr>
<td>Sample size and age (years)</td>
<td>79 men (E = 35, C = 44), median age 64 in E and in C</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>24-h pad-test (&lt;4 g = continent) and 1-h pad-test (&lt;1 g = continent)</td>
</tr>
<tr>
<td>Training protocol</td>
<td>E: maximum 9 guided PFME 30 min sessions postoperatively at week 2, 3, 4, 6, 8, 12, 16, 20 and 26 + 150 home exercises daily</td>
</tr>
<tr>
<td>Drop-out results</td>
<td>C: verbal instructions and information folder of PFME</td>
</tr>
<tr>
<td>Results</td>
<td>13/79</td>
</tr>
</tbody>
</table>
Table 8.1 Randomized controlled studies of physical therapy for incontinence after prostatectomy—cont’d

| Results | No significant difference in incontinence between E and C group: at 1, 4, 8, 12 and 26 weeks after catheter removal for 24-h pad-test at 1, 12 and 26 weeks for 1-h pad-test |
| Author | Filocamo et al., 2005 |
| Design | 2-arm RCT |
| Experimental group (E): PFME programme |
| Control group (C): no instructions in PFME |
| Sample size and age (years) | 300 men after RP (E = 150, C = 150), mean age for E = 65.0 (range 51–75) and C = 66.8 (range 45–75) |
| Diagnosis | 1-h pad-test, 24-h pad test, number/pads daily, ICS-male questionnaire |
| Training protocol | PFME started after catheter removal (Kegel exercises only, no rectal ES or BF). Contractions were evaluated by digital anal control. At home (10 contractions of 5s and 10s rest in between). PFME in all positions, PFME before any effort or activity that might induce UI |
| Drop-out | 2/300 (1%) |
| Results | Significantly more patients in the E group were continent at 1 and 6 months after surgery compared to the C group. Patient age correlated with continence in the E group, but not in the C group. 93.3% of the total population achieved continence after 1 year |
| Author | Floratos et al., 2002 |
| Design | 2-arm RCT |
| Experimental group (E): PFMT + EMG BF |
| Control group (C): verbal instructions about PFMT |
| Sample size and age (years) | 42 men (E = 28, C = 14), mean age 63.1 in E (SD = 4) and 65.8 in C (SD = 4.3) |
| Diagnosis | ICS 1-h pad-test and questionnaire |
| Training protocol | E: 15 sessions EMG BF with surface electrodes, 3x/week, 30 min, at home: 50 to 100 contractions/day |
| C: verbal instructions on PFMT, 1 session anal control; at home: 80–100 contractions/day 3–5s with submaximal strength of 70% |
| Drop-out | 0% |
| Results | No significant difference in incontinence (ICS 1-h pad-test and number of pads) between E and C group at baseline 1, 2, 3 and 6 months after surgery (p > 0.05) |
| Author | Franke et al., 2000 |
| Design | 2-arm RCT |
| Experimental: PFMT + BF |
| Control: no treatment |
| Sample size and age (years) | 30 men (E = 15, C = 15); mean age E = 62.3; C = 60.7 |
| Diagnosis | Voiding diary, 48-hour pad-test |
| Training protocol | Experimental: 5 sessions of 45 min BF behavioural therapy |
| Control: no therapy |
| Drop-out | 6 at 6 weeks, 7 at 12 weeks, 15 at 24 weeks |
| Results | No significant difference E and C group in pad-test and incontinence episodes at 6 weeks, 3 and 6 months |
| Author | Geraerts et al., 2013 |
| Design | 2-arm RCT |
| Experimental group (E): pre- and postoperative PFME + BF |
| Control group (C): postoperative PFME + BF |
| Sample size and age (years) | 180 men (E = 91, C = 89); mean age E = 61.8; C = 62.0 |
| Diagnosis | Test 24 h (0 g 3 days = continent) and 1-h pad-test, VAS, IPSS, KHQ |

(Continued)
Table 8.1 Randomized controlled studies of physical therapy for incontinence after prostatectomy—cont’d

<table>
<thead>
<tr>
<th>Training protocol</th>
<th>E: preop: 3 guided PFME + BF preoperatively + 21 days home: 60 exercises/day preoperatively, postoperative after catheter removal: weekly guided PFME + BF till continence</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Home: PFME 10 contractions 1 s, 10 contractions 10 s, 3x daily</td>
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<tr>
<td></td>
<td>C: same postoperative programme</td>
</tr>
<tr>
<td>Drop-out</td>
<td>5%</td>
</tr>
<tr>
<td>Results</td>
<td>No significant difference E and C group in time to continence by pad-test, and incontinence episodes at 1, 3, 6 and 12 months after catheter removal</td>
</tr>
<tr>
<td>Author</td>
<td>Glazener et al., 2011</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Design</th>
<th>2 two-arm RCTs (trial 1: men after RP; trial 2: men after TURP)</th>
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<tbody>
<tr>
<td>Sample size and age (years)</td>
<td>Men incontinent after RP (trial 1), TURP (trial 2)</td>
</tr>
<tr>
<td>Trial 1: n = 411/1158 (E = 205, C = 206); mean age for E = 62.4 (SD = 5.8) and C = 62.3 (SD = 5.6)</td>
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<tr>
<td>Trial 2: n = 442/5986 (E = 220, C = 222); mean age for E = 68.2 (SD = 7.7) and C = 67.9 (SD = 8.1)</td>
<td></td>
</tr>
<tr>
<td>Diagnosis</td>
<td>ICIQ-UI SF questionnaire, measure of cost-effectiveness (QALY), use of pads and catheters, day and night urinary frequency and UI, EQ-5D and SF-12 (QoL)</td>
</tr>
<tr>
<td>Training protocol</td>
<td>E: one-to-one therapy sessions including PFMT and BT if OAB/urgency symptoms + PFMT and lifestyle leaflet (4 treatment sessions in 3 months starting 6 weeks after surgery)</td>
</tr>
<tr>
<td>Drop-out</td>
<td>Trial 1: 20/411 (5%); trial 2: 45/442 (10%)</td>
</tr>
<tr>
<td>Results</td>
<td>Trial 1: the rate of UI did not significantly differ between E and C at 12 months after surgery. There were no significant differences in the prevalence of UI or the mean ICIQ score between the groups at any of the time points</td>
</tr>
<tr>
<td>Author</td>
<td>Goode et al., 2011</td>
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<table>
<thead>
<tr>
<th>Design</th>
<th>3-arm RCT</th>
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<tr>
<td>Experimental (E):</td>
<td>E1: 8 weeks of behavioural therapy (PFMT, bladder control strategies)</td>
</tr>
<tr>
<td>Sample size and age (years)</td>
<td>208 men with UI, 1–17 years after RP (age 51–84), (E1 = 70, E2 = 70, C = 68)</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Percentage reduction in mean number of UI episodes after 8 weeks of treatment (7-day bladder diaries), AUA-7 symptom index, IPSS-QoL question, IQ, EPIC, SF-36, global perception of improvement and the patient satisfaction question</td>
</tr>
<tr>
<td>Training protocol</td>
<td>The behavioural therapy (PFMT, bladder control strategies) consisted of (4 visits, ± 2 weeks apart): explanation of anatomy and PFME (anal palpation); home exercises included 3 daily sessions (lying, sitting and standing position) with 15 repetitions of a 2–10 s contraction and an equal relaxation period. The contraction and relaxation duration was advanced by 1 second each week to a max of 10–20 s. Once-daily participants had to interrupt voiding for the first 2 weeks. Participants kept daily bladder diaries and exercise logs during 8 weeks of treatment. Patients received a fluid management handout. Strategies to avoid stress and urge urinary incontinence were clarified</td>
</tr>
<tr>
<td>Drop-out</td>
<td>32/208 (15%)</td>
</tr>
<tr>
<td>Results</td>
<td>Mean incontinence episodes decreased significantly more in E1 and E2 compared to C. There was no significant difference in incontinence reduction between treatment groups. Improvements were durable to 12 months in the active treatment groups</td>
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<tr>
<td>Author</td>
<td>Ip, 2004</td>
</tr>
<tr>
<td>Design</td>
<td>2 arm-RCT</td>
</tr>
<tr>
<td>Sample size</td>
<td>16 men with UI after TURP or RP</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Self-developed questionnaire and St George Urinary Incontinence Score</td>
</tr>
<tr>
<td>Training protocol</td>
<td>E: non-guided PFMT with information on a refrigerator magnet. PFMT: 6 contractions of 5 s contraction, 5 s rest; 6 times a day</td>
</tr>
<tr>
<td>Drop-out</td>
<td>2/16 after 2 weeks; 0/16 after 3 months</td>
</tr>
<tr>
<td>Results</td>
<td>Unable to conclude that men in magnet group had a higher compliance with PFMT when compared with the paper copy group</td>
</tr>
<tr>
<td>Author</td>
<td>Liu et al., 2008</td>
</tr>
<tr>
<td>Design</td>
<td>2 arm-RCT</td>
</tr>
<tr>
<td>Sample size</td>
<td>24 men with UI after RP</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>QoL scale and ICIQ-SF questionnaire</td>
</tr>
<tr>
<td>Training protocol</td>
<td>E: ExMI10Hz for 10 min, followed by 3 min of rest and a 2nd treatment of 50Hz for 20 min, 2x/week</td>
</tr>
<tr>
<td>Drop-out</td>
<td>No information</td>
</tr>
<tr>
<td>Results</td>
<td>At 1 month after surgery the QoL and ICIQ-SF scores were ameliorated in both groups, but did not significantly differ. At 3 and 6 months, E had a significantly better score than C</td>
</tr>
<tr>
<td>Author</td>
<td>Manassero et al., 2007</td>
</tr>
<tr>
<td>Design</td>
<td>2-arm RCT</td>
</tr>
<tr>
<td>Sample size and age (years)</td>
<td>107 incontinent (24-h pad-test &gt;2 g) men after retropubic RP with bladder neck preservation (E = 54, C = 53); mean age 66.8 (SD = 6.3) in E and 67.9 (SD = 5.5) in C</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>24-h pad-test, VAS, a single question of QoL</td>
</tr>
<tr>
<td>Training protocol</td>
<td>E: a pelvic floor re-education programme for as long as any degree of UI persisted (within a 1 year period). The programme involved active PFE with verbal feedback. In case of weak PFM, ES was given at home with an anal probe. Home practice comprised 45 contractions (3 sessions of 15) per day at home, progressively increasing until 90 per day</td>
</tr>
<tr>
<td>Drop-out</td>
<td>13/107 (12%)</td>
</tr>
<tr>
<td>Results</td>
<td>The overall spontaneous continence rate after catheter removal was 23.6%. The proportion of men still incontinent was significantly higher in C compared to E at 1, 3, 6 and 12 months after surgery. VAS and QoL also significantly differed between E and C at 12 months after RP</td>
</tr>
<tr>
<td>Author</td>
<td>Marchiori et al., 2010</td>
</tr>
<tr>
<td>Design</td>
<td>2-arm RCT</td>
</tr>
<tr>
<td>Experimental group (E): intensive PFMT + BF + ES for 2–3 weeks on daily basis</td>
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<tr>
<td>Control group (C): PFMT teaching and oral advice</td>
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</table>

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### Table 8.1 Randomized controlled studies of physical therapy for incontinence after prostatectomy—cont’d

<table>
<thead>
<tr>
<th>Study</th>
<th>Sample size and age (years)</th>
<th>Diagnosis</th>
<th>Training protocol</th>
<th>Drop-out</th>
<th>Results</th>
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<tr>
<td>Mariotti et al., 2009</td>
<td>332 incontinent (&gt;1 pad/daily) men at 30 days after radical prostatectomy (E = 166, C = 166), mean age 67 years in (E) and 66.5 years in (C)</td>
<td>ICIQ-male, RAND 36-item health survey, use of pads</td>
<td>(E): intensive PFMT + BF teaching of correct contraction, 10 sets of ES of 15 minutes each; for 2–3 weeks on daily basis</td>
<td>Not mentioned</td>
<td>Patients enrolled in the (E) group achieved continence earlier than the (C) group (44±2 days versus 76±4 days)</td>
</tr>
<tr>
<td>Mathewson-Chapman, 1997</td>
<td>60 men (E = 30, C = 30), median age 61 in E and in C</td>
<td>24-h pad-test (2 g or less = continent), number of pads and ICS questionnaire</td>
<td>E: BF (15 min) and ES (20 min: 30 Hz 10 min and 50 Hz 10 min) 12 sessions 2x/week, start 7 days postoperatively home exercises daily</td>
<td>No</td>
<td>Significant difference in incontinence between E and C group: at 2, 4 weeks and 2, 3, 4, 5, 6 months after catheter removal for 24-h pad-test and number of pads (p &lt; 0.05)</td>
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<tr>
<td>Moore et al., 1999</td>
<td>53 men (E = 27, C = 26), mean age 60</td>
<td>Voiding diary, perineal muscle strength; number of pads</td>
<td>E: information preoperatively PFME, home exercises with BF</td>
<td>4%</td>
<td>No significant difference between E and C group in number of pads</td>
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<tr>
<th>Study</th>
<th>Sample size and age (years)</th>
<th>Diagnosis</th>
<th>Training protocol</th>
<th>Drop-out</th>
<th>Results</th>
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Table 8.1 Randomized controlled studies of physical therapy for incontinence after prostatectomy—cont’d

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<th>Diagnosis</th>
<th>Training protocol</th>
<th>Drop-out</th>
<th>Results</th>
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<tbody>
<tr>
<td>Moore et al., 2008</td>
<td>2-arm RCT</td>
<td>205 men (E = 106, C = 99), mean age?</td>
<td>24-hour pad-test (≤ 8g = continent) and questionnaires (Incontinence Impact Questionnaire IIQ7 and IPSS) and perception of urine loss as a problem preoperatively and at week 4, 8, 12, 16, 28, 52 after surgery</td>
<td>E: information (written and verbal information) 4 weeks postoperatively; treatment 30min PFMT and BF in outpatient clinic weekly till continence but for maximum 24 weeks+home exercises C: written and brief verbal information+same home exercises Home exercises: 10–12 contractions 3x/day</td>
<td>8%</td>
<td>No significant difference in incontinence between E and C group for all assessments</td>
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<tr>
<td>Nilssen et al., 2012</td>
<td>2-arm RCT, same study as Overgard</td>
<td>85 men (E = 42, C = 43), median age 60 in E and 62 in C</td>
<td>QoL: UCLA-PCI, SF-12</td>
<td>E: guided PFME 45min session, once weekly starting immediately after catheter withdrawal till continence; contractions of 6–8s; 30 home exercises daily. Training recorded in training diary. C: verbal instructions and written information on PFME</td>
<td>5.9%</td>
<td>No significant difference in HR QoL between E and C group at 6 weeks, 3, 6 and 12 months after surgery</td>
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<tr>
<td>Overgard et al., 2008</td>
<td>2-arm RCT</td>
<td>85 men (E = 42, C = 43), median age 60 in E and 62 in C</td>
<td>Number of pads (0 = continent), self-reported continence, 24-h pad-test (&lt; 2 g = continent) and muscle strength by anal pressure (cmH2O)</td>
<td>E: guided PFME 45min session, once weekly, starting immediately after catheter withdrawal till continence; contractions of 6–8s; 30 home exercises daily. Training recorded in training diary. C: verbal instructions and written information on PFME</td>
<td>5/85</td>
<td>No significant difference in incontinence between E and C group: at 1, 4, 8, 12 and 26 weeks after catheter removal for number of pads and self-report, 24-h pad-test, strength; at 1 year Significant difference in incontinence between E and C group (p = 0.028) only on number of pads and self-report</td>
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<td>Parekh et al., 2003</td>
<td>2-arm RCT</td>
<td>38 men (E = 19, C = 19), mean age 61.6 in E and 55.5 in C</td>
<td>Number of pads/day at 6, 12, 16, 20, 28 and 52 weeks</td>
<td>E: two treatments pre- and postoperatively 1x/3 weeks for 3 months PFME (+ BF depending the patient), exercises described C: no formal education on PFMT Home: 6 months or longer functional re-training (2x/d)</td>
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<tr>
<td>Author</td>
<td>2012 Park et al.</td>
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<td>2-arm RCT</td>
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<tr>
<td>Sample size and age (years)</td>
<td>66 men (E = 33, C = 33), mean age 69 in E and in C</td>
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<tr>
<td>Diagnosis</td>
<td>Physical function (functional fitness: sit-ups, grip strength, flexibility and balance ability: chair stand); continence and QoL (24-hour pad-test (&lt;1 g = continent), continence rate, ICIQ, Beck Depression Inventory, SF-36</td>
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<td>Training protocol</td>
<td>Assessment: preoperatively, at start of treatment and after 12 weeks</td>
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<td>Results</td>
<td>Significant difference in achievement of continence between E and C group at week 12 (p &lt; 0.05)</td>
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<td>Significant difference in median time to regain continence between E and C group (p &lt; 0.05)</td>
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<td>Design</td>
<td>2-arm RCT</td>
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<tr>
<td>Sample size and age (years)</td>
<td>58 men after TURP (E = 30, C = 28), mean age 67.5 in E (range 55–73) and 66 in C (range 53–71)</td>
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<td>Diagnosis</td>
<td>Questionnaires of LUTS and QoL, voiding diary and post-micturition dribble, digital test for strength of PFM</td>
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<td>Training protocol</td>
<td>E: 4 treatments postoperatively 1x/week PFMT+home exercises</td>
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<tr>
<td>Results</td>
<td>Significant difference in functional physical fitness (p &lt; 0.001), flexibility (p = 0.027) and balance ability (p = 0.015). NS for grip strength (p = 0.49)</td>
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<tr>
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<td>Significant difference in 24-hour pad-test in favour of E group at week 12 (p = 0.02) and ICIQ (p = 0.03)</td>
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<td>Significant difference in SF-36 in favour of E group (p &lt; 0.01)</td>
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<td>Design</td>
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<td>Sample size and age (years)</td>
<td>73 men (E = 36, C = 37), mean age 62.2 in E and 65.6 in C</td>
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<td>Diagnosis</td>
<td>Questionnaires of LUTS and QoL, voiding diary and post-micturition dribble, digital test for strength of PFM</td>
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<tr>
<td>Training protocol</td>
<td>E: PFMT and BF</td>
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<tr>
<td>Results</td>
<td>Significant difference in functional physical fitness (p &lt; 0.001), flexibility (p = 0.027) and balance ability (p = 0.015). NS for grip strength (p = 0.49)</td>
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<td>Significant difference in 24-hour pad-test in favour of E group at week 12 (p = 0.02) and ICIQ (p = 0.03)</td>
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<td>Significant difference in SF-36 in favour of E group (p &lt; 0.01)</td>
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**Table 8.1 Randomized controlled studies of physical therapy for incontinence after prostatectomy—cont’d**
Male pelvic floor dysfunctions and evidence-based physical therapy

### Table 8.1 Randomized controlled studies of physical therapy for incontinence after prostatectomy—cont’d

| Diagnosis | Incontinence severity measured by 24-h pad-test; continence = use of 1 pad or less daily. Questionnaires (ICS male short form and ICIQ and PFM strength by Oxford scale at 1, 3, 6 and 12 months after surgery) |
| Training protocol | E: information (written and verbal information) 4 weeks postoperatively; treatment 30 min PFMT and BF in outpatient clinic from postop day 15, weekly till continence but with a maximum of 3 months + home exercises. Exercises in right lateral decubitus: 3 series of 10 rapid contractions, 3 sustained contractions of 5, 7 or 10 s; supine position: 10 contractions during prolonged expiration. Home exercises: 10–12 contractions 3x/day while lying, sitting and standing |
| Drop-out | C: brief verbal information from the urologist to contract the pelvic floor |
| Results | Significant difference in incontinence between E and C group for all incontinence assessments and strength (p < 0.05). For QoL only significant difference at 1 month |
| Author | Robinson et al., 2008 |

### Design
- **Experimental group (E):** one preoperative and one session of PFMT at 1 month after surgery
- **Control group (C):** (E) + additionally 4 BF sessions immediately after catheter withdrawal
- **Size of sample**
  - 126 men (E = 62, C = 64)

### Diagnoses
- **LUTS intensity, LUTS distress and health-related quality of life**

### Training protocol
- **E:** brief verbal instruction in PFMT before surgery and one BF session at 2 months after surgery plus PFMT for 4 weeks with BF immediately after catheter withdrawal
- **C:** brief verbal instruction in PFMT before surgery and one BF session at 2 months after surgery

### Drop-out
- Not mentioned

### Results
- Both groups reported steady declines in intensity and distress associated with LUTS, but no between-group differences were reported. No between-group differences were found in impact on health-related quality of life

### Author
- Sueppel et al., 2001

### Design
- **Experimental group (E):** preoperative instructions about PFMT and one session PFMT + BF with rectal probe + PFMT + BF 6 weeks postop and 3, 6, 9, 12 months after surgery + home exercises
- **Control group (C):** PFMT + BF 6 weeks postop and 3, 6, 9, 12 months after surgery + home exercises
- **Sample size and age (years):**
  - 16 men (E = 8, C = 8), mean age 61.8 years in E (range 45–69) and 61.1 year in C (range 55–69)

### Diagnosis
- **Pad-test 45 min, bladder diary, number of incontinence episodes, number of pads/day, QoL, AUA and leakage index before surgery and at 6 weeks, 3, 6, 9, 12 months after surgery + home exercises**

### Training protocol
- **E:** information on PFMT (written and brief verbal information) and PFMT + BF with rectal pressure probe pre- and postoperatively 6 weeks, 3, 6, 9, 12 months after surgery + home exercises
- **C:** PFMT + BF with rectal pressure probe 6 weeks, 3, 6, 9, 12 months after surgery + same home exercises

### Drop-out
- Not reported

### Results
- Only descriptive statistics but better improvement in incontinence in E group

### Author
- Tienforti et al., 2012

### Design
- **Experimental group (E):** 1 preoperative PFMT + EMG BF
- **Control group (C):** pre- and postoperative verbal information
- **Sample size and age (years):**
  - 34 men (E = 17, C = 17), mean age 64 in E and 67 in C

### Diagnosis
- **ICIQ-UI; incontinence episodes, pad use; ICIQ-OAB, IPSS-QoL**

(Continued)
## Table 8.1 Randomized controlled studies of physical therapy for incontinence after prostatectomy—cont’d

| Training protocol | E: one treatment session preoperatively PFME+BF+home exercises pre- and postoperatively Home: 15 contractions of 2–10 s, 3x/day Postop: monthly treatment session+home exercises C: oral and written instructions for PFME | Drop-out Results | 5.8% Difference in incontinence between E and C group at 1, 3 and 6 months after catheter removal (p = 0.02/0.01/ 0.02) for ICIQ-UI; at 3 and 6 months after catheter removal (p < 0.04) for incontinence episodes, pad use, ICIQ-OAB No significant difference in QoL between E and C group at 1, 3 and 6 months after catheter removal |
| Author | Van Kampen et al., 2000 |
| Design | 2-arm RCT Experimental group (E): PFMT+BF Control group (C): placebo treatment |
| Sample size and age (years) | 102 men (E = 50, C = 52), mean age 64.36 in E (SD = 0.81) and 66.58 in C (SD = 0.80) |
| Diagnosis | 24- and 1-hour pad-test, VAS, voiding volume charts, IPSS, number of pads/day (0 pads = continent) |
| Training protocol | E: treatment 30 min PFME and BF in outpatient clinic once a week till continence+home exercises C: 30 min placebo ES, once a week till continence Home: total of 90 contractions/day: 40 contractions of 1 s and 50 contractions of 10 s/day in supine, sitting or standing position |
| Drop-out Results | 4% Significant difference between E and C group in duration and degree of incontinence at 1, 6 and 12 months after RP |
| Author | Wille et al., 2003 |
| Design | 3-arm RCT Experimental group 1 (E1): information+PFMT and ES Experimental group 2 (E2): information+PFMT+ES+BF Control group (C): information about PFME |
| Sample size and age (years) | 139 men (E1 = 46, E2 = 46, C = 47), mean age 64.6 in E1 and E2 and 65.9 in C |
| Diagnosis | 20-min pad-test, diary with number of pads/day (≤ 1 pad/day = continent) and urine symptom inventory at baseline, 3,12 months |
| Training protocol | Started after catheter withdrawal, duration: 3 months E1: PFMT+ES: ES = surface electrodes, 27 Hz, biphasic 1 s bursts, 5 s pulse width and 2 s pulse trains, 15 min 2x/day home device E2: PFMT, ES (5 s stimulation time, 5 s contracting and 15 s relaxing), BF: 15 min 2x/day same home device C: information (written and brief verbal information) and 3 days therapy 20–30 min+home exercises (2x/day for 3 months) |
| Drop-out Results | Not specified No significant difference between E1, E2 and C group in incontinence at 3 and 12 months (Questionnaire: p = 0.8 at 3 months, 0.5 at 12 months; pad-test: p = 0.5 at 3 months, 0.2 at 12 months) |
| Author | Yamanishi et al., 2010 |
| Design | 2-arm RCT Experimental group (E): PFMT+ES Control (C): PFMT+sham ES |
| Sample size and age (years) | 56 men with severe UI after RP, mean 66.6 (SD = 6.2); E: n = 26; C: n = 30 |
| Diagnosis | 3-day pad-test, ICIQ-SF, KHQ |
Table 8.1 Randomized controlled studies of physical therapy for incontinence after prostatectomy—cont’d

| Training protocol | E: standard PFMT + ES (50 Hz square waves with a 300 μs pulse duration and a maximum output of 70 mA (5 s on, 5 s off))  
C: standard PFMT + sham ES (50 Hz square waves with a 300 μs pulse duration and a maximum output of 3 mA (2 s on, 13 s off))  
9/56 (16%)  
Drop-out Results | There was a significant difference in the number of continent patients between both groups at 1, 3 and 6 months. The time to achieve continence was significantly shorter in E than in C (2.7 ± 2.6 months vs 6.8 ± 3.9 months)  
Changes in the amount of leakage, the ICIQ-SF and the KHQ score were significantly larger in E at 1 month compared to C, but there was no difference at 12 months |
| Author | Yokoyama et al., 2004 |
| Design | 3-arm RCT  
Experimental group 1 (E1): FES  
Experimental group 2 (E2): ExMI  
Control (C): PFMT |
| Sample size and age (years) | 36 (12 patients each in the FES, ExMI and PFMT group)  
Mean age was 67.2, 68.2 and 66.2 respectively |
| Diagnosis | Bladder diaries, 24-h pad weight testing, a validated QoL survey |
| Training protocol | E1: FES with an anal probe, 20 Hz square waves (300 μs pulse duration and a maximal output current of 24 mA), 15 min, 2x/day for 1 month  
E2: 10 Hz, intermittently for 10 min, rest of 2 min, and a 2nd treatment at 50 Hz intermittently for 10 min. Treatment session duration was 20 min, 2x/week for 2 months  
C: PFMT with anal digital palpation, verbal and written instructions for home practice of these exercises were given to the patients. |
| Drop-out Results | Not reported  
The leakage weight during the 24 hours after removing the catheter was not significantly different between groups  
At 1 month, the leakage weight significantly differed between E1 and C and at 2 months between E2 and C. At 6 months after surgery the average 24-h leakage weight was <10 g in all groups. QoL measures decreased after surgery, but gradually improved over time in all groups |
| Author | Zhang et al., 2007 |
| Design | 2-arm RCT for patients who were incontinent longer than 6 months with an average of 18–21 months after RP  
Experimental group (E): postoperative PFME + BF one session of 45 min + groups meetings  
Control group (C): PFME + BF one session, PFME at home |
| Sample size and age (years) | 29 men (E = 14, C = 15), mean age 62 in E and 61 in C |
| Diagnosis | VAS and use of pads; Prostate Cancer Index and AUASI, Illness Intrusiveness Rating Scale (IIRS) at start and after 3 months |
| Training protocol | E: postoperative PFME + BF with rectal sensor, one session postoperatively of 45 min by a physical therapist, six biweekly meetings over 3 months by psychologist + home exercises 2–3 times a day for 5–10 min  
C: postoperative PFME + BF one session of 45 min + home exercises same as E group  
2/29 (6%) |
| Drop-out Results | Significant difference in continence VAS in favour of E group after 3 months (p = 0.001)  
Borderline significant difference in use of pads between E and C group (p = 0.057)  
Significant difference in QoL in favour of E group after 3 months (p = 0.037)  
Compliance of PFME: 86% in E group, 46% in C group |

For abbreviations, see text.
Table 8.2 PEDro quality score of (R)CTs in systematic review of physical therapy for incontinence after prostatectomy

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Evidence-Based Physical Therapy for the Pelvic Floor

Chapter

Male pelvic floor dysfunctions and evidence-based physical therapy

In all studies, pelvic floor exercises were started within 7 days after catheter withdrawal. The treatment of Chang et al. (1998) and Goode et al. (2011) took 4 and 8 weeks, respectively. In the studies of Van Kampen et al. (2000), Manassero et al. (2007) and Filocamo et al. (2005) patients were treated as long as any degree of incontinence persisted, with a time frame of 1 year.

In two trials (Franke et al., 2000; Glazener et al., 2011) no significant difference between the experimental and control group was found. Franke et al. started 6 weeks postoperatively with pelvic floor exercises and biofeedback and five sessions were given. Remarkable was the high rate of drop-outs in this study. Glazener et al. (2011) included one group after TURP and one group after RP. The authors gave 1–4 sessions with a therapist over 3 months to the patients in the experimental group.

Preoperative and postoperative PFMT is better than only postoperative PFMT (6 trials)

Six studies attempted to investigate the effect of preoperative PFMT on the duration, and in some studies on the severity, of UI after RP. Four studies found positive results of preoperative PFMT (Sueppel et al., 2001; Burgio et al., 2006; Centemero et al., 2010; Tienforti et al., 2012). One study (Parekh et al., 2003) found a significant difference in incontinence at 3 months after RP in favour of the early start of PFMT but not at 1 year. Only one study (Geraerts et al., 2013) could not find in favour of early PFMT. However, due to the multitude of existing bias, results must be interpreted with caution. Parekh et al., Burgio et al and Tienforti et al altered both pre- and postoperative treatment, which made defining the effect of preoperative PFMT impossible. Sueppel et al compared only one preoperative session with a control group who completed PFMT 6 weeks after surgery and only included 16 patients. Furthermore, follow-up was usually only 3 or 6 months. Finally, a wide range of continence criteria was used among studies, which made it difficult to compare results.

Only Centemero et al and Geraerts et al gave the control and the experimental group the same postoperative therapy. Centemero et al found a positive effect in favour of the experimental group, Geraerts et al found no significant difference, while three preoperative sessions were given to the experimental group. Both studies started postoperative training directly after catheter removal.

Preoperative PFMT and biofeedback is better than only preoperative information about PFMT (1 trial)

Bales et al. (2000) could not find a significant difference in incontinence postoperatively by adding one session of EMG biofeedback 2–4 weeks before surgery. In the control group only information on PFMT was given. Both groups had to do home exercises both pre- and postoperatively. The authors suggested that instead of one biofeedback

<table>
<thead>
<tr>
<th>Study</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
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<td>Zhang, 2007</td>
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<td>+</td>
<td>–</td>
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<td>+</td>
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<td>–</td>
<td>4</td>
</tr>
</tbody>
</table>

+, criterion is clearly satisfied; –, criterion is not satisfied; ?, not clear if the criterion was satisfied. Total score is determined by counting the number of criteria that are satisfied, except that ‘eligibility criteria specified’ score is not used to generate the total score. Total scores are out of 10.
training, more intensive biofeedback training might have led to a better outcome.

Postoperative PFMT is better than only information about PFMT before and after surgery (11 trials)

In one study (Porru et al., 2001), one group received PFMT after TURP for 4 weeks. The control group was only given information about PFMT before and after surgery. A significant difference in incontinence episodes was found between the experimental and control group at 1, 2 and 3 weeks after surgery but not at 4 weeks. The authors concluded that early pelvic floor exercises should be recommended to all cooperative patients after TURP.

Four trials could not find results in favour of a training programme compared with information (Moore et al., 1999, 2008; Wille et al., 2003; Dubbelman et al., 2010). The study of Overgard et al. (2008) is the only study that found results in favour of PFMT for incontinence at 1 year, while most patients benefit from PFMT in the early period after catheter removal. However Nilssen et al. (2012) demonstrated, using the same study population, that QoL was not affected by guided exercises. Four studies (Zhang et al., 2007; Mariotti et al., 2009; Marchiori et al., 2010; Ribeiro et al., 2010) proved that a structured programme decreased the duration of incontinence significantly.

Adding biofeedback to PFMT is better than PFMT alone or information alone (4 trials)

Four trials (Mathewson-Chapman, 1997; Floratos et al., 2002; Wille et al., 2003; Robinson et al., 2008) could not prove an additional effect by adding biofeedback to exercises alone or verbal instructions only.

Adding rectal stimulation to PFMT is better than PFMT alone or information alone (5 trials)

Two trials (Moore et al., 1999; Wille et al., 2003) could not prove any additional effect by adding electrical stimulation to exercises alone or instructions only. One trial (Yamanishi et al., 2010) could not confirm this result and demonstrated a shorter time to continence in the experimental group. Two trials (Yokoyama et al., 2004; Liu et al., 2008) examined the effect of extracorporeal magnetic innervation (ExMI) compared to functional electrostimulation (FES) and/or standard PFMT. Liu et al found a significant improvement in quality of life and ICIQ score at 3 and 6 months after surgery. Yokoyama et al indicated a significant effect of FES and ExMI compared to standard PFMT, but not between FES and ExMI.

PFMT with adherence strategies give better continence results (1 trial)

Methods to increase adherence with the exercises in male patients are rarely described. Only one study compared two different methods to improve compliance (Ip, 2004). The purpose of this study was to validate a new education tool, a refrigerator magnet, in comparison to a paper copy with the same information, to determine if patient compliance with the exercises increased. Results of this study made it unable to conclude that men in the magnet group had a higher compliance with PFMT when compared to the paper copy group. The patient group was very small, no statistical data were available and the methodological quality of the study was very low.

Adding biofeedback and electrostimulation to PFMT gives better results than PFM exercises alone (1 trial)

Goode et al. (2011) found no additional effect of supplementary biofeedback and electrostimulation to PFMT concerning the reduction of incontinence.

Adding general exercises to PFMT for incontinence after surgery is better than PFMT alone (1 trial)

Park et al. (2012) investigated the additional effect of general exercises to PFMT in patients after RP. Both the physical function and the continence rate were significantly better in the experimental group.

Guided PFMT for incontinence an average of 18 months after surgery is better than PFMT alone (1 trial)

Zhang et al. (2007) investigated the efficacy of guided PFMT compared to a group receiving only one session of PFMT and additional home exercises an average of 18 months after surgery. The continence rate and QoL were significantly better in the experimental group.

Adverse effects

In one study (Moore et al., 1999), one patient complained of rectal pain by contracting the pelvic floor muscles and discontinued the therapy. No other author described adverse effects of PFMT after prostatectomy.

Health economics

Information on the total cost of the intervention of physical therapy after prostatectomy was never given. One study (Wille et al., 2003) gave details of the costs of a home biofeedback and electrical stimulation device. In another study (Van Kampen et al., 2000), the number of physical
therapy sessions (an average of 8 in the experimental and 16 in the control group) were calculated and the authors concluded that the cost of treatment was low.

**Discussion**

Urinary incontinence is a common problem after prostatectomy and the role of physical therapy as a first-line treatment option provides only a small base for evidence because of the different results in the studies.

There were few data to determine the effect of pelvic floor training after transurethral resection of the prostate because of lack of good studies. Only two studies described a clear benefit on the recovery of incontinence with PFMT (Chang et al., 1998; Porru et al., 2001). However Glazener et al. (2011) found no beneficial effect of 1–4 sessions of PFMT compared to standard care and lifestyle advice. Furthermore incontinence rates were much higher in this study at 12 months after surgery compared to other studies described in the literature.

*After radical prostatectomy*, conclusions about physical therapy for incontinence are difficult to make because of the heterogeneity of the results. Four of the six trials showed that PFMT was significantly more effective than no treatment or sham treatment in the immediate postoperative period (Van Kampen et al., 2000; Filocamo et al., 2005; Manassero et al., 2007). The results of preoperative PFMT on incontinence were positive in five of the six trials (Sueppel et al., 2001; Parekh et al., 2003; Burgio et al., 2006; Centemero et al., 2010; Tienforti et al., 2012). Half of the studies (Zhang et al., 2007; Overgard et al., 2008; Mariotti et al., 2009; Marchiori et al., 2010; Ribeiro et al., 2010) proved that a structured programme decreased the duration of incontinence significantly in comparison with information only. No additional effect of biofeedback was found in men undergoing a radical prostatectomy in four studies (Mathewson-Chapman, 1997; Floratos et al., 2002; Wille et al., 2003; Robinson et al., 2008). The role of FES and ExMI was confirmed in three of the five studies (Yokoyama et al., 2004; Liu et al., 2008; Yamanishi et al., 2010). Last, the efficacy of guided PFMT even 18 months after radical prostatectomy has been proved in one study (Zhang et al., 2007).

Several limitations should be considered in the different studies. A variety of outcome measurements are used to assess UI. The most widely used assessment is the number of pads (Bales et al., 2000; Floratos et al., 2002). In most studies 0 or 1 pad per 24 hours is defined as continent. Clinical experience revealed that some men wear 1 pad but have a urine loss over 10 g. The severity of incontinence was objectively assessed by the ICS 1-hour pad-test (Van Kampen et al., 2000; Floratos et al., 2002) or during 24 hours (Moore et al., 1999; Van Kampen et al., 2000; Geraerts et al., 2013). In most studies no effort was made to assess pelvic floor muscle strength prior to surgery. At this moment, we do not know if men with a weaker pelvic floor might have more benefit from biofeedback or electrical stimulation. Some studies described a limited number of treatments. We currently do not know if positive effects might be found if patients were treated more frequently.

Many hypotheses were not investigated and as a result conclusions on male incontinence after prostatectomy are limited. The effect of lifestyle changes like weight loss, smoking cessation, adequate fluid intake and regular bowel movements on incontinence after prostatectomy remain undetermined as no trial involved these interventions. Other questions are the competence level of the physical therapist, the programme of the training, especially for endurance of the muscles and functional exercises, the motivation and the adherence to the programme of the patient. No studies were found to investigate these questions.

**SUMMARY AND CLINICAL RECOMMENDATIONS**

The value of PFMT for the treatment of incontinence after prostatectomy remains debated. There may be some benefit offering PFMT preoperatively or PFMT immediately after catheter withdrawal after prostatectomy. The therapy is non-invasive and avoids the side-effects that can occur with medical or surgical treatments. There is no consensus on the efficacy of information on PFMT in comparison with effective treatment. The efficacy of additional biofeedback training has not been proved. On the other hand, some studies have showed positive effect adding electrical stimulation or extracorporeal magnetic innervations.

**Terminal and post-void dribble**

A prolonged final part of micturition when the flow has slowed to a dribble is a troublesome and common problem in older men. In a recent Australian survey, 12% of older men reported frequent terminal dribble (Sladden et al., 2000), mostly associated with obstruction of the urethra. Post-voiding dribble is the involuntary loss of urine, usually after leaving the toilet. Authors suggested that the condition is caused by pooling of urine in the bulb urethra for unknown reasons (Millard, 1989; Denning, 1996) or because of failure of the bulbocavernous muscle to empty the urethra (Dorey, 2008). A small group of patients reported post-micturition dribble in the early postoperative period after prostatectomy because of urethral dysfunction (Wille et al., 2000). A decreased or absent post-void urethral milking results in residual unexpelled urine in the bulb urethra (Wille et al., 2000).

Bulbar urethral massage, with the finger behind the scrotum and moving in a forward and upward direction
to evacuate the remaining urine from the urethra, is not perceived as the optimal long-term treatment strategy by many men. Pelvic floor muscle training (PFMT) can eliminate the urine left in the bulbular urethra after voiding and provide men with a more acceptable option for management (Paterson et al., 1997).

**EVIDENCE FOR EFFECT OF PFMT FOR TREATMENT OF POST-MICTURITION Dribble**

A systematic review of treatment of post-micturition dribble in men was done by Dorey (2008). Effectiveness of a physical therapy approach for terminal and post-micturition dribble is only investigated in four controlled studies (Paterson et al., 1997; Chang et al., 1998; Porru et al., 2001; Dorey et al., 2004). One study (Paterson et al., 1997) recruited participants with pure post-micturition dribble without history of surgery of bladder, prostate or urethra nor a history of urgency or stress incontinence. Two other studies investigated the efficacy of PFME after transurethral resection of the prostate on terminal (Chang et al., 1998) and post-micturition dribble (Porru et al., 2001) (Table 8.3). One study investigated post-micturition dribble in patients with erectile dysfunction (Dorey et al., 2004). The methodological quality of all identified studies concerning post-micturition dribble based on PEDro was rather low and ranged between 3 and 6 out of 10 (Table 8.4).

Paterson et al. (1997) compared PFMT and bulbular urethral massage with only counselling on drinking and toileting. Assessment was done by pad-test <4 hours stored in two sealed plastic bags during 72 hours and improvement in pad weight gain was measured. The best results were obtained by PFMT, eliminating an average of 4.9 g urine while the effect of urethral massage was 2.9 g. The counselling group showed no improvement. The outcome measure was strongly influenced by the degree of urine loss at the start of the study (p <0.001); if the initial urine loss was too small then it would not be possible to detect a treatment effect.

Chang et al. (1998) and Porru et al. (2001) investigated the efficacy of PFMT after TURP for 4 weeks. The control group was only given information about PFMT before and after surgery. Both studies have already been discussed in the above section on postprostatectomy. A significant difference for post-micturition dribble was found in favour of the experimental group 4 weeks after surgery. The others concluded that early PFMT should be considered in alleviating the problem of post-micturition dribble after TURP.

Dorey et al. (2004) concluded that PFMT including a post-void ‘squeeze out’ pelvic floor muscle contraction is an effective treatment for post-micturition dribble in men with erectile dysfunction.

**SUMMARY AND CLINICAL RECOMMENDATIONS**

The evaluation of the efficacy of physical therapy for men with post-void dribble was hampered by the paucity and the methodological quality of published reports in the field. At the present time we can consider that PFMT is effective for post-micturition dribble based on three studies reporting positive results of PFMT in comparison with information only. Bulbar massage can give an additional effect to PFMT for post-micturition dribble, as shown in one study.

**CONCLUSION**

Many male patients with incontinence and lower urinary tract symptoms were referred for physical therapy based on the results of pelvic floor physical therapy in women. Despite the high number of referrals, evidence for physical therapy in men is focused only on incontinence after prostatectomy and post-void dribble. Physical therapy is a non-invasive treatment modality and adverse effects or complications of physical therapy are very rare in contrast with pharmacological treatment and surgery.

Concerning incontinence and physical therapy after transurethral resection of the prostate, conclusions are limited because of the lack of a sufficient number of studies. However two studies described a positive effect on incontinence when patients were treated with pelvic floor muscle training during 4 weeks postoperatively.

Concerning physical therapy after radical prostatectomy, there was some evidence that PFMT was significantly more effective than no treatment or sham treatment preoperatively or in the immediate postoperative period. A treatment programme of additional biofeedback-enhanced PFMT did not affect continence after RP. Conclusions on the efficacy of information on PFMT in comparison with guided PFMT or the efficacy of electrical stimulation cannot be drawn because of the heterogeneity of the results.

For post-micturition dribble, physical therapy is effective as shown in four studies. All reported the positive results of PFMT in comparison with bulbular massage, lifestyle changes or no treatment.

Our knowledge on the effect of physical therapy for UI and lower urinary tract dysfunction remains limited. Considering the absence of side-effects, the low cost and no risk level, pelvic floor re-education as an option in alleviating the problem of incontinence after prostatectomy remains debated. Future research is required to determine at what time men are most likely to benefit from which treatment modality of physical therapy.
<table>
<thead>
<tr>
<th>Author</th>
<th>Chang et al., 1998</th>
</tr>
</thead>
<tbody>
<tr>
<td>Design</td>
<td>2-arm CT not randomized</td>
</tr>
<tr>
<td>Research design</td>
<td>Experimental (E): PFME</td>
</tr>
<tr>
<td>Sample size and age (years)</td>
<td>50 men after TURP (E=25, C=25), mean age 65 in E (range 51–74) and 66 in C (range 45–79)</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Digital evaluation for strength of pelvic floor (0–4), questionnaires, voiding diary, uroflow</td>
</tr>
<tr>
<td>Training protocol</td>
<td>E: PFMT 4 weeks</td>
</tr>
<tr>
<td></td>
<td>Home: 30 exercises 4x/day</td>
</tr>
<tr>
<td></td>
<td>C: no treatment</td>
</tr>
<tr>
<td>Drop-out</td>
<td>Not reported</td>
</tr>
<tr>
<td>Results</td>
<td>Significant difference in strength of pelvic floor between E and C group only at week 4 (p &lt;0.05)</td>
</tr>
<tr>
<td></td>
<td>Significant difference in length of between-void interval between E and C group at week 1 till 4 (p &lt;0.01)</td>
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<td></td>
<td>Significant difference in incontinence between E and C group at week 3 and 4 (p &lt;0.05)</td>
</tr>
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<td></td>
<td>Significant difference in terminal dribbling between E and C group at week 4 (p &lt;0.05)</td>
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<tr>
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<td>Significant difference in QoL between E and C group at week 4 (p &lt;0.01)</td>
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<td></td>
<td>No significant difference in uroflow between E and C group at week 4</td>
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<table>
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<th>Author</th>
<th>Dorey et al., 2004</th>
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<td>Design</td>
<td>2-arm RCT</td>
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<td>Research design</td>
<td>Experimental group (E): PFMT including a strong post-void ‘squeeze out’ pelvic floor muscle contraction, BF, suggestions for lifestyle changes</td>
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<td>Sample size and age (years)</td>
<td>55 men (E=28, C=27), mean age 53.9 years in E (SD=13.0), 59.2 years in C (SD=8.62)</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Interview, digital anal measurements, anal manometric measurements</td>
</tr>
<tr>
<td>Training protocol</td>
<td>E1: education of the anatomy of the pelvic floor, PFMT with BF + post-void ‘squeeze out’ pelvic floor muscle contraction. Five 30-minute periods in consecutive weeks. Advice on lifestyle changes and a list of home exercises</td>
</tr>
<tr>
<td></td>
<td>C: advice on lifestyle changes only in five 30-minute periods in consecutive weeks. These men were offered to cross over to the intervention group at 3 months</td>
</tr>
<tr>
<td>Drop-out</td>
<td>13.9%</td>
</tr>
<tr>
<td>Results</td>
<td>36 (65.5%) of 55 subjects reported post-micturition dribble (PMD) at baseline. At 3 months, there was significant reduction in PMD after intervention compared to the control group. In both groups combined after 3 months of PFMT, 75% became asymptomatic, 8.3% improved and 2.8% still reported PMD</td>
</tr>
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<thead>
<tr>
<th>Author</th>
<th>Paterson et al., 1997</th>
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<td>Design</td>
<td>3-arm RCT</td>
</tr>
<tr>
<td>Research design</td>
<td>Experimental group 1 (E1): PFMT</td>
</tr>
<tr>
<td>Sample size and age (years)</td>
<td>49 men (E1 = 14, E2 = 15, C = 15), mean age 70.8 in E1 (SD=2.7), 69.3 in E2 (SD=3.1) and 69.5 in C (SD=2.4)</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Pad-test, pelvic muscle strength by Oxford Grading System from 0 to 4, bladder chart</td>
</tr>
<tr>
<td>Training protocol</td>
<td>E1: PFMT for 12 weeks with control at 5, 7, 13 weeks</td>
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<td></td>
<td>Home exercises: 5 contractions of 1s, contractions of endurance gradually extending the number of repetitions, spread exercise sessions throughout the day in lying, sitting and standing position</td>
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<td>E2: urethral milking by bulbar massage</td>
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<td></td>
<td>C: counselling about drinking and toileting, relaxation therapy</td>
</tr>
<tr>
<td>Drop-out</td>
<td>12%</td>
</tr>
<tr>
<td>Results</td>
<td>Significant difference in incontinence between E1 and C group for small pad-test (p &lt;0.01), significant difference in incontinence between E2 and C group for small pad-test (p &lt;0.01)</td>
</tr>
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(Continued)
Evidence-Based Physical Therapy for the Pelvic Floor

Table 8.3 Randomized controlled studies of physical therapy for incontinence for terminal and post-micturition dribble—cont’d

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<td>Experimental group (E): PFMT+BF</td>
</tr>
<tr>
<td></td>
<td>Control group (C): preoperative information about PFMT</td>
</tr>
<tr>
<td>Sample size and age (years)</td>
<td>58 men after TURP (E = 30, C = 28), mean age 67.5 in E (range 55–73) and 66 in C (range 53–71)</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Voiding diary</td>
</tr>
<tr>
<td>Training protocol</td>
<td>E: 4 treatments postoperatively 1x/week PFMT+home exercises</td>
</tr>
<tr>
<td></td>
<td>C: information (written and verbal information) postoperatively+same home exercises</td>
</tr>
<tr>
<td></td>
<td>Home: 15 contractions on strength and endurance, 3x/day</td>
</tr>
<tr>
<td>Drop-out</td>
<td>3/58 (5%)</td>
</tr>
<tr>
<td>Results</td>
<td>Post-micturition dribble significant difference between E and C group (p &lt;0.01)</td>
</tr>
</tbody>
</table>

For abbreviations, see text.

Table 8.4 PEDro quality score of (R)CTs in systematic review of physical therapy for post-micturition dribble

<table>
<thead>
<tr>
<th>Study</th>
<th>E</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>Total score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chang, 1998</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>+</td>
<td>–</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>3</td>
</tr>
<tr>
<td>Dorey, 2004</td>
<td>–</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>+</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>+</td>
<td>6</td>
</tr>
<tr>
<td>Paterson, 1997</td>
<td>+</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>+</td>
<td>+</td>
<td>–</td>
<td>+</td>
<td>+</td>
<td>5</td>
</tr>
<tr>
<td>Porru, 2001</td>
<td>+</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>+</td>
<td>+</td>
<td>–</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>6</td>
</tr>
</tbody>
</table>

+, criterion is clearly satisfied; –, criterion is not satisfied; ?, not clear if the criterion was satisfied. Total score is determined by counting the number of criteria that are satisfied, except that ‘eligibility criteria specified’ score is not used to generate the total score. Total scores are out of 10.
REFERENCES


Filocamo, M.T., Li Marzi, V., Del Popolo, G., et al., 2005. Effectiveness of early pelvic floor rehabilitation treatment for post-
Evidence-Based Physical Therapy for the Pelvic Floor


8.2 Male sexual dysfunction

CLASSIFICATION, PREVALENCE AND PATHOPHYSIOLOGY OF MALE SEXUAL DYSFUNCTION AND ROLE OF THE PFM

Sexual function in normal men is dependent on satisfactory libido, erectile function, ejaculation and orgasm. Sexual dysfunction occurs when there is a problem in any of these events. Sexual dysfunction embraces low libido, erectile dysfunction, premature ejaculation, retrograde ejaculation, retarded ejaculation, anorgasmia, anejaculation and sexual pain.

Low libido

Definition and classification

A low libido can be defined as ‘a reduced sexual urge’. As men age, there is a partial androgen decline. Men can be classified as having low or absent libido.

Prevalence and aetiology

The exact prevalence of men who have low libido remains unknown. It is estimated that at 40 years of age, there will be a 10% decline of total testosterone every decade, though the mechanisms are not fully understood (First Latin American Erectile Dysfunction Consensus Meeting, 2003a).

The cause of diminished libido is a result of ageing and a gradual decline in androgen production. The testis produces 95–98% of androgen, with the adrenal glands producing the remaining 2–5% (First Latin American Erectile Dysfunction Consensus Meeting, 2003a).

Erectile dysfunction

Erectile dysfunction is a common condition linked to increasing age and age-related diseases. Men with erectile dysfunction suffer from depression and low self-esteem and experience difficulties establishing and maintaining relationships.

Definition and classification

Erectile dysfunction is defined as ‘the inability to achieve or maintain an erection sufficient for satisfactory sexual performance (for both partners)’ (National Institutes of Health (NIH) Consensus Development Panel on Impotence, 1993).

The severity of erectile dysfunction has been classified as mild, moderate or severe. Men who achieve satisfactory sexual performance 7–8 attempts out of 10 are classified as having mild erectile dysfunction, those who achieve 4–6 out of 10 are classified moderate, and those who achieve 0–3 out of 10 are classified severe (Albaugh and Lewis, 1999).

Prevalence and aetiology

The exact prevalence of erectile dysfunction is unknown. It is common and strongly age-related (Feldman et al., 1994), affecting more than 20% of men under 40 years of age, more than 50% of men over 40 years of age, and more than 66% of men over 70 years of age (Feldman et al., 1994; Heruti et al., 2004). It may affect 10% of healthy men and significantly greater numbers of men with existing comorbidities such as hypertension (15%), diabetes mellitus (28%) and heart disease (39%) (Feldman et al., 1994; Wagner et al., 1996). The prevalence of erectile dysfunction immediately following prostatectomy ranges from 11% to 87% depending largely on the meticulous surgical technique (Alivizatos and Skolarikos, 2005). The number of men with erectile dysfunction is predicted to rise with increased life expectancy and with a growing population of elderly people.

The causes of erectile dysfunction are listed in Table 8.5.

Anatomy of the penis

The internal structure of the penis consists of three cylindrical bodies: dorsally, the two corpora cavernosae communicate with each other for three-quarters of their length and ventrally the corpus spongiosum surrounds the penile portion of the urethra (Fig. 8.5). The proximal end of the corpus spongiosum forms a bulb attached to the urogenital diaphragm and at the distal end expands to form the glans penis (Kirby et al., 1999). The tunica albuginea, which is composed of two layers of elastic and collagen fibres, surrounds the erectile bodies.

The erectile tissue in the corpora cavernosa and the corpus spongiosum is comprised of vascular lacunar spaces, which are surrounded by smooth muscle (Fig. 8.6). The lacunar spaces derive blood from the helicine arteries, which open directly into these sinusoids. Subtunical veins between the inner and outer tunica albuginea form a network, which drains blood from the erectile tissue.

Neurophysiology of penile erection

From a neurophysiological aspect, erection can be classified into three types (Brock and Lue, 1993).

- Reflexogenic erection: Reflexogenic erection originates from tactile stimulation to the genitalia. Impulses reach the spinal erection centre via sacral sensory nerves (S2–S4) and thoracic nerves (T10–L2)
<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Possible components</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Psychological</td>
<td>Marital conflict</td>
<td>Feldman et al., 1994</td>
</tr>
<tr>
<td></td>
<td>Depression</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Poor body image</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Performance related</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Bereavement</td>
<td></td>
</tr>
<tr>
<td>Vascular</td>
<td>Arterial</td>
<td>Feldman et al., 1994</td>
</tr>
<tr>
<td></td>
<td>Venous</td>
<td></td>
</tr>
<tr>
<td>Neurological</td>
<td>Spinal cord trauma</td>
<td>Feldman et al., 1994</td>
</tr>
<tr>
<td></td>
<td>Multiple sclerosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Spinal tumour</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Parkinson’s disease</td>
<td></td>
</tr>
<tr>
<td>Endocrinological</td>
<td>Hormonal deficiency</td>
<td>Feldman et al., 1994</td>
</tr>
<tr>
<td>Diabetic</td>
<td>Peripheral neuropathy</td>
<td>Benet and Melman, 1995</td>
</tr>
<tr>
<td></td>
<td>Hypertension</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Renal failure</td>
<td></td>
</tr>
<tr>
<td>Drug-related</td>
<td>Some antihypertensives</td>
<td>Benet and Melman, 1995</td>
</tr>
<tr>
<td></td>
<td>Some psychotropics</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hormonal agents</td>
<td></td>
</tr>
<tr>
<td>Surgical trauma</td>
<td>Transurethral and RP</td>
<td>Lewis and Mills, 1999</td>
</tr>
<tr>
<td></td>
<td>Pelvic surgery</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Radiotherapy</td>
<td></td>
</tr>
<tr>
<td>Lower urinary tract symptoms (LUTS)</td>
<td>Severity of LUTS, particularly incontinence</td>
<td>Frankel et al., 1998</td>
</tr>
<tr>
<td>Prostatic</td>
<td>Benign prostatic hyperplasia</td>
<td>Daniel et al., 2000</td>
</tr>
<tr>
<td>Lifestyle related</td>
<td>Trauma to the perineum</td>
<td>Bortolotti et al., 1997</td>
</tr>
<tr>
<td></td>
<td>Bicycling</td>
<td>Andersen and Bowim, 1997</td>
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<tr>
<td></td>
<td>Nicotine abuse</td>
<td>Rosen et al., 1991</td>
</tr>
<tr>
<td></td>
<td>Drug abuse</td>
<td>Lewis and Mills, 1999</td>
</tr>
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<td></td>
<td>Alcohol abuse</td>
<td>Fabra and Porst, 1999</td>
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<tr>
<td>Weak pelvic floor musculature</td>
<td>Weak pelvic floor muscles</td>
<td>Dorey et al., 2004</td>
</tr>
<tr>
<td></td>
<td>Ageing</td>
<td>Colpi et al., 1999</td>
</tr>
</tbody>
</table>

**Table 8.5 Risk factors for erectile dysfunction**

**Figure 8.5** Anatomy of the penis.

**Figure 8.6** Cross-section of the penis.
Pathophysiology of penile erection

Penile erection occurs following a series of integrated vascular processes culminating in the accumulation of blood under pressure and end-organ rigidity (Moncada Iribarren and Sáenz de Tejada, 1999). This vascular process can be divided into six phases:

- **Flaccidity**: A state of low flow of blood and low pressure exists in the penis in the flaccid state (Fig. 8.7, top). The ischiocavernosus and bulbocavernosus muscles are relaxed.

- **Filling phase**: When the erection mechanism is initiated, the parasympathetic nervous system provides excitatory input to the penis from efferent segments S2–S4 of the sacral spinal cord; the penile smooth arterial muscle relaxes and the cavernosal and helicine arteries dilate enabling blood to flow into the lacunar spaces.

- **Tumescence**: The venous outflow is reduced by compression of the subtunical venules against the tunica albuginea (corporal veno-occlusive mechanism) causing the penis to expand and elongate but with a scant increase in intracavernous pressure.

- **Full erection**: The intracavernous pressure rapidly increases to produce full penile erection.

- **Rigidity**: The intracavernous pressure rises above diastolic pressure and blood inflow occurs with the systolic phase of the pulse enabling complete rigidity to occur. Contraction or reflex contraction of the ischiocavernosus and bulbocavernosus muscles produce changes in the intracavernous pressure. When full rigidity is achieved, no further arterial flow occurs (Fig. 8.7, below).

- **Detumescence**: The sympathetic nervous system is responsible for detumescence via thoracolumbar segments (T10–T12, L1–L2) in the spinal cord. Contraction of the smooth muscle of the penis and contraction of the penile arteries lead to a decrease of blood in the lacunar spaces and contraction of the smooth trabecular muscle leads to a collapse of the lacunar spaces and detumescence.

**Pathophysiology of erectile dysfunction**

Three types of erectile dysfunction are acknowledged: psychogenic, organic and mixed. They may be primary or secondary after a period of normal erectile function (First Latin American Erectile Dysfunction Consensus Meeting, 2003b). In organic erectile dysfunction, the events leading to full erection do not happen due to insufficient blood reaching the penis or blood escaping from the penis.

**Role of the pelvic floor muscles**

The ischiocavernosus and bulbocavernosus muscles are active during penile erection (Fig. 8.8).

Contractions of the ischiocavernosus muscles increase intracavernous pressure and influence penile rigidity. The area of the corpora cavernosum compressed by the ischiocavernosus muscle ranges from 35.6 to 55.9% (Claes et al., 1996). The middle fibres of the bulbocavernosus muscle assist in erection of the corpus spongiosum penis by compressing the erectile tissue of the bulb of the penis. The anterior fibres spread out over the side of the corpus cavernosum and are attached to the fascia covering the dorsal vessels of the penis and contribute to erection by compressing the deep dorsal vein of the penis, thus preventing the outflow of blood from the penis.
Weak pelvic floor muscles compromise penile erection (Colpi et al., 1999; Dorey et al., 2004).

**Orgasmic and ejaculatory disorders**

The final phase of sexual response in men culminates in orgasm and ejaculation. Although erection and ejaculation are coordinated, they are produced by different mechanisms.

**Classification**

Orgasmic and ejaculatory disorders may be classified as anejaculation, anorgasmia, premature ejaculation, retrograde ejaculation and delayed ejaculation (Hendry et al., 2000). Ejaculatory disorders such as anejaculation, delayed ejaculation and premature ejaculation may lead to complete or partial loss of the ejaculate needed for impregnation of the female partner.

**Anejaculation**

Anejaculation is defined as ‘the absence of ejaculation during orgasm’ (Hendry et al., 2000). Anejaculation may be classified as congenital or acquired and/or psychological (First Latin American Erectile Dysfunction Consensus Meeting, 2003c).

The prevalence of anejaculation is unknown. Ejaculatory dysfunction can be due to congenital abnormalities, surgical trauma following imperforate anus, para-aortic lymphadenectomy or prostate surgery, genital infections such as gonorrhoea or non-specific urethritis, spinal cord injury, antidepressants, antipsychotics and polycystic kidney associated with dilatation of the seminal vesicles (Hendry, 1999).

**Retrograde ejaculation**

Retrograde ejaculation is defined as ‘backward passage of semen into the bladder after emission usually due to failure of closure of the bladder neck mechanism, demonstrated by presence of spermatozoa in the urine after orgasm’ (Hendry et al., 2000). Retrograde ejaculation can be classified as congenital or acquired and/or psychological (First Latin American Erectile Dysfunction Consensus Meeting, 2003c).

The prevalence of retrograde ejaculation is unknown. Retrograde ejaculation can be due to damage of the bladder neck during prostate surgery, bladder neck disorder from alpha-adrenergic, neuroleptic or antidepressant blocking agents, diabetes mellitus and some neuropathies (First Latin American Erectile Dysfunction Consensus Meeting, 2003c).

**Retarded ejaculation**

Retarded ejaculation is defined as ‘undue delay in reaching a climax during sexual activity’ (Hendry et al., 2000). Retarded ejaculation can be classified as drug-related or psychological (Hendry et al., 2000).

The prevalence of retarded ejaculation is unknown. Retarded ejaculation can be due to emotional suppression, an inability to relax, relationship difficulties, medications, societal and religious attitudes, and the use of alcohol and recreational drugs (First Latin American Erectile Dysfunction Consensus Meeting, 2003c).

Regarding the role of the pelvic floor muscles, during sexual activity rhythmic contractions of the bulbocavernosus muscle along with the other pelvic floor muscles result in ejaculation (Gerstenberg et al., 1990). The external urethral sphincter and deep pelvic floor muscles relax rhythmically to allow the ejaculate to pass through the urethra. An ability to relax may compromise this process.

**Premature ejaculation**

Premature ejaculation is one of the commonest forms of sexual dysfunction (Rosen, 2000). It is characterized by a lack of ejaculatory control and is associated with significant effects on sexual functioning and satisfaction (Rowland et al., 2004).
Definition and classification
Premature ejaculation has been defined as ‘recurrent ejaculation that occurs with minimal stimulation and earlier than desired, before or soon after penetration, which causes bother or distress, and upon which the sufferer has little or no control’ (World Health Organization, 1992). It is also defined as ‘the inability to delay ejaculation sufficiently to enjoy lovemaking. Persistent or recurrent occurrence of ejaculation with minimal sexual stimulation before, on, or shortly after penetration and before the person wishes it’ (Hendry et al., 2000).

Premature ejaculation is typically defined by three characteristics: short latency to ejaculation, lack of self-efficacy regarding the rapid ejaculation, and distress or dissatisfaction with the condition (Rowland, 2003). In some men ejaculation occurs before or within 1 minute of the beginning of intercourse (Waldinger et al., 1998). Rowland et al. (2001) believed that the latency for men with premature ejaculation varied from 1 to 5 minutes. However, a study investigating the ejaculatory time of ‘normal’ men in different countries showed that the average time to ejaculation varies between 7 and 14 minutes (Montorsi, 2005). Therefore, the definition of premature ejaculation should not be counted in minutes but acknowledge three core components: short ejaculatory time, lack of control over ejaculation and lack of sexual satisfaction (Montorsi, 2005). It may occur in the absence of sufficient erection and the problem is not the result of prolonged abstinence from sexual activity.

There are several different subtypes of biogenic and psychogenic premature ejaculation according to aetiological features (Metz and Pryor, 2000). Physiological types of premature ejaculation are due to neurological constitution, acute physical illness, physical injury and pharmacological side-effects. Psychological types are due to psychological constitution, acute psychological distress, relationship distress and deficient psychosexual skills. Premature ejaculation may be labelled psychogenic when the physical cause is unknown.

Prevalence and aetiology
The prevalence of premature ejaculation is 16.3 to 32.5% (Rowland et al., 2004). There is no evidence that ejaculation latency increases with age. In a stopwatch study of 110 men aged 18 to 65 years, 76% reported their ejaculation to be as rapid at their first sexual contacts, with 23% reporting increasing rapidity and only 1% reporting a delay (Waldinger et al., 1998).

The aetiology of premature ejaculation is unknown, but psychological, behavioural and biogenic components are likely (Montague et al., 2004). There may be an organic basis for some forms. The causes can be congenital or acquired and/or psychological (First Latin American Erectile Dysfunction Consensus Meeting, 2003c).

Pathophysiology
Data suggest that men with premature ejaculation have hypersensitivity and hyperexcitability of the glans penis and the dorsal nerve (Xin et al., 1996, 1997).

Role of the pelvic floor muscles
During sexual activity, rhythmic contractions of the bulbocavernous muscle along with the other pelvic floor muscles result in ejaculation (Gerstenberg et al., 1990). Contraction of the pelvic floor muscles combined with intermittent relaxation of the external urinary sphincter and urogenital diaphragm allows ejaculation (Krane et al., 1989). The bladder neck sphincter under involuntary control remains closed.

It is hypothesized that weak pelvic floor musculature affords little control to delay ejaculation and that the voluntary use of the pelvic floor muscles could delay ejaculation.

Sexual pain
Sexual pain is any pain that affects the ability to gain and maintain an erection and achieve orgasm and ejaculation.

Evidence for the Role of Physical Therapy in the Treatment of Male Sexual Dysfunction
The treatment of male sexual dysfunction by physical therapists has been based on the evidence from a few trials. These trials were limited to the treatment of erectile dysfunction and premature ejaculation.

Erectile Dysfunction
A literature review was undertaken to ascertain if physical therapy had merit as a conservative treatment for erectile dysfunction.

Literature search strategy
A search of the following computerized databases from 1980 to 2005 was undertaken: Medline, AAMED (Allied and Alternative Medicine), CINAHL, EMBASE – Rehabilitation and Physical Medicine and the Cochrane Library Database. The keywords chosen were erectile dysfunction, impotence, conservative treatment, physical therapy, pelvic floor exercises, biofeedback, electrical stimulation and electrotherapy. A manual search was undertaken
of identified manuscripts reporting on research studies gained from the references of this literature.

**Selection criteria**

A study was included if the trial reported the results of physical therapy for men with erectile dysfunction and the outcome measures were reliable and relevant to the problem under investigation (Table 8.6).

### Methodological quality

Methodological rigor was assessed by a PEDro quality score (see Table 8.7).

### Evidence for the effect

Only three randomized controlled trials (RCTs) provided evidence that pelvic floor muscle exercises (PFME) cured erectile dysfunction.

---

**Table 8.6 Literature review of physical therapy for erectile dysfunction**

<table>
<thead>
<tr>
<th>Author</th>
<th>Mamberti-Dias and Bonierbale-Branchereau, 1991</th>
</tr>
</thead>
<tbody>
<tr>
<td>Design</td>
<td>Not random; no control</td>
</tr>
<tr>
<td>Sample size</td>
<td>210 men with erectile dysfunction; some with venous leakage; some psychological</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Some with venous leakage; some psychological erectile dysfunction</td>
</tr>
<tr>
<td>Training protocol</td>
<td>PFME+ES sacral and penile or perineal electrode 5–25 Hz then 50–400 Hz intermittent Visual stimulation and penile temperature 15 treatments</td>
</tr>
<tr>
<td>Drop-out</td>
<td>Not reported</td>
</tr>
<tr>
<td>Adherence</td>
<td>Not reported</td>
</tr>
<tr>
<td>Results</td>
<td>At 3 months: 111 (53%) cured; 44 (21%) improved; 55 (26%) failed; 67% attained 4/10 to 8/10 ISMR (index of subjective mean rigidity) Subjective outcome</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Author</th>
<th>Claes and Baert, 1993</th>
</tr>
</thead>
<tbody>
<tr>
<td>Design</td>
<td>Randomized; no control</td>
</tr>
<tr>
<td>Sample size and age (years)</td>
<td>150 men with venogenic erectile dysfunction; age 23–64; median age 48.7</td>
</tr>
<tr>
<td>Group 1: 72 surgery</td>
<td></td>
</tr>
<tr>
<td>Group 2: 78 PFME</td>
<td></td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Venogenic erectile dysfunction</td>
</tr>
<tr>
<td>Training protocol</td>
<td>Group 1: surgery deep dorsal vein Group 2: patient education 5-weekly PFME; home exercises; digital anal assessment baseline, 4 and 12 months; 40mg papaverine+needle; EMG ischiocavernosus muscle+maximum PFM contraction</td>
</tr>
<tr>
<td>Drop-out</td>
<td>Not reported</td>
</tr>
<tr>
<td>Adherence</td>
<td>Not reported</td>
</tr>
<tr>
<td>Results</td>
<td>At 4 months: Group 1: 44 (61%) cured; 17 (23.6%) improved; 11 (15.2%) failed Group 2: 36 (46%) cured; 22 (28%) improved; 20 (25.6%) failed At 12 months: Group 1: 30 (42%) cured; 23 (32%) improved Group 2: 33 (42%) cured; 24 (31%) improved; 45 (58%) refused surgery Subjective and objective outcomes</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Author</th>
<th>Colpi et al., 1994</th>
</tr>
</thead>
<tbody>
<tr>
<td>Design</td>
<td>Not random; controlled</td>
</tr>
<tr>
<td>Sample size and age (years)</td>
<td>59 men; age 20–63; mean age 39</td>
</tr>
<tr>
<td>Group 1: 33 men: PFME+Bf</td>
<td></td>
</tr>
<tr>
<td>Group 2: 26 men: controls</td>
<td></td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Venogenic erectile dysfunction</td>
</tr>
<tr>
<td>Training protocol</td>
<td>30 of 59 deep dorsal vein surgery; 29 of 59 psychological therapy No information on which intervention; no information on type of BF</td>
</tr>
<tr>
<td>Drop-out</td>
<td>Not reported</td>
</tr>
<tr>
<td>Adherence</td>
<td>Not reported</td>
</tr>
</tbody>
</table>

(Continued)
### Table 8.6 Literature review of physical therapy for erectile dysfunction—cont’d

| Results | At 11 months:  
|---------|----------------
|         | Group 1: 21 (63%) cured or improved; 9 refused surgery  
|         | Subjective outcome  
| Author  | Claes et al., 1995  
| Design  | Not random; no control  
| Sample size | 122 men with venogenic erectile dysfunction  
| Diagnosis | Venogenic erectile dysfunction  
| Training protocol | Patient education; PFME; EMG or pressure BF; ES with anal or surface electrode, symmetrical biphasic low frequency 50 Hz pulse 100μs; 6s stimulation, 12s rest maximum intensity  
| Drop-out | 14/122 drop-outs (11.5%)  
| Adherence | 88.5% adhered  
| Results | At 4 months: 53 (43%) cured; 37 (30%) improved; 32 (26.2%) failed including 14 drop-outs  
|         | At 12 months: 44 (36%) cured; 41 (33.6%) improved; 37 (30.3%) failed including 14 drop-outs; 65 (53.4%) refused surgery  
| Subjective outcome |  
| Author  | Stief et al., 1996  
| Design  | Not random; controlled  
| Sample size | 22 men with erectile dysfunction who were vasoresponders  
| Diagnosis | Venogenic erectile dysfunction  
| Training protocol | Transcutaneous ES to smooth muscle corpus cavernosum: low-frequency symmetrical trapezoidal 100–200μs 12 mA alternating 10–20Hz and 20–35Hz 5s stimulation 2–5 days for 20min  
| Drop-out | Not reported  
| Adherence | Not reported  
| Results | At 5 days: 5 (23%) cured; 3 (13.6%) responded to vasoactive drugs; 14 (63%) failed  
| Subjective outcome |  
| Author  | Derouet et al., 1998  
| Design  | Not random; no control  
| Sample size | 48 men with erectile dysfunction  
| Diagnosis | Erectile dysfunction  
| Training protocol | Transcutaneous ES penile or perineal electrodes bipolar pulsed 85μs 30Hz 20–120mA 3-s stimulation, 6-s rest 20min daily for 3 months  
| Drop-out | 10/48 drop-outs (20.8%)  
| Adherence | 79.2% adhered  
| Results | At 3 months: 5 (10.4%) cured; 20 (41.6%) improved; 23 (47%) failed, including 10 drop-outs  
| Subjective improvement |  
| Author  | Sommer et al., 2002  
| Design  | Randomized controlled; PEDro score 7/10  
| Sample size and age (years) | 104 men with venogenic erectile dysfunction; aged 21–72, mean age 43.7  
| Group 1: 40 men  
| Group 2: 36 men  
| Group 3: 28 men  
| Diagnosis | Venogenic erectile dysfunction  
| Training protocol | Group 1: 3 weekly PFME  
| Group 2: oral PDE5 inhibitor  
| Group 3: placebo  
| At baseline, 4 weeks and 3 months: KEED erectile dysfunction questionnaire, IIEF Q 3 and 4, GAQ.  
| At baseline and 3 months: caversonography  
| Drop-out | Not reported  
| Adherence | Not reported  

---

302
Table 8.6 Literature review of physical therapy for erectile dysfunction—cont’d

| Results | At 3 months:  
|         | Group 1: 80% improved significantly; 46% improved penile rigidity  
|         | Group 2: 74% improved  
|         | Group 3: 18% improved  
|         | Subjective and objective |
| Author  | Van Kampen et al., 2003 |
| Design  | Not random; no control |
| Sample size and age (years) | 51 men with erectile dysfunction with mixed aetiology; age 25–64; mean age 46 |
| Diagnosis | Erectile dysfunction |
| Training protocol | Patient education; PFME in lying, sitting and standing; anal pressure BF; ES anal or surface electrode 50Hz 200μs: 6 s stimulation 12 s rest once a week for 4 months; home exercise 90 contractions  
| Drop-out | 9/51 drop-outs (18%) |
| Adherence | 82% adhered |
| Results | At 4 months: 24 (46%) cured; 12 (24%) improved; 15 (31%) failed including drop-outs |
| Subjective and objective outcome | |

| Author  | Dorey et al., 2004 |
| Design  | Randomized controlled; PEDro score 8/10 |
| Sample size and age (years) | 55 men with erectile dysfunction with mixed aetiology. Age 22–78; mean age 59 |
| Diagnosis | Erectile dysfunction |
| Training protocol | Intervention group (I): 28 men – PFME+lifestyle changes  
|                  | Control group (C): 27 men – lifestyle changes  
|                  | At 3 months, control group given intervention  
| Drop-out | 5/55 drop-outs (9%) at 3 months |
| Adherence | Adherence 91% at 3 months |
| Results | At 3 months: erectile function domain of IIEF: I group significantly improved, p = 0.001; C group p = 0.658; anal pressure: I group significantly improved, p < 0.001  
|                  | At 6 months: blind assessment: 22 (40%) normal function including drop-outs; 19 (34.5%) improved including drop-outs; 14 (25.5%) failed including drop-outs |
| Subjective and objective outcomes | |

| Author  | Prota et al., 2012 |
| Design  | Randomized controlled; PEDro score 8/10 |
| Sample size and age (years) | 52 men with post-RP erectile dysfunction. Age 62–64 (±8); mean age 63 |
| Diagnosis | Post-prostatectomy erectile dysfunction |
| Training protocol | Intervention group (I): 26 men PFME+EMG  
|                  | Control group (C): 26 men with instruction in pelvic floor contraction by urologist  
|                  | At 15 days after surgery, weekly PFME and anal EMG for 12 weeks+home exercises  
|                  | C: instruction in PFM contraction by urologist  
| Drop-out | Before first month: 9/26 (35%) in I group and 10/26 (38%) in C group (19 not included in study) |
| Adherence | Adherence of subjects included in study: 100% |
| Results | At 1 month: IIEF-5: I group potency 5.88%; C group 1%  
|                  | At 3 months: IIEF-5: I group potency 11.76%; C group potency 4%  
|                  | At 6 months: IIEF-5: I group potency 23.53%; C group potency 6.25%  
|                  | At 12 months: IIEF-5: Intervention group potency 47.1% (8/17) (p = 0.032); control group potency 12.5% (2/16)  
|                  | A strong association was found between potency and continence |

ED-EQOL, Erectile Dysfunction Effect on Quality of Life; IIEF, International Index of Erectile Function; KEED, Kölner Erfassungsbogen für Erektile Dysfunktion; PDE5 inhibitor, phosphodiesterase type 5 inhibitor. For other abbreviations, see text.
or improved erectile function (Sommer et al., 2002; Dorey et al., 2004; Prota et al., 2012). The trials by Sommer et al. (2002) and Prota et al. (2012) scored 7/10 using a PEDro quality score and the trial by Dorey et al. (2004) scored 8/10 (Table 8.7).

Five trials that were either non-randomized or uncontrolled provided weak evidence (Mamberti-Dias and Bonierbale-Branchereau, 1991; Claes and Baert, 1993; Colpi et al., 1994; Claes et al., 1995; Van Kampen et al., 2003). Two non-randomized uncontrolled trials solely used electrical stimulation and provided only weak evidence (Stief et al., 1996; Derouet et al., 1998).

The trial by Sommer et al. (2002) used a large sample size of 124 men with venogenic erectile dysfunction. Men were randomized into three groups with one group receiving PFME, one group receiving Viagra and one group receiving a placebo. At 3 months the PFME group improved more than the Viagra group and significantly more than the placebo group. In the trial by Dorey et al. (2004) 55 men were randomized into two groups with one group receiving PFME and one group receiving lifestyle changes. At 3 months the PFME group improved significantly compared to the control group. The control group was then given PFME and they improved significantly when compared to their erectile function at baseline. Both groups continued home exercises for a further 3 months.

The study by Prota et al. (2012) was the first RCT for men with erectile dysfunction post-radical prostatectomy which compared PFMEs and EMG to a control group receiving pelvic floor muscle contraction instruction by a urologist. Significant return to potency was seen in the intervention group at 12 months.

### Effect size

The three randomized controlled trials all showed significantly improved erectile function with PFME.

Dorey et al. (2004) found at 3 months using the erectile function domain of the International Index of Erectile Function (IIEF) that the PFME group improved significantly ($p=0.001$) compared with the control group ($p=0.658$) (Fig. 8.9). At 3 months, when the control group were given PFME they improved erectile function significantly ($p<0.001$). This trial also found that anal pressure in the intervention group significantly improved after 3 months PFME ($p<0.001$) when compared to the control group.

Sommer et al. (2002) found that the group of men who performed PFME improved more than the group of men receiving oral phosphodiesterase type 5 (PDE5) inhibitor (Viagra) and significantly more than the group receiving a placebo (Fig. 8.10).

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<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>Total score</th>
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<tr>
<td>Sommer et al., 2002</td>
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<td>Dorey et al., 2004</td>
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<td>8</td>
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<td>Prota et al., 2012</td>
<td>+</td>
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<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>7</td>
</tr>
</tbody>
</table>

+, criterion is clearly satisfied; −, criterion is not satisfied; ?, not clear if the criterion was satisfied. Total score is determined by counting the number of criteria that are satisfied, except that ‘eligibility criteria specified’ score is not used to generate the total score. Total scores are out of 10.
Prota et al. (2012) found that men receiving PFMEs, anal EMG and home exercises showed significantly improved potency at 12 months compared to the control group (p = 0.032). They also found that there was a strong association between potency and continence.

**Clinical significance**

Both Sommer et al. (2002) and Dorey et al. (2004) found that PFME improved erectile function clinically. Sommer's study found that 46% of men had improved penile rigidity and Dorey's study found that 40% of men regained normal erectile function, and a further 34.5% improved erectile function.

Prota et al. (2012) found that PFMEs significantly improved erectile dysfunction after radical prostatectomy. Both Sommer et al and Dorey et al had excluded men following radical prostatectomy so the trial by Prota et al presented new encouraging data.

**Study methodology**

The methodological quality was good in the three randomized controlled trials. The sample size was larger in the study by Sommer et al. (2002). Sommer et al. (2002) studied men with proven venogenic erectile dysfunction. Dorey et al. (2004) studied men with a wide range of aetiology. Both used validated subjective outcome measures and unlike the other trials both used objective outcome measures. Prota et al. (2012) studied 33 men with erectile dysfunction after radical prostatectomy and used a validated outcome measure.

The results from the uncontrolled or non-randomized trials should be interpreted with caution due to poor methodology. Only one of these trials used randomization, five of the trials lacked controls, and seven provided only subjective outcomes. The lack of control groups in these trials impinged on the validity of evidence provided. The lack of randomization is an important methodological limitation that fundamentally limits any definitive interpretation and translation of the findings from these trials.

**Type of intervention**

Only one trial used PFME alone (Sommer et al., 2002). This trial provided good results without biofeedback and questions the need for biofeedback. Three trials included biofeedback as the only other modality (Colpi et al., 1994; Dorey et al., 2004; Prota et al., 2012), while two combined PFME with biofeedback and electrical stimulation (Claes et al., 1995; Van Kampen et al., 2003). It is impossible to determine which modality has caused the effect when three modalities are used.

The amount of PFME varied. Colpi et al. (1994) expected men to perform daily home exercises for 30
minutes a day for 9 months as a realistic alternative to surgery. Dorey et al. (2004) instructed men to perform 18 strong contractions a day with an emphasis on functional work. Three trials performed a long-term follow-up (Claes and Baert, 1993; Claes et al., 1995; Prota et al., 2012) and followed up subjects for 12 months with encouraging results.

Two trials used electrical stimulation alone. Derouet et al. (1998) found electrical stimulation to the ischiocavernosus muscle produced a cure rate of only 10.4% while Stief et al. (1996) in a controlled trial explored transcutaneous electrical stimulation to the smooth muscle of the penile corpus cavernosum and effected a 23% cure rate. Whatever effect was achieved, both cure rates were low compared to the PFME trials.

**Frequency and duration of training**

The amount of treatment varied from between five and 20 treatment sessions, though some papers did not provide this information. Sommer et al. (2002) treated men in three-weekly PFME sessions and monitored the men at 4 weeks and 3 months. Dorey et al. (2004) treated men in five-weekly PFME sessions and monitored the men at 3 months and at 6 months. In both studies men performed home exercises. Prota et al. (2012) treated men weekly for 3 months.

**Short- and long-term effects**

From the available data, it appears that patients were assessed initially and then at between 3 and 12 months. The exception to this was the trial by Stief et al. (1996) where outcomes were assessed after 5 days.

Sommer et al. (2002), Dorey et al. (2004) and Prota et al. (2012) used the subjective validated IIEF, which is used extensively for trials using oral medication for erectile dysfunction. Sommer et al. (2002) used the validated Kölner Erfassungsbogen für Erektile Dysfunktion (KEED). Dorey et al. (2004) used an assessor who was blinded to the subject group to report trial outcomes. Mamberti-Dias and Bonierbale-Branchereau (1991) used an index of subjective mean rigidity (ISMR) and reported an increase from 4 out of 10 to 8 out of 10 mean ISMR.

Most outcomes used patient reported ‘cure’, ‘improved’ or ‘failure’. ‘Cure’ was defined as an erection suitable for satisfactory sexual performance with vaginal penetration in all studies. ‘Improvement’, however, was defined in a number of ways from ‘a significant increase of erection quality and performance’ (Colpi et al., 1994) to ‘partial response for those patients who reported some increase in quality (duration or rigidity) of erections but not sufficient for sexual intercourse’ (Claes et al., 1995).

Three trials used objective outcome measurements. Claes and Baert (1993) injected 40 mg papaverine to achieve penile rigidity and tested with needle electromyography (EMG) while contracting the ischiocavernosus muscle maximally. Sommer et al. (2002) used Rigiscan® as an objective measurement of penile rigidity and Dorey et al. (2004) used anal manometric biofeedback readings.

Sommer et al. (2002) used a quality of life instrument and Dorey et al. (2004) used the validated Erectile Dysfunction – Effect on Quality of Life (ED–EQoL) (MacDonagh et al., 2002). Dorey et al. (2004) found there was poor correlation of the IIEF with the ED–EQoL in the intervention group, but significant correlation in the control group. This finding showed that erectile dysfunction may have impacted on men in different ways and demonstrated a clear reason for the clinical usefulness of a quality of life questionnaire.

The short-term effects were good in all trials of PFME for erectile dysfunction. The three randomized controlled trials have provided good results at 3 months (Dorey et al., 2004; Sommer et al., 2002) and 6 months (Dorey et al., 2004) and 12 months (Prota et al., 2012). The results were good at 12 months in the trial by Claes and Baert (1993).

**Psychosexual issues**

All the trials used a sample of heterosexual men. No study mentioned any cultural factors although the study by Prota was conducted in Brazil. The perceptions of sexual activity vary from one man to another and impact on the expectations and the subjective measurement of sexual performance. Not all men wish to practise penetrative sex. There were no studies that identified and addressed the difficulties and needs of homosexual men who practise anal intercourse.

**Conservative management for the prevention of erectile dysfunction**

There were no publications using preventative conservative treatment. However, if the pelvic floor musculature is poor and PFME can relieve erectile dysfunction, then it seems reasonable to suppose that preventative muscle strengthening may help to prevent erectile dysfunction.

**SUMMARY AND CLINICAL RECOMMENDATIONS**

Based on evidence, PFME should be the first-line treatment for erectile dysfunction (Fig. 8.11). They may be performed in conjunction with other treatment for erectile dysfunction such as oral therapy, vacuum devices, intracavernous injections, intraurethral medication, constriction bands and counselling.
A literature review was undertaken to ascertain whether PFME may have merit as a treatment for premature ejaculation.

**Literature search strategy**

A search of the following computerized databases from 1980 to 2005 was undertaken: Medline, AAMED (Allied and Alternative Medicine), CINAHL, EMBASE – Rehabilitation and Physical Medicine and the Cochrane Library Database. The keywords chosen were premature ejaculation, conservative treatment, physical therapy, pelvic floor muscle exercise, biofeedback, electrical stimulation and electrotherapy. A manual search was also undertaken.

**Selection criteria**

A study was included if it reported the results of physical therapy for men with premature ejaculation.

**Methodological quality**

No RCTs were identified.

**Results**

Only two non-randomized uncontrolled trials were found, providing weak evidence (Table 8.8).

---

**Table 8.8 Trials of physical therapy for premature ejaculation**

<table>
<thead>
<tr>
<th>Author</th>
<th>Design</th>
<th>Subjects</th>
<th>Protocol</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>La Pera and Nicastro, 1996</td>
<td>Non-randomized; no control</td>
<td>18 men with premature ejaculation</td>
<td>PFME Pressure BF ES</td>
<td>At 7 weeks: 11 (61%) cured; 7 (39%) no improvement Subjective and objective outcomes</td>
</tr>
<tr>
<td>Claes and van Poppel, 2005</td>
<td>Non-randomized; no control</td>
<td>29 men with premature ejaculation</td>
<td>PFME ES</td>
<td>After treatment: 19 (65.5%) improved; 10 (34.5%) failed to improve At 12 months: most of the 19 who improved still showed a positive response</td>
</tr>
</tbody>
</table>

For abbreviations, see text.

**Study methodology**

La Pera and Nicastro (1996) used PFME, pressure biofeedback and electrical stimulation in a non-randomized and uncontrolled trial of 18 patients with a mean age of 34 years (range 20–52 years) to treat premature ejaculation; 15 had experienced the problem for over 5 years. The results showed that 11 patients (61%) were cured and able to control the ejaculatory reflex associated with improved PFM control and seven (39%) had no improvement. The biofeedback readings were not given. This non-randomized, uncontrolled study has shown that there may be merit in strengthening the PFM to control the ejaculatory reflex to prevent premature ejaculation.

In a non-randomized uncontrolled trial, Claes and van Poppel (2005) investigated the action of PFME and electrical stimulation on 29 men with premature ejaculation. After treatment, they found that 19 men (65.5%) showed improvement, which was verified by their partner. At 12 months, most of the men who had improved still showed a positive result.

The methodology used in these two trials without randomization or the use of a control group and with a small sample size provided only weak evidence. However, results from these trials indicated that this subject is worth further exploration.
SUMMARY AND RECOMMENDATIONS

RCTs need to be undertaken to investigate PFME for premature ejaculation before any conclusions can be made.

CONCLUSION

There is good-level evidence that PFME seem to have merit as a treatment for erectile dysfunction. For those patients who appear to have been cured or improved with PFME, it may be prudent to continue these simple exercises for life and possibly avoid a return of erectile dysfunction. However, long-term compliance may be a problem. Following initial pelvic floor training, it may be possible to maintain muscle performance with a minimal exercise programme.

There was no strong evidence that electrical stimulation was effective. No studies demonstrating preventative conservative treatment were found.

A multicentre randomized controlled trial with larger sample numbers is needed to explore the use of PFME as a first-line treatment for men with erectile dysfunction. Similar trials are also needed to ascertain the role of PFME as prophylaxis for erectile dysfunction.

No conclusions can yet be made concerning PFME for premature ejaculation and further investigation by means of RCTs needs to be undertaken.

REFERENCES


Chapter 9

Evidence-based physical therapy for pelvic floor dysfunctions affecting both women and men

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9.1 Anal incontinence

Epidemiology, anatomy and pathophysiology, and risk factors

Bary Berghmans, Esther Bols, Ylva Sahlin, Espen Berner

INTRODUCTION

Anal incontinence (AI) is a major healthcare problem that can be particularly embarrassing and affects 2–24% of community-dwelling adults (Macmillan et al., 2004), with 1–2% experiencing significant impact on daily activities (Farage et al., 2008). The actual prevalence is likely to be higher due to underreporting (Markland et al., 2008).

AI as a symptom of anorectal dysfunction can be defined as the complaint of involuntary loss of faeces or flatus (Haylen et al., 2010). AI covers a wide spectrum from involuntary but recognized passage of gas, liquid or solid stool (urgency incontinence) to unrecognized anal leakage of mucus, fluid or stool (passive incontinence) (Miner, 2004). Anal continence is based on a combined interplay of faeces consistency, sensory, motor, reservoir functions and mental components (Madoff et al., 2010).
Incontinence occurs if one or more of these components fail and when compensatory mechanisms fall short. Vaginal delivery has been reported to be one of the major causes of AI in women (Madoff et al., 2004), and in accordance with this finding, Bols et al. found that risk factors for postpartum AI appeared to be a third- or fourth-degree sphincter rupture and AI during pregnancy (Bols et al., 2013). Several colorectal, urological or gynaecological interventions can cause AI as well. Specific neurological diseases associated with AI include diabetes, multiple sclerosis, Parkinson’s disease, stroke and spinal cord injury. AI is mostly associated with advancing age and disability (Potter et al., 2002). However, younger patients are often affected as well, resulting in difficulties participating in school, work or social life. It is not hard to imagine what it is like to experience loss of faeces in the middle of a shop, workplace, bus or school. Often, it is difficult to explain your problem to others, like family or a partner. The implications of AI are huge and the social restriction in many patients is severe: staying at home nearby a toilet, having to avoid social contacts including relationships or sexual contact, having feelings of depression and low self-confidence (Nelson et al., 1995). A lot of these implications are due to the unpredictable character of AI and the fear of odour. Despite this huge impact, because of fear, embarrassment and insufficient knowledge that their problem can be treated, it is striking that only one-third of all patients with faecal incontinence (FI, incontinence of stool only) report their problem to a doctor (Kalantar et al., 2002; Whitehead, 2005).

AI often coincides with other pelvic floor, pelvic or abdominal health problems, like constipation, prolapse or urinary incontinence (Siekert-ten Hove et al., 2010). As discussing all kinds of possible patient profiles would be too complicated, this chapter will focus merely on AI.

### Epidemiology

Prevalence figures of AI are often influenced by the use of different definitions and target populations. In a systematic literature review, based on cross-sectional studies, the prevalence in the general population was estimated to be 2–24% for AI and 0.4–18% for FI (Macmillan et al., 2004). Another systematic review reported prevalence of AI to be 0.8% and 1.6% for men and women <60 years respectively, and 5.1% and 6.2% for men and women ≥60 years. Based on clinical studies AI seems to be more prevalent among women, although epidemiological studies report a more equal distribution. This discrepancy might be related to the age and gender of individuals who actively seek help (Madoff et al., 2004). Moreover, the prevalence is higher in postpartum women and patients with cognitive problems or a neurological disease (Tjandra et al., 2007). Approximately 50% of FI patients also suffer from urinary incontinence, most likely based on a dysfunctional levator ani muscle (Teunissen et al., 2004).

Only a few studies report on FI incidence rates. The 5- and 10-year incidence rate among community-dwelling persons is 5.3–7% (women) and 4.1–5.3% (men) and increases with age: 13–15.3% (women) and 13.2–20% (men) (Ostbye et al., 2004; Markland et al., 2010; Rey et al., 2010).

Among elderly persons living in nursing homes, the 10-months incidence rate was 20% (Chassagne et al., 1999).

### Anatomy and Pathophysiology

Continence depends on several anatomic and physiologic entities: anal sphincter function, pelvic floor function, rectal distensibility, ano-rectal sensation, ano-rectal reflexes, intact nervous system, mental function, stool volume, stool consistency and colonic transit. Deficiency of one or more of these factors can lead to incontinence (Rey et al., 2010).

The anal sphincter muscles are located in the distal part of the anal canal, which arises from the sigmoid colon and rectum. The anal sphincter mechanism involves the internal anal sphincter, external anal sphincter and puborectalis muscle. The internal anal sphincter is a circular smooth muscle layer under involuntary control and is mainly contracted in rest. This sphincter represents 80% of the basal resting pressure (Ostbye et al., 2004; Rey et al., 2010). Internal anal sphincter dysfunction is often associated with faecal seepage (passive AI). Besides during childbirth, the sphincter can be damaged by anorectal surgery (sphincterotomy or fistulotomy), anal stretch (Deutekom et al., 2005) and primary degeneration (Xu et al., 2012).

The external anal sphincter is a striated muscle, innervated by the pudendal nerve (S2–S4) and comprising of three parts: a subcutaneous, a superficial and a deep part. At rest, the external sphincter is submaximally contracted and only modestly contributing to basal pressure. Basal pressure normally redoubles in case of voluntary sphincter contraction (Dunivan et al., 2010). Besides, a sudden rise in intra-abdominal pressure initiates a spinal reflex which causes the external sphincter to contract. In addition to both sphincters, the haemorrhoidal plexus also contributes to basal pressure (15%).

The puborectalis muscle is part of the levator ani muscle, joined with the pubococcygeus and iliococcygeus muscle. The puborectalis muscle is anatomically and functionally closely related to the external anal sphincter. The puborectalis muscle forms a muscular sling around the anorectal junction and creates an angulation between the anal canal and rectum due to its...
attachment anterior to the pubic bone. At rest, the ano-
rectal angle is 90°, increasing during straining and def-
ecation to about 135°, which facilitates the passage of
stool (Lucas et al., 1999). The puborectalis muscle sling
and anorectal angle seem to contribute to maintain con-
tinence, although uncertainty exists as to which extent
(Madoff et al., 1992).

In response to rectal distension the external sphincter
contracts, which coincides with reflex inhibition of the
internal anal sphincter (Whitehead et al., 1982). Further
accommodation of the rectum occurs when defecation
is not appropriate and the internal sphincter regains
its tone. Consequently, the possibility to postpone def-
cecation is dependent on rectum distensibility, reservoir
function and efficiency of the anal-sphincter mecha-
nism. Adequate rectal sensation is necessary to notice
rectal contents. Presence of a small amount results in
some relaxation of the internal anal sphincter and the
anal mucosa is able to discriminate gas, solid or liquid
faeces (Baeten, 1985). Rectal sensation is often impaired
in patients with diabetes, spinal disease or constipation
(Madoff et al., 1992).

Voluntary relaxation of the external anal sphincter
and puborectal sling causes opening of the anal canal.
Straining further decreases anal pressure due to relaxation
of the internal and external sphincter, and defecation is
possible.

<table>
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<th>Table 9.1 The aetiology of anal incontinence</th>
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<tr>
<td><strong>Trauma</strong></td>
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ageing and the menopause overcome compensatory mechanisms (Eason et al., 2002; Hay-Smith et al., 2002).

**Other aetiological factors**

Several colorectal, urological or gynaecological interventions can cause AI as well. The surgical procedures most related to AI are sphincterotomy for anal fissure, sphincter dilatation, haemorrhoidectomy, fistulotomy, ileal pouch reconstruction and hysterectomy (Madoff et al., 1992).

Children who are born with congenital abnormalities, such as imperforate anus or Hirschprung’s disease (encopresis), often experience lifelong problems with incomplete evacuation of faeces and soiling despite anatomical correction (Nelson, 2004).

Specific neurological diseases associated with AI include stroke, Parkinson’s disease, spinal cord injury, multiple sclerosis and diabetes. Denervation or neuropathy is frequently present in patients with diabetes, vaginal delivery, descending perineum syndrome, chronic straining at stool and rectal prolapse (Rao, 2004).

AI disproportionately affects individuals with physical and mental disabilities, especially in nursing homes. Characteristics associated with the development of FI in the institutionalized elderly are: a history of urinary incontinence, impaired mobility, poor cognitive function, older age, neurological disease, core stability problems, non-Caucasian and problems with daily living activities (Chassagne et al., 1999; Madoff et al., 2004). In these homes, the association between urinary incontinence and AI is also well known. This so-called ‘double incontinence’ can be explained by the same underlying causes of poor mobility and cognitive impairment (Chassagne et al., 1999).

Demented residents are at high risk of developing faecal impaction because of neglect of the call to stool, impaired awareness of rectal fullness and coexistent limited mobility (Tobin and Brocklehurst, 1986). Moreover, an association was found between poor health and patients with long-lasting FI (Chassagne et al., 1999). Furthermore, FI is often related to irritable bowel syndrome and constipation, which is thought to be more prevalent amongst women (Palsson et al., 2004).

Symptoms associated with AI are sometimes related to other causes, known as pseudo-incontinence (Madoff et al., 1992). Clinically significant AI should be differentiated from perianal leakage of material other than stool (due to fistulas, prolapsing haemorrhoids, anorectal neoplasms, sexually transmitted diseases and poor hygiene) and frequent defecation moments and urge sensations without loss of anal contents (due to inflammatory bowel disease, pelvic irradiation, irritable bowel syndrome, and low anterior resection of the rectum). Careful diagnostics should differentiate between pseudo-incontinence and clinically important AI (Madoff et al., 1992).

Men, aged ≥85 years or suffering from kidney problems have a higher risk of developing FI (Lucas et al., 1999). Besides, radiotherapy as a result of prostate cancer increases the risk of flatus incontinence (Geinitz et al., 2011). Lower radiotherapy doses do not seem to avoid FI. In both men and women kidney problems, diarrhoea, feeling of incomplete evacuation, pelvic radiation in the past, development of urgency complaints (Rey et al., 2010) and urinary incontinence (Markland et al., 2010) contribute to the development of FI.

**REFERENCES**


Evidence-based physical therapy for pelvic floor dysfunctions

Assessment of the nature and severity of AI

Bary Berghmans, Esther Bols

To determine the nature and severity of AI, different subjective and objective diagnostic procedures are available. Inquiry on some aspects of AI can be obtained by a defecation or stool diary. A diary is useful to determine the severity of AI, regarding frequency of unintentional bowel movements and constitution of lost faeces. Unfortunately, the use of diaries in patient management is often uncommon, despite the fact that it can offer important information to guide selection of diagnostics or treatment. It is very important to promote and stimulate the patient to keep a record of his or her stool behaviour and to provide the physician with this information as the results can contribute...
to the treatment of AI. History taking and physical examination precede the additional diagnostic investigations of the rectum, anus and pelvic floor. Additional diagnostic investigations, performed by or within the physician’s area of responsibility, are anal manometry, rectal capacity measurement, endoanal sonography, anorectal sensation, neurophysiological testing, defecography and magnetic resonance imaging (MRI).

**DIAGNOSTIC ASSESSMENT**

**History taking**

During history taking the following topics should be addressed:

- Status of pelvic floor muscles, reservoir function, consistency of faeces and combined interplay.
- Nature and severity of AI: the nature of AI is classified as either passive incontinence, urgency (Haylen et al., 2010), or a combination of both (mixed incontinence) (Soffer and Hull, 2000; Baeten, 2003; Rao, 2004; Teunissen et al., 2004).
- Proctological, gynaecological, obstetric (number of deliveries, duration of birth, birth weight, instrumental delivery, episiotomy or sphincter rupture), urological and sexological history.
- Co-morbidity.
- Coping strategies: e.g. pad and medication use.
- Psychosocial complaints.
- Defecation and micturition pattern.
- Diet and fluid intake.
- Local and/or general barriers.
- Above all, the physician should specifically ask in what way the patient is socially disabled, since relevant social and personal limitations caused by AI are a main focus in the intervention programme (Madoff et al., 1992; Lucas et al., 1999).

**Physical examination**

- General inspection: breathing pattern, mobility and gait analysis.
- Local inspection perianal area in rest: to assess presence of faecal matter, scars, fistulas, dermatitis, gapping anus, keyhole deformity, haemorrhoids and skin tags. Scars can be indicative of previous episiotomies or perianal lacerations and a gapping anus for major loss of sphincter function. A gapping anus is often associated with rectal prolapse (Madoff et al., 2004). Deformities in the anal region may be due to previous haemorrhoidectomy, fistulotomy or fissurectomy. Sometimes, chronic skin irritation is present.
- Local inspection of perianal area during contraction, coughing or straining: to assess the performance of contracting/relaxing and to demonstrate mucosal or rectal prolapse, perineal descent, or paradoxal straining.
- Rectal palpation: to assess perianal sensation, anal pressure at rest, pressure during contraction and during straining, and relaxation. Sphincter defects can be located and palpated, especially during contraction. In women, the presence of a rectocele can be established. External anal sphincter and puborectal muscle strength can be assessed by digital rectal examination according to the modified Oxford grading system (Madoff et al., 1992; Laycock and Jerwood, 2001; Enck and Klosterhafen, 2005) (Table 9.2). The Oxford scale is internationally accepted and most frequently used muscle grading method, with the intra-observer variability reported to be high (Messelink et al., 2005). The score ranges from 0 (no muscle contraction) to 5 (strong contraction). Endurance of submaximal strength and exhaustion of these muscles are also determined (Table 9.2).
- Vaginal examination: can give a good impression of the pelvic floor musculature and may detect abnormalities of the recto-vaginal wall (Madoff et al., 1992).
- During rectal palpation and vaginal examination the PERFECT assessment is often used to direct a chosen patient-specific treatment protocol (Laycock and Jerwood, 2001). Table 9.2 explains the characters of the PERFECT acronym. The power represents the strength of the pelvic floor muscles and is measured using the modified Oxford grading system, as mentioned above. Assessment of endurance strength and peak force strength helps to prescribe an appropriate exercise programme. Patients graded 0, 1 or 2 on the modified Oxford scale seem to be more suitable for rectal balloon training and/or electrical stimulation (ES) instead of pelvic floor muscle training (PFMT) (Lucas et al., 1999; Terra et al., 2006).

**Table 9.2 The PERFECT assessment scheme and modified Oxford scale**

<table>
<thead>
<tr>
<th>Character</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>P</td>
<td>Power (pressure)</td>
</tr>
<tr>
<td>0</td>
<td>No muscle contraction</td>
</tr>
<tr>
<td>1</td>
<td>Flickering contraction</td>
</tr>
<tr>
<td>2</td>
<td>Weak contraction</td>
</tr>
<tr>
<td>3</td>
<td>Moderate contraction</td>
</tr>
<tr>
<td>4</td>
<td>Good contraction</td>
</tr>
<tr>
<td>5</td>
<td>Strong contraction</td>
</tr>
<tr>
<td>E</td>
<td>Endurance</td>
</tr>
<tr>
<td>R</td>
<td>Repetitions</td>
</tr>
<tr>
<td>F</td>
<td>Number of fast maximum, 1 s contractions</td>
</tr>
<tr>
<td>C</td>
<td>Co-contractions</td>
</tr>
<tr>
<td>T</td>
<td>Timing and coordination</td>
</tr>
</tbody>
</table>
ADDITIONAL DIAGNOSTIC TESTS

After history taking and physical examination additional tests for the assessment of the anorectal region in relation to AI might be performed.

Additional tests performed by either the physician or the pelvic physical therapist

Discrepancy exists between the perception of the patient and the clinician regarding severity of symptoms (De Backer, 1998). Therefore, it is recommended during screening, diagnostic assessment or evaluation to use at least one patient-reported outcome that enables the patient to report severity of symptoms and consequences of the health problem (Avery et al., 2007).

Defecation diary

As stated before, the use of a defecation diary is important at onset of assessment and makes it possible to assess the defecation pattern (De Backer, 1998). It is recommendable to fill out a diary until the point that consistency and frequency are normalized. The Bristol Stool Form Scale (Lewis and Heaton, 1997; Rogers et al., 2006) seems to be a good instrument to map out faeces consistency and can be integrated in a defecation diary (Table 9.3).

Wexner and Vaizey scores

Severity of AI, including social impact, can be assessed with the frequently used grading system of Wexner (Cleveland Clinic score), ranging from 0 (perfect continence) to 20 (complete incontinence) (Jorge and Wexner, 1993) and of Vaizey (St Mark’s score), which is a modification of the Wexner score (with the items ‘urgency’ and ‘medication use’ and a lower weighting for ‘pad use’, ranging from 0 (perfect continence) to 24 (complete incontinence) (Vaizey et al., 1999) (Table 9.4).

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### Table 9.3 The Bristol Stool Form Scale

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Separate hard lumps, like nuts</td>
</tr>
<tr>
<td>2</td>
<td>Sausage-shaped but lumpy</td>
</tr>
<tr>
<td>3</td>
<td>Like a sausage or snake but with cracks on its surface</td>
</tr>
<tr>
<td>4</td>
<td>Like a sausage or snake, smooth and soft</td>
</tr>
<tr>
<td>5</td>
<td>Soft blobs with clear-cut edges</td>
</tr>
<tr>
<td>6</td>
<td>Fluffy pieces with ragged edges, a mushy stool</td>
</tr>
<tr>
<td>7</td>
<td>Watery, no solid pieces</td>
</tr>
</tbody>
</table>

### Table 9.4 Grading system according to Wexner and Vaizey

<table>
<thead>
<tr>
<th>Incontinent</th>
<th>Never</th>
<th>Rarely</th>
<th>Sometimes</th>
<th>Weekly/usually</th>
<th>Daily/always</th>
</tr>
</thead>
<tbody>
<tr>
<td>Solid stool</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Liquid stool</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Gas</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Alteration in lifestyle</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Wears pad</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Need to wear a pad or plug</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Taking constipating medication</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Lack of ability to defer defecation for 15 min</td>
<td>0</td>
<td>4</td>
</tr>
</tbody>
</table>

Wexner: never = 0; rarely = < 1/month; sometimes = < 1/week and ≥ 1/month; usually = ≥ 1/week and < 1/day; always = ≥ 1/day.
Vaizey: never = no episodes in the past 4 weeks; rarely = 1 episode in the past 4 weeks; sometimes = > 1 episode in the past 4 weeks, but < 1 a week; weekly = 1 or more episodes a week but < 1 a day; daily = 1 or more episodes a day.

*Vaizey and Wexner items.
*Only Wexner item.
*Only Vaizey item.
Quality of life evaluation

The Faecal Incontinence Quality of Life scale (FIQL) is designed for use in clinical trials and outcome research in adults with AI. Furthermore, it has been developed to be responsive to the condition in question and is based on a clearly described conceptual framework (Rockwood et al., 2000; Rockwood, 2004). Rockwood et al. (2000) concluded that the FIQL subscales demonstrated acceptable internal consistency, test–retest reliability, and adequate discriminate and convergent validity. Another psychometric study (Bols et al., 2012b) showed that the depression subscale had inadequate internal responsiveness and longitudinal construct validity. The FIQL total score had acceptable responsiveness, test–retest reliability, and longitudinal construct validity. Practical issues related to the 29-item FIQL include the complicated addition of items and time-consuming completion, which holds consequences for clinical use. Based on these results and in the absence of other recommendable disease-specific quality of life (QoL) scales, it is recommendable to use the FIQL for evaluating QoL of patients with AI (Avery et al., 2007).

Global Perceived Effect

The Global Perceived Effect (GPE) questionnaire, reflecting patients’ global perceived change or extent of improvement of subjective health status, is attractive due to its simplicity and practicality (Jaeschke et al., 1989; Veldhuyzen-van Zanten et al., 1999).

Biofeedback

EMG/pressure

An intra-anal electromyographic (EMG) sensor, a perianal surface EMG electrode (both measuring change of motor unit activity), or an anal manometric probe (measuring intra-anal pressure change), connected to a biofeedback device/machine, is used to inform the patient about the activity of the pelvic floor and sphincter ani muscles by way of a visual display and/or an auditory signal. This (feedback) data can be collected and stored for further counselling and evaluation (Norton, 2004).

Rectal balloon

A rectal balloon, attached to a syringe, can be introduced in the rectum and slowly inflated with air, while the patient is lying in lateral position. This enables assessment of sensory threshold, urge sensation, change in distinction of and response to rectal volume related to distension, maximal tolerated volume and recto-anal inhibitory reflex (Norton and Chelvanayagam, 2004; Bols et al., 2012a).

Additional tests performed by the physician

The following diagnostic tests should be performed according to a standard procedure, by physicians (or technicians) specialized in performing the specific tests.

Anal manometry determines resting pressure (mmHg) and maximal squeeze pressure (mmHg) of the anal sphincter and puborectalis muscle and rectal capacity can be measured (Diamant et al., 1999).

With endoanal sonography (Stoker et al., 2001, 2002), the integrity of the anal sphincter complex can be demonstrated.

Neurophysiological testing, in casu pudendus nerve terminal motor latency (PNTML), measures the conduction time of the left and right pudendal nerves. Uncertainty exists on the accuracy of PNTML and its predictive value on outcome (Kiff and Swash, 1984; Madoff et al., 2004).

Defecography is a dynamic radiologic study of attempted defecation. Abnormalities can be detected during rest, straining and squeezing, such as rectocele and rectal intussusception (incomplete rectal prolapse) (Terra et al., 2008).

Endoanal magnetic resonance imaging (MRI) provides an impression of the anatomy of the anus and pelvic floor and can adequately demonstrate sphincter defects (Stoker et al., 2001, 2002).

The pelvic physical therapist should receive the conclusions from all diagnostic tests to gain more insight into the aetiology and prognosis of improvement or recovery of AI. Ultimately, a functional or anatomic diagnosis is formulated after evaluating the results of the assessment. The outcome of diagnostics defines the relevant problem area and guides decision-making concerning therapy, and at the same time it provides objective baseline data to evaluate the selected treatment.

REFERENCES


Conservative interventions for treatment of AI

Bary Berghmans, Esther Bols

Treatment of patients with AI consists of conservative as well as surgical interventions. Conservative interventions incorporate lifestyle interventions like dietary adaptations, medication, bowel management, smoking behaviour, absorbent materials and pelvic physical therapy. Table 9.5 summarizes the therapeutic options and goals regarding conservative treatment of FI.

The International Consultation on Incontinence (ICI) (Norton et al., 2009) states that physical therapy should be tried before any surgical treatment. Moreover, national guidelines in the UK recommend maximal education, lifestyle and dietary interventions preceding pelvic floor muscle training (PFMT) or biofeedback (Norton et al., 2007).
Patients with such an embarrassing condition as AI must be treated carefully. Therefore, prior to pelvic physical therapy, education and information should be given about the disorder. One determinant in success of physical therapy is a reliable relationship with the therapist and motivation of both the therapist and the patient (Norton and Cody, 2012). After diagnostic assessment, manually directed techniques or specifically designed equipment are used to treat the motor, sensory or reservoir component of the disorder.

### LIFESTYLE INTERVENTION

#### Information and education

Patients with AI often lack knowledge about bowel function, working mechanism and the anatomy of pelvic organs related to stool and bowel function. Many of them have an inefficient or wrong toilet behaviour, defecation pattern and are not well educated or trained. They have developed attitudes toward defecation based on what they learned from their family and societal environment (Norton, 2004).

Therefore, every treatment programme should start with information and education on general health, and more specific advice and teaching about lifestyle changes related to toilet behaviour and bowel function (Norton and Chelvanayagam, 2004). Although the literature does not consistently report on the strength of association of relevant risk factors for AI, like weight loss, toilet behaviour, use and facilities, and smoking, patient and healthcare provider attitudes, information and education should incorporate these topics.

#### Weight loss

Although obesity is considered a risk factor for AI by some authors, others did not find a significant correlation between obesity and AI (Bliss et al., 2013). Markland et al. (2010) found improvement on reducing...
Evidence-based physical therapy for pelvic floor dysfunctions

Evidence-based physical therapy for pelvic floor dysfunctions includes pelvic floor muscle training (PFMT), biofeedback (BF) (including rectal balloon training, RBt) and electrical stimulation (ES) and is offered by pelvic physical therapists (see Table 9.5). Often, one or more physical therapy interventions are combined, depending on the underlying cause of AI.

As pelvic physical therapy, in general, is simple, inexpensive and mostly without unfavourable physical side-effects, it is an appealing conservative treatment option in patients with AI (Madoff et al., 2004).

Pelvic floor muscle and sphincter training

Pelvic floor muscle and sphincter training are recommended as an early intervention in the treatment of AI as part of an integrated conservative management approach (Norton et al., 2006, 2009). The pelvic floor muscles support the abdominal organs and work tonically and reflexively to maintain continence. Approximately 70% slow-twitch and 30% fast-twitch fibres are present in the pelvic floor muscles to serve this purpose (Laycock and Jerwood, 2001). Patients suffering from AI often show weakened muscle function. PFMT aims to restore the muscular strength, relaxation, coordination and timing of contractions. Exercises consist of selective (maximal) voluntary contractions and relaxations of the pelvic floor muscles and external anal sphincter with a repetitive character. Exercises can activate latent motor units to the point that the muscle becomes functional again (MacLeod, 1983). Exercises in a progressive resistance programme adhere to the basic muscle training principles. The principle of ‘overload’ is based on stimulation of the muscle beyond its normal level of performance. To evaluate this principle the PERFECT assessment prior to training is necessary. The principle of ‘specificity’ refers to training a muscle in the way the muscle needs to be used. Exercises are adapted to slow-twitch fibres (endurance exercises) and fast-twitch fibres (power and speed exercises). The principles ‘maintenance’ and ‘reversibility’ alert the patient to train regularly.

Smoking

Anecdotally smoking has been claimed to stimulate onset of defecation. This might be due to stimulation of distal colonic motility and faecal urgency (Rausch et al., 1998; Bliss et al., 2013). The scientific evidence for such an association is weak. No association has been found between antenatal smoking and postnatal AI (Chaliha et al., 1999). In another study, involving twin sisters, no significant association was found between smoking and AI (Abramov et al., 2005). In a longitudinal observational study of community-living elderly men and women, smoking was not predictive of prevalence or incidence of FI (Ostbye et al., 2004).

EVIDENCE FOR EFFECTIVENESS OF PATIENT EDUCATION

In an RCT, nurse-led education and advice about conservative AI management (e.g. advice on diet, medication titration and bowel retraining) alone and as part of a combined intervention that added exercises and/or biofeedback were compared (Norton et al., 2003). All four study groups showed effect in reducing frequency of AI (Norton et al., 2003). Another study, reported as abstract only, underlined the benefits of systematic education and standard medical care for a group of AI patients who had failed prior attempts at medical management, leading to a successful outcome in 38% (Heymen et al., 2001). Success was defined as a patient’s report that they had experienced adequate relief of bowel symptoms.

The overall conclusion of the Fifth International Consultation on Incontinence (ICI) (Bliss et al., 2013) is that there is insufficient evidence to recommend or discourage most lifestyle modifications either for the prevention or treatment of AI. The ICI recommends patient education about the causes of AI and a systematic effort to remove barriers to effective toileting as an intervention that is likely to be beneficial based on the consensus of experts (Bliss et al., 2013). This may be provided at relatively low cost and involves no significant risk to the patient.

Apart from education and information on lifestyle interventions, some other relevant subjects to discuss with the patient are:

- Influence of stress and relaxation on pressure resilience of the pelvic floor
- General and local relaxation exercises
- Relation with other pelvic floor-related symptoms, such as prolapse, urinary incontinence
- Optimization of defecation frequency, consistency, position during toileting
sometimes lifelong. Inactivity will convert the muscle to its pre-trained status and symptoms can occur again (Kuijpers, 1997). Awareness of the different muscles involved in maintaining continence is necessary to be sure of avoidance of co-contractions of surrounding muscles (abdominals, buttocks, thighs and back) and activation of the relevant muscles. Sometimes, when patients find and use the relevant muscles at the appropriate time, symptoms can reduce at once (Norton, 2004). Ultimately, exercises should be practised in different starting positions, from lying to sitting to standing, simulating everyday situations as much as possible (Lucas et al., 1999).

Biofeedback and rectal balloon training

Haskell and Rovner made the first attempt at biofeedback (BF) for AI in 1967 (MacLeod, 1983). They reported the successful use of electrodes and electromyography in 71% of patients treated for AI. Cerulli et al. (1979) were the first to use BF by way of insertion of three balloons: one intra-rectal (to allow rectal distension) and two intra-anal (to record internal and external sphincter contractions separately).

Many authors estimate BF to be a useful adjunct to PFMT or ES in patients with AI (Norton and Kamm, 2001). BF is a technique that monitors biological signals and electrically amplifies these to provide feedback to the patient. BF intends to control physiological processes, normally being under involuntary control.

Nowadays, three modalities of BF in the treatment of AI can be recognized (Norton and Cody, 2012):

1. An intra-anal electromyographic (EMG) sensor, an anal manometric probe (measuring intra-anal pressure change), or perianal surface EMG electrode is used to inform the patient about the activity of the pelvic floor muscles by way of a visual display and/or an auditory signal. The patient attempts to align the response (the patient’s pelvic floor muscle activity) to the ideal response (pre-set, visualized on screen). The goal of this treatment modality is to create awareness of the squeezing musculature and strengthen it without rectal distension. In addition, the correct muscle response and progress of the patient can be demonstrated.

Training can focus on endurance force (a submaximal contraction sustained for prolonged time) or increase of squeeze amplitude (peak force). The exercises are based on exercise programmes, originally used for urinary incontinence (Schissler et al., 1994).

2. The second modality involves the use of a manometric rectal balloon (rectal balloon training). The rectal balloon is filled with air to imitate rectal contents. The patient with an elevated sensory threshold is trained to discriminate smaller rectal volumes, resulting in an earlier warning from stool entering the rectum and earlier external sphincter response to counteract reflex inhibition of the internal sphincter (Miner et al., 1990; Heymen et al., 2009).

However, progressive distension of the rectal balloon is used in patients with a hypersensitive rectum to resist feelings of urgency.

3. The third modality is a 3-balloon system (a balloon-tipped water-perfused catheter or a Schuster-type three-balloon probe) used to train a forceful external anal sphincter contraction after a stimulus of rectal distension (Engel et al., 1974; Heymen et al., 2000).

In this way, external sphincter contraction counteracts relaxation of the internal anal sphincter due to rectal distension. This treatment was originally described by Engel et al. in 1974 (Latimer et al., 1984). Some authors feel that sensory delay is an important factor in FI (Miner et al., 1990).

Overall, BF provides feedback about the possibility, degree and quality of contracting and relaxing the pelvic floor, and gives feedback on the coordination between rectal distension and contracting the anal closing system (Heymen et al., 2009).

The use of BF as a treatment for AI is recommended by the ICI when other behavioural and medical management has been tried and inadequate symptom relief obtained (Bliss et al., 2013). Moreover, its use is promoted given the numerous positive outcomes from uncontrolled trials, limitations in the current RCTs and low morbidity associated with its application. Patients most likely to benefit from BF include those who have motivation, intact cognitive skills, some rectal sensation and nearly intact sphincters and innervation (Loening-Baucke, 1990; Jorge and Wexner, 1993; Heymen et al., 2000). It is reported that patients with neurological deficits (diabetes, spina bifida, multiple sclerosis) are less likely to be treated successfully (Heymen et al., 2000).

Even though continence is achieved after pelvic physical therapy, the rectosphincteric reflexes sometimes remain abnormal, implicating that the external sphincter response to rectal distension is an unreliable predictor of treatment outcome (Latimer et al., 1984).

Electrical stimulation

The use of electrical stimulation (ES) for the treatment of urinary and anal incontinence spans more than a 35-year period with apparent success (Hosker et al., 2007). ES is the application of an electrical current, thereby passively stimulating the pelvic floor muscles, sphincters and accompanying nerve structures. The purpose of ES is to re-educate weakened and poorly functioning pelvic floor muscles by means of increasing awareness and isolated contraction of the stimulated structures (Hosker et al., 2007). ES is often used as an adjunct to pelvic floor muscle and sphincter exercises and BF to assist with identification and isolation of pelvic floor muscles and to increase contraction strength.

In ES, the number of motor units recruited is dependent on a number of factors. These include the parameters of
the electrical stimulus, impedance (resistance to the flow of the current) and the size and orientation of the electrodes. The electrodes should be placed as close as possible to the pelvic floor muscles. The electrical stimulus should be stimulating enough to depolarize the nerve, whereas uncomfortable sensations should be avoided (Laycock et al., 1994). The precise mechanisms by which ES can restore faecal control are not well understood: Salmons and Vrbova (1969) suggested that ES improves muscle function by transforming fatigable fast-twitch muscle fibres to less-fatigable slow-twitch fibres and Hudlicka et al. (1982) reported an increase of capillary density, which supports the efficient working of these slow-twitch, oxidative fibres. Some studies have shown an increase in axonal budding following denervation (Laycock et al., 1994). Changes in fibre diameter may be important. However, apart from physiological changes, it may be that the predominant mechanism of improved faecal control is an enhanced awareness of the anal sphincter (Haskell and Rovner, 1967). A possible working mechanism of neuromodulation through efferent or afferent nerve stimulation needs to be further investigated.

Contraindications for ES are anal infections, rectal bleeding, complete denervation of the pelvic floor (will not respond), swollen/painful haemorrhoids, deficient sensation, atrophy of mucosa, 6-week period after surgery, pacemaker, dementia, pregnancy and pain during palpation (Newman and Giovannini, 2002).

### Evidence for Effectiveness of Pelvic Physical Therapy

A literature review was undertaken to ascertain the effectiveness of different pelvic physical therapy interventions, i.e. pelvic floor muscle training (PFMT), biofeedback (BF) and electrical stimulation (ES), as conservative treatment for Al.

### Literature search strategy

A search of the following computerized databases from 1980 to November 2012 was undertaken: Cochrane Library, PubMed, EMBASE, PEDro and CINAHL. Furthermore, reference lists of included studies were screened for unidentified articles. Only randomized trials were included, with full reports in English, German or Dutch reporting on PFMT, BF or ES as conservative treatment in adults with Al. The characteristics of the 18 included RCTs are detailed in Table 9.6.

<table>
<thead>
<tr>
<th>Table 9.6 Characteristics of included randomized controlled studies of pelvic physical therapy for Al</th>
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<tbody>
<tr>
<td><strong>Author</strong></td>
</tr>
<tr>
<td><strong>Sample size</strong></td>
</tr>
<tr>
<td><strong>Mean age (range) (years)</strong></td>
</tr>
<tr>
<td><strong>Study population</strong></td>
</tr>
<tr>
<td><strong>Methods</strong></td>
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<td></td>
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<tr>
<td><strong>Training protocol</strong></td>
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<tr>
<td><strong>Outcome</strong></td>
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<tr>
<td></td>
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<tr>
<td><strong>Results (between-group analyses)</strong></td>
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<tr>
<td><strong>Author</strong></td>
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<td><strong>Training protocol</strong></td>
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(Continued)
| Outcome | CRO: manometry, first sensation, first feeling of urge, rectal/anal sensation, Oxford EAS/PF, endurance EAS/PF, fatigue PF  
MTV, fatigue EAS  
PRO: GPE, FIQL subscale ‘Lifestyle’  
Vaizey, remaining subscales FIQL |
| Results (between-group analyses) | CRO (manometry): PI NS  
CRO (Oxford): PI 1 better  
PRO (GPE): PI 1 better  
PRO (Vaizey): PI NS |
| Author | Davis et al., 2004 |
| Sample size | 39 |
| Mean age (years) | 60.5 |
| Study population | FI (liquid and solid stool during ≥12 mth) and scheduled for sphincter repair |
| Methods | 1: sphincter repair  
2: sphincter repair + BF (manometry balloon) + PFMT at home |
| Training protocol | Start 3 mth after surgery, 1x/week 30 min for 6 weeks (5 series max long duration contractions 10 s, submax 5 s, and series quick contractions)  
PFMT at home: 2x/day |
| Outcome | CRO: manometry  
PRO: VAS, FIQL, Wexner |
| Results (between-group analyses) | CRO: PI NS |
| Author | Fynes et al., 1999 |
| Sample size | 40 |
| Mean age (range) (years) | 32 (18–48) |
| Study population | AI after obstetric anal sphincter damage |
| Methods | 1: vaginal manometry BF (perineometer) + PFMT at home  
2: anal EMG-BF + ES + PFMT at home |
| Training protocol | 1: 1x/week 30 min for 12 weeks (20 short max contractions of 6–8 s, 10 s rest + long duration contractions 30 s) + PFMT at home (standard Kegel PFMT, instructions not reported)  
2: 1x/week for 12 weeks (audiovisual EMG feedback + ES + PFMT at home (standard Kegel PFMT, instructions not reported) |
| Outcome | CRO: manometry, vector symmetry index  
PRO: modified Pescatori scale |
| Results (between-group analyses) | CRO: PI (12 weeks) 2 better  
PRO: PI (12 weeks) 2 better |
| Author | Glazener et al., 2001 |
| Sample size | 747 |
| Mean age (years) | 1: 29.6; 2: 29.4 |
| Study population | Self-reported AI 3 mth postnatal |
| Methods | 1: education + PFMT + visit nurse  
2: standard care |
| Training protocol | Advice PFMT at 5, 7 and 9 mth postnatal (8–10 sessions daily 80–100 short and long duration contractions + bladder training in case indicated) |
| Outcome | PRO: symptom questionnaire |
| Results (between-group analyses) | 12 mth: 1 better  
AI 4 vs 10% |
| Author | Glazener et al., 2005 |
| Sample size | 747 |
| Mean age (years) | 1: 29.6; 2: 29.4 |
| Study population | Self-reported AI 3 mth postnatal |
Table 9.6 Characteristics of included randomized controlled studies of pelvic physical therapy for AI—cont’d

| Methods | 1: education + PFMT + visit nurse  
2: standard care  
Training protocol | Advice PFMT at 5, 7 and 9 mth postnatal (8–10 sessions daily 80–100 short and long duration contractions + bladder training in case indicated)  
Outcome | PRO: symptom questionnaire  
Results (between-group analyses) | 6 yr: NS, AI 12 vs 13%  
Author | Healy et al., 2006  
Sample size | 58  
Mean age (range) (years) | 1: 25 (41–68); 2: 23 (40–74)  
Study population | AI without severe sphincter damage  
Methods | 1: endo-anal ES at home  
2: ES + EMG-BF (supervised)  
Training protocol | Treatment duration: 3 mth  
1: daily 1 hr with portable device. Different frequencies 3–10–20–30–40 Hz  
2: 30 min/week: (1) = alternated ES + EMG-BF supervised, 30 min/week. (2) = ES 2 x 15 min: 15 min 10 Hz and 15 min 40 Hz, both without EMG  
Outcome | CRO: manometry, PNTML  
PRO: Wexner, RAND-36  
Results (between-group analyses) | CRO and PRO: PI NS  
Author | Heymen et al., 2000  
Sample size | 40  
Mean age (range) (years) | 74 (36–88)  
Study population | Patients not suitable for surgery  
Methods | 1: EMG-BF + education  
2: EMG-BF + RBT (sensory)  
3: EMG-BF + EMG-BF at home  
4: EMG-BF + RBT (sensory) + EMG-BF at home  
Training protocol | BF: 1 x/week 1 hr  
EMG at home: 5 sets/day. 1 set = 20 cycles of 10 s contraction followed by 10 s rest  
Outcome | PRO: incontinence episodes  
Results (between-group analyses) | PI NS  
Author | Heymen et al., 2009  
Sample size | 108  
Mean age (years) | 59.6  
Study population | Weekly FI after failure education, medication and lifestyle changes  
Methods | 1: PFMT + manometry BF (coordination training + sensory)  
2: PFMT  
Training protocol | 1 x/2 weeks 1 hr for 12 weeks  
PFMT at home: 5 x/day + during ADL activities  
Outcome | CRO: squeeze pressure; first sensation  
PRO: FISI, subjective improvement  
Diary | FIQL, BDI, STAI-1, STAI-2  
Results (between-group analyses) | CRO: 3 mth, 1 better, NS  
PRO: 3 mth/1 yr, 1 better  
Diary: 3 mth, 1 better,  
FIQL, BDI, STAI-1, STAI-2: NS  
(Continued)
<table>
<thead>
<tr>
<th>Author</th>
<th>Sample size</th>
<th>Mean age (range) (years)</th>
<th>Study population</th>
<th>Methods</th>
<th>Training protocol</th>
<th>Outcome</th>
<th>Results (between-group analyses)</th>
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<tbody>
<tr>
<td>Ilnyckyj et al., 2005</td>
<td>54</td>
<td>59 (26–75)</td>
<td>Chronic and weekly idiopathic FI</td>
<td>1: education + PFMT</td>
<td>3x/week for 1 week, followed by 2 weeks PFMT at home, then 4th treatment session (1st 45 min, 2–4 30 min)</td>
<td>PRO: incontinence episodes</td>
<td>PI NS</td>
</tr>
<tr>
<td>Mahony et al., 2004</td>
<td>60</td>
<td>1: 35 (23–39); 2: 32 (22–42)</td>
<td>FI symptoms after obstetric traumata</td>
<td>1: intra-anal EMG-BF + PFMT at home</td>
<td>For 12 weeks: intra-anal BF: 10 min 3 quick max contractions in 5 s and 8 s rest, and long duration contractions of 5 s and 8 s rest</td>
<td>CRO: manometry</td>
<td>CRO: Wexner, FIQL</td>
</tr>
<tr>
<td>Miner et al., 1990</td>
<td>25</td>
<td>M: 17–64; F: 30–76</td>
<td>FI</td>
<td>Phase 1:</td>
<td>3x 20 min for 3 days</td>
<td>CRO: rectal sensation; manometry</td>
<td>CRO manometry: PI 1 better</td>
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<tr>
<td>Naimy et al., 2007</td>
<td>49</td>
<td>36 (22–44)</td>
<td>AI after 3rd/4th grade rupture</td>
<td>1: EMG-BF (anal electrode)</td>
<td>1: 2x instructional session 30 min. At home: 5x 3 s, 10 s and submax as long as possible contractions 20 min, 2x/day for 8 weeks 2: 2x instructional session 30 min. At home: 30–40 Hz, &lt;80 mAmp (max tolerance), 3 s stimulation, 3 s rest, 20 min, 2x/day for 8 weeks</td>
<td>PRO: Wexner, FIQL, RQL, VAS</td>
<td>PI NS</td>
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Table 9.6 Characteristics of included randomized controlled studies of pelvic physical therapy for AI—cont’d

<table>
<thead>
<tr>
<th>Author</th>
<th>Norton et al., 2003</th>
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<tr>
<td>Sample size</td>
<td>171</td>
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<tr>
<td>Mean age (range) (years)</td>
<td>56 (26–85)</td>
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<td>Study population</td>
<td>FI</td>
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<tr>
<td>Methods</td>
<td>1: standard care (advice) 2: 1 + PFMT 3: 2 + clinical manometry BF 4: 3 + intra- anal EMG-BF at home</td>
</tr>
<tr>
<td>Training protocol</td>
<td>PFMT: 50 max sustained sphincter contractions + 50 fast-twitch contractions/day Manometry BF: sensory, coordination and strength training protocol. 9x 40–60 min sessions for 3–6 mth</td>
</tr>
<tr>
<td>Outcome</td>
<td>CRO: manometry</td>
</tr>
<tr>
<td>PRO: subjective improvement (range 0–11), diary, Vaizey, SF-36, HADS and disease-specific questionnaire</td>
<td></td>
</tr>
<tr>
<td>Results (between-group analyses)</td>
<td>CRO: PI 1 yr, NS</td>
</tr>
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<table>
<thead>
<tr>
<th>Author</th>
<th>Norton et al., 2006</th>
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<td>Sample size</td>
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<tr>
<td>Mean age (range) (years)</td>
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<td>Study population</td>
<td>FI and on waiting list for BF</td>
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<tr>
<td>Methods</td>
<td>1: ES (35 Hz) 2: placebo ES (1 Hz)</td>
</tr>
<tr>
<td>Training protocol</td>
<td>3 weeks 20 min/day, week 4–8 40 min/day</td>
</tr>
<tr>
<td>Outcome</td>
<td>CRO: manometry</td>
</tr>
<tr>
<td>PRO: subjective improvement VAS, diary, symptom questionnaire PRO: FIQL</td>
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<tr>
<td>Results (between-group analyses)</td>
<td>CRO: PI NS  PRO: PI NS</td>
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<tr>
<th>Author</th>
<th>Osterberg et al., 2004</th>
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<tr>
<td>Sample size</td>
<td>59</td>
</tr>
<tr>
<td>Mean age (range) (years)</td>
<td>68 (52–80) 64 (43–81)</td>
</tr>
<tr>
<td>Study population</td>
<td>Idiopathic (neurogenic) AI, despite dietary advice</td>
</tr>
<tr>
<td>Methods</td>
<td>1: levatorplasty 2: ES (anal electrode)</td>
</tr>
<tr>
<td>Training protocol</td>
<td>2–7 weeks (median = 4), 12 sessions 20 min, 25 Hz, duration 1.5 s, pulse-train interval 3 s, up to max tolerance</td>
</tr>
<tr>
<td>Outcome</td>
<td>CRO: physiological variables PRO: Miller’s incontinence score Physical and social limitations</td>
</tr>
<tr>
<td>Results (between-group analyses)</td>
<td>CRO: 3, 12, 24 mth, NS PRO: 3 mth, 1 better Physical and social limitations: 3, 12, 24 mth: 1 better</td>
</tr>
</tbody>
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<table>
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<tr>
<th>Author</th>
<th>Schwandner et al., 2010</th>
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<tbody>
<tr>
<td>Sample size</td>
<td>158</td>
</tr>
<tr>
<td>Mean age (range) (years)</td>
<td>1: 62.0 2: 63.6</td>
</tr>
<tr>
<td>Study population</td>
<td>AI</td>
</tr>
<tr>
<td>Methods</td>
<td>1: 3 T (triple target regimen): amplitude-modulated middle-frequency ES (AM-MF)+ EMG-BF 2: EMG-BF</td>
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(Continued)
Methodological quality

The PEDro rating score was used to classify the methodological quality of all included trials (Table 9.7), resulting in low (2/10) to high (8/10) methodological quality. It should be noted that the two criteria related to blinding of the therapist and patient are almost impossible to meet in physical therapy trials.

Pelvic floor muscle training

PFMT has been proven effective in the treatment of stress urinary incontinence (Berghmans et al., 1998). Since the anal sphincter and puborectalis muscle form a part of the same pelvic floor as the closing urethral system, expectations are raised for the same positive results of pelvic floor muscle re-educative techniques in AI. Hay-Smith et al. (2009) concluded that PFMT, properly taught, is still the mainstay of physical therapy.

Norton and Cody (2012) compared a group receiving PFMT with advice with a group with only advice, and found no statistical difference between the groups post-treatment and at one year follow-up. However, this study was executed by specialized nurses who were not pelvic physical therapists. Moreover, the PFMT programme left doubts about adequacy of the dose–response relationship.

Bartlett et al. (2011) found no difference between BF either combined with a PFMT programme of prolonged submaximal pelvic floor muscle (PFM) contractions or combined with the same contractions and quick repetitive maximal PFM contractions. Glazener et al. (2001) found that PFMT 3 months after delivery in women with urinary incontinence did not significantly decrease the risk of AI one year post partum. At 6-year follow-up, AI rates were similar between the active PFMT group and the standard care group (Glazener et al., 2005). Currently, no definite statement is available on the role of anal sphincter and PFM exercises as an intervention for AI patients. It is suggested that some aspects of PFMT have a therapeutic effect, but this is not proven so far (Norton and Cody, 2012).

PFMT and anal sphincter exercises might be recommended as part of an integral approach based on all treatable components, such as education/advice, awareness, BF and rectal balloon training (Bliss et al., 2013). This recommendation is also based on the low costs and lack of side-effects of this intervention.

Biofeedback

Success rates of physical therapy in AI are generally based on numerous uncontrolled trials, mainly focusing on BF therapy. There are over 60 uncontrolled trial reports in the literature on the use of BF for the management of

<table>
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<th>Table 9.6 Characteristics of included randomized controlled studies of pelvic physical therapy for AI—cont’d</th>
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<tr>
<td>Training protocol</td>
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<tr>
<td>3T: 25 kHz+biphasic modulations of 40 Hz. 1 pulse=5–8s with pause of 10–15s, ≥80–100 mA, 20 min, 2/day for 9 mth</td>
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<tr>
<td>EMG-BF: 20 min, 2/day for 9 mth, contraction 3–8s, pause 10–15 s</td>
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<tr>
<td>PRO: Vaizey, Wexner; FIQL; Park score; Continence</td>
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<tr>
<td>Training protocol</td>
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<tr>
<td>1x/mth 30 min for 5 mth</td>
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<tr>
<td>Outcome</td>
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<tr>
<td>PFMT at home: 10 sessions 10x 5s sphincter contractions</td>
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<tr>
<td>Outcome</td>
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<tr>
<td>PRO: Pescatori, St Mark’s Hospital FI score, VAS, questionnaire results of preset aims</td>
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<tr>
<td>Results (between-group analyses)</td>
</tr>
<tr>
<td>CRO: manometry, fatigue time, fatigue contractions</td>
</tr>
<tr>
<td>PRO: Pescatori, St Mark’s Hospital FI score, VAS, questionnaire results of preset aims</td>
</tr>
<tr>
<td>Results (between-group analyses)</td>
</tr>
<tr>
<td>CRO: PI NS</td>
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</table>

BDI, Beck Depression Inventory; CRO, clinician-reported outcome; EAS, external anal sphincter; HADS, Hospital Anxiety and Depression Scale; MTV, maximal tolerable volume; NS, not significant difference; PF, pelvic floor; PI, post-intervention; PNTML, pudendus nerve terminal motor latency; PP, per protocol; PRO, patient-reported outcome measures; RQL, Reduced Quality of Life scale; STAI, Spielberger State–Trait Anxiety Inventory. For other abbreviations, see text.
Evidence-based physical therapy for pelvic floor dysfunctions

Evidence (Norton and Kamm, 2001; Norton and Cody, 2012). Some authors conclude that BF is the treatment of choice for AI on the basis of these observational studies (Enck et al., 1994) and controlled clinical trials (Enck et al., 1994; Guillemot et al., 1995; Heymen et al., 2009). An overall cure and improvement rate of about 70% has been reported (Norton et al., 2003; Solomon et al., 2003).

With regard to controlled trials, some authors report on the hypothesis that BF together with another intervention

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<th>Study</th>
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<tr>
<td>Bols, Berghmans et al., 2012</td>
<td>+</td>
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+, criterion is clearly satisfied; −, criterion is not satisfied; ?, not clear if the criterion was satisfied. Total score is determined by counting the number of criteria that are satisfied, except that ‘eligibility criteria specified’ score is not used to generate the total score. Total scores are out of 10.
is more effective than another intervention alone. Healy et al. (2006) found no statistical difference in effect of supervised EMG-BF and endo-anal ES in comparison with endo-anal ES at home. Naimy et al. (2007: 124) compared EMG-BF with ES (anal probe) in patients with AI after third/fourth-degree sphincter rupture. After treatment, there were no differences between the study groups. It should be noticed that the intervention lasted no more than 2 months.

Heymen et al. (2009) and Bols et al. (2010) evaluated patients with FI and AI respectively, who failed earlier conservative treatment (dietary adaptations, medication) in a stepwise approach. Heymen et al. (2009) concluded that manometric BF as add-on therapy to PFMT significantly improved squeeze pressure, Faecal Incontinence Severity Index scores and subjective improvement post-intervention compared to PFMT alone.

Bols et al. (2010) compared PFMT alone against PFMT and rectal balloon training. Adding rectal balloon training significantly improved maximal tolerable volume, subjective improvement and the subscale ‘Lifestyle’ of the Faecal Incontinence Quality of Life scale (FIQL), although loss of power in this study should be taken into account.

Davis et al. (2004) compared anal sphincter repair with and without subsequent manometric BF and home-based PFMT commenced 3 months postoperatively in a small group of women with obstetric sphincter trauma during 6 weeks only. There were no statistically significant differences between both groups.

Ilnyckýj et al. (2005) found no difference between a group with PFMT and manometric BF and a group with PFMT only. This intervention contained only four treatment sessions over 4 weeks.

Some authors studied whether one BF modality would be more effective than other BF modalities. Solomon et al. (2003) found no difference between groups using PFMT (feedback by digital palpation) combined with anal manometric BF or PFMT combined with transanal ultrasound BF. Heymen et al. (2000) found no difference between clinical EMG-BF, clinical EMG-BF with rectal balloon training, clinical EMG-BF with EMG-BF at home and clinical EMG-BF with rectal balloon training and EMG-BF at home. Miner et al. (1990) compared a group with sensory BF with feedback and without feedback. The group with feedback significantly improved more regarding rectal sensation, incontinence episodes frequency and regaining continence.

There is little evidence which method of BF is superior due to the small study samples and the doubtful training intensity, especially in the two last-mentioned studies. The results of a Cochrane review on the effects of BF and/or PFMT for the treatment of AI in adults, based on 21 randomized controlled trials, showed that some elements of BF therapy, like rectal balloon training and sphincter exercises, might have a therapeutic effect (Norton et al., 2006). However, a recent meta-analysis of Enck et al. (2009) concluded that BF for AI is not different in efficacy from non-BF therapy and no differences were observed comparing various modes of BF. In summary, the use of BF as a treatment for AI is recommended after other behavioural and medical management has been tried if inadequate symptom relief has been obtained. This recommendation is based on the numerous positive outcomes from uncontrolled trials, limitations in the current RCTs and low morbidity associated with its application (Norton et al., 2010; Bliss et al., 2013).

**Electrical stimulation**

A second Cochrane review by Hosker et al. (2007) evaluated ES in adult patients with AI. Four eligible trials with 260 participants were identified. Findings from one trial suggested that ES with anal BF and exercises provides more short-term benefits than vaginal BF and exercises for women with obstetric-related AI (Fynes et al., 1999).

Another study found contradictory results, with no added benefit from ES over BF and exercises alone (Mahony et al., 2004; Osterberg et al., 2004; Naimy et al., 2007). Although all trials included in this Cochrane review reported that patients’ symptoms were generally improved, it was not clear that this was the effect of ES. No further conclusions could be drawn from the data available (Hosker et al., 2007). Norton et al. (2006) examined in a randomized controlled trial whether anal ES, using an anal probe electrode in the absence of any adjunctive exercises or advice, would improve symptoms of AI and anal sphincter pressures when compared with ‘sham’ ES. Patients rated that their bowel control had improved to a modest extent. However, there was no statistically significant difference detected between the groups, suggesting that 1 Hz was as effective as 35 Hz. This raises the possibility that the main effect is not sphincter contraction but sensitization of the patient to the anal area, or simply the effect of intervening per se (Norton et al., 2006). This result is in agreement with Mahony et al. (2004), who concluded that the addition of ES did not enhance symptomatic outcome. Schwandner et al. (2010) compared amplitude-modulated middle-frequency ES as adjunct to EMG-BF and PFMT/anal sphincter training (triple target regimen = 3T) with EMG-BF and PFMT/anal sphincter training only. The 3T was statistically significant as superior on all outcome measures, except for quality of life.

Overall, patients seem to benefit from physical therapy interventions and results are promising (Norton et al., 2009). However, uncertainty exists on the effectiveness of physical therapy interventions in AI, and therefore it needs to be further elucidated which intervention is superior and which intervention is suitable for a particular patient.
PREDICTIVE FACTORS FOR SUCCESS OF PELVIC PHYSICAL THERAPY

Evaluation of predictive factors for success of pelvic physical therapy is hindered by the heterogeneity of studies, especially regarding study population, type of interventions and intensity of therapy.

In general:
• An adequate training dose (train specific muscles, 3x/day, 2–3x/week during at least 5 months, 8–12 nearly maximal contractions) and adherence to therapy, increases the likelihood of recovery (Bo and Aschehoug, 2007).

Biofeedback with PFMT:
• Longer time since AI onset decreases the chance of recovery after BF with PFMT (Bols et al., 2012b).

• Experiencing minor embarrassment, the use of constipation medication and presence of at least one delivery-related risk factor (including high birth weight/episiotomy/instrumental delivery/prolonged second stage of labour/breech delivery) increase the chance of recovery after BF with PFMT (Bols et al., 2012b).

• Necessity of more than three BF sessions is predictive for deterioration during follow-up (Ryn et al., 2000).

Biofeedback with PFMT and electrical stimulation:
• Having passive AI, diarrhoea, primary repair of a rupture after vaginal delivery and perineal and/or perianal scar tissue decreases the chance of recovery (Terra et al., 2008).

Electrical stimulation:
• Less severe FI symptoms and the loss of liquid stool instead of solid stool increases the chance of recovery after ES (Osterberg et al., 1999).

REFERENCES


Evidence-Based Physical Therapy for the Pelvic Floor


Evidence-based physical therapy for pelvic floor dysfunctions

INTRODUCTION, EPIDEMIOLOGY, AND PATHOPHYSIOLOGY/AETIOLOGY

This section considers pelvic pain and/or related dysfunction originating (or considered to be originating) in the pelvic floor muscles (PFM) and their bony attachments, fascia and ligaments, including both deep (levator ani) and superficial (perineal muscle) layers. Dysfunction may be primarily from the PFM, occur secondary to visceral changes (lower urinary tract, reproductive tract or anorectum), or be referred from other pelvic somatic (cutaneous) and visceral (mammary) structures.

Pelvic pain presents a complex and challenging problem to the clinician and researcher alike, arising from a multitude of possible causes and conditions. The Institute of Medicine Pain Consensus Statement recognizes that while pain may arise in the nervous system it represents a complex and evolving interplay of biological, behavioural, environmental and societal factors that go beyond simple explanation (IoM, 2011). The complexity of pelvic pain is compounded by the close relationship between visceral and somatic structures within the pelvis, the complexity of neurogenic innervation of these structures, and by the intimate nature of this body region, affect on personal relationships and sexuality. Further, chronicity of pelvic pain and the potential for central sensitization of pain adds an even more challenging layer over the study of this condition and clinical management of patients who suffer from it.

Pain is defined as ‘an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage’ (Baranowski et al., 2012). Historically, chronic pain was defined as pain persisting for greater than 6 months, however currently it is considered that if non-acute and central sensitization mechanisms are well documented, then the pain may be regarded as chronic, irrespective of the time period (Baranowski et al., 2012). Predisposing factors, causes, central and peripheral mechanisms are illustrated in Figure 9.1. Pain may be a symptom of many syndromes, conditions or diseases, but in chronic pain, pain per se may be considered the disease, in its own right (Siddall and Cousins, 2004).

Acute pelvic pain is usually associated with well-identified pathology (e.g. trauma, tumour, infection) or physiological process (e.g. inflammation) and presents far less of a diagnostic dilemma than chronic pain. The scientific understanding of chronic or persistent pain continues to evolve, and has recently been reviewed by Melzack and Katz (2013), incorporating their Neuromatrix Theory.

Chronic pelvic pain (CPP) is a common condition. The true prevalence and incidence of pelvic floor pain alone, or co-existing with other CPP conditions, is unknown. Prevalence of female CPP – of all types – appears highest in the reproductive years, with prevalence rates varying from 15% in the USA (Mathias et al., 1996) to 21.5% in Australia (Pitts et al., 2008) and 24% in the UK (Zondervan et al., 2001). In the Australian sample, up to 20% of those with pelvic pain described it as severe
Evidence-Based Physical Therapy for the Pelvic Floor

Precise prevalence of male pelvic floor pain is also unknown, however the more specific conditions of bladder pain syndrome or chronic prostate pain syndromes are thought to occur in up to 5% of men (Suskind et al., 2013). In the absence of a specific disease or well-defined condition, chronic pain in structures related to the pelvic floor is currently considered a ‘syndrome’ rather than a condition or disease (Baranowski et al., 2012; Engeler et al., 2012). This diagnosis is usually made after excluding conditions of well-understood aetiology, which have clear diagnostic identification and indications for treatment. One system for classifying CPP syndromes has been proposed by Engeler et al. (2012), as shown in Figure 9.2. This system describes various visceral and somatic CPP syndromes according to phenotyping, terminology and taxonomy. The focus is on defining the end-organ dysfunction; a process that is useful for identification of the tissue which may be the primary pain generator in peripheral dysfunction. While useful as a classification schema, a limitation is the frequently observed overlap of many of these diagnoses (Peters et al., 2008). An aetiological model to explain the connections observed between visceral and myofascial CPP syndromes has been proposed by Hoffman (2011). This model describes the inter-relationships between viscero-visceral convergence; viscero-somatic convergence; increased tension of pelvic floor muscle (PFM) creating visceral symptoms along with somato-visceral convergence; and central sensitization with expansion of receptive fields, as illustrated in Figure 9.3.

To fully understand CPP, an appreciation of pain neurophysiology is required to ensure assessment of the patient moves beyond peripheral tissue dysfunction (nociceptive pain) to include peripheral sensitization, neuropathic pain and central sensitization (Hilton and Vandyken, 2011; Vandyken and Hilton, 2012). This framework builds on a previous pain neurophysiology work (Moseley, 2007), which attempts to explain the continuum of peripheral tissue dysfunction and central sensitization and how pain neuroscience applies to patients with CPP. The International Association for the Study of Pain (IASP) provides the following definitions: peripheral sensitization refers to increased responsiveness and reduced threshold of nociceptive neurons in the periphery to the stimulation of their receptive fields; central sensitization refers to increased responsiveness of nociceptive neurons in the

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(Figure 9.1 Predisposing factors, causes, central and peripheral mechanisms of chronic pelvic pain. Reproduced from Engeler et al., 2012 with permission.)
central nervous system to their normal or sub-threshold afferent input. Clinically, sensitization may only be inferred indirectly from phenomena such as hyperalgesia or allodynia (IASP, 2011). Clinicians need to be prepared and able to move between the ‘peripheral’ and the ‘central’, not dismissing one or the other (Edwards and Jones, 2013).

**Pelvic floor pain**

Chronic pelvic floor pain syndrome of musculoskeletal origin – in common with chronic pelvic pain of various visceral origins – does not have a well-understood aetiology; there are no clear diagnostic markers. Furthermore, patient management suffers from insufficient evidence-based investigations. This uncertainty is reflected in the rather broad and imprecise terminologies that are proposed for this syndrome. One such term is ‘pelvic floor muscle pain syndrome’ (Baranowski et al., 2012; Engeler et al., 2012), which is defined as:

the occurrence of persistent or recurrent episodic pelvic floor pain. There is no proven well-defined local pathology. The pain may be associated with negative cognitive, behavioural, sexual or emotional consequences, as well as with symptoms suggestive of lower urinary tract, sexual, bowel or gynaecological dysfunction. This syndrome may be associated with overactivity of or trigger points within the pelvic floor muscles. Trigger points may also be found in several muscles, such as the abdominal, thigh and paraspinal muscles and even those not directly related to the pelvis. (Baranowski et al., 2012; Engeler et al., 2012.)
This classification system includes further description of characteristics associated with the pain syndrome (such as referral and temporal patterns, psychological and associated pelvic symptoms) but does not describe how these symptoms and signs are evaluated. Future research may well add certainty to our understanding of this syndrome and lead to a well-defined condition with clear indications for treatment.

Another phenotyping system for CPP syndromes which incorporates muscle-related pain has been developed by Shoskes et al. (2009). This system has been developed for male urological/prostate pain syndromes. Six domains make up the classification system: Urological, Psychosocial, Organ specific, Infection, Neurologic/systemic conditions, and Tenderness of skeletal muscles, giving the acronym UPOINT. The last domain, tenderness, is defined as ‘palpable muscle spasm or trigger points in abdomen and pelvic floor’ (Shoskes et al., 2009), and measurement is dichotomous, with each domain scored as Yes or No. The method of measurement for muscle tenderness is not described, however the authors acknowledge the domains remain open to further sub-categorization as new mechanisms and biomarkers are discovered.

The following descriptions of terms are provided to assist understanding of what chronic PFM syndrome is. They are primarily based on clinical examination, and originate from outside of the PFM literature.

## Muscle pain

Muscle pain or tenderness is known as myalgia and may be a symptom of many diseases or disorders (Mense and Simons, 2001). Myalgia can be described as below, however the distinctions between these definitions may be arbitrary:

- A trigger point is defined as a tender, taut band of muscle that can be painful spontaneously or when stimulated (Gerwin, 2010). Local or referred pain may be reproduced. An active trigger point is said to have a characteristic ‘twitch’ response when stimulated, however the twitch response to palpation has been shown to be unreliable (Lucas et al., 2009). The most reliable sign is sensitivity to applied pressure, however due to the limited number of studies available, and significant methodological problems, physical examination is not currently recommended as a reliable test for the diagnosis of trigger points (Lucas et al., 2009). There have been no reports of reliability testing of trigger points in the PFM.

- Myofascial pain is localized pain or tender spots within the muscles or their fascial linings, with or without trigger points being identified. For a further overview of myofascial pain syndromes and their evaluation, the reader is directed to Giamberadino et al. (2011).

- Fibromyalgia, which refers to the constellation of widespread pain and muscle tenderness, affecting multiple body sites, accompanied by fatigue, unrefreshed sleep and cognitive problems (Wolfe et al., 2010). Trigger points found in fibromyalgia may have a specific body localization.

Pelvic floor muscle pain or tenderness may therefore also be called pelvic floor myalgia, or perineal or levator ani myalgia if the location of the pain is specific to the superficial or deep pelvic floor musculature.

## Pelvic floor muscle tension

Resting muscle tension is determined as the resistance to stretch, passive movement or deformation when the tissue is digitally palpated. Mense et al. (2001b) suggest this can be determined by the compliance (compressibility) of a muscle. This is assessed by pressing a finger into a muscle belly to determine how easily it indents and how ‘springy’ it is. Normal PFM function includes the ability to remain in a ‘normal’ rested state when required, the ability to contract when required and the ability to relax fully following a contraction. The difficulty with interpreting and measuring these states is that little normative data exist on ‘normal’ PFM activity, hence it is difficult to describe
and quantify deviations from normal. These states of PFM activity each exist on a continuum: (1) low to high resting tension; (2) low to high contractile activity; and (3) absent, partial or incomplete ability to relax post contraction. The first and the third states have proven to be the most challenging to describe and measure.

Pelvic floor muscle ‘overactivity’ is a term applied (Messelink et al., 2005; Messelink, 2007) to a condition in which the PFM do not relax, or may even contract when relaxation is functionally needed, for example during micturition, defecation or sexual intercourse. Initially the term ‘overactivity’ was based on a symptom (such as pain) plus a physical examination sign (such as inability to relax the PFM or an increased resting level of tension).

Various terms can be found throughout the literature referring to this sign, including: tone, tension, stiffness, firmness, hardness, rigidity, compliance, resting pressure, compressibility, elasticity; with the terms hypertonic, high-tone, shortened pelvic floor, overactive, non-relaxing and spasm used to refer to the finding of increased activity in the PFM. It is important to note that the term ‘tone’ has a very specific neurological meaning in upper (hypertonic: rigidity) and lower (hypotonic: flaccidity) motor neuron lesions (Bhidayasiri et al., 2005), and as such, would seem incorrect when applied to muscle tension in a patient without a neurogenic condition.

Muscle tension, stiffness or tightness may be the preferred term when referring to the state of muscle activity (at rest, on contraction or post-contraction relaxation) in the non-neurogenic pelvic floor and encompasses:

- muscle spasm: persistent contraction of striated muscle that cannot be released voluntarily. If the contraction is painful, this is usually described as a cramp. Spasm would be associated with electromyographic (EMG) evidence of contraction in the muscle;
- compliance: the compressibility of a muscle, clinically assessed by pressing a finger into it to determine how easily it is indented and how ‘springy’ it is. This may be synonymous with stiffness or hardness.

Physiologically, resting muscle tension is the combination of the elastic and viscoelastic stiffness in the muscle, in the absence of contractile activity (Simons and Mense, 1998). Muscle tension can be affected by exogenous factors, such as the amount of pressure applied, and endogenous factors, such as muscle activation, tissue thickness, cross-sectional area of the muscle itself, and fluid present within the muscle (swelling, inflammation) (Mense et al., 2001b). No consensus exists regarding objective quantification of this finding.

**Diagnostic terminology**

If features of both muscle pain and increased tension at rest are present, the diagnosis of ‘PFM pain syndrome’, or the ‘overactive pelvic floor’ is often made. If both pain and increased tension in the PFM are present at rest or post contraction, the alternative diagnosis of PFM (or perineal/levator ani) tension myalgia can be made. This may be a more accurate syndrome description as both the symptom and the sign are present in the diagnostic term.

Historically, the terminology that has been used to describe this syndrome of pelvic floor pain plus increased tension is varied, and includes coccydynia (Thiele, 1937; De Andrés and Chaves, 2003), levator ani (spasm) syndrome (Smith, 1959; McGivney and Cleveland, 1965), levator ani syndrome as a subset of chronic proctalgia (Drossman, 2006), tension myalgia of the pelvic floor (Sinaki et al., 1977), pelvic floor spasticity (Kuipers and Bleijenberg, 1985) urethral/anal sphincter dysynergia (Whitehead, 1996), vaginismus (Masters and Johnson, 1970) and shortened pelvic floor (Fitzgerald and Kotarinos, 2003). These terms suggest various involvement of bladder, bowel and sexual function, in addition to pain and muscle tension. Considering the more recent recommendations of pelvic pain syndrome terminology, if the primary generator of pelvic pain appears to be the levator ani, and both muscle pain and muscle tension are identified, the diagnostic term levator ani tension myalgia, or chronic PFM pain syndrome may be preferred. If bladder, sexual and/or bowel dysfunction accompanies the muscle pain and increased tension, CPP syndrome may be the most appropriate term.

**Summary**

It is often difficult to differentially diagnose pain originating from deep muscular structures from that emanating from visceral structures, as the symptoms are often similar due to anatomic proximity and shared innervation, as well as viscerosomatic convergence and projection, hence to the patient, pain may be perceived in either location. Pelvic floor muscle pain and altered tension are often present in patients with CPP syndromes and are currently considered the diagnostic feature of chronic PFM syndrome, or pelvic floor tension myalgia. Further research is needed to determine the extent to which muscle dysfunction can
lead to or exacerbate pain, and the extent to which pain may lead to muscle dysfunction.

**ASSESSMENT**

This section focuses on the steps required to effectively evaluate the PFM in a patient who presents with suspected PFM pain ± increased resting muscle tension. The subjective history and objective physical assessment of PFM pain should be comprehensive and relevant to the presenting symptoms and signs. A thorough biopsychosocial history should be taken, to include assessment of all biological, medical, psychological and social factors known to affect pelvic floor dysfunction and pain. Embracing the psychosocial components in tandem with the biomedical aspects early in assessment can be challenging for clinicians trained in a traditional biomedical model of healthcare. A comprehensive assessment also assists the clinician to evaluate the relative contribution of peripheral tissue dysfunction, peripheral sensitization and central sensitization to the patient’s condition. Expert multidisciplinary input may be required to effectively investigate suspected depression, anxiety, catastrophization, hypervigilance, fear, anxiety and poor pain self-efficacy, which have been linked with higher pelvic pain scores in men (Shoskes et al., 2009), and poorer outcomes of treatment for pelvic pain in women (Desrochers et al., 2010). Psychosocial factors judged to be potential barriers to a patient’s recovery need to be addressed in the overall management with consideration of referral for assistance when the scope of psychosocial involvement is beyond the skills of the treating clinician (Edwards and Jones, 2013). Assessment of peripheral tissue dysfunction and central sensitization are not mutually exclusive approaches, although philosophically they may appear as conflicting standpoints (Engeler et al., 2013). The clinical process of peripheral tissue assessment does not distinguish between acute and chronic PFM pain, as the assessment of the peripheral tissues – when indicated – should be undertaken with the same degree of methodological rigour and clinical sensitivity, regardless of chronicity.

**Confirmation of primary pain generator**

If the clinician suspects PFM tension myalgia is the primary complaint the patient describes, the PFM must be fully evaluated to confirm the primary tissue pain generator. You may be the first clinician whom the patient consults, hence adequate clinical investigation is required to exclude serious pathology, avoid missing obvious visceral dysfunction and to ‘rule in’ or ‘rule out’ local tissues as primary pain generators, so that treatment is effectively targeted. This is the biomedical part of the biopsychosocial model of CPP evaluation. Tissue assessment does not dictate treatment approach, but may provide the clinician with symptoms and signs to use in re-assessment. It may not be possible to measure whether local nociceptive factors or central factors are the dominant pain mechanism, and it is possible the patient may present with both.

**Subjective assessment: symptoms**

**Pain history**

As pain is often the predominant symptom, a thorough pain history should include: the site and duration of pain, nature of onset or precipitating event, pain characteristics, and response of pain to activity and associated symptoms (Hopwood, 2000). The clinician must be aware of the terminology the patient uses to describe his/her ‘pain’ symptom, which may include discomfort, tenderness, pressure, sensitivity, soreness, and refer to this terminology accordingly. Pelvic floor muscle pain may be present at rest or appear mechanical in nature, e.g. altered with change of posture, movement, activity or state of muscle activity. The clinician needs to determine factors that suggest peripheral dysfunction, such as a mechanical onset/trauma as the precipitating initial event, as well as be aware of the clinical state in which the patient presents at the time of consultation. From the pain history, factors that suggest central sensitization include patient description of disproportionate and non-mechanical pain, fear avoidance and catastrophization, poor sleep and multiple system involvement. If these symptoms are present, Hilton and Vandyken (2011) make a case for addressing these aspects before consideration of an internal examination, to establish the presence and extent of central sensitization and any fear associated with the perceived threat of an internal examination. Clinicians should always be mindful of the association between CPP and prior history of sexual abuse or trauma in women (Lampe et al., 2000) and men (Hu et al., 2007).

**Pain rating scales**

Pain may be assessed simply on a dichotomous scale: present or absent; however, severity of pain cannot be differentiated, which poses a challenge for measuring graded response to treatment. Commonly used ordinal pain scales include a numerically rated (0–10) scale (NRS) and a visual analogue pain intensity scale (VAS) (Wallerstein, 1984). As pain levels may vary between most pain experienced, average pain levels and pain at rest, it can be useful for the patient to complete a pain scale for each state.

**Pain mapping**

Pain mapping may include a pain checklist or a pain chart. A pain checklist is a list of anatomical locations from which the patient selects relevant sites to his/her complaint.
While in use in paediatric and cognitively impaired populations, there are no reports in the literature of the use of pain checklists in pelvic pain assessment. A pain chart/body map is a simple line drawing of an outline of the human body, onto which the patient sketches or ticks or marks areas of bodily pain to demonstrate the site and extent of perceived pain. They are considered robust for use in the general musculoskeletal population (Ohnmeiss, 2000). While in clinical use, there are no reports of psychometric testing of pain charts for suitability of use in patients with pelvic pain. In addition, as the PFM are located internally, a patient may have difficulty identifying these muscles as the site of their pain on a unidimensional diagram.

Pain questionnaires

General pelvic floor or specific organ dysfunction questionnaires may also be used to record pain co-existing with other pelvic floor dysfunction. These include the Australian Pelvic Floor Questionnaire (Baessler et al., 2009), the Pelvic Floor Distress Inventory (Barber et al., 2005), the Urogenital Distress Inventory (Shumaker et al., 1994), the O’Leary–Sant Indexes (O’Leary et al., 1997; Lubeck et al., 2001), the National Institutes of Health Chronic Prostatitis Symptom Index (NIH-CPSI) (Litwin et al., 1999), the Prolapse–Incontinence Sexual Questionnaire-12 (Rogers et al., 2003) and the Female Sexual Function Index (Rosen et al., 2000). All of these questionnaires have acceptable psychometric properties, so choice will be determined according to the patient’s predominant clinical presentation. Pain may also be measured descriptively: CPP has been assessed using more generic questionnaires such as the McGill Pain Questionnaire, Short-Form McGill Pain Questionnaire, Chronic Pain Grade Scale and the Short Form-36 Bodily Pain Scale, summarized well by Hawker et al. (2011).

Objective assessment: signs

Examination

Firstly, obvious pathology which may present with primary or referred PFM pain or altered tension should be assessed and excluded. Following this, much information may be gathered by objective assessment of the PFM, but the validity, reliability, responsiveness and generalizability of this information is limited by a lack of normative data especially in patients with increased PFM tension. Despite this, the information that is gathered through assessment is of prime clinical utility, and may lead the way to the development of robust assessment tools and normative values of PFM activity. Basic PFM observation and objective assessment should be performed as described in Chapter 5.

The distinguishing features of chronic PFM pain syndrome – or levator tension myalgia – are pain and altered tension (± trigger points) in the PFM. Hence in the assessment of a patient with pelvic pain, particular emphasis is placed on assessment of pain and muscle resting tension, contractile activity and the ability to relax. Sensitive and careful examination is vital. A suggested process to elicit useful information in the physical assessment is outlined below.

Visual inspection of the perineum at rest. The genital hiatus may appear reduced in size and the perineal body displaced anteriorly if there is increased tension in the PFM at rest.

Observation of contraction, relaxation and bearing down. Movement associated with PFM contraction may be absent due to an increased level of resting tension, or display obvious change in recruitment patterning, timing and proprioception. Incomplete, abnormal (discordant) or absent relaxation may be observed following attempted contraction, or the patient may need to attempt relaxation several times before the PFM activity subsides. Bearing down may be absent or result in an in-drawing. Rapid onset of fatigue may be noted. On attempted location and isolation of a PFM contraction, accessory muscle activity may be observed in the thigh and buttock muscles and trunk. It may be helpful to note the presence or absence of PFM in-drawing with a volitional cough, however, this is difficult to scale objectively.

Sensation/neurological integrity. In the absence of spinal or neurological injury, the anal wink reflex may be absent due to an involuntary contraction or increased level of resting tension in the PFM.

Assessment of the external tissues, sites of possible pain referral and the perineum. Digital palpation of the external genitalia and perineum is systematic and considers tissue quality, sensation, temperature and tenderness. Thickening of subcutaneous connective tissue around perineal or suprapubic trigger points, or their regions of pain referral, may be identified on palpation as altered tissue bulk, contour, elasticity and temperature, along with variation in colour (Fitzgerald and Kotarinos, 2003).

Assessment of the internal vagina/rectum. This should always be performed gently and sensitively. In this population, careful attention is required to assess the symptom of myalgia and the signs of altered tension (at rest, on contraction and relaxation), and trigger points in the levator ani.

- **Myalgia:** Digital evaluation should always be performed very gently, with a single, well-lubricated digit. The clinician should evaluate: the presence of pain/pelvic floor myalgia, identifying and recording the site, nature and whether it is localized or diffuse. The aim is to reproduce the patient’s pain, at a mild intensity only. Pain may also be assessed on PFM contraction, and on relaxation following contraction. Pain in the PFM should be assessed and recorded bilaterally.
It is uncommon to find pain on digital palpation of the PFM in asymptomatic women (Tu et al., 2008b; Montenegro et al., 2010; Kavvadias et al., 2013), whereas PFM tenderness is commonly observed in women (Fitzgerald et al., 2011) and men (Shoskes et al., 2008) with CPP. Hence pain on palpation of the PFM should be considered a positive finding.

Four studies have tested the reliability of a scale designed to record patient-reported pain on clinician’s digital palpation of the PFM, as shown in Table 9.8. Differences in reliability findings may be explained by variance in populations tested (age, parity, symptom status), pelvic muscle topographical sites assessed, pressure applied by tester, scales and scoring methods, resulting in large differences in reliability found within and between studies. Only one scale reported results for levator ani alone, however the reliability values varied widely between anterior and posterior, and left and right levator ani. Furthermore, this study investigated only pain-free women, therefore this scale requires further testing in a symptomatic cohort.

Muscle tension: Pelvic floor muscle tension may be present during the examination, and may be associated with pain. Tension may be invoked by overly-vigorous examination, therefore it is paramount to assess the PFM carefully. Increased tension may be present at rest, or be provoked by a voluntary PFM contraction attempt, and thus confound measurement of the voluntary contraction. Tension may be detected generally throughout the PFM, or concentrated in discrete areas, or taut bands (which may themselves harbour a trigger point). The location and type of altered tension should be described as clearly as possible; marking on an anatomical sketch may assist accuracy. It is important to use a valid and reliable scale for assessing muscle tension, as this sign is often the target of treatment, in order to effect a reduction in pain.

While many studies have observed increased PFM tension in patients with pelvic floor pain, only one study has investigated the reliability of a scale for measuring levator ani tension: Kavvadias et al. (2013) investigated the intra- and inter-rater reliability of PFM tension in 17 nulliparous asymptomatic women, using a 3-point digital palpation scale (high, normal or low tone). Exact description of pressure applied when assessing ‘tone’ was not stated. Their results revealed very low (poor) and non-significant scores for reliability (ICC = −0.36, p = 0.92 – 0.03, p = 0.45). Dietz and Shek (2008) tested the reliability of a 6-point scale they devised to test the elasticity of the PFM in women with POP. The method of assessment involved passive distension of the levator hiatus. While their scale demonstrated moderate reliability (κ = 0.55, 95% CI 0.44–0.66), it has not been tested in a population with pain.

Trigger points: the sign of increased resting tension in the PFM may be accompanied by the presence of a trigger point.

Contractile activity of the PFM. This is covered in detail in Chapter 5.

Relaxation of the PFM. Pelvic floor muscle relaxation has been defined as the diminution or termination of PFM contraction (Messelink et al., 2005) and is always tested after a contraction. The ICS proposed qualitative rating scale has three levels: absent (no relaxation palpable), partial (return to resting state), complete (relaxation beyond the resting level). Reliability testing of this scale by Sliker-ten Hove et al. (2009) revealed substantial intra-rater reliability (κ = 0.76; 95% CI: 0.59–0.87), however inter-rater reliability was only fair (κ = 0.39; 95% CI: −0.01–0.38) indicating that further refinement of the scale is required. These authors proposed an improvement to the scale may occur with an additional level of relaxation added, to be termed ‘incomplete’, which indicates relaxation that does not (quite) reach the resting level. Another scale for PFM relaxation in a neurogenic population has been described as: 3 for active (good) relaxation after active contraction; 2 denotes hypertonic muscle with temporary relaxation after elongation; and 1 indicates a spastic muscle, unable to relax even after passive elongation (De Ridder et al., 1998). This scale has been reported to be reliable in a population of multiple sclerosis patients. It is assumed that elongation is achieved by stretching the PFM, however the technique to perform this assessment has not been described.

Further evaluation/investigations

Instrumented methods of measuring PFM pain/sensitivity and PFM tension have been described, and potentially offer more promise than digital scales as objective measures of these aspects.

Pressure-pain thresholds

- Vulval tissue: A vulvalgesiometer has been reported to reliably measure the genital pressure-pain threshold in the vulval tissues of women with urogenital pain (Pukall et al., 2007), however this device is available in the research setting only.
- Vaginal pressure algometer: Tu et al. (2008a) investigated the reliability of vaginal pressure-pain
### Table 9.8  Reliability of digital palpation scales to assess pelvic floor muscle pain

<table>
<thead>
<tr>
<th>Study</th>
<th>Population</th>
<th>Scale tested</th>
<th>Tissue assessed</th>
<th>Method of measurement</th>
<th>Scaling and score</th>
<th>Reliability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tu et al. 2008</td>
<td>20 asymptomatic and 19 women with ‘chronic pelvic pain’; mixed parity</td>
<td>‘Muscle hyperalgesia scale’</td>
<td>Iliococcygeus, pubococcygeus, coccygeus, obturator internus</td>
<td>‘Small rotating movements of the index finger’</td>
<td>4-point pain scale, 8 sites = composite score 0–24</td>
<td>$\kappa$ = 0.02–0.35 (collapsed to 2-pt pain scale, $\kappa$ = 0.04–0.63)</td>
</tr>
<tr>
<td>Slieker-ten Hove et al. 2009</td>
<td>41 women, with and without pelvic floor disorders; mixed parity</td>
<td>‘Pain scale’</td>
<td>Vaginal walls: anterior, posterior, left and right</td>
<td>Amount of pressure not stated</td>
<td>Dichotomous pain scale: present vs absent</td>
<td>Intra-reliability: $\kappa_w = 0.79$; inter-reliability: $\kappa_w = 0.85$</td>
</tr>
<tr>
<td>Montenegro et al. 2010</td>
<td>48 asymptomatic and 108 women with ‘chronic pelvic pain’; mixed parity</td>
<td>‘Pelvic muscle tenderness’</td>
<td>Levator ani, obturator internus, piriformis</td>
<td>‘As comfortable and delicate a manner as possible’</td>
<td>No pain; painful discomfort; intense pain; maximum total score of 12</td>
<td>$\kappa$ = 0.91 for tenderness (levator ani not assessed independently)</td>
</tr>
<tr>
<td>Kavvadias et al. 2013</td>
<td>17 asymptomatic, nulliparous women</td>
<td>‘Pelvic muscle tenderness’</td>
<td>Levator ani (anterior; posterior), obturator internus, piriformis</td>
<td>‘Pressure was steadily applied’</td>
<td>0–10 VAS</td>
<td>ICC = 0.28–0.87 for levator ani</td>
</tr>
</tbody>
</table>

ICC, intraclass correlation; VAS, visual analogue scale.
Pelvic floor muscle tension

- **Pressure manometry:** While this method has established reliability in an asymptomatic population (Frawley et al., 2006) and has been used in CPP intervention studies to measure pre-/post-treatment values of resting pressure (Palsson et al., 2004; Abbott et al., 2006; Rogalski et al., 2010), reliability of the tool in a pain population has not been established. Thomson et al. (2005) used a cut-off value of 40 cmH₂O to determine ‘high’ resting tension in their cohort of pelvic pain participants, however there are no published normative resting pressure values of levator ani, so the threshold for resting tension is not known.

- **Surface EMG:** This tool provides a potentially useful surrogate measure of muscle tension as myoelectrical activity is a component of the definition of muscle tone. However, despite proposed cut-off values reported for determining the threshold of normal versus increased muscle resting tension (Tu et al., 2008b; Voorham-van der Zalm et al., 2008), no widely accepted ‘normal’ resting value sEMG of the PFM exist. Further, this tool has only poor–fair reliability in a non-pain population (Auchincloss and McLean, 2005).

- **Real-time ultrasound:** Transperineal ultrasound has been used by Davis et al. (2011) to assess anorectal angle (ARA) and levator plate angle (LPA) in men with urological chronic pelvic pain syndromes (UCPPS) compared with controls. They found men with UCPPS had more acute ARAs than controls both at rest and during contraction. The two groups did not differ in LPA at rest; however, men with UCPPS had significantly more acute angles during contraction and levator plate excursion. Reliability testing and normative data are required using real-time ultrasound in a PFM pain population.

- **Dynamometry:** Morin et al. (2010) have investigated passive properties of the PFM using a dynamometer, by stretching the vaginal tissues and recording passive resistance and elastic stiffness. Values from a pelvic pain population, reliability testing and normative data are required for further application of this tool, however at present, dynamometry is only available in a research setting.

- **Elastometry:** A further device, an elastometer, is also in early testing phases as a measurement tool of the passive properties of the PFM (Kruger et al., 2011). Reliability in this small cohort was reported (ICC = 0.92; 95% CI 0.89–0.93; ICC = 0.86; 95% CI 0.82–0.89).

**Examination beyond the pelvic floor muscles**

If no positive findings emerge from PFM assessment to confirm that the PFM is responsible for the pain, and the symptoms cannot be clinically reproduced, assessment should expand to structures that may refer pain to the PFM. These include nearby muscles, joints, viscera and the central nervous system. It has been stated that ‘the muscle in which the pain and tenderness is located often serves only as a starting point for finding the source of the pain’ (Mense and Simons, 2001: 84). A detailed explanation of the mechanisms of how pain is referred from other muscles, nearby joints, viscera and the central nervous system is beyond the scope of this chapter but is addressed comprehensively by Mense et al. (2001a). It is common to find obturator internus, psoas, gluteal and piriformis muscle abnormality and pain in patients with PFM pain (Fitzgerald and Kotarinos, 2003; Hetrick et al., 2002; Tu et al., 2008b).

The importance of assessing beyond the immediate pelvic floor structures for dysfunction located in other musculoskeletal structures that may impact on the local system has been highlighted (Fitzgerald and Kotarinos, 2003; Prendergast and Weiss, 2003). Once neurophysiological adaptation to the chronic local pain has occurred, the sensitized spinal cord is vulnerable to, and influenced by, other organs and muscles that converge onto the same nerves (somatovisceral convergence), or by general factors that increase nerve sensitivity. Thus dysfunction in other pelvic muscles (abdominal, gluteal, thigh) may perpetuate PFM pain and dysfunction. If no somatic or visceral structure can be identified as contributing to the patient’s symptom(s), further end-organ investigation should be minimized (Stacy et al., 2012) and consideration of pain as the primary disease is appropriate (Siddall and Cousins, 2004).

**Summary of assessment findings**

Assessment of PFM tension myalgia requires a comprehensive biopsychosocial assessment. Reference data of normal PFM activity against which abnormalities may be compared, is lacking. Useful clinical information of the presence and extent of local tissue dysfunction in a patient presenting with suspected PFM pain or altered tension can be gained from a careful and comprehensive perineal and levator ani evaluation. However, valid and reliable scales for measuring PFM pain, tension or trigger points are scarce. The clinician and researcher should choose the most robust scale available for the parameter under measure.
OUTCOME MEASURES

It is recommended that management of patients with chronic pain should include patient-reported outcomes and clinician-reported outcomes, with the former being the more important as the patient is the primary judge of whether changes are important or meaningful (Turk et al., 2006). Patient-reported outcomes are unmodified patient responses and should include the following domains: assessment of pain, physical functioning, emotional functioning, patient rating of improvement and satisfaction with treatment (Turk et al., 2006).

Using numeric rating scale/visual analogue scale, reductions in chronic pain intensity in individuals of at least 10–20% appears to reflect minimally important changes; reductions of 30% appear to reflect at least moderate clinically important differences; and reductions in chronic pain intensity of 50% appear to reflect substantial improvements (Dworkin et al., 2008). The role played by the costs and side-effects of treatment and the anticipated duration of the change (e.g. a 10% decrease that lasts for several years might be more important than a 10% decrease that lasts for a few months) should be carefully evaluated.

There are a limited number of questionnaires that measure physical and emotional functioning specific to pelvic pain conditions or syndromes, such as the O’Leary–Sant indexes (O’Leary et al., 1997; Lubeck et al., 2001) specific to Interstitial Cystitis/Bladder Pain Syndrome; the National Institutes of Health Chronic Prostatitis Symptom Index (NIH-CPSI) (Litwin et al., 1999); and the Female Sexual Function Index (Rosen et al., 2000). Patient/participant ratings of improvement and satisfaction with treatment may be chosen as the primary outcome measure. These scales may be called the Patient Global Impression of Change scale/Patient Global Impression of Improvement (Yalcin and Bump, 2003; Srikrishna et al., 2010), or Global Response Assessment (Propert et al., 2002). These scales use a 7-point rating scale with the options: very much improved, much improved, minimally improved, no change, minimally worse, much worse, very much worse. Patient’s Overall Rating of Symptoms Index is a 6-point scale, with anchors that address the overall change after treatment as worse, no better (0% improvement), slightly improved (25%), moderately improved (50%), greatly improved (75%), or symptoms gone (100% improvement), and has been used in bladder pain trials (Parsons et al., 1993).

Clinician-reported outcomes include scales or outcomes either observed by a clinician/researcher or requiring interpretation. They also include laboratory, behavioural and device measurements, and include objective and usually quantitative behavioural or physiological measures often performed by devices or raters. As outlined above, very few of these are yet available to measure PFM pain/tension.

There are no current guidelines for the selection of outcome measures appropriate for the assessment and treatment of patients with PFM pain or altered tension. Due to the heterogenous nature of PFM pain syndrome presentations, outcome measure selection may be guided by somatic or visceral perspectives. The clinician and researcher should carefully consider the appropriateness of the selected outcome measure to the patient or population under review, considering reliability, validity and responsiveness to change of the particular tool being used. Outcomes should be selected that reflect both subjective and objective measures. However, as the patient experience of pain is the most important outcome, a pain scale or global rating of change will remain the outcome measure of choice to measure the effect of the intervention on the patient’s condition.

Summary

Development and testing of robust assessment tools and outcome measures will facilitate well-designed, high-quality intervention studies to measure effects of treatment for PFM pain and altered tension. This will also allow mechanistic studies, which aim to describe the effect of the intervention on the target tissue – PFM pain or altered tension – to be accurately interpreted. In the absence of robust measures, an explanation of what effect the therapy has had on the PFM pain/tension is not possible.

TREATMENT

Clinicians currently utilize a wide range of conservative therapies to treat CPP, as summarized in Box 9.1, however there are no clear evidence-based guidelines as to the most appropriate treatment for PFM pain or altered tension. Despite the plethora of options, there is a paucity of evidence from primary research supporting any particular modality. Effective treatment of any chronic pain condition is challenging by the very nature of chronic pain. The complexity of multisystem involvement in patients with CPP and the lack of clear diagnostic markers to assist evaluation increase the difficulty of selecting effective therapies for these patients. In contrast to other areas of pelvic floor dysfunction treated by conservative therapies, CPP suffers from a lack of evidence overall, due to poor quality methodological studies and inconclusive results to date, and even fewer robust studies investigating more clearly defined sub-sets of CPP, such as PFM tension myalgia. Despite this, it is encouraging that research to guide conservative therapies in this area is slowly emerging.
Box 9.1 Conservative therapy treatment options for pelvic floor pain

**Education**
- Neurophysiology of pain
- Structure and function of affected tissues

**Lifestyle modification**

**Dietary advice**
- Guided imagery

**Cognitive behavioural therapy**
- Whole body postural correction
- General fitness advice and exercise prescription
- Pelvic floor muscle exercises:
  - awareness, relaxation, proprioception,
  - re-education, contraction

**Manual therapy**
- Myofascial release, trigger point therapy, local massage, muscle stretching, scar tissue mobilization:
  - to pelvic floor muscles
  - to pelvic, buttock, abdominal and spinal muscles

**Voiding and defecation retraining**
- Postural mechanics
- Pelvic floor muscle awareness and function in appropriate position, simulated action

**Adjunctive therapies**
- Biofeedback:
  - electromyography
  - manometry
  - real-time ultrasound
- Electrical stimulation:
  - pain relief
  - muscle re-education
- Dry needling, trigger point injection therapy (saline), acupuncture, electroacupuncture
- Magnetic field therapy
- Therapeutic ultrasound
- Laser therapy
- Dilator therapy
- Application of heat
- Application of cold

**Pain management strategies**
- Pain education
- Goal-setting
- Multidisciplinary approach

Databases were searched for systematic reviews and randomized controlled trials (RCTs) for evidence of conservative therapy treatment for chronic PFM pain syndrome/PFM tension myalgia over the date range 2003–2013, in women or men, with full publication in English. No results emerged specific to this diagnostic term, hence the search was broadened to CPP, then narrowed to reviews or studies which may have included PFM pain or altered PFM tension in their population, or outcomes which measured PFM pain or tension in a CPP population. Systematic reviews by Loving et al. (2012) and Yunker et al. (2012) were identified. The protocol for a Cochrane review to evaluate non-surgical interventions for the management of CPP has been published (Cheong et al., 2010), however to date the review is not available. Thirteen RCTs relevant to PFM pain or altered tension in women or men were identified (Chiarioni et al., 2006; Haugstad et al., 2006; Heyman et al., 2006; Giubilei et al., 2007; Carrico et al., 2008; Haugstad et al., 2008; Lee et al., 2008; Sikiru et al., 2008; Fitzgerald et al., 2009; Lee and Lee, 2009; Bernardes et al., 2010; Chiarioni et al., 2010; Samhan et al., 2011; Fitzgerald et al., 2012) and are summarized in Table 9.9.

The interventions in these RCTs predominantly focused on treatments directed at local/peripheral tissue dysfunction or sensitization, using manual therapy or adjunctive therapy techniques to pelvic floor tissues (Chiarioni et al., 2006; Heyman et al., 2006; Lee et al., 2008; Sikiru et al., 2008; Lee and Lee, 2009; Fitzgerald et al., 2009; Bernardes et al., 2010; Chiarioni et al., 2010; Samhan et al., 2011; Fitzgerald et al., 2012). Two of the RCTs targeted more general whole body therapies (Haugstad et al., 2006; Giubilei et al., 2007), which may also influence local tissue functioning, and one RCT (Carrico et al., 2008) targeted a more central desensitization approach through guided imagery, although the aim was to relax tension in the PFM in order to reduce bladder pain.

The methodological quality scores of these RCTs varied, and are summarized in Table 9.10. There was considerable heterogeneity across these trials (sex, condition, pain mechanism targeted, description of PFM pain or tension, modality applied, range of outcomes reported, contradictory results, follow-up period), small numbers of participants and not all were powered to show a treatment effect. These limitations prevent firm conclusions regarding effectiveness of any particular modality for any particular condition. What is helpful to reduce PFM pain or increased tension in one condition or syndrome may not be helpful in another, if the pain and tension are due to different causes. Overall the evidence for conservative therapy treatment of CPP or PFM tension myalgia remains sparse, highlighting the need for much more research to be undertaken in this field.

Despite the limitations, cautious conclusions may be drawn from these studies to assist clinicians to make evidence-informed decisions regarding treatment. Most studies reported positive effects of treatment, although this did not reach between-group significance in the primary outcome measure for guided imagery in women with bladder pain syndrome (Carrico et al., 2008) and TENS for men with CPP (Samhan et al., 2011). The longest follow-up of treatment effectiveness was reported for somatocognitive therapy (Haugstad et al., 2008), with the benefits of this therapy on pain relief durable at 12 months, which is clinically relevant. Manual therapy trials appeared to
### Table 9.9 Randomized controlled trials on conservative therapy treatments for pelvic floor muscle pain and/or pelvic floor muscle increased tension

<table>
<thead>
<tr>
<th>Author</th>
<th>Chiarioni et al., 2006</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Design</strong></td>
<td>2-arm RCT</td>
</tr>
<tr>
<td><strong>Study population</strong></td>
<td>109 patients (men and women) with pelvic floor dyssynergia (paradoxical contraction of PFM, as measured by anal canal pressures and sEMG)</td>
</tr>
<tr>
<td><strong>Intervention</strong></td>
<td>TG 1: 5-weekly 30-min BF sessions (effective expulsion efforts, PFM relaxation training with sEMG BF, balloon defecation training); TG 2: polyethylene glycol 14.6–29.2 g/day, plus 5-weekly counselling sessions to prevent constipation</td>
</tr>
<tr>
<td><strong>Adherence/Drop-out</strong></td>
<td>6 months: 0 missing. 12 months: TG 1, missing data 2 patients; TG 2, missing data 4 patients</td>
</tr>
<tr>
<td><strong>Outcome measures</strong></td>
<td>5-point GRA; bowel symptoms reported in patient diary; anal canal pressure, sEMG measures, balloon expulsion</td>
</tr>
</tbody>
</table>
| **Results**     | 6 and 12 months: TG 1 more likely (80%) than TG 2 (22%) to report GRA improvement p <0.001. TG 1: less straining, fewer incomplete bowel movements than TG 2 p <0.01.
24 months: TG 1 only, 81.5% reported ‘major’ satisfaction in treatment effect
Physiologic outcomes: TG 1 reduced paradoxical contraction (16.7% at 6, 12 and 24 months, compared with TG 2, 96.4% at 6 and 12 months, p <0.001) and improved ability to evacuate balloon (18.5% at 6, 12 months, 16.7% at 24 months, compared with TG 2, 96.4% at 6 and 12 months, p <0.01) |

<table>
<thead>
<tr>
<th>Author</th>
<th>Haugstad et al., 2006</th>
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</thead>
<tbody>
<tr>
<td><strong>Follow-up study</strong></td>
<td>Haugstad et al., 2008</td>
</tr>
<tr>
<td><strong>Design</strong></td>
<td>RCT</td>
</tr>
<tr>
<td><strong>Study population</strong></td>
<td>40 women. CPP (deep pelvic pain) 1–10 years’ duration. PFM palpated as part of vaginal examination to exclude obvious pathology, findings of pain/tension not reported</td>
</tr>
</tbody>
</table>
| **Intervention** | Standard gynaecological treatment (hormonal, analgesic treatment as required, dietary and bowel advice, sexological advice)
TG: Mensendieck somatocognitive therapy (a cognitive-based approach to increase awareness of body movements, tension, relaxation, posture, gait, respiration) for 3 months (10 treatment sessions)
CG: nil additional treatment |
| **Adherence/Drop-out** | 1 drop-out from each group at 3 months; 1 further drop-out at 1 year |
| **Outcome measures** | Therapist rating of Mensendieck score (in five domains) of motor function evaluated by video recording |
| **Results**     | Significant improvement in VAS, Mensendieck score and GHQ-30 in TG (VAS: 48% reduction in score, p <0.000)
Non-significant changes in CG (VAS: 8% reduction in score, p=0.07) |

<table>
<thead>
<tr>
<th>Author</th>
<th>Heyman et al., 2006</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Design</strong></td>
<td>RCT</td>
</tr>
<tr>
<td><strong>Study population</strong></td>
<td>50 women. CPP of &gt;6 months; inclusion criterion of pain on firm palpation of PFM (rated as present)</td>
</tr>
</tbody>
</table>
| **Intervention** | TG: ‘forceful’ distension of PFM via per rectal digital palpation. Pressure applied for 60s; procedure repeated after 2–4 weeks
CG: counselling only |
| **Adherence/Drop-out** | 6 drop-outs |
| **Outcome measures** | Participant self-rating of pain intensity on a VAS, scale=100
TG: decrease in pain intensity of 35 (±31)/100
CG: no change on pain scale
Difference: p=0.001, OR 18.37 (95% CI: 3.39–99.64) |

(Continued)
### Table 9.9 Randomized controlled trials on conservative therapy treatments for pelvic floor muscle pain and/or pelvic floor muscle increased tension—cont’d

<table>
<thead>
<tr>
<th>Author</th>
<th>Giubilei et al., 2007</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Design</strong></td>
<td>Double-blinded RCT</td>
</tr>
<tr>
<td><strong>Study population</strong></td>
<td>103 sedentary men with CP/CPPS, &lt;50 years of age, pain in pelvic region for ≥3 months, score of ≥15 points on NIH-CPSI scale plus ≥6 points on pain subscale (subjective pain report) (PFM examination findings not reported)</td>
</tr>
<tr>
<td><strong>Intervention</strong></td>
<td>18-week exercise programme, three sessions per week, self-directed, with monthly telephone calls to verify adherence</td>
</tr>
<tr>
<td><strong>TG (n=52):</strong></td>
<td>active exercise group (40 min fast-paced walking, specific postural muscle and isometric strengthening exercises)</td>
</tr>
<tr>
<td><strong>CG (n=51):</strong></td>
<td>flexibility group (stretching exercises)</td>
</tr>
<tr>
<td><strong>Adherence/Drop-out</strong></td>
<td>1st analysis at 6 weeks: TG=41 (85%), CG=44 (90%)</td>
</tr>
<tr>
<td><strong>Outcome measures</strong></td>
<td>NIH-CPSI; anxiety scale; participant self-rating of pain intensity (0–10)</td>
</tr>
<tr>
<td><strong>Results</strong></td>
<td>6 weeks: significant decreases in NIH-CPSI (p=0.02), pain (p=0.009), and QoL impact (p=0.006), subscales and pain VAS scores (p=0.03), in favour of TG</td>
</tr>
<tr>
<td></td>
<td>18 weeks: significant decreases in NIH-CPSI (p=0.006), pain (p=0.0009), and QoL impact (p=0.02), subscales and pain VAS scores (p=0.003), in favour of TG</td>
</tr>
<tr>
<td><strong>Adverse events</strong></td>
<td>(predominantly treatment-related pain): TG – initial aggravation of pain, tended to be self-limiting within 2–4 weeks</td>
</tr>
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<table>
<thead>
<tr>
<th>Author</th>
<th>Carrico et al., 2008</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Design</strong></td>
<td>Pilot RCT</td>
</tr>
<tr>
<td><strong>Study population</strong></td>
<td>30 women with interstitial cystitis. Included in study if patient reported pain on levator ani palpation (0–10 VAS) by researcher (values not reported)</td>
</tr>
<tr>
<td><strong>Intervention</strong></td>
<td>TG: listened to a 25-min guided imagery CD (focused on healing the bladder, relaxing the PFM and quieting the nerves), twice per day for 8 weeks</td>
</tr>
<tr>
<td><strong>CG:</strong></td>
<td>rested in lying or sitting position for 25 min twice per day for 8 weeks</td>
</tr>
<tr>
<td><strong>Adherence/Drop-out</strong></td>
<td>TG: 4 drop-outs; CG: 1 drop-out</td>
</tr>
<tr>
<td><strong>Outcome measures</strong></td>
<td>7-point GRA; 2-day voiding diary; IC-SIPI; VAS for pain; Interstitial Cystitis Self-Efficacy Scale</td>
</tr>
<tr>
<td><strong>Results</strong></td>
<td>Improvements in all measures in favour of TG, but none statistically significant between groups</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Author</th>
<th>Lee et al., 2008</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Design</strong></td>
<td>2-arm RCT</td>
</tr>
<tr>
<td><strong>Study population</strong></td>
<td>89 men diagnosed with CP/CPPS, symptoms for &gt;3 months, score of ≥15 points on NIH-CPSI scale</td>
</tr>
<tr>
<td><strong>Intervention</strong></td>
<td>TG 1: acupuncture (20 sessions, 2x 30 min per week for 10 weeks)</td>
</tr>
<tr>
<td><strong>TG 2: sham acupuncture (same protocol as TG 1, with needles placed at shallower depth and 15 mm lateral to acupoints used in TG 1)</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Adherence/Drop-out</strong></td>
<td>TG 1: 5 drop-outs; TG 2: 2 drop-outs</td>
</tr>
<tr>
<td><strong>Outcome measures</strong></td>
<td>NIH-CPSI total score after 20 sessions; NIH-CPSI at follow-up time points; NIH-CPSI sub-scales; GRA</td>
</tr>
<tr>
<td><strong>Results</strong></td>
<td>At cessation of treatment: significant reduction in NIH-CPSI score in 73% of TG 1 compared to 47% of TG 2 (p=0.02)</td>
</tr>
<tr>
<td></td>
<td>24 weeks after treatment: significant reduction in NIH-CPSI score in 32% of TG 1 compared to 13% of TG 2 (p=0.04)</td>
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</tbody>
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<table>
<thead>
<tr>
<th>Author</th>
<th>Sikiru et al., 2008</th>
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<tbody>
<tr>
<td><strong>Design</strong></td>
<td>3-arm RCT</td>
</tr>
<tr>
<td><strong>Study population</strong></td>
<td>24 men, aged 24–50 years, diagnosed with CP/CPPS</td>
</tr>
<tr>
<td><strong>Intervention</strong></td>
<td>TG 1: antibiotics + TENS (20 min daily, 5x per week, 4 consecutive weeks; TENS parameters: 100 Hz, 100 μs, 25 mA, external electrodes)</td>
</tr>
<tr>
<td><strong>TG 2: antibiotics + analgesics</strong></td>
<td></td>
</tr>
<tr>
<td><strong>TG 3: antibiotics + placebo tablets.</strong></td>
<td></td>
</tr>
<tr>
<td>Table 9.9</td>
<td>Randomized controlled trials on conservative therapy treatments for pelvic floor muscle pain and/or pelvic floor muscle increased tension—cont’d</td>
</tr>
<tr>
<td>Adherence/Drop-out</td>
<td>No drop-outs</td>
</tr>
<tr>
<td>Outcome measures</td>
<td>NIH-CPSI pain scale (location, frequency, severity of pain)</td>
</tr>
<tr>
<td>Results</td>
<td>TG 1: significant reduction in pain score compared with TG 2 and TG 3, p=0.000</td>
</tr>
<tr>
<td>Author</td>
<td>Fitzgerald et al., 2009</td>
</tr>
<tr>
<td>Design</td>
<td>RCT</td>
</tr>
<tr>
<td>Study population</td>
<td>23 men and 24 women with urological CPP syndrome: IC/PBS or CP/CPPS ≤3 years duration, PFM tenderness (not rated) on digital (vaginal or rectal) palpation but excluded if no tenderness or intolerant to palpation</td>
</tr>
<tr>
<td>Intervention</td>
<td>10 sessions, 1 hour each over 12 weeks with a physical therapist</td>
</tr>
<tr>
<td>TG: myofascial therapy treatments to internal and external pelvic myofascial structures</td>
<td></td>
</tr>
<tr>
<td>CG: global therapeutic massage therapy</td>
<td></td>
</tr>
<tr>
<td>Adherence/Drop-out</td>
<td>6 drop-outs (TG 4; CG 2)</td>
</tr>
<tr>
<td>Outcome measures</td>
<td>7-point GRA; NRS for pain, urgency and frequency; 24 hour voiding diary; IC-SIPI; NIH-CPSI (males); SF-12; Sexual Function Index (FSFI for females; Sexual Health Inventory for males)</td>
</tr>
<tr>
<td>Results</td>
<td>At completion of intervention phase: Significant improvement in GRA between groups: 57% in TG, 21% in CG, p=0.03</td>
</tr>
<tr>
<td>TG: improved pain and bladder symptom scores and PFM tenderness scores compared with CG, p &lt;0.05</td>
<td></td>
</tr>
<tr>
<td>Adverse events (predominantly treatment-related pain) – TG: 52%; CG: 21%</td>
<td></td>
</tr>
<tr>
<td>Author</td>
<td>Lee &amp; Lee 2009</td>
</tr>
<tr>
<td>Design</td>
<td>3-arm, parallel group RCT</td>
</tr>
<tr>
<td>Study population</td>
<td>36 men aged 18–50 years, with CP/CPPS &gt;3 months’ duration, score of &gt;15 on NIH-CPSI scale</td>
</tr>
<tr>
<td>Intervention</td>
<td>Advice and exercise, plus:</td>
</tr>
<tr>
<td>TG 1: electroacupuncture (2x per week for 20 min for 6 weeks; 6 acupuncture points over sacral foramina S2 and S3, and piriformis muscle, stimulated at 4 Hz and 5–10 mA)</td>
<td></td>
</tr>
<tr>
<td>TG 2: sham electroacupuncture (same as TG 1 but at sites 15 mm lateral to TG 1 sites and no current applied, sound only)</td>
<td></td>
</tr>
<tr>
<td>TG 3: advice and exercise alone</td>
<td></td>
</tr>
<tr>
<td>Adherence/Drop-out</td>
<td>4 drop-outs (TG 1: 1; TG 2: 2; TG 3: 1)</td>
</tr>
<tr>
<td>Outcome measures</td>
<td>3 weeks and 6 weeks: NIH-CPSI total and subscale scores: changes in pain, voiding and QoL</td>
</tr>
<tr>
<td>Results</td>
<td>3 weeks: significant decrease in NIH-CPSI pain subscale in TG 1 compared to TG 2 and TG 3, p &lt;0.05; 6 weeks: significant decrease in NIH-CPSI total score in TG 1 compared to TG 2 and TG 3, p &lt;0.001; significant decrease in NIH-CPSI pain subscale in TG 1 compared to TG 2, p &lt;0.001, and TG 3, p &lt;0.01; no other significant differences between groups</td>
</tr>
<tr>
<td>Author</td>
<td>Bernardes et al., 2010</td>
</tr>
<tr>
<td>Design</td>
<td>Double-blind, crossover RCT</td>
</tr>
<tr>
<td>Study population</td>
<td>26 women with CPP ≥6 months; VAS &gt;3; absence of well-defined pelvic pathologies</td>
</tr>
<tr>
<td>Intervention</td>
<td>10x 30-min, twice-weekly sessions of intra-vaginal electrical stimulation (IVES) (8 Hz, intensity variable), followed by a crossover period of a further 10 sessions of IVES:</td>
</tr>
<tr>
<td>Phase 1 TG (n=15): active IVES</td>
<td></td>
</tr>
<tr>
<td>Phase 1 CG (n=11): sham IVES</td>
<td></td>
</tr>
<tr>
<td>Adherence/Drop-out</td>
<td>At completion of crossover phase: CG – 1 drop-out (4%)</td>
</tr>
<tr>
<td>Outcome measures</td>
<td>Pain intensity on VAS 0–10, divided into three categories (0 = no pain, 1–3 = slight, 4–7 = moderate, 8–10 = intense); dyspareunia (present, absent)</td>
</tr>
<tr>
<td>Results</td>
<td>At completion of crossover phase: active IVES more likely to have reduced pain scores (≤3) compared to sham IVES (p=0.036); no change in reduction of dyspareunia between groups (p=0.317)</td>
</tr>
</tbody>
</table>

(Continued)
show benefit from targeted myofascial treatment in men and women with pain on PFM palpation (Heyman et al., 2006; Fitzgerald et al., 2009; Fitzgerald et al., 2012). Electroacupuncture appeared to be more effective than sham electroacupuncture, and acupuncture more effective than sham acupuncture for men with CPP in the trials by Lee et al. (Lee et al., 2008; Lee and Lee, 2009). Intra-vaginal electrical stimulation (IVES) appeared to be more effective than sham IVES for pain relief in women with CPP (Bernardes et al., 2010), however the result was affected
by the crossover design. Biofeedback using intra-anal sEMG electrode was effective for global improvement in symptoms compared to laxatives in men and women with paradoxical PFM contraction (Chiarioni et al., 2006), and more effective than electrical stimulation or PFM massage for men and women with pain on palpation of levator ani (Chiarioni et al., 2010). Both these trials also showed reduction in anal canal pressures in responders, therefore provided a physiological explanation of the mechanism of effect of treatment. No study reported on PFM pain levels (measured from digital palpation), before or after treatment.

The risk of adverse events was low/absent in most studies, with the exception of the manual therapy trials, where treatment-related pain was not uncommon, reported by 30% (Fitzgerald et al., 2009) and 14% (Fitzgerald et al., 2012) of participants. Drop-outs were high in this study (>50%), but not reported as attributable to treatment-related

Table 9.10 PEDro quality score of RCTs in systematic review of conservative therapy treatments for pelvic floor muscle pain and/or pelvic floor muscle increased tension

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<td>Fitzgerald et al., 2012</td>
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</table>

+, criterion is clearly satisfied; –, criterion is not satisfied; ?, not clear if the criterion was satisfied. Total score is determined by counting the number of criteria that are satisfied, except that ‘eligibility criteria specified’ score is not used to generate the total score. Total scores are out of 10.
pain. Heyman et al. (2006) also reported ‘mild temporary increased local pain’, resulting in two drop-outs after the first session. Treatment-related discomfort also occurred in 74% of participants in the active arm of the physical activity study (Giubilei et al., 2011), however these participants were previously sedentary and their discomfort was short-lived.

As recommended for assessment, treatment should also be set within a biopsychosocial framework. If changes associated with central sensitization are dominant, somatic treatment may need to be deferred. Treatment may be commenced with pain education, relaxation therapy and guided imagery in order to downregulate the central nervous system’s responsiveness and threshold to stimuli. This approach has been recommended in the chronic musculoskeletal pain literature (Nijs et al., 2011), and interpreted for CPP by Hilton and Vandyken (2011), although a methodologically robust clinical trial is required to validate and demonstrate clinical effectiveness. Building rapport with the patient, or forming a ‘working alliance’ appears to be associated with improved patient outcomes (Hall et al., 2010; Ferreira et al., 2013) and has been incorporated in the somatocognitive therapy approach (Haugstad et al., 2006).

**CONCLUSION**

To guide clinicians in providing optimal care for patients with CPP, there is no better recommendation than the definition of evidence-based practice as described by Sackett and Haynes (2002). The three pillars can be applied to CPP in the following way:

- **Best research evidence:** This review agrees with the cautious recommendations issued by recent systematic reviews (Loving et al., 2012; Yunker et al., 2012). In the absence of clear evidence from CPP research to guide conservative treatment for chronic PFM syndrome at present, the clinician relies heavily on input from the other two pillars.
- **Clinical expertise:** This is gained from cumulative experience, education and clinical skills, to provide an understanding of the individual patient’s biological, psychological and social state and circumstance. Chronic pelvic pain is complex and clinicians need to develop advanced clinical reasoning skills to identify which presentations may respond to which conservative therapy.
- **Patient values and preferences:** As the patient brings his or her own personal experience and unique concerns, expectations and values to the encounter, the clinician needs to provide scientifically based and clinically sound options to enable the patient to choose his/her preferred treatment. From there, the clinician is advised to work collaboratively with the patient towards his/her goals.

Further research of chronic PFM syndrome is urgently required. Intervention studies should clearly state the condition under investigation, use standardized terminology, describe how PFM pain and tension are evaluated, and measure response to therapy using robust tools. Mechanistic studies will add to our understanding of the role of conservative therapies in this field.

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Evidence-based physical therapy for pelvic floor dysfunctions
Evidence-Based Physical Therapy for the Pelvic Floor


a device to measure genital pressure-pain threshold. Physiol. Meas. 28, 1543–1550.


Thiele, G.H., 1937. Coccygodynia and pain in the superior gluteal region and down the back of the thigh: causation by tonic spasm of the levator ani, coccygeus and piriformis muscles and relief by massage of these muscles. JAMA 109, 1271–1275.


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DYSFUNCTION OF BLADDER CONTROL

Lower urinary tract dysfunction can be classified as either congenital or acquired. Acquired dysfunction is often secondary to an underlying abnormality or the result of learned behaviours, such as during the toilet training process or in response to dysuria or an unacceptable voiding environment (Ellsworth et al., 1995; Greenfield & Wan, 2000).

CLASSIFICATION: URINARY INCONTINENCE DURING THE DAY

Urinary incontinence is most often associated with underlying detrusor overactivity and accompanied by the symptoms of an overactive bladder (OAB), such as frequency of micturition, urgency to find a toilet, posturing to prevent leakage during urgency, small volume voids, nocturnal enuresis or nocturia (Chiozza, 2002). On bladder ultrasound the detrusor wall may be thickened; a cross-section of over 3–4 mm at 50% of the expected bladder capacity is suspicious of detrusor overactivity (Nijman et al., 2005). On uroflowmetry a high flow rate early in the void and an overall shortened flow is often seen, and referred to as the ‘tower’ pattern. If urodynamic investigation is carried out a small bladder capacity can be confirmed and detrusor overactivity noted during filling (Van Gool & de Jonge, 1989), towards end of fill only, or just preceding the voiding contraction.

Recently, incontinence in a subgroup of children has been attributed to volitional voiding postponement and found to be associated with a significant number of behavioural symptoms (Lettgen et al., 2002). Post-void leakage in the presence of a normal foreskin or adequate labial separation is likely to indicate dysfunctional voiding mechanics. In some children laughter triggers partial to complete bladder emptying and although various hypotheses have been proposed to explain the symptom, no definitive aetiology is yet accepted (Nijman et al., 2005). Alteration in the frequency of micturition is another common symptom of bladder dysfunction. Although increased frequency is often associated with an underlying OAB, it is also a hallmark of sensitivity of the urothelium. Current bacteriuria, post-infection inflammation, chronic inflammatory changes, oestrogen and prostaglandin swings, elevated caffeine and acidic urine can all precipitate increased voiding frequency (Martini & Guignard, 2001). A less common presentation relates to the sudden onset of extraordinary urinary frequency, with voiding intervals as short as 15 minutes. This self-limiting condition is not associated with incontinence, nocturnal enuresis or nocturia, but may be linked to alterations in renal solute handling (Parekh et al., 2000).
Dysfunctional voiding refers to an inability to fully relax the bladder neck, urinary sphincter or pelvic floor during voiding. Alternatively it may be associated with dys-synergia of the external urinary sphincter, engendering an inappropriate response to a detrusor contraction. Once urethral resistance is encountered, the detrusor may:

1. continue to contract and effect emptying;
2. reduce contractile activity and prolong bladder emptying; or
3. be completely inhibited by urethral or pelvic floor muscle (PFM) activity, with bladder emptying achieved by abdominal straining.

A child with either of the last two presentations is said to have detrusor underactivity (Neveus et al., 2005).

Altered patterns of voiding are independent of underlying neurological impairment, and may be learned during the toilet training years, adopted following episodes of dysuria or constipation, or occur secondary to sexual abuse. The child’s environment, in particular toilet conditions and privacy issues, can trigger or exacerbate voiding symptoms. Micturition is often achieved by abdominal straining. Alternatively it may be associated with dys-synergia of the external urinary sphincter, engendering an inappropriate response to a detrusor contraction. Once urethral resistance is encountered, the detrusor may:

- the PF EMG trace is silent until the void is initiated and then becomes active;
- the PF EMG is intermittently active during the void; or
- the abdomen is used to generate voiding pressure.

In conjunction with a significant volume of urine remaining in the bladder post void, such a trace would indicate voiding dysfunction. A video-urodynamic study will further elucidate whether there is specific dysfunction at the bladder neck (Grafstein et al., 2005).

### Physical Therapy Intervention for Children with Urinary Incontinence or Dysfunctional Voiding

Children with bladder dysfunction require a multidisciplinary approach for both investigation and intervention. Box 10.1 outlines the components of a physical therapy programme for children with bladder dysfunction during the day. Treatment efficacy can be evaluated by reduction in number of wet episodes, improvement in bladder emptying and resolution of associated symptoms.

**Box 10.1 Components of a physical therapy programme for children with bladder dysfunction during the day**

- Educate normal bladder behaviour and specific changes underlying the child’s symptoms
- Implement voiding routine so that child passes urine at regular intervals
- Teach pelvic floor muscle (PFM) awareness (+ mirror, surface perineal EMG/anal probe EMG, transabdominal or perineal ultrasound) and coordination to achieve PFM recruitment and relaxation with minimal accessory muscle activity
- Train optimal voiding mechanics and posture (+ biofeedback during voiding)
- Bowel management and optimal defecation dynamics if indicated
- Normalize PFM capabilities if necessary
- Adjunctive neuromodulation for overactive bladder symptoms
- Consider clean intermittent catheterization if large post-void residual volumes of urine persist (Pohl et al., 2002)
A systematic review of randomized controlled trials (RCTs) of non-pharmacological intervention in children with urinary incontinence of any non-neurological/structural aetiology identified two studies (Sureshkumar et al., 2003), one reporting the efficacy of daytime alarms (Halliday et al., 1987) and the other included an arm that used biofeedback therapy for children with proven urgency syndrome (Van Gool et al., 1999).

From the details in Table 10.1 it appears that:

- there was no difference between the proportions of children with persistent wetting in either alarm group;
- there was no decrease in the frequency of wetting episodes in children receiving clinic-based biofeedback; and
- anal electrical stimulation (ES) may be effective in the treatment of daytime incontinence and OAB in girls.

A review of non-pharmacological intervention for dysfunctional voiding revealed two randomized uncontrolled trials of biofeedback training (van Gool et al., 1999; Klijn et al., 2003). Other therapies described in cohort studies included PFM awareness training, ES and electromagnetic stimulation and clean intermittent catheterization. From the trial summaries in Table 10.2 it appears that:

- home-based uroflow training for 8 weeks significantly increased daytime continence at 6-month follow-up;
- clinic-based biofeedback did not improve daytime wetting when compared to standard therapy alone (Van Gool et al., 1999).

Quality aspects of the RCT or non-randomized controlled trials are reported in Table 10.3. Clearly further controlled studies of the various interventions for both incontinence and voiding dysfunction in children are needed. The techniques reported favourably in cohort studies may be effective, but to date have been incompletely evaluated.

### Nocturnal Enuresis

Nocturnal enuresis (NE) is defined as emptying of the bladder during sleep (Neveus et al., 2005). Enuresis in children without any other lower urinary tract symptoms (LUTS) or history of bladder dysfunction is subclassified as monosymptomatic. Where the symptoms of increased or decreased voiding frequency, incontinence, urgency,
Table 10.2 Trial details for intervention in children with dysfunctional voiding

<table>
<thead>
<tr>
<th>Author</th>
<th>Van Gool &amp; de Jonge, 1989</th>
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<tbody>
<tr>
<td>Design</td>
<td>(1) Biofeedback and standard therapy vs (2) standard therapy</td>
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<tr>
<td>n</td>
<td>104</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>U/D proven dysfunctional voiding</td>
</tr>
<tr>
<td>Protocol</td>
<td>Ongoing with 6- and 9-month evaluation</td>
</tr>
<tr>
<td>Drop-outs/adherence</td>
<td>Not reported</td>
</tr>
<tr>
<td>Results</td>
<td>Improved at 6 months: (1) 20/34; (2) 18/25; RR 1.47 (0.67–1.79)</td>
</tr>
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<td>9 months: (1) 25/45; (2) 25/42; RR 1.10 (0.67–1.79)</td>
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Author | Klijn et al., 2003 |
Design | (1) Standard therapy vs (2) additional personalized home video vs (3) standard therapy, home video and home uroflowmeter |
| n | 143 |
| Diagnosis | U/D proven voiding dysfunction of non-neurogenic origin |
| Protocol | 8 weeks, 4-month outpatient follow-up |
| Drop-outs/adherence | Not reported |
| Results | Daytime continence: (1) 46%; (2) 54%; (3) 61% |
|         | PVR <10%: (1) 60%; (2) 77%; (3) 73% NS |

NS, not significant; PVR, post-void residual volume of urine; U/D, urodynamically.

Table 10.3 PEDro quality score of trials of non-pharmacological treatment of paediatric bladder dysfunction

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<td>−</td>
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+, criterion is clearly satisfied; −, criterion is not satisfied; ?, not clear if the criterion was satisfied. Total score is determined by counting the number of criteria that are satisfied, except that “eligibility criteria specified” score is not used to generate the total score. Total scores are out of 10.
hesitancy, straining, weak or intermittent urine flow, incomplete emptying, post-void dribble or dysuria coexist with NE, the condition is defined as non-monosymptomatic (Neveus et al., 2005). A child with primary nocturnal enuresis (PNE) has never been dry for at least 6 months, whereas secondary enuresis implies initial reliable night dryness that has been lost.

As can be seen from Table 10.4, prevalence of NE differs by gender in children under 12 years of age, but shows no gender bias in older adolescents and adults. From the age of 16 years onward, prevalence remains constant at around 2.3%, but most sufferers wet more than three nights per week (Yeung et al., 2004a). Underlying urinary tract pathology (OAB, functional bladder outlet obstruction, congenital obstructive lesions) is associated with up to 93% of cases of enuresis in adulthood (Yeung et al., 2004b).

Detrusor overactivity is often present in non-monosymptomatic NE; in fact enuretic children who do not respond to first-line therapy have been shown to have reduced nocturnal bladder capacity, one of the hallmarks of overactive bladder (Yeung et al., 2004a). Overactivity during the day is associated with small voided volumes and a reduced functional bladder capacity (Kruise et al., 1999); however, in up to one-third of all enuretic children there may be isolated nocturnal detrusor overactivity (Watanabe, 1995; Watanabe et al., 1997). This has been reported in 44% of patients whose NE failed to respond to standard treatment (Yeung et al., 1999, 2002).

NE is related to an inability to rouse from sleep to void. A correlation has been reported between low functional bladder capacity and a high sleep arousal threshold so that affected children show less frequent arousal during the night (Yeung et al., 2005).

Renal urine production and its circadian rhythm contribute to nocturnal enuresis. Diuresis during sleep should be approximately 50% of daytime levels (Rittig et al., 1995) and be regulated by free water excretion (arginine vasopressin, AVP) or solute excretion (angiotensin II and aldosterone) (Rittig et al., 1999). Scandinavian studies have demonstrated that two-thirds of patients with monosymptomatic nocturnal enuresis produce large amounts of nocturnal urine, exceeding bladder capacity (Norgaard et al., 1985; Rittig et al., 1989). Polyuria in children with NE is defined as >130% of expected bladder capacity (EBC) for age when the formula (age +2)×30 is applied (Koff, 1983). It is not known whether these patients have impaired renal sensitivity to vasopressin or require supranormal levels to achieve a circadian rhythm of urine production. Children with nocturnal enuresis and nocturnal polyuria may also have sodium retention that generates hypovolaemia and inhibits vasopressin production (Kamperis et al., 1983; Vande Walle et al., 2004). In addition, children with NE and nocturnal polyuria are likely to have reduced functional bladder capacity for age (Yeung, Diao et al., 2004b).

In summary, the interplay of pathological changes in children with nocturnal enuresis remains elusive. There is clearly a mismatch between nocturnal urine production volume, bladder functional capacity and a disturbance of arousal mechanisms. Not all disturbances are present in each child and the relative vulnerability to each remains largely undetermined. The only independent variables conclusively associated with nocturnal enuresis to date are non-pathophysiological and include gender (males more at risk) (Cher et al., 2002), a positive family history (Fergusson et al., 1986) and co-existing behavioural problems.

Children with NE can be classified into one of three groups, facilitating a treatment approach that targets underlying pathology.

1. Polyuria will be proven when there is a monosymptomatic presentation and overnight urine production >130% of EBC. It is likely that no bladder wall changes will be demonstrated on ultrasound and that normal bladder emptying will be observed. Children may have either age-expected or reduced bladder capacity.

2. Underlying bladder dysfunction will be suggested by a small bladder capacity revealed on ultrasound, and confirmed functionally by the frequency–volume chart (FVC). The bladder will empty appropriately and with a normal flow, but commonly displays hypertrophy. Specific urodynamic findings in this group may include moderate or severe OAB, sphincter and pelvic floor discoordination during voiding and a small cystometric capacity. There is generally no evidence of polyuria.

3. In the third diagnostic group, children show normal day FVC, acceptable voiding dynamics and appropriate ultrasound parameters. However, there is nocturnal onset of covert detrusor overactivity and an associated reduction in nocturnal bladder capacity. This category of patients has persistent symptoms not responding to therapy (Yeung et al., 2002).

Table 10.4 Prevalence of enuresis by gender at different ages

<table>
<thead>
<tr>
<th></th>
<th>5 years (%)</th>
<th>7 years (%)</th>
<th>9 years (%)</th>
<th>Mid to late teens (%)</th>
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<tbody>
<tr>
<td>Boys</td>
<td>13–19</td>
<td>15–22</td>
<td>9–13</td>
<td>1–2</td>
</tr>
<tr>
<td>Girls</td>
<td>9–16</td>
<td>7–15</td>
<td>5–10</td>
<td>1–2</td>
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In the third diagnostic group, children show normal day FVC, acceptable voiding dynamics and appropriate ultrasound parameters. However, there is nocturnal onset of covert detrusor overactivity and an associated reduction in nocturnal bladder capacity. This category of patients has persistent symptoms not responding to therapy (Yeung et al., 2002).
Given that nocturnal enuresis is either monosymptomatic or associated with underlying bladder dysfunction, optimal care of the child involves multidisciplinary evaluation and multimodal management. Physical therapy strategies offer adjunctive intervention and are best used as part of a combined and tailored therapeutic approach, particularly for children with non-monosymptomatic presentation. Whilst initial treatment should be targeted at the lower urinary tract symptoms in non-monosymptomatic presentations, after resolution of daytime symptoms residual NE responds in a similar way to monosymptomatic enuresis (Rittig et al., 2013).

Box 10.2 outlines the treatment strategies available to the therapist for children with non-monosymptomatic NE (i.e. proven filling or emptying bladder dysfunction). Non-monosymptomatic NE is addressed once the hallmarks of daytime urinary dysfunction have been managed. Accumulated stool in the rectum, faecal soiling, slow transit through the colon or dyssynergic defecation dynamics will also have been evaluated and treated (Franco et al., 2013). Physical therapists (PTs) may augment management of an overactive bladder with neuromodulation, the rationale being that inhibiting detrusor overactivity facilitates age-expected storage of urine thus reducing the mismatch of production to storage during sleep. Placebo-controlled trials of surface neuromodulation delivered over the sacrum or posterior tibial nerve have shown an improvement in urodynamic parameters and, when therapy is given in excess of 3 months, control of day symptoms and enuretic episodes (Bower et al., 2001; Raheem et al., 2013). The effect of neuromodulation in children with bladder dysfunction is additive to other modalities and plasticity changes induced over long periods (Bower & Yeung, 2004). There is a strong evidence base to support pelvic floor muscle relaxation training for children with voiding dysfunction, however strength and endurance training of the pelvic floor muscles does not improve nocturnal enuresis (Van Kampen et al., 2009).

The bedwetting alarm is the most effective intervention for monosymptomatic NE (Glazener et al., 2003, 2004). Children are 13 times more likely to become dry with an alarm than without treatment, with a 43% last- ing cure rate (Houts et al., 1994; Nijman et al., 2005). Optimal results appear to be associated with high levels of motivation of the child and family and a high initial frequency of wet nights (Nijman et al., 2005), whereas failure was associated with a low functional bladder capacity and an inability to be woken by the alarm (Butler & Robinson, 2002).

Cochrane reviews evaluating different interventions for NE conclude that:

- Simple changes to behaviour by the child or family (e.g. charting, rewards, lifting at night, waking, bladder training) are better than doing nothing.
- Simple behavioural strategies are less effective than enuresis alarm or pharmacotherapy.
- There is weak evidence from low level studies re any benefit from hypnosis, psychotherapy, acupuncture or medicinal herbs.
- Combination therapy with anticholinergic medication and enuresis alarm/antidiuresis reduces relapse rates.
- Compared to no treatment, about two-thirds of children became dry during alarm use.
- While antidiuresis may have a more immediate effect, alarms appeared to be as effective by the end of a course of treatment.

(Huang et al., 2011; Deshpande et al., 2012; Caldwell et al., 2013)

**Box 10.2 Components of a physical therapy programme for children with non-monosymptomatic nocturnal enuresis**

- Institute regular voiding schedule and appropriate hydration
- Develop pelvic floor muscle (PFM) proprioception, awareness and timing (+ biofeedback)
- Train specific PFM muscle relaxation during voiding
- Train unopposed bladder emptying and normalize voiding mechanics
- Intervention to reduce urgency and strategies to prevent urge leak (+ neuromodulation and antimuscarinic medication)
- Treat underlying bowel dysfunction

**Bowel Dysfunction in Children**

Children with bladder dysfunction often present with bowel symptoms such as constipation or faecal soiling. Slow gut transit and abnormal defecation dynamics are also important symptoms. However, few families present complaining of these issues and they are usually unmasked only during interview and assessment. Bladder bowel dysfunction (BBD) is the term used for co-existing symptoms in both systems. The frequently used terms in childhood bowel dysfunction are presented in Box 10.3.

Functional chronic constipation occurs in around 64% of children with constipation and is not associated with organic/neurological causes. Diagnosis requires
Evidence for pelvic floor physical therapy in children

**Box 10.3 Definitions of childhood bowel dysfunction**

- Faecal impaction: faecal mass in rectum/abdomen that cannot be passed on demand
- Organic constipation: congenital/anatomical structural defects that obstruct the colon, metabolic and endocrine disorders, connective tissue disease, neurological causes, slow colonic transit, infections and degenerative conditions
- Functional constipation: no underlying organic cause; subclassified into functional constipation, functional faecal retention and constipation-predominant irritable bowel syndrome
- Faecal incontinence: passage of stools in an inappropriate place for at least 8 weeks; sub-classified into organic or functional
- Functional faecal incontinence is further classified depending on the presence or absence of associated constipation
- Pelvic floor dyssynergia: lack of pelvic floor relaxation during attempts to defecate – paediatric PTs have much to offer the child with this dysfunction

Rasquin-Weber et al., 1999; Benninga et al., 2005

**UNDERLYING PATHOPHYSIOLOGY OF BOWEL DYSFUNCTION**

In the newborn, meconium is passed within the first 24 hours, with lower birth weight children having delayed passage of stool (Weaver & Lucas, 1993). Bowel actions occur up to six times daily for the first few weeks of life, but decline in frequency and increase in size and weight until by 4 years a child will defecate once daily (Weaver, 1988). Defecation frequency is highly variable, with a 4-year-old being as likely as an adult to pass stool three times daily to three times a week (Hatch, 1988).

Stool arriving in the rectum distends rectal and pelvic floor stretch receptors leading to relaxation of the internal anal sphincter and movement of stool into the anal canal. Contraction of the external anal sphincter follows perception of the call to stool. At a convenient time and place the external anal sphincter and pelvic floor are voluntarily relaxed, intra-abdominal pressure is generated and defecation follows. Many children achieve bowel control around 18 months, but the age at which complete control is evidenced varies widely.

Up to 70% of constipated children have blunted or absent rectal sensitivity (Loening-Baucke, 1984; Benninga et al., 2004b), related to increased rectal compliance, a lack of daily routine, unacceptable toilets or inadequate privacy. Poor perception of rectal filling can trigger increased rectal capacity, impaired stool quality, an increased rectoanal inhibitory reflex threshold and incomplete emptying at eventual defecation. Although stool consistency in constipation is generally assumed to be hard and dry, it may be soft and unformed, and therefore difficult to perceive and fully evacuate.

Children can voluntarily suppress the urge to defecate, a behaviour that may be due to an impairment of learning, distress, trauma, a disruption of routine, inattention, or cognition difficulties. Toilet refusal is often associated with the memory or expectation of pain at defecation. Causes include having passed a large or hard stool, the presence of an anal fissure, an anal streptococcal infection, anxiety or irrational fears associated with the toilet (Chase et al., 2004). Longitudinal studies of toilet training have identified that constipation precedes both stool withholding and hiding before defecation (Taubman et al., 2003; Blum et al., 2004). The signs of stool withholding in a toddler include squatting, crossing of the legs, stiffening of the body, forcefully contracting the gluteal muscles, hiding and holding onto furniture. During this time of stool urge, the rectum accommodates stool content until the urge to defecate passes. Over time the stool accumulates, becoming harder and drier.

Constipation can be broadly considered to be due to an abnormal contraction pattern of colonic motor function or to an inability to relax the pelvic floor and anal sphincter during defecation. These underlying causes may coexist.
in 13% of adolescent subjects (Chitkara et al., 2004), or have no overlap (Gutierrez et al., 2002).

In a Dutch sample normal colonic transit time was found in 56% of children with chronic constipation, with the remainder showing both significantly longer segmental and total transit time when compared to children with either non-retentive soiling or abdominal pain (Benninga et al., 2004a). The presence of a postprandial gastrocolonic response implies normal colonic motility. It has been suggested that increased colonic transit time may be secondary to chronic faecal retention in the rectum (Benninga et al., 2004a).

Abnormal contraction of the external anal sphincter was observed during attempted defecation in 64% of children with chronic constipation (Gutierrez et al., 2002). There may also be a lack of increased intra-abdominal pressure or a partial or non-relaxation of the internal anal sphincter in children with pelvic floor dyssynergia.

PTs are most commonly involved in treating bowel dysfunction in children who have pelvic girdle muscle dyssynergia during elimination, slow transit of stool through the gut, or require rehabilitation after anorectal surgery.

When children show inappropriate pelvic floor contraction, or fail to relax the pelvic musculature during attempts to defecate, pelvic floor dyssynergia may be suspected. A diagnosis identifies lack of perineal descent, maintenance of an acute anorectal angle and poor rectal funnelling during attempted defecation. Treatment sessions may involve anal manometry with use of a rectal balloon to produce rectal sensation and train appropriate volume sensation. Subsequent perineal anal plug electrode EMG can then record and feedback activity in the external anal sphincter. The PT uses this visual message to teach the child specific recruitment and relaxation of the external anal sphincter and global pelvic musculature. As a stand-alone treatment biofeedback has not been shown to be efficacious for constipation that is not associated with dysfunctional defecation (Poenaru et al., 1997). However, in the subset of children with constipation and identified pelvic floor dyssynergia, abnormal defecation dynamics improve significantly after biofeedback therapy but long-term bowel function is not superior (Van der Plas et al., 1996).

Since biofeedback is always offered as part of a multimodal package of care for children with bowel dysfunction ± lower urinary tract symptoms, studies investigating the isolated effect of pelvic muscle motor control training using only biofeedback techniques are rare. Further, the difficulty in interpreting study findings is exacerbated by a lack of subclassification of constipation and poor description of defecation mechanics at baseline, particularly when children present with predominant bladder problems. In addition, most studies are case series and although data of constipation as a secondary measure favours adjunctive biofeedback, study design and patient heterogeneity limits endorsement. A protocol has been published describing an upcoming RCT of physical therapy intervention and medical care versus medical care alone in children with functional constipation and results are keenly awaited (van Engelenburg-van Lonkhuyzen et al., 2013).

Two systematic reviews of the use of biofeedback for dysfunctional elimination have identified six randomized trials of biofeedback in children with pelvic floor dyssynergia ± constipation (Table 10.5). One RCT in adults with constipation and pelvic floor dyssynergia comparing anorectal biofeedback with placebo shoulder musculature biofeedback showed non-significant improvement in constipation severity after intervention (Hart et al., 2012). A further RCT stated constipation in children with urinary tract infections as a secondary outcome measure, but did not report change after biofeedback (Klijn et al., 2006). Quality aspects of the studies are shown in Table 10.6.

---

**Box 10.4 Intervention cascade for functional incontinence in children**

- Comprehensive bowel history including 2-week stool chart
- Neurological screen, including inspection of lumbar region
- Abdominal palpation to identify faecal mass
- Evaluation of rectal diameter or identification of impacted rectum
- Perineal inspection to confirm anal position, identify any descent, soiling, dermatitis, fissures, haemorrhoids or excoriation
- Rectal emptying of impacted stool
- Maintenance of regular soft stools (stool softeners/laxatives for at least 4–6 months)
- Training optimal defecation mechanics
- Toilet habit training, desensitization of toilet phobias, environmental management
- Strategies to manage faecal incontinence episodes
<table>
<thead>
<tr>
<th><strong>Table 10.5</strong></th>
<th>RCTs for biofeedback intervention in children with constipation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Author</strong></td>
<td>Wald et al., 1987</td>
</tr>
<tr>
<td><strong>Design</strong></td>
<td>(1) Pressure biofeedback (2) Mineral oil therapy</td>
</tr>
<tr>
<td><strong>n</strong></td>
<td>50</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>Faecal soiling; 18 had abnormal defecation dynamics</td>
</tr>
<tr>
<td><strong>Drop-outs/Adherence</strong></td>
<td>Not reported</td>
</tr>
<tr>
<td><strong>Results</strong></td>
<td>No difference in soiling (1) vs (2) post treatment or at follow-up Normal defecation dynamics: (1) 6/9; (2) 3/9</td>
</tr>
</tbody>
</table>

| **Author**     | Loening-Baucke, 1990                                       |
| **Design**     | (1) Coordination biofeedback plus medical care vs (2) Medical care |
| **n**          | 43                                                           |
| **Diagnosis**  | Contraction of EAS and pelvic floor during defecation Faecal incontinence |
| **Protocol**   | 6 sessions of weekly EMG anal sphincter and rectal biofeedback |
| **Drop-outs/Adherence** | 2 patients lost to follow-up                               |
| **Results**    | Symptoms resolved at 7 and 12 months: (1) 55%, 50%; (2) 5%, 16% Normal defecation dynamics: (1) 77%; (2) 13% |

| **Author**     | Van der Plas et al., 1996                                  |
| **Design**     | (1) Standard medical management with biofeedback (2) Standard medical management |
| **n**          | 192                                                          |
| **Diagnosis**  | <3 stools/week; soiling >2x/month; laxative use; 60% abnormal defecation dynamics |
| **Protocol**   | 5 clinic visits for both groups                              |
| **Drop-outs/Adherence** | 5 and 8 patients lost to follow-up at 6 months and 1 year respectively |
| **Results**    | Symptom resolution: (1) 32%; (2) 33% Normal defecation dynamics: (1) 86%; (2) 52% (p<0.001) |

| **Author**     | Nolan et al., 1998                                         |
| **Design**     | (1) Standard medical management with biofeedback (2) Standard medical management |
| **n**          | 29                                                           |
| **Diagnosis**  | Soiling resistant to treatment with proven pelvic floor dysfunction |
| **Protocol**   | 3–4 sessions of weekly anal EMG biofeedback                 |
| **Drop-outs/Adherence** | 3 children lost to repeat manometry at 6 months           |
| **Results**    | Symptom improvement: (1) 4/14; (2) 6/15 NS Normal defecation dynamics: (1) 7/13; (2) 2/13 |

| **Author**     | Sunic-Omejc et al., 2002                                    |
| **Design**     | (1) Standard medical management with biofeedback (2) Standard medical management |
| **n**          | 49                                                           |
| **Diagnosis**  | Non-organic chronic constipation in children <5 years. Abnormal defecation in 57% |
| **Protocol**   | Biofeedback in clinic and home pelvic floor exercises for 12 weeks |
| **Drop-outs/Adherence** | Not reported                                                |
| **Results**    | Improved constipation: (1) 84%; (2) 62.5%                   |

| **Author**     | Kajbafzadeh et al., 2011                                    |
| **Design**     | Randomized controlled trial A: animated biofeedback via perianal EMG electrodes + standard care B: standard care of timed voiding, high fibre diet and hydration |

(Continued)
Evidence-Based Physical Therapy for the Pelvic Floor

Neuromodulation of the central nervous system has long been known to alter function in the urinary system. Over the last 5 years electrical therapy has been shown to have an application for colonic inertia in children with constipation. An Australian study of children with slow transit constipation randomized to a total of 8 weeks of real or placebo interferential therapy reported a significant reduction in soiling, abdominal pain and laxative use in the active group (Clarke et al., 2009; Ismail et al., 2009). In addition, the group reported a decreased colonic transit

<table>
<thead>
<tr>
<th>Table 10.5</th>
<th>RCTs for biofeedback intervention in children with constipation—cont’d</th>
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</thead>
<tbody>
<tr>
<td>n</td>
<td>80</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Simultaneous constipation±faecal soiling and dysfunctional voiding</td>
</tr>
<tr>
<td>Protocol</td>
<td>A: animated anal sphincter biofeedback for 6–12 sessions; two sessions/week with continuation until normalization of uroflow EMG</td>
</tr>
<tr>
<td></td>
<td>Follow-up at 6 and 12 months</td>
</tr>
<tr>
<td>Drop-outs/Adherence Results</td>
<td>Full attendance and no drop-out at follow-up</td>
</tr>
<tr>
<td>Constipation:</td>
<td>A: 25/40 pre→8/40 6 mth→8/40 12 mth</td>
</tr>
<tr>
<td></td>
<td>B: 20/40 pre→12/40 6 mth→12/40 12 mth</td>
</tr>
<tr>
<td>Anal incontinence:</td>
<td>A: 15/40 pre→0/40 6 mth→0/40 12 mth</td>
</tr>
<tr>
<td></td>
<td>B: 20/40 pre→16/40 6 mth→9/40 12 mth</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Table 10.6</th>
<th>PEDro quality score of trials of biofeedback treatment for functional childhood constipation</th>
</tr>
</thead>
<tbody>
<tr>
<td>E – Eligibility criteria specified</td>
<td></td>
</tr>
<tr>
<td>1 – Subjects randomly allocated to groups</td>
<td></td>
</tr>
<tr>
<td>2 – Allocation concealed</td>
<td></td>
</tr>
<tr>
<td>3 – Groups similar at baseline</td>
<td></td>
</tr>
<tr>
<td>4 – Subjects blinded</td>
<td></td>
</tr>
<tr>
<td>5 – Therapist administering treatment blinded</td>
<td></td>
</tr>
<tr>
<td>6 – Assessors blinded</td>
<td></td>
</tr>
<tr>
<td>7 – Measures of key outcomes obtained from &gt;85% of subjects</td>
<td></td>
</tr>
<tr>
<td>8 – Data analysed by intention to treat</td>
<td></td>
</tr>
<tr>
<td>9 – Statistical comparison between groups conducted</td>
<td></td>
</tr>
<tr>
<td>10 – Point measures and measures of variability provided</td>
<td></td>
</tr>
<tr>
<td>Study</td>
<td>E</td>
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<td>-------------</td>
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</tbody>
</table>

+, criterion is clearly satisfied; –, criterion is not satisfied; ?, not clear if the criterion was satisfied. Total score is determined by counting the number of criteria that are satisfied, except that ‘eligibility criteria specified’ score is not used to generate the total score. Total scores are out of 10.
Evidence for pelvic floor physical therapy in children


Evidence-Based Physical Therapy for the Pelvic Floor


Pelvic floor physical therapy in the elderly: where’s the evidence?

Adrian Wagg

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INTRODUCTION

The majority of developed countries of the world face profound demographic change. The greatest expansion will be in the proportion of the oldest old in the population, those in their ninth decade of life (Kinsella and Wan He, 2009). Forecasts suggest that for many developed countries, the number of people over the age of 65 will shortly outnumber those under the age of 20. Older people typically present with multimorbidity in addition to the physiological changes associated with ageing and research into, and the development of, age-appropriate interventions is required to meet the needs of older people. The major focus of healthcare for the elderly should be to ensure an integrated and comprehensive approach to the needs of older people and their caregivers. The quest for adequate and cost-effective healthcare for the growing number of older persons has received increased attention from health service providers, national governments and international organizations as the need to improve quality of services and at the same time constrain healthcare spending becomes paramount.

Of particular relevance to continence in older people, the International Consultation on Urological Diseases, in conjunction with the European Association of Urology, has taken the lead on developing evidence-based guidelines into the causes and management of incontinence in the elderly and frail elderly through the International Consultation on Incontinence (Abrams et al., 2010; DuBeau et al., 2010).

PREVALENCE

The prevalence of urinary incontinence (UI) increases with increasing age, affects women more than men and is associated with significant personal stress, shame and social stigma (Irwin et al., 2006) with considerable morbidity (Coyne et al., 2012; Milsom et al., 2012) and cost (Stothers et al., 2005; Thom et al., 2005; Milsom et al., 2013). The social and psychological consequences associated with incontinence are such that sufferers become less socially and, in the case of overactive bladder, less physically active (Coyne et al., 2013).

The presence of comorbidities and functional impairment in the elderly may lead practitioners to overlook incontinence and thus leave it untreated, and merely contained by continence products. The influence of co-existing diseases on the impact of incontinence in older people has been well described; there are however few data that describe the treatment of these co-existing comorbidities in terms of the impact on lower urinary tract symptoms or incontinence. One area that has received attention is the effect of exercise, either as musculoskeletal exercises to increase gait speed and stamina alone or combined with pelvic floor muscle re-education, in a variety of older people, from community dwelling to nursing home residents (Kim et al., 2007; van Houten et al., 2007; Sugaya et al., 2007).
However, it has not yet been shown whether high-intensity pelvic floor muscle training (PFMT) alone in the frail elderly is associated with reduced UI.

**Classification of incontinence**

The prevalence of different types of incontinence, unchanged in older persons, does alter in association with increasing age. Storage symptoms – nocturia, nocturnal polyuria, urinary urgency, frequency and urgency incontinence – become more common, as do the voiding symptoms (Irwin et al., 2006). Voiding symptoms are more common in men, probably due to the increased prevalence of bladder outflow tract obstruction, but this difference is less than is traditionally taught. In addition to the ‘classical’ subtypes of UI, functional incontinence occurs more frequently in older than young people; this is where the incontinence is not necessarily due to a lower urinary tract disorder, but is a reflection of either physical or cognitive disability such that the affected individual fails to maintain continence or voids in an inappropriate place or at a socially inappropriate time.

**Who are the elderly and ‘frail’ elderly?**

Societal ageing has been described as one of the greatest challenges of the 21st century. Whereas ageing for many is characterized as ‘a progressive, generalized impairment of function resulting in a loss of adaptive response to stress (loss of biological reserve) and in a growing risk of age-associated disease’ (Kirkwood, 1995), there has been a change in the physical wellness of older people in the ‘baby boomer’ generation which has led to reductions in late life disability (Martin et al., 2010). Chronological age is simply too unsophisticated a marker with which to label such a heterogeneous group. A simple distinction might be drawn between the robust and frail elderly. Frailty as a concept has a number of definitions that centre on the concept of biological reserve. The frailty phenotype combines impaired physical activity, mobility, balance, muscle strength, motor processing, cognition, nutrition and endurance (Fried et al., 2001; Centers for Disease Control National Center for Health Statistics, 2004; Ferrucci et al., 2004). It is not identical to disability and the presence of co-existing disease (comorbidity). In a study of older people meeting strict ‘phenotypic’ criteria for frailty, only 22% of the sample also had both comorbidity and disability; 46% had comorbidity without disability; 6% disability without comorbidity; and 27% had neither (Fried et al., 2001). Frailty may, however, also be defined in a more mechanistic fashion, by adding up the total number of pre-existing biomedical and social comorbidities; frail people, however defined, do have a higher risk of recurrent disease, increased disability, hospitalization and death than those without frailty (Lacas and Rockwood, 2012). Definitions of ‘old’ and ‘elderly’ vary within the literature. Definitions often reflect the theory of ageing that each definition is attempting to explain, or occasionally, merely convenience. A balanced approach to understanding the ageing process includes not only the understanding of the physiological changes that occur, but the social context in which they occur and the attitudinal changes of ageing persons themselves (Stein and Moritz, 1999). The ‘life course’ conceptual framework considers the influence of modifiable factors of lifestyle, such as not smoking or abusing alcohol, regular exercise, good social supports and of non-modifiable factors such as economic circumstances and depressive disorders; all of which are independently predictive of healthy ageing in a 50-year prospective cohort study (Vaillant and Mukamal, 2001). Functional status of an individual therefore depends on the interaction of all an individual’s life course events and is independent of chronological age.

Chronological age is in fact a very poor indicator of functional status. Individuals of the same age show great variability in social, psychological and physical changes. However, the chronological age of retirement, commonly 65 years in many nations although this is changing across Europe, is the age when ‘old age becomes operationalized. The WHO (Stein and Moritz, 1999) uses the ‘life course’ framework, to define ageing as ‘the process of progressive change in the biological, psychological and social structure of individuals’ without prescribing any chronological ages associated with this process. When ages are, however, superimposed on this definition, ‘mid-life’ is defined as beginning at 50 years or after the menopause in women, ‘young old’ at 60 years and ‘very old’ at 80+ years (Stein and Moritz, 1999).

‘Incontinence’ as a diagnosis or as a symptom fits well into the ‘life course’ model, where comorbidities or life events have an impact on the course of the disease process or symptoms, and chronological age may not be a significant factor in either symptom presence or severity although age is an immutable risk factor associated with UI.

The first general assumption, made by the committee on the frail elderly for the Fifth International Consultation on incontinence, is that there is no reason to suspect that interventions proven to be effective in the management of community dwelling older people should not be effective in the frail elderly and that such interventions should not be withheld for reasons of age alone. However, due regard should be given to remaining life expectancy, the wishes of the patient and caregiver, and the potential for benefits and harms of the proposed treatment (Wagg et al., 2013). Incontinence in older people most often has multiple underlying causes and, as such, forms a true geriatric syndrome in a similar fashion to falls and delirium. Cure, as defined by total absence of symptoms, as for most other chronic conditions, is the exception rather than the rule when dealing with frail older people but there is great opportunity to relieve the burden of symptoms and greatly
improve quality of well-being for the majority of older people affected by incontinence (Ouslander, 2000).

**Prevalence of incontinence in the elderly**

Estimates of prevalence of UI vary widely, depending upon the definition used in the study (the ICS definition ‘any involuntary loss of urine’ says nothing of severity, frequency, duration or impact) and the setting in which the study took place. Generally, the more functionally dependent the study population, the higher the prevalence of UI such that the highest prevalence occurs on frail older people in residential long-term care. Crude prevalence estimates for the most inclusive definitions of UI in women (‘ever’, ‘any’ or ‘at least once in the past 12 months’) range from 5% to 69%, with most studies reporting a prevalence of any UI in the range of 25–45%. There are, however, still gaps in our knowledge; for instance, in a systematic review of incontinence in people with a dementia diagnosis living at home, rates of incontinence varied between 1.1% in a general community population to 38% in those receiving homecare services (Drennan et al., 2013). Men appear to have overall half the prevalence of UI than women. The increasing prevalence in association with age is largely due to the contribution of urgency incontinence (UUI) rather than stress incontinence (SUI). One study demonstrated an increasing rate of UUI from 0.7% between age 50 and 59, 3.4% for 70 years and older men. SUI prevalence was steady at 0.5% and 0.1%, respectively (Ueda et al., 2000). A similar trend of increasing proportions of urgency and UI with increasing age was shown in the United States and a smaller population-based Canadian study (Finkelstein, 2002; Diokno et al., 2007). Conversely, Maral and co-workers showed increasing prevalence of SUI with age, from 0.9% between age 35 and 44, to 4.9% at age 65 and older (Maral et al., 2001).

Faecal incontinence is also more prevalent in the elderly although more difficult to measure due to a lack of standard definitions. Prevalence rates were reported to vary between 5% and 10% in community-dwelling elderly aged over 60 years in The Netherlands (88% response rate) (Teunissen et al., 2004). In a single, relatively bias-free study using a standardized instrument to ascertain faecal incontinence, rates varied between 11% and 15% (Macmillan et al., 2004). Faecal incontinence rates show no sex difference, and increase in association with increasing age for both men and women.

With regards to elderly people seeking assistance for symptoms of incontinence, rates of use of health services have been shown to be consistently low across three nationalities, but higher in one study. Andersson et al. (2004) investigated by questionnaire how UI affects daily activities and help-seeking behaviours in a Swedish regional population (n = 2129), and found that only 18% of 65–79-year-olds requested treatment – those with the worst leakage and level of distress. Hannestad et al. (2000) found that only 25% of symptomatic older Norwegian women sought help – again those who were older and with worse symptoms. In the UK, a similar mailed questionnaire to an elderly regional population (n = 915) found that 15% of those with incontinence had used continence services. The most significant factor for continence service usage was being asked about their symptoms by a health professional (OR 15.7, 95% CI 7.3, 33.9). Other significant factors were more severe and bothersome symptoms, and worse general health (Peters et al., 2004). These figures were similar to another UK study, in which only 9% of all adults with severe symptoms sought a consultation, which the authors found was associated with an acceptance of incontinence as normal in older women (McGrother et al., 2004). However, in an Australian study, 73% of women aged 70–75 years had sought help or advice about their incontinence, and these were women with more severe symptoms (Miller et al., 2003).

Although urinary incontinence is widely purported to be a predictor of nursing home admission, data supporting this assertion are few. Whereas Holroyd-Leduc and co-workers investigated the relationship of UI to key adverse outcomes (death, nursing home admission, functional decline) in 5500 community-dwelling elderly, mean age 77 years (69–103 years) and concluded that UI was not an independent risk factor for these adverse outcomes, but higher levels of illness severity and functional impairment were (Holroyd-Leduc et al., 2004).

**AETIOLOGY AND PATHOPHYSIOLOGY**

In the elderly, UI may be associated with reversible factors. These can be remembered using the mnemonic ‘DIPPER’S’ (Box 11.1) (DuBeau et al., 2010). These factors are useful to consider in the initial assessment of an older person with recent onset UI and describe those areas to which attention might be paid, leading to an amelioration of symptoms without any intervention aimed specifically at the lower urinary tract. UI in an older person manifests as a typical geriatric syndrome, with a complex interaction of underlying factors.
pathophysiological changes, risk factors and modifying factors. Addressing incontinence in the elderly then usually requires a multifactorial intervention to achieve benefit.

**Central neurological factors affecting control of continence**

1. **Diseases that affect central neurological control:** stroke, brain tumour, Parkinson's disease, multiple sclerosis, diabetes mellitus, cerebral atrophy, multi-system atrophy, normal pressure hydrocephalus, dementia, depression (De Ridder et al., 1998; Gariballa, 2003).

2. **Neurological disorders that affect suprasacral spinal cord pathways,** with deficits affecting both somatic and autonomic nervous systems: multiple sclerosis, dorsal column neuropathies, spinal cord injury (Blok et al., 1997) leading to sphincter dyssynergia through upper motor neuron damage, or conversely sphincter underactivity via sympathetic dysfunction (Corcos and Schick, 2001).

3. **Progressive sympathetic nervous system activation:** occurs in older age and may be a causal component in urinary tract pathophysiology, though the underlying central nervous system mechanisms mediating this increase in activity are unknown (Esler et al., 2002).

4. **Peripheral nerve root compression (S2–S4)** from musculoskeletal injury or degeneration, leading to decreased lower limb mobility, impaired sensation and reflexes, and PFM and striated sphincter weakness (Corcos and Schick, 2001).

**Non-neurological disease**

**Ageing urinary tract**

Physiological changes in the lower urinary tract of men and women in association with age have been well described but are limited by their cross-sectional nature and in that most studies have been done in people with pre-existing lower urinary tract symptoms (Malone-Lee and Wahedna, 1993; Resnick et al., 1995; Collas and Malone-Lee, 1996; Pfisterer et al., 2006). Urodynamic changes associated with age have typically included smaller voided volume, increased residual volume, smaller bladder capacity, and increased rates of detrusor overactivity. Reported age-related changes in the lower urinary tract are shown in Table 11.1 and detailed below.

<table>
<thead>
<tr>
<th>Age-related change</th>
<th>Potential effects on continence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bladder overactivity and urgency UI</td>
<td>Impaired bladder contractility, increased residual urine, and decreased functional bladder capacity</td>
</tr>
<tr>
<td>Bladder function: Decreased capacity Decreased sensation of filling Increased detrusor overactivity Decreased bladder contractile function Increased residual urine</td>
<td>Increased likelihood of urinary symptoms and UI</td>
</tr>
<tr>
<td>Urethra: Decreased closure pressure in women</td>
<td>Increased likelihood of stress and urgency UI</td>
</tr>
<tr>
<td>Prostate: Increased incidence of benign prostatic obstruction Increased incidence of prostate cancer</td>
<td>Increased likelihood of urinary symptoms and UI</td>
</tr>
<tr>
<td>Decreased oestrogen (women)</td>
<td>Increased incidence of urogenital atrophy and related symptoms Increased incidence of recurrent urinary tract infections</td>
</tr>
<tr>
<td>Increased night-time urine production</td>
<td>Increased likelihood of nocturia and night-time UI</td>
</tr>
<tr>
<td>Altered central and peripheral neurotransmitter concentrations and actions</td>
<td>Increased likelihood of lower urinary tract dysfunction</td>
</tr>
<tr>
<td>Altered immune function</td>
<td>Increased likelihood of recurrent urinary tract infections</td>
</tr>
<tr>
<td>Increased prevalence of white matter hyperintensities in brain</td>
<td>Increased prevalence of severe urge/urgency, link to cognitive impairment and impaired mobility</td>
</tr>
</tbody>
</table>
Evidence-Based Physical Therapy for the Pelvic Floor investigated

Similarly, an age-related decline in detrusor contractility can be demonstrated, in line with the observed reduction in acetylcholine release from nerve stimulation (Susset et al., 2001). The detrusor smooth muscle contractility itself appears to be unaffected by age in the absence of bladder dysfunction. The observation of impaired emptying appears to be the result of a dampening of generated contractile force by the connective tissue (Susset et al., 1978). However, where there is detrusor activity, a reduction in detrusor contractility can be demonstrated, in line with the observed reduction in acetylcholine release from nerve stimulation (Yoshida et al., 2004).

2. Sphincter integrity: In an intraurethral ultrasound investigation, Klauser et al. (2004) found that with increasing age the striated urethral sphincter showed a linear decline in thickness and ability to produce urethral closure pressure. In ageing para-urethral tissue, the connective tissue component has been shown to increase, altering in composition to be more fibrous relative to other components, and show a decrease in the vascularity of the mucosa and urethral nerve supply (Verelst et al., 2002). With regard to decreased urethral muscle function, Perucchini et al. (2002b) showed a decline in the number of striated muscle fibres, fibre density and total cross-sectional area of striated muscle in a dissection study of the anterior and posterior walls of the urethra in 25 female cadavers aged 15–80 years. A seven-fold variance was seen, however, between the specimens with the most and fewest muscle fibres anteriorly, and localized losses were found in the proximal posterior striated sphincter muscles (Perucchini et al., 2002a). Similarly, an age-related apoptosis of human rhabdosphincter myocytes has been described (Strasser et al., 2000).

3. PFM ageing: Pelvic floor muscle isometric strength measured by maximum volitional vaginal closure force in the mid-sagittal plane shows no significant decline with increasing age (Trowbridge et al., 2007). This is somewhat unexpected as a 30–40% loss of skeletal muscle volume and cross-sectional area is the usual finding in striated muscle with advancing age. Changes in skeletal muscle occur with age in endocrine, neural, enzymatic and energy systems, partially genetically driven, and result in a decrease in muscle mass by fibre, vascular and mitochondrial degradation and loss associated with the sarcopenia of ageing (Powers and Howley, 2001). Many of the studies on pelvic floor muscular changes in association with age have not controlled for parity, confounding the results. Dimpfl et al. (1998) showed histomorphological changes in the PFM in older women compared to women under 40 years, such as decreased fibre circumference and fibrosis. Gunnarsson and Mattiasson (1999), using surface electromyography (EMG), showed that older women without incontinence did not show as much of a decline in PFM strength as women with SUI or urgency or mixed incontinence. In this study, the investigators hypothesized that neuromuscular changes in the pelvic floor were progressive and present for a long time before symptoms appeared. However, Constantinou and coworkers (2002), using magnetic resonance imaging (MRI), showed that older women displaced the pelvic floor significantly less than younger women on voluntary contraction. More recently, in an MRI study comparing nulliparous young and old women, no change in volume of the levator ani in association with age could be demonstrated, whereas the obturator internus, used as a comparator, did demonstrate such an effect (Morris et al., 2012).

4. Ageing changes in the fascial supports of the urinary tract: Ageing connective tissue shows evidence of fewer and more immature collagen cross-linkages, resulting in a two- to three-fold reduction in the maximum load to failure, decreased plasticity and elasticity (Frankel and Nordin, 1980).

Other aetiologies

1. Side-effects of prescription and over-the-counter drugs.

2. Social and environmental status: relating to the characteristics of the living environment (ease of access to washroom), mobility, need for social support of care from either formal or informal caregiver.

3. Disturbance of the vasopressin system, or diseases causing a shift in diuresis: diseases such as diabetes mellitus, congestive heart failure and sleep apnoea cause a shift in diuresis: from daytime to night (i.e. nocturnal polyuria [Asplund, 2004]).

4. Functional impairment: Described as the difference between environmental demand and functional capability (Eekhof et al., 2000) and can be modified by treatable factors such as intercurrent illnesses, medications, nutritional status, vision and hearing status, mobility and dexterity, pain, anxiety and depression (Harari et al., 2003). Within functional impairment, strength impairment and lower limb
mobility are the critical domains (Jenkins and Fultz, 2005) for predicting UI. In stroke, functional impairment, particularly needing help to access the toilet, is the strongest independent factor associated with new-onset faecal incontinence after a stroke (Harari et al., 2003).

5. Obesity: Obesity is a risk factor, more for SUI than for UIUI but is increasingly important as the proportion of either very overweight or obese people in the population of developed countries increases. The mechanism behind the increased risk may be simply mechanical but may also reflect the influence of the metabolic syndrome on the development of incontinence in later life (Chu et al., 2013). Central obesity in women appears to be a significant risk factor over and above that attributable to increased body mass index (Krause et al., 2010).

Factors in females

Factors in females are:

- becoming oestrogen-deficient postmenopausally (Schaffer and Fantl, 1996; Davila et al., 2003);
- high parity (Simeonova et al., 1999);
- certain types of intrapelvic surgery, including hysterectomy (Sherburn et al., 2001);
- female circumcision (Stein and Moritz, 1999).

Becoming oestrogen-deficient leads to:

- loss of collagen, thinning epithelium in vagina, caused by decreased collagen synthesis (Falconer et al., 1996) and increased collagenase activity (Kushner et al., 1999);
- decreased vascular plexi in the submucosa of the urethra – this submucosal vascular bed gives passive urethral control and loss can lead to a loss of up to 30% of urethral closure pressure (Corcos and Schick, 2001);
- less acidic urethral and vaginal environments (increased pH), leading to changes in the vaginal flora and more risk of colonization with Gram-negative bacteria, which in turn leads to a higher risk of atrophic vaginitis and urinary tract infections (Nilsson et al., 1995; Notelovitz, 1995; Samsoe, 1998; Bachmann and Nevadunsky, 2000).

Factors in males

Factors in males are increased prostatic size in:

- benign prostatic enlargement (Madersbacher et al., 1999; Blanker et al., 2000);
- prostatic carcinoma.

In benign prostatic enlargement (BPE), the outer zones of the prostate progressively atrophy while the inner zones begin to grow again until death. In carcinoma, the outer glandular epithelium enlarges. This leads to:

- impaired urinary flow;
- strangulation of the urethra;
- urinary retention;
- urinary frequency;
- detrusor overactivity;
- incomplete emptying and retrograde filling of the ureters (Timiras and Leary, 2003).

Treatment of these disorders by prostatectomy, whether simple, radical or transurethral, carries a risk of urethral vascular bed destruction and nerve damage, even in ‘nerve sparing’ surgery (Corcos and Schick, 2001).

Faecal incontinence and constipation

Faecal incontinence and constipation in the elderly has many risk factors, including lifestyle issues such as lack of mobility, inadequate fluid and fibre intake, use of common medications and systemic diseases such as diabetes mellitus, diarrhoeal disease, functional impairment, obesity, systemic sclerosis, neuromuscular diseases and psychiatric disorders. Epidemiological risk factors are increasing age, being female and low socioeconomic status.

Functional obstruction can be caused by pelvic floor dysfunction, including rectocele, enterocele, perineal descent and anismus, and disorders of colonic motility (Cundiff et al., 2000). In a review of constipation, irritable bowel syndrome and diverticulosis in the ageing gastrointestinal tract, the prevalence of constipation and diverticulosis was found to be greater in the elderly, but the aetiology was unclear. Ageing changes of increased fibrous connective tissue within the gut wall and a decreased neural supply to the colon are likely causes (Camilleri et al., 2003). On high spatial resolution endo-anal MRI in normal ageing in continent elderly (Rociu et al., 2000) found there was a thinning of the external anal sphincter (EAS) and longitudinal muscle of the anus, and compensatory thickening of the internal anal sphincter. Likewise, in a recent ultrasound and manometric study, ageing was associated with a thickening of the internal anal sphincter. Older incontinent women had a thinner EAS, had decreased maximum squeeze pressures, and were hypersensitive to rectal distension with decreased tolerable rectal volumes and urge to defecate at lower volumes (Lewicky-Gaupp et al., 2009).

EVIDENCE FOR EFFECT OF PFMT IN PREVENTION OF UI IN OLDER PERSONS

Overall, evidence for the utility of pelvic floor muscle training (PFMT) in older people is lacking. Studies are in general of poor or moderate quality, non-comparable and require some extrapolation of their findings to be applicable (see Table 11.2).
### Table 11.2 Reviews of conservative interventions for prevention of UI in older persons

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Prompted voiding</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Authors</strong></td>
<td>Palmer, 2005</td>
</tr>
<tr>
<td><strong>Design</strong></td>
<td>Systematic literature review</td>
</tr>
<tr>
<td><strong>Sample</strong></td>
<td>1 quasi-experimental, 1 repeated measures, 1 prospective case series and 1 systematic (Cochrane) review</td>
</tr>
<tr>
<td><strong>Methods</strong></td>
<td>Sample, methods, and results were examined to address: is prompted voiding effective in reducing wetness episodes and increasing requests for toileting?</td>
</tr>
<tr>
<td><strong>Results</strong></td>
<td>Different prompted voiding protocols were used limiting comparison across studies. Sample sizes were small and mainly white elderly female long-term care residents participated. Staff adherence to the protocol was important to its success. Little evidence exists that self-initiated requests for toileting is increased. Wetness episodes decreased in the short term</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Authors</th>
<th>Eustice et al., 2000</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Design</strong></td>
<td>Cochrane review update</td>
</tr>
<tr>
<td><strong>Sample</strong></td>
<td>Nine trials included, n = 674 older adults</td>
</tr>
<tr>
<td><strong>Methods</strong></td>
<td>Literature searched according to protocol (all randomized or quasi-experimental studies). Two reviewers evaluated studies for methodological quality; third reviewer proof read the review</td>
</tr>
<tr>
<td><strong>Results</strong></td>
<td>Insufficient evidence to reach firm conclusions for practice. Suggestive evidence exists for short-term benefit from prompted voiding, longer effects are not known</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Authors</th>
<th>Schnelle et al., 2010</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Design</strong></td>
<td>RCT</td>
</tr>
<tr>
<td><strong>Sample</strong></td>
<td>112 nursing home residents in 12 nursing homes in USA</td>
</tr>
<tr>
<td><strong>Methods</strong></td>
<td>Prompted voiding, exercise and mobility endurance, food and fluid intake administered by trained research staff for 5 days a week from 7 am to 3:30 pm over 12-week period. Frequency of urinary and faecal incontinence and constipation were outcome variables</td>
</tr>
<tr>
<td><strong>Results</strong></td>
<td>Urinary incontinence frequency and number of appropriate toiletings improved</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Authors</th>
<th>Ostaszkiewicz et al., 2004a</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Design</strong></td>
<td>Cochrane review</td>
</tr>
<tr>
<td><strong>Sample</strong></td>
<td>Four trials included, n = 378</td>
</tr>
<tr>
<td><strong>Methods</strong></td>
<td>Literature searched and evaluated per protocol. Trials too heterogeneous for meta-analysis</td>
</tr>
<tr>
<td><strong>Results</strong></td>
<td>Adherence to habit retraining protocols is difficult for staff. Evidence is too limited to judge if improvements in continence make habit re-training protocols worth investment</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Authors</th>
<th>Nikoletti et al., 2004</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Design</strong></td>
<td>RCT</td>
</tr>
<tr>
<td><strong>Sample</strong></td>
<td>41 elderly incontinent patients on acute care rehabilitation units in Australian hospitals</td>
</tr>
<tr>
<td><strong>Methods</strong></td>
<td>Patients in the treatment group were monitored for 72 hours with an electronic device. Patients in the control group received standard habit training. Prescribed voiding times for both groups (control and monitoring group) were developed and continence outcomes were measured</td>
</tr>
<tr>
<td><strong>Results</strong></td>
<td>No statistically significant improvements in self-reported or carer reported UI frequency. Significant reduction in self-reported and carer reported severity of UI in one-month follow-up of treatment group. Missing data and problems with using the electronic monitoring device were noted</td>
</tr>
</tbody>
</table>

(Continued)
Primary prevention

There is no evidence to support the use of PFMT in the primary prevention of urinary or faecal incontinence in older men and women.

Secondary prevention

A Dutch study that investigated the effect of a preventative screening programme for UI in 1121 subjects over the age of 75 years from 12 general practices, found that early screening and detection did not result in a measurable effect in reduction of prevalence of urinary incontinence compared to the control group who did not receive the preventative screening. The authors recommended that preventative assessment of the elderly is individually targeted, started before the age of 75 years and offered in the primary healthcare setting (Eekhof et al., 2000). A systematic review of physical therapies for prevention of urinary and faecal incontinence in adults of 2002 (Hay-Smith et al., 2002) and 2007 (Hay-Smith et al., 2007 [now withdrawn]) failed to find evidence for the use of PFM exercises in the prevention of UI in adults, and recommended further research, largely due to the quality of the included studies.

Diokno et al. (2004) reported the results of an RCT of a preventive behavioural modification programme in a
Evidence-Based Physical Therapy for the Pelvic Floor

A group of continent older women, aged 55 years and over. The intervention consisted of a 2-hour classroom presentation, and an individual session 2–4 weeks later to reinforce the home programme, measure PFM strength per vaginum and check bladder training concepts. The main outcome measure of number of incontinent episodes per year measured via a validated questionnaire showed that this programme was significantly effective in maintaining continence in the treatment group compared to the control group (OR 1.97, 95% CI 1.15, 3.38, p = 0.01). In addition, voiding frequency significantly decreased, PFM strength significantly increased and all improvements remained at 12 months after the intervention. For men prior to prostate surgery, a non-randomized trial of preoperative PFMT produced a significant improvement in pelvic floor muscle endurance after transurethral prostatectomy, but clinically relevant storage or voiding improvements did not occur (Tibaek et al., 2007). A Behavioural programme to prevent incontinence in community-dwelling older women resulted in increased awareness of bladder control and pelvic floor muscle training and demonstrated changes in pelvic muscle function and voiding interval (Sampselle et al., 2005).

Tertiary prevention

In a Cochrane review, Hay-Smith et al. (2002) found no studies of the elderly available for inclusion, and concluded that there is little evidence for tertiary prevention. However, the Cochrane review of 2010 changed emphasis in that it compared pelvic floor therapy regimens in terms of efficacy in treatment. The conclusions of this review were that pelvic floor muscle therapy was effective versus no treatment, a placebo drug or an inactive control treatment for women with either stress, urgency or mixed urinary incontinence and that there appeared to be no diminution of effect with increasing age. However, the studied samples were of community-dwelling women and there were no reports stratified according to age (Hay-Smith et al., 2012). There are no studies that included frail older persons and, as such, the principles of the Fifth International Consultation on Incontinence in the treatment of frail older people should apply.

EVIDENCE FOR EFFECT OF PFMT IN OLDER PERSONS

Although pelvic floor muscle rehabilitation has not been studied extensively in frail older persons, age and frailty alone should not preclude their use in appropriate patients with sufficient cognition to participate (Perrin et al., 2005; Hagglund, 2010). Similarly, a combined behavioural modification programme involving pelvic floor muscle exercises, bladder training and information about lifestyle modifications that prevented UI in community-dwelling older women aged between 55 and 80 years (Diokno et al., 2004), could be effective in some frail older women, but we located no studies that reported on promotion or maintenance of urinary continence in frail older adults. Likewise, a combined Behavioural modification programme involving pelvic floor muscle exercises, bladder training and information about lifestyle modifications that prevented UI in community-dwelling older women aged between 55 and 80 years could be effective in some frail older women (Kim et al., 2007). Additionally, few outcome measures have been validated in the elderly population (Fonda et al., 1998). A satisfactory outcome may be achieved using individualized treatment goals, and broad principles defining success were first defined by Fonda. For some ‘independent continence’ may be the desired outcome, but ‘dependent continence’ (dry with the assistance or reminders of a caregiver) or ‘social continence’ (dry with the use of appropriate aids and devices) can be achieved with suitable management.

The International Continence Society (ICS) Standardization Committee recommends that outcome measures specific to frail elderly people should be described in the same categories as for all adults:

1. patient observations and symptoms (e.g. patient and caregiver report of symptom response);
2. documentation of the symptoms (e.g. bladder diaries, wet checks, pad-weight tests);
3. anatomical and functional measures (e.g. urodynamics, post-void residual, PFM strength, timed up and go [TUG] test) (Podsiadlo and Richardson, 1991);
4. quality of life (e.g. condition-specific and generic measurement, validated for elderly populations);
5. socioeconomic measures (e.g. cost, cost–benefit, cost-effectiveness).

The Standardization Committee commented that while PFM training ‘may be of value in the management of incontinence in the frail elderly … there are no valid data on the measurement of PFM strength before and after treatment as a useful outcome measure for frail older patients’ (Fonda et al., 1998). Despite considerable effort on the part of physical therapists (PThs) engaged in validation and reliability studies of their muscle measurement instruments, there has still been little, if any, work in frail older persons (Bo and Sherburn, 2005).

There is no direct evidence for the effectiveness of PFMT in elderly women. When available data regarding conservative management in adults are stratified by age, they do not take into account age associated comorbidity. Therefore, it is not known whether age or comorbidities affect treatment outcome, or whether the benefits of different interventions are applicable to older people. To investigate whether age was a factor in being able to successfully achieve continence after a PFMT programme, Truijen et al. (2001) performed a retrospective analysis of 104 women (mean 55 years, 28–79 years) who had achieved continence after PFMT and compared them to those who had
Specific treatments

Functional activity training

Progressive resistance training has been shown to be effective in improving strength and functional mobility in older populations. Many exercise programmes for the elderly focus on flexibility and light aerobic exercise with low-dose resistance training, despite growing evidence for progressive resistance training being effective (Fiatarone et al., 1994).

Studies that have examined the effects of mobility alone on continence outcomes have been performed in both community dwelling and frail older residents of care homes. An RCT comparing a flexibility programme and a progressive resistance programme of 10 weeks’ duration, both groups consisting of a 1 hour, twice-weekly class (n = 40, mean age 68 years), found, at the end of the programme, a significant difference between groups in lower and upper limb muscle strength and gait and balance in favour of the progressive resistance training group (Barrett and Smerdely, 2002). There is a strong association between functional improvement and reduced urinary incontinence (Jenkins and Fultz, 2005).

In a frail nursing home population (n = 190, mean age 88 years (Schnelle et al., 2002) showed that even a low-intensity functional exercise (rather than PFM exercise) and prompted voiding programme was successful in improving mobility endurance, physical activity, limb strength and decreasing leakage episodes compared to those who received normal nursing home care. Subjects were guided, with minimal assistance, through repetitions of sit-to-stand, arm curls and/or arm raises, and walking or wheeling their chairs. Their exercise target goals were reset weekly. Despite the activity dosage in this study being a low intensity, good adherence was maintained by the intervention being instituted with prompted toileting and fluid intake at 2-hourly intervals during the daytime for 8 months.

More recently, a randomized controlled trial including 98 care home residents allocated to either a 3-month training programme (n = 48) or a control group (n = 50). The programme accommodated physical activity and ADL training, and the control group received ‘usual care’. The main outcome measure was UI as measured by a 24-hour pad-weight test. Only 68 participants were included in the analysis, 35 in the intervention group and 33 in the control group of average age 84 years. There was a statistically significant between-group change in urinary leakage, favouring the intervention group. Leakage increased in the control group residents (Vinsnes et al., 2012). However, in a Dutch study that compared a weekly group training session and homework exercises for 6 months versus care ‘as usual’, no between-group differences in participants with UI (intervention – 40%; control – 28%) and in frequency of incontinence episodes (intervention – 51%; control – 42%) were found (Tak et al., 2012).

PFMT alone or within a ‘package’ of treatment

There are few data that support the use of PFMT in frail older persons (McDowell et al., 1999). The majority of study results support the use of PFMT in the community-dwelling elderly population, and are incorporated in current best practice guidelines. Whether these results can be extrapolated to those with frailty is unknown, but there is perhaps no reason to suspect that intervention will have some benefit, provided that PFMT is based upon sound principles of exercise training. The presence of significant cognitive impairment in the frail elderly will, however, limit the applicability of PFMT given the need to learn and adhere to the prescribed exercise.

In the prospective RCT by McDowell et al. (1999), a crossover design of homebound elderly with high levels of comorbidities (n = 105, mean age 77 years), the authors found a statistically and clinically significant decrease in both urge and stress accidents (74% reduction) as recorded on bladder diaries immediately after 8 weeks of PFMT with biofeedback. Exercise adherence was the best predictor of success. These results, however, are short term due to the crossover design of the study, and are limited by the lack of intention-to-treat analysis and a 19% dropout rate.

Weinberger et al. (1999), however, reported that elderly incontinent women (mean age 76±8 years) derive long-term clinical benefit from non-surgical incontinence therapy (mailed questionnaire follow-up at 21±8 months post intervention), and that the overall likelihood of improvement was greatest in participants with the most severe incontinence at baseline. They included PFMT with or without biofeedback, bladder training, education and lifestyle management, oestrogen replacement, functional electrical stimulation and pharmacological therapy in their ‘package’ of interventions. At follow-up, participants reported that PFMT, delayed voiding and caffeine restriction were the most effective interventions within the ‘package’. The series of randomized controlled and crossover trials from Kim et al reported on the efficacy of a multicomponent intervention comprising strength training of the thigh and abdominal muscles performed between PFM exercises, including chair exercises, weight-bearing exercises and ball exercises in stress, mixed and urgency incontinence in community-dwelling older Japanese women.
Evidence-Based Physical Therapy for the Pelvic Floor (Kim et al., 2007, 2011b). In the 2007 study of 70 women of mean age 76 years, a 3-month initial programme was maintained with monthly sessions and followed up for a year. In the intervention group, maximum walking speed and adductor muscle strength increased significantly after the intervention; there were no significant changes in the control group. After 3 months of exercise, 54.5% of the intervention group and 9.4% of the control group reported being continent. These women had also significantly increased their walking speed and had lost weight. The 2011 study (Kim et al., 2011b) included 127 women with either stress, urgency or mixed UI. There were significant differences in changes of functional fitness and incontinence variables between the intervention and control groups. The multidimensional exercise treatment was significantly effective in decreasing all three types of urinary incontinence. However, the effects of the exercise treatment were greater on SUI than on UUI or MUI. At 7-month follow-up, the UUI and MUI groups statistically significantly deteriorated from their post-intervention state. The cure rate of urine leakage after the follow-up was significantly associated with compliance (OR = 1.13, 95% CI = 1.02–1.29) and reduction in body mass (OR = 0.78, 95% CI = 0.60–0.96). The most recent study reported on the additional effect of a heat and steam generating sheet (HSGS) worn on the lower backs of participating women (Kim et al., 2011a). In this study, 147 community-dwelling women aged 70 years and older with stress, urge and mixed UI were randomly assigned to exercise+HSGS (n = 37), exercise only (n = 37), HSGS only (n = 37) or an education group (n = 36). Exercise+HSGS and exercise groups received exercise training twice a week for 3 months. The HSGS group used one sheet per day continuously for 3 months. The intervention groups showed significant improvements in muscle strength and walking speed compared to the education group. Exercise and HSGS showed urine loss cure rates of 54.1%, exercise 34.3% and HSGS 21.6% after treatment; whereas, the education group (2.9%) showed no significant improvement (X² = 21.89, p < 0.001). Combining the HSGS to the exercise intervention showed a 61.5% cure rate for SUI, 50.0% UUI and 40.0% MUI.

A retrospective chart review of women ≥60 years old who underwent PFM for urinary incontinence examined the long-term efficacy of PT-delivered exercises 1–5 years after the therapy. Eighty-nine older women (mean age 70 years; range 60–81) were treated and 40 were followed up to 5 years. At 5 years of follow-up, 27.5% had improved, 57.5% remained stable and 15% had deteriorated compared with their post-treatment continence status. Twenty-nine patients (72.5%) were continuing their PFM exercises, and 42.5% were performing the exercises daily. Improvement was more common in those who remained actively exercising (Simard and Tu, 2010).

In 1990 an RCT comparing PFMT with or without EMG biofeedback to a control group for 8 weeks with weekly visits for evaluation of progress or biofeedback training in a population of community-dwelling older women with SUI (n = 118, mean 62 years) was assessed. There was a significant decrease in the number of leakage episodes for both intervention groups compared to the control group (F[2,118] = 15.60, p = 0.001), with the addition of EMG biofeedback significantly improving EMG readings for ‘fast’ contractions but not for those held for 3 seconds.

Wells et al. (1991) compared PFMT to an α-adrenergic agonist in an RCT of 157 women between 55 and 90 years (mean 66 years). The exercise group performed daily muscle awareness, strength and functional protocol for 6 months (drop-outs 34%) while the pharmacological group took the medication for up to 4 weeks (drop-outs 15%). Muscle strength was measured on a five-point scale by digital vaginal testing and by intravaginal EMG by non-blinded assessors. Both pharmacology and PFMT improved incontinence similarly, but PFMT strength was found to be significantly better for the exercise group. The authors suggest that those participants who performed higher levels of exercise (>80 contractions/day) produced better, though not significant, results on continence measures, and that further investigation into exercise adherence was required.

Bladder training and behavioural techniques

There is evidence for the efficacy of behavioural and bladder training in older persons (Roe et al., 2007a, 2007b) and because behavioural interventions have no side-effects, they have been the mainstay of UI treatment in the elderly and the frail elderly.

Interventions involving PFMT in community-dwelling elderly include (Wyman et al., 1998) a comparison of bladder training, PFMT and a combination ‘package’ in 204 older, but not frail, women (mean age 61 ±10 years) who had stress, urgency or mixed incontinence. The participants undertook 12 weeks of intervention with a follow-up 3 months later. At 3-month follow-up, no differences in outcomes between groups were observed. The authors concluded that specific treatment might not be as important as having a structured intervention programme with frequent patient contact, or that as PFMT was a common intervention in all treatment groups, all groups improved equally because of the PFMT component. However, the training intensity of the PFMT in this study was not high enough to cause a change in muscle strength. In a study examining the effects of a combined bladder retraining and PFMT regimen on UI in older women living in a rest home, 25 women of mean age 78 underwent 6–8 weeks’ treatment and the results versus the usual care group evaluated at 8 weeks and 6 months post intervention. Evaluations at 8 weeks and 6 months showed urinary incontinence with urgency, frequency and nocturia complaints statistically significantly decreased in the treatment group compared to the control group.
A significant increase in pelvic floor strength was observed in the treatment group compared to the control group. The frequency or intensity of the exercise regimen was not described in detail, unlike the bladder retraining programme (Aslan et al., 2008).

Burgio et al. (1998) reported bladder training to be more effective than anticholinergic medication or placebo in an RCT of 198 women aged 68±8 years with either UI or MUI. After 8 weeks of intervention, behavioural training resulted in an 81% reduction in the number of incontinent episodes per week, compared to 69% in the drug treatment group and 40% in the placebo group. The bladder training protocol in this study included PFMT three times daily, the ‘knack’, as well as behavioural training.

Behavioural interventions, also called voiding programmes, used predominantly in frail adults, all of which require active caregiver participation, include:

- **Prompted voiding**, which involves prompts to toilet with social approval, which is designed to increase patient requests for toileting and self-initiated toileting, and decrease the number of UI episodes. It was first used in the 1980s for incontinent nursing home residents (Palmer, 2005).

- **Habit retraining**, which requires the identification of the incontinent person’s individual toileting pattern, including UI episodes, usually by means of a bladder diary. A toileting schedule is then devised to pre-empt UI episodes. There is no attempt in habit retraining to alter an individual’s voiding pattern (Palmer, 2004).

- **Timed voiding** involves toileting an individual at fixed intervals, such as every 3 hours. This is considered a passive toileting programme; no attempts are made at patient education or reinforcement of behaviours, or to re-establish voiding patterns (Ostaszkiewicz et al., 2005). Other terms used to describe timed voiding are scheduled toileting, routine toileting and fixed toileting.

All these interventions might be combined with PFMT. A systematic literature review aimed to identify conservative interventions for reducing urinary incontinence (UI) in non-institutionalized frail older adults identified seven studies with 683 participants and concluded that multicomponent behavioural interventions, which include pelvic floor muscle exercises and bladder training, had the strongest evidence for reducing UI (Talley et al., 2011).

Randomized trials in nursing home settings have overwhelmingly depended on research staff to supervise or conduct the interventions. Studies repeatedly show that, once a trial ends, the staff rarely maintain the intervention at the same level, if at all. Many of the published interventions are of such intensity or frequency that they are unsustainable given locally available resources. The frequency of the intervention varied across studies as well, with toileting conducted every 2 hours over 12-hour, 14-hour and 24-hour schedules (Palmer, 2005). There is still much work to be done in assessing the minimum dose of intervention required to achieve and maintain a benefit in the institutionalized or home-bounded frail older person as few intervention studies have been conducted with incontinent hospitalized and home-bounded frail elders. Limitations in many studies include: small samples with low power to detect significant differences; variable terminology and operational definitions making comparisons across studies difficult. The majority of data are limited to women, especially in nursing home trials. Whilst some data have accumulated in the last decade there is still much to be done in treatment trials for frail older people.

### CLINICAL RECOMMENDATIONS

An active case finding approach is recommended for urinary incontinence in frail older people. Acute-onset incontinence should be assessed and treated immediately. The DIPPER mnemonic can be a useful reminder for the potential reversible causes of recent onset incontinence in older people. Refer on to appropriate medical personnel for this.

No patient should receive physical therapy treatment until these causes of incontinence have been addressed. Follow the appropriate clinical guidelines when assessing a patient in an aged-care facility.

### Physical therapy assessment

- Take a complete history to determine causative factors and their possible interaction. This may require information to be gathered from caregivers as well as the patient, regarding cognition, musculoskeletal problems, activities of daily living (ADL), pain and neurological symptoms and including bowel function.

- Complete a subjective and objective continence and pelvic floor assessment (including per vaginum examination where appropriate) following the guidelines in Chapter 6.

- Undertake appropriate assessment measures that can also be used as outcome measures, if possible including a bladder diary, PFM strength measure(s), functional mobility tests such as timed-up-and-go (TUG) test, and quality of life measures.

### Physical therapy treatment/management

- Institute general balance exercises, and lower limb strength training (sit to stand, walking and stair-climbing training) to address functional and mobility impairment. Prescribe, apply and train in the use of appropriate gait aids or other assistive devices such as
PFMT with cognitively intact patients, specifically a progressive resistance protocol and functional use of PFM during activities (‘the Knack’). If appropriate, include electrical stimulation to assist a strengthening programme where there is marked weakness or poor sensation of PFM.

• Institute bladder training where required, including education about ideal bladder habits and hygiene. Include in this the amount and type of fluid and fibre intake, redistribution of fluid and food intake during the day, relaxation and urgency suppression techniques, toilet positioning to facilitate complete emptying, timed or prompted voiding.

• Include PFM exercises and education about good bladder habits in all general exercise classes for older people, whether in community or residential aged-care facilities.

REFERENCES


Constantinou, C.E., Hvistendahl, G., Ryhnamer, A., et al., 2002. Determining the displacement of the pelvic floor and pelvic organs during voluntary contractions using magnetic resonance imaging in younger and older women. BJU Int. 90, 408–414.


Evidence-Based Physical Therapy for the Pelvic Floor


Kim, H., Yoshida, H., Suzuki, T., 2011a. Effects of exercise treatment with or without heat and steam generating
Evidence-Based Physical Therapy for the Pelvic Floor


Evidence-Based Physical Therapy for the Pelvic Floor


Evidence for pelvic floor physical therapy for neurological diseases

Marijke Van Kampen, Inge Geraerts

CHAPTER CONTENTS

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INTRODUCTION

Several neurological disease processes can cause changes in bladder and bowel function (Chancellor and Blaivas, 1995; Skeill et al., 2001). Bladder and bowel problems cause much anxiety and may reduce quality of life (Chancellor and Blaivas, 1995).

Treatment procedures of neurological patients with genitourinary and bowel problems are largely based on empirical evidence with a limited research base (Chancellor and Blaivas, 1995; Harari et al., 2004; Leboeuf and Gousse, 2004). An assessment of the patient’s physical, psychological, cognitive and emotional limitations may influence the treatment strategy. Although many options exist for therapy, a stepwise approach with initially non-invasive treatment is important considering the course of the disease (Chancellor and Blaivas, 1995; Leboeuf and Gousse, 2004). The role of pelvic floor physical therapy for bladder and bowel problems in specific neurological diseases is actually more and more investigated. Eleven randomized controlled studies of pelvic floor physical therapy for stroke (Tibaek et al., 2004, 2005, 2007) and MS patients (Vahtera et al., 1997; Prasad et al., 2003; McClurg et al., 2006, 2008, 2010; Khan et al., 2010; Lucio et al., 2010, 2011) with bladder and bowel problems are published. Other neurological pathologies like Parkinson’s disease, spina bifida, syringomyelia, peripheral neuropathies, Huntington’s disease, multiple system atrophy, dementia, spinal cord injuries, disc prolapse and tumours of the spinal cord might also be responsible for the development of a neurogenic bladder and bowel dysfunctions. No randomized controlled trials on evidence for pelvic floor physical therapy in those neurological diseases can be found, although physical therapy of the pelvic floor in Parkinson’s disease and spinal cord injury patients with partial lesion has been undertaken (Ishigooka et al., 1996; Vaughan et al., 2011).

This chapter is limited to treatment of stroke and MS patients with genitourinary and/or bowel problems.

STROKE

Definition

Stroke or cerebrovascular accident (CVA) is the clinical manifestation of ischaemia or infarction of brain tissue caused by arterial occlusion, intracerebral and subarachnoid haemorrhage or congenital malformation (Flisser and Blaivas, 2004).

Incidence and prevalence

Each year a typical health authority of 1000 men and women can expect two new stroke cases and four recurrent stroke patients and there will be approximately six survivors of stroke living in the community (Chancellor and Blaivas, 1995).

Urologic and bowel symptoms and urodynamic investigation

Urinary incontinence (UI) was reported in 32–83% in the early period after stroke. A review of prevalence of incontinence was given by Brittain et al. (1998). The natural history
of UI following stroke knows a gradual, spontaneous improvement from 19% at 3 months, 15% at 1 year to 10% at 2 years (Patel et al., 2001b). Jorgensen et al. (2005) found a prevalence of 17% UI among long-term survivors and 7% of the control subjects without stroke. Sakakibara et al. (1996) performed micturitional histories and urodynamic investigation in 72 stroke patients. A total of 53% of the patients performed micturitional histories and urodynamic investigation in 72 stroke patients. A total of 53% of the patients had one or more urinary symptoms within 3 months after stroke. Thirty-six per cent had nocturia followed by 29% with urgency incontinence and 25% with difficulty of voiding. Urodynamic investigation shows that initially after stroke the bladder is often areflexic (Flisser and Blaivas, 2004). Detrusor hyperreflexia and urgency incontinence generally follow. Sphincteric incontinence in the recovery phase is normally not a consequence of the stroke but is almost always a pre-morbid condition (Flisser and Blaivas, 2004).

Anal incontinence in stroke patients was reported in 23–40% of the cases on admission and 7–9% 6 months after stroke (Brocklehurst et al., 1985; Nakayama et al., 1997; Brittain et al., 1998; Krogh et al., 2001).

Initial incontinence is associated with age older than 75 years, visual field defect, dysphagia, motor weakness, severity of stroke, diabetes, hypertension and comorbidity of other diseases (Nakayama et al., 1997; Gross et al., 2000; Sze et al., 2000; Patel et al., 2001a). Furthermore, UI in the acute stage is a predictor of survival and closely associated with disability severity (Patel et al., 2001a). UI emerged as a risk factor for nursing home replacement (Patel et al., 2001a; Pettersen et al., 2002).

Pathophysiology

Not all incontinence after stroke is directly related to neurologic injury of the micturition pathways. Other mechanisms are general impairment, cognitive deficits and overflow incontinence unrelated to stroke (Flisser and Blaivas, 2004). The neurophysiologic explanation for detrusor areflexia in the initial phase after stroke is unknown. Detrusor-hyperreflexia was noted in lesions of the frontal lobe as well as the basal ganglia. Uninhibited sphincter relaxation is typical for frontal lobe lesions and detrusor sphincter dyssynergia are common in the basal ganglia lesions (Sakakibara et al., 1996). The location of the injury, the extent of the damage and the role of the affected area determine the precise urologic impact (Flisser and Blaivas, 2004). Rationale for physical therapy after stroke can be explained because patients have problems of urgency, stress and urge incontinence. The aim of physical therapy is to strengthen or to relax the pelvic floor muscles and to reduce frequency, urgency and nocturia.

Treatment: evidence for effect (prevention and treatment)

There has been little research into treatment of urinary and faecal incontinence and constipation in stroke survivors. Wikander et al. (1998) concluded that incontinence reduced significantly after a special multidisciplinary programme in comparison with a control group treated with a conventional rehabilitation programme. The special multidisciplinary programme contained physical training (dressing, transfer in the hospital and at home with attention to bladder and bowel management), social and cognitive interaction (memory training, problem solving, social interaction, expression and comprehension). Harari et al. (2004) concluded that a single clinical/educational nurse intervention in stroke patients effectively improved bowel dysfunctions up to 6 months later and bowel modifying lifestyle behaviours up to 12 months later.

The effect of pelvic floor muscle training on incontinence in stroke patients was evaluated in three randomized controlled studies (Tibaek et al., 2004, 2005, 2007). In fact, this is one RCT of 26 incontinent women reported in two publications because two different assessment tools were used. A third study investigated the 6-month long-term effect of 24 of these women on quality of life (QoL) (Table 12.1). The effect of pelvic floor exercises in women with UI after stroke was measured by QoL parameters (Tibaek et al., 2004) and by diary for the frequency of voiding, incontinence episodes and number of pads, 24-hour home pad-test and vaginal palpation of pelvic floor muscles (Tibaek et al., 2005). The intervention included group treatment during 12 weeks comprising of 12–24 standardized pelvic floor exercises. The control group followed the normal standard programme of stroke rehabilitation without specific treatment of UI.

In the first study, QoL measured with the Short Form 36 Health Survey Questionnaire (SF-36) and Incontinence Impact Questionnaire (IIQ-7) did not show significant difference between the two groups after 12 weeks (Tibaek et al., 2004).

In the second study, a significant improvement of frequency of voiding (p = 0.028), 24-hour home pad-test (p = 0.013) and endurance of pelvic floor muscles (p = 0.028) was demonstrated in the treatment group compared with the control group (Tibaek et al., 2005).

In the third study, QoL was measured with the Short Form 36 Health Survey Questionnaire (SF-36) and the Incontinence Impact Questionnaire (IIQ-7). A trend to a long-lasting effect regarding role limitations because of emotional problems (SF-36) and a tendency to a decreased impact of UI compared with the control group (IIQ-7) were found (Tibaek et al., 2007).

The methodological quality was 5 out of 10 on the Pedro scale; neither the patients nor the therapist or assessor were blind to the study (Table 12.2). Other limitations of the study are a small sample size, 12 women in each group. In the first study (Tibaek et al., 2004), the instruments to document the effect are not the optimal choice because the SF-36 gives an indication of general health and the IIQ turned out to be rather insensitive towards women with urge urinary incontinence. Remarkable is the fact that only 8% of the 339 stroke patients were potential candidates for pelvic floor physical therapy, mostly because of their neurological status (Tibaek et al., 2004, 2005).
### Table 12.1  Randomized controlled studies of physical therapy for bladder and bowel dysfunctions in neurogenic patients

<table>
<thead>
<tr>
<th>Stroke</th>
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<tbody>
<tr>
<td><strong>Author</strong></td>
<td>Tibaek et al., 2004</td>
</tr>
<tr>
<td><strong>Design</strong></td>
<td>2-arm RCT</td>
</tr>
<tr>
<td>Experimental group (E): PFMT</td>
<td></td>
</tr>
<tr>
<td>Control group (C): no treatment for incontinence</td>
<td></td>
</tr>
<tr>
<td><strong>n</strong></td>
<td>26 women (E = 14, C = 12), mean age 60 years (range: 56–74) with stroke</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>Short Form 36 Health Survey Questionnaire (SF-36) and Incontinence Impact Questionnaire (IIQ-7)</td>
</tr>
<tr>
<td><strong>Training protocol</strong></td>
<td>E: PFMT 6s contraction, 6s rest, 3s contraction, 3s rest, 30s contraction, 30s rest; every contraction: 4–8 times in different positions, group-treatment (6–8 patients) 1 h/week during 12 weeks as outpatient; individual: vaginal palpation 2–3 times over 12 weeks</td>
</tr>
<tr>
<td>Home exercises: 1–2 times daily</td>
<td></td>
</tr>
<tr>
<td><strong>Drop-out</strong></td>
<td>C: no treatment for UI but normal standard programme for rehabilitation 8%</td>
</tr>
<tr>
<td><strong>Results</strong></td>
<td>No significant difference between E and C group in SF-36 and IIQ-7</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Author</th>
<th>Tibaek et al., 2005</th>
</tr>
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<tbody>
<tr>
<td><strong>Design</strong></td>
<td>2-arm RCT</td>
</tr>
<tr>
<td>Experimental group (E): PFMT</td>
<td></td>
</tr>
<tr>
<td>Control group (C): no treatment for incontinence</td>
<td></td>
</tr>
<tr>
<td><strong>n</strong></td>
<td>26 women (E = 14, C = 12), mean age 60 years (range: 56–74) with stroke</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>Voiding diary, UI 24-h pad-test, number of pads, digital palpation of pelvic floor muscles</td>
</tr>
<tr>
<td><strong>Training protocol</strong></td>
<td>E: PFMT 6s contraction, 6s rest, 3s contraction, 3s rest, 30s contraction, 30s rest</td>
</tr>
<tr>
<td>Every contraction: 4–8 times in different positions; group-treatment (6–8 patients) 1 h/week during 12 weeks outpatient; individual: vaginal palpation 2–3 times in 12 weeks</td>
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<tr>
<td>Home exercises: 1–2 times daily</td>
<td></td>
</tr>
<tr>
<td><strong>Drop-out</strong></td>
<td>C: no treatment for UI but normal standard programme for rehabilitation 8%</td>
</tr>
<tr>
<td><strong>Results</strong></td>
<td>Significant difference between E and C group in frequency of voiding (p = 0.028), 24-h home pad-test (p = 0.013) and endurance of pelvic floor muscles (p = 0.028)</td>
</tr>
</tbody>
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<table>
<thead>
<tr>
<th>Author</th>
<th>Tibaek et al., 2007</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Design</strong></td>
<td>2-arm RCT</td>
</tr>
<tr>
<td>Experimental group (E): PFMT</td>
<td></td>
</tr>
<tr>
<td>Control group (C): no treatment for incontinence</td>
<td></td>
</tr>
<tr>
<td><strong>n</strong></td>
<td>24 women (E = 12, C = 12), mean age 60 years (range: 56–74) with stroke</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>Short Form 36 Health Survey Questionnaire (SF-36) and Incontinence Impact Questionnaire (IIQ7) &gt; 6 mth later</td>
</tr>
<tr>
<td><strong>Training protocol</strong></td>
<td>Identical to Tibaek et al., 2004 and 2005</td>
</tr>
<tr>
<td><strong>Drop-out</strong></td>
<td>8%</td>
</tr>
<tr>
<td><strong>Results</strong></td>
<td>No significant difference between E and C group in SF-36 and IIQ-7: only trend</td>
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<thead>
<tr>
<th>MS</th>
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<tbody>
<tr>
<td><strong>Author</strong></td>
<td>McClurg et al., 2006</td>
</tr>
<tr>
<td><strong>Design</strong></td>
<td>3-arm RCT</td>
</tr>
<tr>
<td>Control group (C): PFMT and advice</td>
<td></td>
</tr>
<tr>
<td>Experimental group (E): E1: + BF; E2: + BF + NMES</td>
<td></td>
</tr>
<tr>
<td><strong>n</strong></td>
<td>30 women with MS: C = 10, mean age 49 years; E1 = 10, mean age 52 years; E2 = 10, mean age 49 years (range: 33–67)</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>Leakages on voiding diary, 24-h pad-test, uroflowmetry, PF assessment (PERFECT scheme) and IIQ, UDI, KHQ, MSQoL. Assessment at week 0, 9, 16, 24</td>
</tr>
</tbody>
</table>

(Continued)
### Table 12.1 Randomized controlled studies of physical therapy for bladder and bowel dysfunctions in neurogenic patients—cont’d

| Training protocol | Treatment during 9 weeks  
|-------------------|-------------------------|
| **C** | advice + PFMT gradually, 5 times a day; E1: + BF, E2: + BF + NMES  
| ES = biphasic CC, two parameter settings | 250μs, 40Hz, 5s on, 10s off/450μs, 10Hz, 10s on, 3s off; duration from 5 to 30 minutes daily  
| **ES** | biphasic CC  
| Two parameter settings: 250μs, 40Hz, 5s on, 10s off/450μs, 10Hz, 10s on, 3s off; duration from 5 to 30 minutes daily  
| Drop-out | 2/30  
| Results | Significant difference in favour of E2 compared with C at 9, 16 and 24 weeks for leakages, 24-h pad-test, digital assessment in some parts of the questionnaires (p < 0.05)  
| Author | McClurg et al., 2008  

| Design | 2-arm RCT  
| **C** | PFMT, BF and placebo ES  
| **E** | PFMT, BF and ES  
| n | 74 women with MS: C = 37, mean age 52 years; E = 37, mean age 48 years (range: 27–72)  
| Diagnosis | Leakages on voiding diary, 24-h pad-test, uroflowmetry, PF assessment (PERFECT scheme), EMG, VAS, IIQ, UDI, IPSS  
| Training protocol | Treatment during 9 weeks  
| ES = biphasic CC  
| Two parameter settings: 250μs, 40Hz, 5s on, 10s off/450μs, 10Hz, 10s on, 3s off; duration from 5 to 30 minutes daily  
| Drop-out | 2/74  
| Results | Significant difference in favour of E compared with C for leakages, 24-h pad-test, digital assessment in some parts of the questionnaires  
| Author | McClurg et al., 2010  

| Design | 2-arm RCT  
| **C** | Advice  
| **E** | Advice + abdominal massage  
| n | 30 women with MS and constipation; E = mean age 52 years; C = 37, mean age 48 years (range: 27–72)  
| Diagnosis | CSS, neurogenic bowel dysfunction, bowel diary  
| Training protocol | Advice on bowel management  
| Abdominal massage daily for 4 weeks  
| Drop-out | 1/30  
| Results | Significant difference in favour of E compared with C for constipation symptoms (p = 0.003)  
| Author | Khan et al., 2010  

| Design | 2-arm RCT  
| **C** | control group: received the usual care  
| **E** | Experimental group (E): bladder rehabilitation programme  
| n | 74 women with MS; E = 40, mean age 49 years; C = 34, mean age 51 years (range: 29–65)  
| Diagnosis | Questionnaires: UDI-16, NDS, AUA, IIQ-7  
| Training protocol | E: multidisciplinary bladder rehabilitation programme during 1 year: or individualized inpatient (IP) or outpatient (OP) programme  
| IP = 3 h/day over 6 weeks, OP = 30 min 2–3×/week  
| Therapy: individual, assessment of bladder type, diary with strict fluid, PFME, timed voiding  
| C: waitlist group with usual care  
| Drop-out | 22%  
| Results | Significant difference between E and C group in all questionnaires (p < 0.01), improvement in bladder function, overactivity and QoL  
| Author | Khan et al., 2010 |
### Table 12.1  Randomized controlled studies of physical therapy for bladder and bowel dysfunctions in neurogenic patients—cont’d

<table>
<thead>
<tr>
<th>Author</th>
<th>Lucio et al., 2010</th>
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<tbody>
<tr>
<td><strong>Design</strong></td>
<td>2-arm RCT</td>
</tr>
<tr>
<td><strong>n</strong></td>
<td>27 women with MS: E = 13, mean age 36 years; C = 14, mean age 34.7 years (range: 20–49)</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>Urodynamics, 24-h pad-test, voiding diary, PF assessment (PERFECT scheme)</td>
</tr>
<tr>
<td><strong>Training protocol</strong></td>
<td>E: PFME with perineometer: 30 slow contractions, 3 min of fast contractions in supine position</td>
</tr>
<tr>
<td><strong>Drop-out</strong></td>
<td>Not reported</td>
</tr>
<tr>
<td><strong>Results</strong></td>
<td>Significant difference between E and C group in 24-h pad-test (p = 0.00), number of pads (p = 0.01), nocturia (p &lt; 0.00) and improvement of pelvic floor muscle power, endurance, resistance and fast contractions (p &lt; 0.00), no significant difference in urodynamics</td>
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<table>
<thead>
<tr>
<th>Author</th>
<th>Lucio et al., 2011</th>
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<tr>
<td><strong>Design</strong></td>
<td>2-arm RCT</td>
</tr>
<tr>
<td><strong>n</strong></td>
<td>35 women with MS: E = 18, mean age 36 years; C = 17, mean age 34.7 years (range: 20–49)</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>OAB questionnaire, medical outcomes study, ICIQ, Qualiveen questionnaire</td>
</tr>
<tr>
<td><strong>Training protocol</strong></td>
<td>E: PFME with perineometer: 30 slow contractions, 3 min of fast contractions in supine position</td>
</tr>
<tr>
<td><strong>Drop-out</strong></td>
<td>Not reported</td>
</tr>
<tr>
<td><strong>Results</strong></td>
<td>Significant difference between E and C group in all questionnaires</td>
</tr>
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<table>
<thead>
<tr>
<th>Author</th>
<th>Prasad et al., 2003</th>
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<tr>
<td><strong>Design</strong></td>
<td>3-arm RCT</td>
</tr>
<tr>
<td><strong>n</strong></td>
<td>18 women and 10 men with MS and post-void RV</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>Post-void RV</td>
</tr>
<tr>
<td><strong>Training protocol</strong></td>
<td>T1 = lower abdominal pressure (Crede’s manoeuvre) during 2 weeks</td>
</tr>
<tr>
<td><strong>Drop-out</strong></td>
<td>2/30 after randomization</td>
</tr>
<tr>
<td><strong>Results</strong></td>
<td>No significant difference between T1 and T2 group but results reach significance in favour of vibration (p = 0.059)</td>
</tr>
<tr>
<td><strong>Author</strong></td>
<td>Vahtera et al., 1997</td>
</tr>
<tr>
<td><strong>Design</strong></td>
<td>2-arm RCT</td>
</tr>
<tr>
<td><strong>n</strong></td>
<td>50 women and 30 men with MS (E = 40, C = 40); mean age 43 years (range: 25–68)</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>LUTS by self-administered questionnaire, muscle activity by surface EMG-Bf</td>
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</tbody>
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(Continued)
Table 12.1 Randomized controlled studies of physical therapy for bladder and bowel dysfunctions in neurogenic patients—cont’d

<table>
<thead>
<tr>
<th>Training protocol</th>
<th>Drop-out Results</th>
<th>Results</th>
</tr>
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<tbody>
<tr>
<td>E: PFMT: 3 s contraction, 3 s rest (10 times) 5 s contraction, 3 s rest (5 times) 15 s contraction, 30 s rest (5 times), others: 5 times in different positions</td>
<td>At 2 mth 2/40, at 6 mth 3/40 in the E group, not mentioned in control group</td>
<td>Significant difference between E and C group in LUTS (incontinence, nocturia, urge) p &lt;0.001, QoL (traveling, social shame and need of pads) muscle activity p &lt;0.01.</td>
</tr>
<tr>
<td>ES: interferential currents carrier frequency of 2000 Hz treatment frequency of 5–10 Hz, 10–50 Hz and 50 Hz, 10 min of each frequency, 3 min rest, 6 sessions during 21 days outpatient</td>
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<tr>
<td>BF: same PFMT after ES during 2 sessions; home exercises: 20 contractions 3–5 times/week during 6 mth in sitting and standing position</td>
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<td>C: no treatment</td>
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| KHQ, Kings Health Questionnaire; UDI, Urogenital Distress Inventory; CC, Constant Current; VAS, visual analogue score; IPSS, International Prostate Symptom Score; CSS, Constipation Scoring System; NDS, Neurological Disability Scale; AUA, American Urological Association; ICIQ, International Consultation on Incontinence Questionnaire; RV, residual volume. For other abbreviations, see text.

Table 12.2 PEDro quality score of RCTs in systematic review of pelvic floor physical therapy for neurological diseases

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+, criterion is clearly satisfied; –, criterion is not satisfied; ?, not clear if the criterion was satisfied. Total score is determined by counting the number of criteria that are satisfied, except that ‘eligibility criteria specified’ score is not used to generate the total score. Total scores are out of 10.
Conclusions and clinical recommendations

Clinical recommendations based on to-date evidence promote pelvic floor exercises to reduce incontinence with special attention to education and improvement of physical functions and social interaction but conclusions have to be drawn cautiously because of limited research (Tibæk et al., 2004, 2005, 2007).

MULTIPLE SCLEROSIS (MS)

Definition

MS is caused by inflammatory and demyelinating lesions in the white matter of the brain and the spinal cord, leading to a wide variety of neurological deficits (Chancellor and Blaivas, 1995).

Incidence and prevalence

The disease has an incidence of approximately 1 new case per 10,000 people every year, mostly manifesting between the ages of 20 and 50 years. MS is more common in women than in men by a ratio of 2:1. The prevalence is about 1 per 1000 in the United States and 2 per 1000 in North Europe. MS is less common in Orientals (Leboeuf and Gousse, 2004).

Urologic and bowel symptoms

Urologic symptoms in MS patients vary greatly from one study to another. Urgency, reported in 24–86% of the cases, and frequency, between 17% and 82% are the most frequent symptoms in the MS population (Mayo and Chetner, 1992; Leboeuf and Gousse, 2004). Urgency incontinence is reported in 19 to 72% while hesitancy and retention is found in 2 to 49% of MS patients (Leboeuf and Gousse, 2004; Mayo and Chetner, 1992). Constipation was found in 54% of the MS patients while 29% experienced faecal incontinence (Hennessey et al., 1999). All these symptoms are rated as the third most important problem in MS after spasticity and incoordination, limiting patients’ ability to work (Jawad et al., 1999). In men and women with MS not all the urologic dysfunctions can be presumed to be secondary to MS. A poor correlation has been found between subjective symptoms and objective urodynamic evaluations (Chancellor and Blaivas, 1995).

Pathophysiology

Damage to the innervation of the lower urinary tract mostly affects the sphincter and the detrusor. Three main types of pattern of urodynamic dysfunction are described:

- detrusor hyperreflexia without bladder outlet obstruction will occur in 26–99% of MS patients;
- detrusor hyperreflexia with detrusor-external sphincter dyssynergia (DESD) is documented in 23–52% of MS patients;
- MS patients with detrusor hypo- or areflexia are seen in 6–40% of the cases (Chancellor and Blaivas, 1995; Gallien et al., 1998; Leboeuf and Gousse, 2004).

Treatment: evidence for effect (prevention and treatment)

A few authors investigated physical therapy as a treatment modality in MS patients but without a control group (Van Poppel et al., 1985; Primus, 1992; Klarskov et al., 1994; De Ridder et al., 1999; Skeill et al., 2001) and noted a good subjective improvement in incontinent episodes and pad use after electrical stimulation or biofeedback training. Primus (1992) gave maximal vaginal electrical stimulation on 27 MS patients and found an initial efficacy of 85% but a decrease during follow up to 18% after 3 months. They concluded that long-term treatment is necessary to minimize symptoms in the MS population. De Ridder et al. (1999) offered a practical tool in the selection of MS patients to predict good prognosis: pelvic floor physical therapy should be restricted to patients with mild MS, without pelvic floor spasticity or detrusor sphincter dysynergia. They designed a digital scoring system for pelvic floor spasticity based on experience: 1 = spastic muscle unable to relax even after passive elongation; 2 = hypertonic muscle with temporary relaxation after elongation; 3 = active relaxation after active contraction.

This digital test has proven to be reliable between examiners (r = 0.90).

Bowel management in MS patients is empirical with a lack of evidence. Only one study investigated the effect of physical therapy but without a control group. Wiesel et al. (2000) offered biofeedback training as treatment in 13 patients with MS complaining of constipation or faecal incontinence. Five patients experienced a beneficial effect of biofeedback. Treatment was more likely to be successful in patients with limited disability and a non-progressive disease course.

Evidence-based medicine on MS and pelvic floor physical therapy

Evidence-based medicine on pelvic floor physical therapy for MS was researched in eight studies (see Table 12.1). Seven studies described the efficacy of physical therapy to reduce urgency, frequency, incontinence, nocturia and bladder emptying. Vahtera et al. (1997) investigated the effect of electrical
Evidence-Based Physical Therapy for the Pelvic Floor

stimulation and pelvic floor muscle exercises on lower urinary tract symptoms in multiple sclerosis patients (MS) with near-normal post-void residual volumes (<100 ml) and mild MS. The control group was not treated nor even tested on activity of the pelvic floor. Electrical stimulation (ES) with interferrional currents in combination with regular pelvic floor exercises improved significantly urgency, frequency, incontinence, nocturia and bladder emptying in comparison with a control group without treatment. The therapy significantly improved the maximal strength and endurance of the pelvic floor muscles. Compliance with the pelvic floor muscle exercises was 62.5% after 6 months; others trained irregularly. Three patients relapsed because of appearance of bladder symptoms or severe relapses in MS. Men may respond more rapidly to the therapy for incontinence. The symptoms of urgency were relatively easy to reduce in women. Lucio et al. (2010) randomized 27 female MS patients in a group that received PFMT with a vaginal perineometer and a sham group where the perineometer was introduced but no contractions were asked. They concluded that PFMT is an effective approach to treat lower urinary tract dysfunction (LUTD) in MS. In a second study of 35 MS patients (Lucio et al., 2011), they assessed QoL and found again results in favour of PFMT compared to sham therapy. Khan et al. (2010) assessed the effectiveness of a 6-week bladder rehabilitation programme in 40 persons with MS with a control waitlist group (n =34). A multifaceted, individualized rehabilitation programme reduced disability and improved QoL in MS patients compared with no intervention after 12 months of follow-up.

McClurg et al. (2006) compared three treatment modalities in 30 people with MS: PFMT and advice; PTMT, advice and EMG biofeedback (BF); and a third group adding neuromuscular electrical stimulation (NMES). They found a statistical significant difference between groups 1 and 3 for number of leaks and pad-test and a statistical benefit for group 2 compared with group 1 for pad-test. In a second study (McClurg et al., 2008), they found a statistical superior benefit by adding NMES to a programme of PFMT and EMG-BF on LUTD in 74 MS patients. Prasad et al. (2003) compared in 28 MS patients lower abdominal pressure with external bladder stimulation and no therapy to aid bladder emptying. All patients received all therapies during 2 weeks but were randomized in the sequence of therapy. Analysis was only done after 6 weeks for the whole group. The difference between abdominal pressure and vibration just failed to reach significance (p = 0.059) but both therapies were more effective than no treatment. There was no significant reduction in either the frequency of micturition or episodes of incontinence (see Table 12.1).

One study investigated the effect of abdominal massage against advice for the alleviation of constipation in 30 patients with MS and found a significant difference in favour of the massage.

Clinical recommendations

Based on evidence to date, electrical stimulation and pelvic floor exercises in MS patients decreases urgency, frequency, incontinence, nocturia and improves bladder emptying and pelvic muscle activity. Abdominal massage is effective to reduce constipation. Further research will establish the efficacy of these interventions.

CONCLUSION

Conclusions and clinical recommendations on the role of pelvic floor physical therapy for genitourinary and bowel problems in specific neurological diseases as stroke have to be taken with care because of the lack of good randomized controlled trials with a sufficient number of patients. A significant improvement of incontinence in stroke patients was demonstrated offering a 12-week PFME group treatment while QoL was the same for the experimental and control group.

For MS patients, a significant difference in lower urinary tract symptoms and pelvic floor muscle activity was found after electrical stimulation, biofeedback and pelvic floor muscle exercises or bladder training compared with a control group without specific treatment or with one modality of treatment. Abdominal massage has a positive effect on the symptoms of constipation.

The methodological quality of recent studies is high. For patients with other neurological disorders, efficacy of physical therapy is not yet investigated.

Research on efficacy and selection criteria for pelvic floor physical therapy is necessary to help neurological patients to prevent urological and bowel complications and to improve quality of life. Future research has to be undertaken not only on stroke and MS but also on other neurological diseases.

REFERENCES


Pelvic floor dysfunction, prevention and treatment in elite athletes

Kari Bø

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INTRODUCTION

Because of its location inside the pelvis, the pelvic floor muscles (PFM) are the only muscle group in the body capable of giving structural support for the pelvic organs and the pelvic openings (urethra, vagina and anus) (see Fig. 7.9, p. 231). Ultrasound and MRI studies have shown that the PFM are ‘stiffer’ and have a more cranial position in nulliparous compared to parous women (Peschers et al., 1996; Miller et al., 2001), and in continent versus incontinent women (Haderer et al., 2002).

Lack of an automatic, unconscious co-contraction or delayed or weak co-contraction of the PFM may lead to urinary and anal incontinence, prolapse of the anterior vaginal wall, posterior vaginal wall, vaginal apex, and uterus, or constipation, pain and sexual dysfunction (Bump and Norton, 1998). Although there are anecdotal reports of pelvic organ prolapse (POP) in young, nulliparous marathon runners and weightlifters, there are very few studies on pelvic organ prolapse in exercising women. In a study comparing nulliparous women before and after 6 weeks of summer military training it was found that women attending paratrooper training were significantly more likely to have stage II prolapse (RR = 2.72, 1.37 < RR < 5.40, p = 0.003). They were also significantly more likely to have worsening in their pelvic support regardless of initial POP stage (Larsen and Yavorek, 2007). In a study of women undergoing surgery for POP, 56 women agreed to admit to the ward one day before and participate in 1 hour of prescribed physical activities (walking for 45 min, including going up and down one flight of stairs, standing up from sitting 5 times, bending down to pick up something off the floor 10 times and jogging/stamping briskly on the spot for 1 minute), and then to remain mostly mobile for 4–6 hours after a POP-Q test. The POP-Q test was repeated the next morning with the same examiner. Seventy per cent maintained the same POP-Q stage, 4% had a lower stage and 26% increased the stage. None of these studies compared the results between exercisers and non-exercisers, and therefore further studies are warranted.

There is also sparse knowledge about anal incontinence during physical activity. One recent study included female students, age 18-40 years, from sport, physical therapy and nursing in Southern France (Vitton et al., 2011). They found a statistically significant higher prevalence of anal incontinence in those performing intensive sport, defined as training more than 8 hours per week compared to all other subjects (14.8% vs 4.9, respectively, p = 0.001). Anal incontinence was mainly represented by loss of flatus (84%).

So far, the focus within the sports literature has been on urinary incontinence (UI) during physical activity (Bø, 2004a). Well-established aetiological factors for UI include older age, obesity, gynaecological surgery, and pregnancy and vaginal childbirth (instrumental deliveries increase the risk). Other factors are less clear, such as strenuous work or exercise, constipation with straining on stool, chronic coughing, or other conditions that increase abdominal pressure chronically (Bump and Norton, 1998; Moore et al., 2013).
The aim of this chapter is to give a systematic review of the literature on UI in connection with participation in sport and fitness activities with a special emphasis on prevalence and treatment of female elite athletes.

**METHODS**

This is a systematic review of the literature covering incidence, prevalence, treatment and prevention of female UI in sport and fitness activities, with focus on stress urinary incontinence (SUI) (see Chapter 7). For epidemiological studies computerized search on ‘Sport’ and ‘Pub Med’ were done. Mesh words of urinary incontinence or pelvic organ prolapse combined with exercise, fitness, physical activity and sport were used. In addition, the chapter on epidemiology from the Fifth International Consultation on Incontinence (ICI) was consulted (Milsom et al., 2013). All studies found on prevalence and incidence were included in this chapter.

For treatment the same computerized search was conducted, together with a manual search of studies reported in the Cochrane Library (Dumoulin and Hay-Smith, 2010; Hay-Smith et al., 2011; Herderschee et al., 2011).

**PREVALENCE OF UI AND PARTICIPATION IN SPORT AND FITNESS ACTIVITIES**

Urinary incontinence is more common in women than in men and may affect women of all ages. Prevalence rates in the general population of women aged between 15 and 64 years vary between 32% and 64% (Milsom et al., 2013). The most common type of UI in women is SUI, followed by urge incontinence (UUI) and mixed incontinence (MUI). Urinary incontinence is often regarded as a problem affecting older, postmenopausal, multiparous women. However, several epidemiological studies have demonstrated that symptoms of SUI are frequent in populations of nulliparous young females (Bo et al., 1989b; Nygaard et al., 1990; Nygaard et al., 1994; Brown and Miller, 2001; Fozzatti et al., 2012).

UI is neither a life-threatening nor dangerous condition. However, it is socially embarrassing, and may cause withdrawal from social situations and reduced quality of life (Norton et al., 1988; Hunskar and Vinsnes, 1991). In the elderly it is a significant cause of disability and dependency. SUI implies that urine loss occurs during increases in abdominal pressure. If present, it is therefore likely that urine loss will occur during physical activity. Thus, sedentary women who are less exposed to physical exertion may not manifest SUI, though the underlying condition may be present. SUI has shown to lead to withdrawal from participation in sport and fitness activities (Bo et al., 1989a; Nygaard et al., 1990) and may be considered a barrier for lifelong participation in health and fitness activities in women (Brown and Miller, 2001). Cross-sectional studies in the general female population reporting that physically active women have less incontinence compared to their sedentary counterparts are difficult to interpret as this may be due to the fact that women with incontinence have stopped exercising (Brown et al., 1996; Van Oyen and Van Oyen, 2002; Hannestad et al., 2003; Østbye et al., 2004; Danforth et al., 2007; Kikuchi et al., 2007; Townsend et al., 2008; Zhu et al., 2008). Contradictory to these studies, Fozzatti et al. (2012) found that 24.6% of nulliparous women attending gyms compared to 14.3% in a group not attending gyms or doing high impact activities (except running) reported UI ($p=0.006$). This supports the results from a study on group fitness instructors, showing a prevalence of 26% with the same prevalence in those teaching yoga and Pilates (Bo et al., 2011). Although UI itself does not cause significant morbidity or mortality, it may lead to inactivity. A sedentary lifestyle is an independent risk factor for several diseases and conditions (e.g. high blood pressure, coronary heart disease, type II diabetes mellitus, obesity, colon and breast cancer, osteoporosis, depression and anxiety [Bouchard et al., 1993]).

**Prevalence of UI in female elite athletes**

An overview of published studies on prevalence of UI in elite athletes is shown in Table 13.1. There is a high prevalence of symptoms of both SUI and UUI in young nulliparous as well as parous elite athletes. Two studies compared the prevalence of incontinence in elite athletes with that of age-matched controls. Bø and Borgen (2001) found equal prevalences of overall SUI and UUI in both groups. However, the prevalence of leakage during physical activities was significantly higher in the elite athletes. Caylet et al. (2006) found a significantly higher prevalence in elite athletes compared to controls.

None of the listed studies characterized incontinence with urodynamic testing (simultaneous measurement of urethral and bladder pressures during increase in abdominal pressure), and it is therefore not possible to confirm whether the leakage represents SUI, UUI or MUI. However, in a study by Sandvik et al. (1993), questions used in a survey were validated against the diagnosis made by a gynaecologist after urodynamic evaluation. The diagnosis of SUI increased from 51% to 77%, MUI decreased from 39% to 11%, and UUI increased from 10% to 12% after urodynamic assessment. In another study of nulliparous physical education students, six of seven who underwent ambulatory urodynamic assessment showed evidence of urodynamic SUI (Bo et al., 1994).
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<th>Table 13.1  Studies of prevalence of urinary incontinence (UI) in elite female athletes</th>
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(Continued)
As seen from Table 13.1, the question on incontinence was posed in a general way with no time restrictions (e.g. leakage during past week or month) and was not always well described. Eliasson et al. (2002) is the only research group adding clinical measurements to the study. They measured urinary leakage in all elite trampolinists who reported the leakage to be a problem during trampoline training. The leakage was verified in all participants with a mean leakage of 28 g (range 9–56) in a 15-minute test on the trampoline. PFM function was measured in a subgroup of 10 women. They were all classified as having strong voluntary contractions by vaginal palpation.

Unlike the current ICS definition of urinary incontinence, the former ICS definition required that the leakage had to be considered a hygienic or social problem. The reported prevalence is reduced when this definition is used (Milsom et al., 2013). Nevertheless a high proportion of athletes report that the leakage is embarrassing, affects their sport performance, or is a social or hygienic problem (Caylet et al., 2006; Eliasson et al., 2008). Bø and Borgen (2001) reported that significantly more elite athletes with eating disorders had symptoms of both SUI and UUI, and Eliasson et al. (2002) showed that incontinent trampolinists were significantly older (16 vs 13 years), had been training longer and more frequently, and were less able to interrupt the urine flow stream by voluntarily contracting the PFM than the non-leaking group.

| Author          | Cross-sectional. Postal survey | 8 Danish sports clubs (including ballet) competing at national level (n=397). Mean age 22.8 years (range 14–51). 8.6% were parous | 73.7% | Do you experience urine loss while participating in your sport or in daily life? | 51.9% experienced urine loss during sport or in daily life; 43% while participating in their sport: gymnastics = 56%; ballet = 43%; aerobics = 40%; badminton = 31%; volleyball = 30%; athletics = 25%; handball = 21%; basketball = 17% |
| Author          | Cross-sectional, case control. Postal survey | 171 elite athletes from sport clubs in Southern France; 513 controls from in the same geographical area. Age 18–35 | Elite athletes (A): 55.6%; controls (C): 70% | Questions not reported | UI: 28% in A vs 9.8% in C (p=0.03). SUI most common type of UI. No significant difference between groups in SUI or UUI |

There are two hypotheses about the pelvic floor in elite athletes, tending in opposite directions.

**Hypothesis one: female athletes have strong PFM**

The rationale would be that any physical activity that increases abdominal pressure will lead to a simultaneous or precontraction of the PFM, and the muscles will be trained. Based on this assumption, general physical activity would prevent and treat SUI. However, women leak during physical activity, and they report worse leakage during high-impact activities. No sports involve a voluntary contraction of the PFM. Many women do not demonstrate an effective simultaneous or precontraction of the PFM during increased abdominal pressure (Bo et al., 2003). In nulliparous...
women this may be due to genetically weak connective tissue, location of the PFM at a lower, caudal level inside the pelvis, lower total number of muscle fibres (especially fast-twitch fibres), or untrained muscles in those leaking.

To date there is little knowledge about PFM function in elite athletes. Bø et al. (1994) measured PFM function in sport and physical education students with and without UI and did not find any difference in PFM strength. The increase in PFM pressure during a voluntary contraction was 16.2 cmH₂O (SD 8.7) in the group with SUI and 14.3 cmH₂O (SD 8.2) in the continent group. However, this study was limited by its small sample size, and no strong conclusion can be drawn. Statistically significant differences in PFM function and strength between continent and incontinent women have been shown in the adult population (Hahn et al., 1996; Gunnarsson, 2002; Mørkved et al., 2002). Bø (unpublished data) assessed PFM strength in four elite female power-lifters and compared them to 20 physical therapy students. Mean muscle strength during voluntary contraction in power-lifters was 22.6 cmH₂O (SD 9.1) and in the physical therapy students 19.3 cmH₂O (SD 6.8) (NS). Only one of the elite athletes in the above-mentioned ongoing study had exercised the PFM systematically. She reported to have trained her PFM regularly to increase low back stability and abdominal pressure during lifting. Her mean PFM strength was 36.2 cmH₂O. She was totally continent even when competing in World championships, but so were those who had not trained the PFM.

**Hypothesis two: female athletes may overload, stretch and weaken the pelvic floor**

Heavy lifting and strenuous work have been listed as risk factors for the development of pelvic organ prolapse and SUI (Bump and Norton, 1998; Milsom et al., 2013; Moore et al., 2013). Nichols and Milley (1978) suggested that the cardinal and uterosacral ligaments, PFM and the connective tissue of the perineum might be damaged chronically because of repeated increase in abdominal pressure due to hard manual work and chronic cough. To date, there are still few data to support the hypothesis. In a study of Danish nursing assistants it was found that they were 1.6 times more likely to undergo surgery for genital prolapse and incontinence than women in the general population (Jørgensen et al., 1994). However, the study did not control for parity. Hence, it is difficult to conclude whether heavy lifting is an aetiological factor. Figure 13.1 shows urinary leakage in a weightlifter.

In the United States Air Force female crew, 26% of women capable of sustaining up to 9 G reported UI (Fischer and Berg, 1999). However, more women had incontinence off duty than while flying and it was concluded that flying high-performance military aircraft did not affect the rate of incontinence. Davis and Goodman (1996) found that nine of 420 nulliparous female soldiers entering the airborne infantry training programme developed severe incontinence. Hence, most women were not negatively affected by this high impact activity. Figure 13.2 shows a parachute jumper in the landing phase.

Hay (1993) reported the maximum vertical ground reaction forces during different sport activities to be 3–4 times body weight for running, 5–12 times for jumping, 9 times for landing from front somersault, 14 times for landing after double-back somersault, 16 times during landing in long jumps, and 9 times body weight in the
lead foot in javelin throwing. Thus, one would anticipate that the pelvic floor of athletes needs to be much stronger than in the normal population to counteract these forces. Figure 13.3 shows a gymnast performing a jump. Several studies have found that coughing and Valsalva (as in defecation) increase intra-abdominal pressure to a significantly higher degree than different daily movements and exercises (Weir et al., 2006; Mouritsen et al., 2007; O’Dell et al., 2007). Many exercises, including abdominal exercise, did not increase the intra-abdominal pressure more than rising up from a chair (Weir et al., 2006; O’Dell et al., 2007). Borin et al. (2013) compared PFM strength in 10 handball, 10 volleyball and 10 basketball players and a non-exercising control group and found weaker muscles in the volleyball and basketball players compared with the controls. They also found that lower strength correlated with increased symptoms of UI.

Although the prevalence of UI is high, many athletes do not leak during strenuous activities and high increases in intra-abdominal pressure. However, from a theoretical understanding of functional anatomy and biomechanics, it is likely that heavy lifting and strenuous activity may promote these conditions in women already at risk (e.g. those with benign hypermobility joint syndrome). Physical activity may unmask and exaggerate the condition (Moore et al., 2013). There is a need for further studies to understand the influence of different exercises and general physical activity on the pelvic floor.

**Prevention**

There are no studies applying PFM training (PFMT) for primary prevention for SUI. Theoretically, one could argue that strengthening the PFM by specific training would have the potential to prevent SUI and POP. Strength training of the PFM has shown to increase the thickness of the muscles, reduce muscle length, reduce the levator hiatus area and lift the levator plate to a more cranial level inside the pelvis in women with POP (Brækken et al., 2010). If the pelvic floor possesses a certain ‘stiffness’ (Ashton-Miller et al., 2001; Haderer et al., 2002), it is likely that the muscles could counteract the increases in intra-abdominal pressures occurring during physical exertion.

**Preventive devices**

Devices that involve external urinary collection, intravaginal support of the bladder neck or blockage of urinary leakage by occlusion are available, and some have been shown to be effective in preventing leakage during physical activity. A vaginal tampon can be such a simple device. In a study by Glavind (1997), six women with SUI demonstrated total dryness when using a vaginal device during 30 minutes of aerobics. For smaller leakage, specially designed protecting pads can be used during training and competition.

**Treatment of SUI in elite athletes**

SUI can be treated with bladder training, PFMT with or without resistance devices, vaginal cones or biofeedback, electrical stimulation, drug therapy or surgery (Dumoulin and Hay-Smith, 2010; Hay-Smith et al., 2011; Herderschee...
One would assume that the elite athletes would respond in the same way to treatment as other women do. However, given the high impact on their pelvic floor they may need stronger PFMT than non-athletes.

To date there are methodological problems assessing bladder and urethral function during physical activity before and after treatment (James, 1978; Kulseng-Hanssen and Klevmark, 1988).

**Surgery**

Elite athletes are young and mostly nulliparous, and it is therefore recommended that PFMT should be the first choice of treatment, and always tried before surgery (Moore et al., 2013). The leakage in athletes seems to be related to strenuous high-impact activity, and elite athletes do not seem to have more UI than others later in life when the activity is reduced (Nygaard, 1997; Bø and Sundgot-Borgen, 2010). Therefore, surgery seems inappropriate in elite athletes who have incontinence only during exercise and sport.

**Bladder training**

Anecdotally, most elite athletes empty their bladder before practice and competition, which was also reported to be common in young nulliparous women attending gyms (Fozzatti et al., 2012). Therefore, it is unlikely that any of them would exercise with a high bladder volume. However, as in the rest of the population elite athletes may have a non-optimal toilet behaviour and the use of a frequency–volume chart may be an important first step to improve this.

**Oestrogen**

The role of oestrogen in incidence, prevalence, and treatment of SUI is controversial. Two meta-analyses of the effect have concluded that there is no change in urine loss after oestrogen replacement therapy (Andersson et al., 2013). Oestrogen given alone therefore does not seem to be an effective treatment for SUI. There is a higher prevalence of eating disorders in athletes compared to non-athletes, and these athletes may be low in oestrogen (Bø and Borgen, 2001). However, most amenorrhoeic elite athletes would be on oestrogen replacement therapy because of the risk of osteoporosis. Oestrogen may have adverse effects such as a higher risk of coronary heart disease and cancer.

**PFMT**

Based on systematic reviews and meta-analysis of RCTs it has been stated that conservative treatment should be first-line treatment for SUI (Moore et al., 2013). Cochrane reviews conclude that PFMT is an effective treatment for adult women with SUI or MUI, and consistently better than no treatment or placebo treatments (Dumoulin and Hay-Smith, 2010; Herderschee et al., 2011; Hay-Smith et al., 2011). Subjective cure and improvement rates after PFMT for SUI or MUI was reported in RCTs to be up to 70% (Moore et al., 2013). Cure rates, defined as ≤2 g of leakage on pad-tests, vary between 44% and 70% in SUI (Bø et al., 1999; Mørkved et al., 2002; Dumoulin et al., 2004). Adverse effects have only been reported in one study (Lagro-Janssen et al., 1992). One woman out of 54 reported pain with PFMT contractions; three had an uncomfortable feeling during exercise and two felt that they did not want to be continually occupied with the problem.

No RCTs on the effect of PFMT on UI have been conducted with elite athletes. However, Bø et al. (1999, 1990) and Mørkved et al. (2002) used tests involving high-impact exercise (running and jumping) before and after treatment, and showed that it is possible to cure or reduce urinary leakage during physical activity. Bø et al. (1989a) demonstrated that after specific strength training of the PFM, 17 of 23 women reported improvement during jumping and running, and 15 during lifting. Significant improvement was also obtained while dancing, hiking, during general group exercise, and in an overall score on ability to participate in different activities. Measured with a pad-test with standardized bladder volume during activities comprising running, jumping jacks and sit ups, there was a significant reduction in urine loss from mean 27 g (95% CI: 8.8, 45.1; range 0–168) to 7.1 g (95% CI: 0.8, 12.4; range 0–58.3), p < 0.01 (Bø et al., 1990). Mørkved et al. (2002) demonstrated a 67% cure rate in a test involving physical activity after individual biofeedback-assisted strength training of the PFM.

Sherman et al. (1997) randomized 39 female soldiers, mean age 28.5 years (SD 7.2), with exercise-induced UI to PFMT with or without biofeedback. All improved subjectively and showed normal readings on urodynamic assessment after treatment. Only eight subjects desired further treatment after 8 weeks of training.

Two small case series on elite athletes and sport students have been published (Rivalta et al., 2010; Da Roza, 2012). Rivalta et al. (2010) reported total relief of reported symptoms and no leakage on pad testing after 3 months of a combination of electrical stimulation, PFMT with biofeedback and vaginal cones. Da Roza et al. (2012) reported that seven nulliparous sport students significantly improved PFM strength and reduced ICQI score, frequency and amount of leakage after 8 weeks of training.

Elite athletes are accustomed to regular training and are highly motivated for exercise. Adding three sets of 8–12 close-to-maximum contractions of the PFM, 3–4 times a week (Pollock et al., 1998) to their regular strength-training programme does not seem to be a big task. However, there is no reason to believe that they are more able than the general population to perform a correct PFMT contraction.
Therefore, thorough instruction and assessment of ability to contract is mandatory. Because most elite athletes are nulliparous, there are no ruptures of ligaments, fascias, muscle fibres, or peripheral nerve damage. Therefore, it is expected that the effect would be equal or even better in this specific group of women. On the other hand, the impact and increase in abdominal pressure that has to be counteracted by the PFM in athletes performing high-impact activities is much higher than what is required in the sedentary population. The pelvic floor therefore probably needs to be much stronger in elite athletes.

There are two different theoretical rationales for the effect of PFMT (Bo, 2004b). Miller et al. (1998) found that a voluntary contraction of the PFM before and during cough reduced leakage by 98% and 73% during a medium and deep cough, respectively. Kegel (1948) first described the PFMT method in 1948 as ‘tightening’ of the pelvic floor. The rationale behind a strength-training regimen is to increase muscle tone and cross-sectional area of the muscles and increase stiffness of connective tissue, thereby lifting the pelvic floor into a higher pelvic position.

It is unlikely that continent elite athletes or participants in fitness activities think about the PFM or pre-contract them voluntarily. A contraction of the PFM most likely occurs automatically and simultaneously or even before the impact or abdominal pressure increase (Constantinou and Govan, 1981). It seems impossible to voluntarily precontract the PFM before and during every increase in abdominal pressure while participating in sport and leisure activities (Fig. 13.4). The aim of the training programme therefore would be to build up the PFM to a firm structural base where such contractions occur automatically.

Most likely very few, if any, athletes have learned about the PFM, and one could assume that none has tried to train them systematically. The potential for improvement in function and strength is therefore huge. PFMT has proved to be effective when conducted intensively and with a close follow-up in the general population (Moore et al., 2013). It is a functional and physiological non-invasive treatment with no known serious adverse effects and it is cost-effective compared to other treatment modalities. However, there is a need for high-quality RCTs to evaluate the effect of PFM strength training in female elite athletes.

CONCLUSION

Stress urinary incontinence may be a barrier to women’s participation in sport and fitness activities and may therefore be a threat to women’s health, self-esteem and well-being. Its prevalence among young, nulliparous elite athletes is high, with the highest prevalence found in those involved in high-impact activities such as gymnastics, track and field, and some ball games. There are no RCTs or reports on the effect of any treatment in female elite athletes. PFMT has been shown to be effective in RCTs, has no serious adverse effects, and has been recommended as first-line treatment in the general population. There is a need for more basic research on PFM function during physical activity and the effect of PFMT in female elite athletes.

CLINICAL RECOMMENDATIONS

- Suggest use of preventive devices or tampons to prevent leakage during physical activity.
- Follow general recommendations for PFMT for SUI (see Chapter 7).
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Evidence-Based Physical Therapy for the Pelvic Floor

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INTRODUCTION

Quality assurance and cost-effectiveness are, worldwide, issues of great concern in modern-day healthcare. The development of clinical practice guidelines (CPGs) is considered as a strategy to guarantee and improve the quality and efficiency of care.

A useful working definition of CPGs is derived from the Institute of Medicine (Field and Lohr, 1992). CPGs are defined as ‘systematically, on the basis of (best) evidence and consensus developed recommendations, drafted by experts, field-tested, and directed at performing diagnostic and therapeutic interventions in persons with definitive, suspected or health-threatening conditions, or directed at areas which have to do with good management and administration of the profession(al)’ (Field and Lohr, 1992; Hendriks et al., 1995; Hendriks et al., 1996, 1998b, 1998c; Grol et al., 2005).

Also, for both national and international physical therapy (physiotherapy) associations, the development and the implementation of CPGs constitute an important part of the quality of physical therapy care (policy) (Van der Wees et al., 2003). This interest is due to pressure from both society (policy-makers, healthcare managers, financiers and patients) on physical therapists (PTs) to ensure quality of care and to justify their position in the healthcare system (Hendriks et al., 1996; Hendriks et al., 2000b) as well as from PTs themselves in order to embed evidence-based practice into their profession (Van der Wees et al., 2003).

The European Region of WCPT (ER-WCPT) has developed a framework for the development of clinical guidelines (Van der Wees and Mead, 2004). According to this organization, in 2010, eight European countries had physical therapy-specific guideline programmes (Clinical Guideline Development Programs in the European Region of WCPT, 2010). In this chapter the ongoing process and development of the clinical practice guidelines (CPGs) of the Royal Dutch Society for Physical Therapy (KNGF) are described.

Methods for guideline development have been harmonized to a certain degree (Burgers et al., 2003; Van der Wees et al., 2007b), for which the AGREE (Appraisal of Guidelines, Research and Evaluation) instrument provides an important framework (Brouwers et al., 2010).

This instrument can be used to assess the quality of CPGs and helps guideline developers to structure and improve the process of guideline development (Table 14.1).

The Dutch programme for guideline development in physical therapy was critically reviewed and evaluated using the AGREE instrument (van der Wees et al., 2007a). Identification of weaknesses was subsequently used to update the programme.

The framework of the updated Dutch guideline programme is shown in Table 14.2.

Nowadays, Dutch CPG programmes comply with almost all AGREE criteria including piloting of the CPGs among target users.

CPGs can be considered as important state-of-the-art documents, which can guide professionals in their daily practice and make explicit what professionals can do in a certain situation or with a specific condition, and why they do it. CPGs should not be applied rigidly but are intended to be more flexible; however, in most cases, they can and should be followed. Yet, it is important to realize that CPGs only reflect the current state of knowledge,
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Table 14.1 The 23 key items of the AGREE instrument

The AGREE II consists of 23 key items organized within six domains followed by two global rating items (‘Overall Assessment’). Each domain captures a unique dimension of guideline quality.

Domain 1: Scope and purpose
1. The overall objective(s) of the guideline is (are) specifically described.
2. The health question(s) covered by the guideline is (are) specifically described.
3. The population (patients, public, etc.) to whom the guideline is meant to apply is specifically described.

Domain 2: Stakeholder involvement
1. The guideline development group includes individuals from all relevant professional groups.
2. The views and preferences of the target population (patients, public, etc.) have been sought.
3. The target users of the guideline are clearly defined.

Domain 3: Rigour of development
1. Systematic methods were used to search for evidence.
2. The criteria for selecting the evidence are clearly described.
3. The strengths and limitations of the body of evidence are clearly described.
4. The methods for formulating the recommendations are clearly described.
5. The health benefits, side-effects, and risks have been considered in formulating the recommendations.
6. There is an explicit link between the recommendations and the supporting evidence.
7. The guideline has been externally reviewed by experts prior to its publication.
8. A procedure for updating the guideline is provided.

Domain 4: Clarity of presentation
1. The recommendations are specific and unambiguous.
2. The different options for management of the condition or health issue are clearly presented.
3. Key recommendations are easily identifiable.

Domain 5: Applicability
1. The guideline describes facilitators and barriers to its application.
2. The guideline provides advice and/or tools on how the recommendations can be put into practice.
3. The potential resource implications of applying the recommendations have been considered.
4. The guideline presents monitoring and/or auditing criteria.

Domain 6: Editorial independence
1. The views of the funding body have not influenced the content of the guideline.
2. Competing interests of guideline development group members have been recorded and addressed.

at the time of publication, and expertise on effective and appropriate care with respect to (a) certain health problem(s). They are subject to a continuous process of integration of new views, based on inevitable changes in the state of scientific information and technology. New evidence is mostly gathered in systematic reviews. However, this kind of research is not a panacea for the problems associated with reviews of the literature. Due to its non-experimental nature it is prone to the flaws that apply to all non-experimental research (de Bie, 1996; Shaneyfelt and Centor, 2009).

So, readers should always keep these facts in mind while studying both systematic reviews and CPGs and must be critical in their appraisal of the information. Especially, statements about efficacy and efficiency of interventions, only based on clinical practice or experience or reflecting opinions of so-called experts in the field, might be biased and the real value discussed.

GUIDING PRINCIPLES IN THE DEVELOPMENT OF CPGs

Important guiding principles in the development of CPGs are (Hendriks et al., 1998c; Hendriks et al., 2000a):
- The subject matter is clearly delineated on the basis of a clear medical diagnosis of health problems and related conditions that can be addressed by physical therapy.
• CPGs should be structured according to the phases of the physical therapy process (Table 14.1 and Fig. 14.1) as laid down in CPGs by the professional organization (KNGF, 1993; Heerkens et al., 2003).

• A uniform professional language is used. Whenever indicated, use is made of available (international) classifications and accepted terminology, in particular the International Classification of Functioning, Disability and Health (WHO, 2001), but also the International Classification of Diseases (WHO, 1993), the Dutch Classification of Procedures (Heerkens et al., 1995) and Medical Terms for Health Professionals (Heerkens et al., 1998) (Fig. 14.1).

• Uniform and valid diagnostic and responsive outcome measurements are used.

• CPGs should be based on the best available clinical evidence, and on consensus between experts if no evidence is available.

• Clinical considerations have priority over cost-effectiveness.

• CPGs should be consistent with CPGs produced by other professions or groups of professions.

• CPGs should be based on integration and coherence of care. Physical therapy may be one of the possible interventions in the total care of a patient. It should be evident at which point and why physical therapy is appropriate.

• CPGs should be patient-orientated and in agreement with the policies of patient organizations. Individual patients also need to have a voice in determining care (NRV, 1998; Newman et al., 2002). Are the expectations and treatment goals of patients the same as those of PTs?

• The necessary expertise and knowledge required of PTs should be made clear.

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Table 14.2 Updated Dutch programme for guideline development in physical therapy

<table>
<thead>
<tr>
<th>Section</th>
<th>Description</th>
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<tbody>
<tr>
<td>1. Structure and organization</td>
<td>Central professional organization in collaboration with other institutes. Monodisciplinary development group (5–10 members). Small group (2–3) of employed staff within development group responsible for review of literature and actual writing of the guideline. Patients involved in external review group and focus groups.</td>
</tr>
<tr>
<td>2. Preparation/initiation</td>
<td>Special interest groups can propose topics using application form. Procedure is described for prioritizing topics. Guideline committee selects. KNGF board makes final decision. Literature orientation on the subject. Barriers and needs of physical therapists and patients are described in application form.</td>
</tr>
<tr>
<td>3. Development</td>
<td>Literature search using systematic strategy. Systematic review or meta-analysis if no (recent) review is available. Quality of studies assessed using different tools for diagnosis, intervention and systematic reviews. Hierarchy of the evidence described in four levels according to Dutch consensus. Grading of recommendations in four levels. Standardized formulation of recommendations according to grading. Outline of guideline divided in physical therapy diagnosis and treatment based on clinical reasoning process. Use of International Classification of Functioning (ICF) as nomenclature.</td>
</tr>
<tr>
<td>6. Evaluation and update</td>
<td>No later than 5 years after publication, decision about update, based on new evidence, results from pilot, professional developments and developments in guideline methodology. Additional (systematic) review of literature. Weighing of the evidence and recommendations adjusted or added if necessary.</td>
</tr>
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</table>
THE DEVELOPMENT PROCESS OF CPGs

The Dutch CPGs are based on the different stages of the physical therapy cure and care process (Hendriks et al., 2000a; Hendriks et al., 2000b; Bernards et al., 2011), the available clinical evidence, and expert consensus. Priority is given to a cost-effective approach and multidisciplinary consensus on diagnosis, intervention and secondary prevention. Recommendations are based on the results of new or recorded systematic reviews or meta-analysis.

Five groups contribute to the development of the CPGs (see Fig. 14.2):

1. The Royal Dutch Physical Therapy Society (KNGF) and four collaborating partners (the Dutch Institute of Allied Health Care (NPI), the Center of Evidence-Based Physiotherapy (CEBP), Department of Epidemiology, Maastricht University, and the Dutch Organization for Quality Assurance (CBO) which initiates and eventually endorses the CPGs.
2. The steering group which plans and coordinates the activities.
3. The task group which develops the CPGs.
4. A group of clinical experts in the subject matter of the CPGs which comments on the guidelines or parts of it during the development.
5. A randomly selected group of PTs who pilot-test the guidelines in clinical practice.

Figure 14.1 The physical therapy process, relevant data and necessary classifications. CMT, Classification of Medical Terms; CVBP, Classification of Interventions and Procedures (for the allied health professions); ICF, International Classification of Functioning, Disability and Health; ICPC, International Classification of Primary Care.
The work flow of the development process consists of: (1) formulation of clinical questions and patient-relevant outcomes, (2) systematic identification and summarizing of relevant evidence, (3) synthesis of the evidence by grading its quality and (4) formulation of recommendations for daily practice (van der Wees et al., 2007a). The different elements in guideline development are described in Table 14.1.

**Phases in development of CPGs**

There are four important phases in the development of clinical practice guidelines:

1. The preparatory phase.
2. The design phase, encompassing the draft guidelines and the authorization phase.
3. The implementation phase.
4. The evaluation and updating phase.

**Method of development of CPGs**

1. **The preparatory phase**

   This phase involves the selection of a topic based on certain criteria (Field and Lohr, 1992; Grimshaw et al., 1995a; van Everdingen et al., 2004; Grol et al., 2005; van der Wees et al., 2011) (Box 14.1).

   The scope and objectives of the guideline are defined. The clinical questions and patient-important outcomes are formulated.

2. **The design phase**

   This phase should guide the task group in the development of the guidelines and is, for educational reasons, based on the different stages of the physical therapy process (Fig. 14.1, Box 14.2). In the process of physical therapy practice a number of interrelated stages can be
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Box 14.1 Possible criteria to select a subject for the development of clinical practice guidelines

- Subject concerns a problem or controversy in healthcare for which healthcare providers are seeking a solution.
- It is anticipated that consensus about the procedure/intervention is possible.
- Healthcare providers are awaiting guidelines because they need a state-of-the-art document about a subject/topic.
- The subject is relevant because it has an impact on the costs of healthcare in terms of prevention of health problems or saving of costs.
- There is enough scientific evidence.
- There is a genuine expectation that the guidelines fit within existing norms, values and routines.
- The subject matter can be reasonably delineated.
- It is possible to collect data about the care.

Box 14.2 The different phases of the process of physical therapy practice

1. Examination of the referral data.
2. History-taking.
4. Formulating the physical therapist’s diagnosis and deciding whether or not physical therapy is indicated.
5. Formulating the treatment plan.
7. Evaluating the (changes in) a patient’s (functional) status and one’s own course of action.
8. Concluding the treatment period and reporting to the referring discipline.

Box 14.3 Guides for selecting articles that are most likely to provide valid results

**Therapy**
- Was the assignment of patients to treatments randomized?
- Were all of the patients who entered the trial properly accounted for and attributed at its conclusion?

**Diagnosis**
- Was there an independent, blind comparison with a reference standard?
- Did the patient sample include an appropriate spectrum of the sort of patients to whom the diagnostic test will be applied in clinical practice?

**Harm**
- Were there clearly identified comparison groups that were similar with respect to important determinations of outcome (other than the one of interest)?
- Were outcomes and exposures measured in the same way in groups being compared?

**Prognosis**
- Was there a representative patient sample at a well-defined point in the course of disease?
- Was the follow-up sufficiently long and complete?

The purpose of these rigorous literature reviews is to document the evidence to justify the recommendations and to minimize potential for any bias (van Tulder et al., 2003; van der Wees et al., 2011). See for an example on SUI, Berghmans et al. (1998c) or Hay-Smith et al. (2001).

Describing the quality of the evidence is important so that users of the guidelines can interpret the relative importance of the evidence. Each type of evidence (e.g. risk factors, diagnostic testing, prognosis, prevention, treatment) should be reviewed against a set of methodological criteria and systematically applied within study types. For evidence on treatments, high-quality randomized clinical trials (RCTs) are considered the strongest evidence, followed by cohort studies, case–control studies and non-analytic studies such as case reports or case series. Based on the quality of individual studies, an overall synthesis of the evidence will result in evidence statements expressed in levels of evidence. The PEDro scale was used in the updated SUI guidelines to study the internal validity of clinical trials, where high scores on the 11-item quality scale indicate a low risk of bias (Maher et al., 2004).

When scientific evidence from systematic reviews or primary trials is not available, the task group and the clinical experts formulate the CPGs on the basis of consensus. The task group first develops the diagnostic
part of the CPGs that may include an algorithm of the process of care and clinical decision-making, to formulate the management goals and an intervention plan. Twenty-five practising physical therapists with special interest and expertise in the problem area review this part of the CPGs.

Following the plan of activities, the task group continues with the therapeutic part of the guidelines that, if indicated and possible, should include the recommended intensity, frequency and duration of the intervention(s). The same group of therapists who were consulted in the previous phase review this part.

When both diagnostic and therapeutic parts of the guidelines are completed, the first draft is sent to 60 randomly selected specialized therapists for pilot testing and comments. Additional comments are obtained from clinical experts in relevant professions. Based on the comments and experiences of the PTs and the clinical experts, the draft is rewritten. The modified draft is then discussed by the ‘Authorization Committee’ (Fig. 14.2). Following approval of this committee, the guidelines are published in a scientific journal and introduced and implemented in the field.

The final product, as a result of the method of development, consists of four parts:

- The practice guidelines themselves.
- A summary or algorithm on an A4 laminated quick reference card.
- A scientific justification with references.
- A specific strategy and instruments for implementation of the guidelines (e.g. a knowledge check to test discrepancies between the actual and the recommended practice as stated in the guidelines).

During and after the course of treatment, the therapeutic process and results are evaluated. Data obtained during the care process are recorded according to the CPGs for documentation that have been developed to ensure systematic and uniform record-keeping (KNGF, 1993; Hendriks et al., 1998b, 1998c; Heerkens et al., 2003).

3. The implementation phase

This phase comprises the dissemination and specific strategy to implement the developed CPGs, according to the general method of implementation (Hendriks et al., 1998c; Hendriks et al., 2000a; Hendriks et al., 2000c).

4. The evaluation and updating phase

The effectiveness of the guidelines needs to be evaluated at the level of professionals and patients (see Fig. 14.2). The CPGs should be updated every 3–5 years after the guidelines are put into practice, or whenever new scientific insights make an update necessary.

**DISCUSSION**

The Dutch CPGs ‘Clinical practice guidelines for physiotherapists’ diagnosis and management of stress urinary incontinence in adult women’ ([www.cebp.nl [guidelines]]) (Berghmans et al., 1998a, 1998b) were the first to be developed for the diagnosis and management by physical therapists of SUI (in adult women). At the time of publication the guidelines provided up-to-date information on diagnosis, intervention, consultation and education on this specific health problem, information that the profession generally accepts as representing the state of the art (Hendriks et al., 1996; Hendriks et al., 1998a, 1998b; Hendriks et al., 2000a; van Everdingen et al., 2004; Grol et al., 2005).

However, subsequent developments led to improvements in the application of physical therapy in this group of patients and had an impact on the knowledge contained in these guidelines. The CPGs mentioned above have now been updated and extended also to adult men, and the revised version was published in 2011 (Bernards et al., 2011). The update of the guideline is in line with the structure and methods for guideline development, implementation and updating of KNGF guidelines which offers practical recommendations for a strategy to collect the relevant literature, including the selection of search terms, sources to be consulted and the period covered by the search (Hendriks et al., 1998a; Hendriks et al., 2000a; van der Wees et al., 2007a,b). The CPG is available in Dutch and English and can be downloaded at the website [www.fysioet-evidencebased.nl](http://www.fysioet-evidencebased.nl).

**Changing practice**

As stated before, an important strategy to improve the quality of physical therapy and to minimize undesirable variability in clinical practice is the development and implementation of evidence-based CPGs. In general, it can be concluded that the provision of explicit CPGs, supported by reinforcement strategies, will improve PTs performance and, in certain situations as a main goal, the patients’ health outcomes.

It is clear that just developing and disseminating of CPGs is not sufficient! Even well-established guidelines, like the example of the updated guidelines for SUI (Bernards et al., 2011), will not contribute to an improved quality unless they are embedded in effective implementation programmes (Grol and Grimshaw, 2003; Grol et al., 2005; Grol and Wensing, 2006). Implementation implies the introduction of a change or innovation such that it becomes a normal component of clinical practice for individual PTs and is no longer considered as new. In other words, a vital element of successful CPGs implementation is changing the individual PTs’ behavioural...
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process that has to take place in the constantly changing environment of evidence-based practice and life-long learning.

CPGs implementation is often laborious and has proved to be the weakest link in the whole process (Grol et al., 2005). Therefore, next to publication, dissemination and implementation of the CPGs, a set of (postgraduate) courses and tools was developed and published in order to facilitate and promote practical use of the guidelines in clinical practice (Van Ettekoven and Hendriks, 1998; Bekkering et al., 2005). Following a standard implementation procedure, the CPGs for the Physiotherapy of Patients with Stress Urinary Incontinence have been successfully implemented in The Netherlands.

A number of systematic reviews by Grimshaw et al. (Grimshaw et al., 1995a; Grimshaw et al., 1995b) described 91 studies and showed that the effect of introducing guidelines, especially in terms of their impact on clinical practice, is greater than had been previously assumed. Also on the basis of the studies of Grimshaw and Russell (1993), Grimshaw et al. (1995a), Grimshaw et al. (1995b), Davis and Taylor-Vaisey (1997) and more recently van der Wees et al. (2011), it can be concluded that thoroughly developed guidelines can alter clinical practice patterns and can lead to positive changes in patient outcomes. However, the studies also showed that the acceptance and use of guidelines are closely connected with the way in which they are developed and introduced. These findings were confirmed by the reviews of Grimshaw et al. (2001) and Grol and Grimshaw (2003).

The CPGs on SUI were developed by an independent multidisciplinary group of experts that represented all concerned professional organizations. The CPGs were based on the results of systematic reviews or meta-analyses (see for example on this topic Hendriks et al., 1998c; Hendriks et al., 2000c; Berghmans et al., 1998c; Hay-Smith et al., 2001; Hay-Smith and Dumoulin, 2006), because CPGs should provide clear clinical recommendations based on scientific and clinical evidence.

To optimize the development of CPGs, it is recommended that future users are involved as much as possible in the developmental process (Grol et al., 1994, 2005; Grimshaw et al., 2001; van Everdingen et al., 2004; van der Wees et al., 2008) and that PTs are able to exert a great deal of influence on guidelines implementation. The use of a top-down approach will engender resistance and, thus, have an adverse effect. However, adopting a bottom-up approach is often inefficient in terms of making the best use of the time invested and of avoiding ambiguity. In order to increase the acceptance and use of CPGs it might, therefore, be helpful to adapt centrally produced guidelines, with the help of a local team, to deal specifically with the local situation or to add a number of complementary agreements or criteria if necessary.

Although guidelines can immediately be put into practice, they may also be adapted to individual situations. Converting guidelines into a locally used protocol is possible and, at times, desirable. The conversion of centrally produced guidelines into a local protocol ensures that there is a local investment in, or ‘buying into’, the guidelines. This will speed up acceptance and, thus, implementation of the guidelines.

FUTURE

Evaluation of the effect of the implementation process of CPGs is needed to draw conclusions about how CPGs can be effectively and efficiently implemented in future. Only by evaluating carefully the effect of developing and implementing the centrally produced guidelines is it possible to identify specific barriers and impediments that need to be overcome in the successful implementation of guidelines, or to identify innovations.

Besides the Dutch CPGs for SUI, an evidence-based statement for anal incontinence has been published recently (Bols et al., 2013) and CPGs for urge urinary incontinence are currently being developed in The Netherlands and will be published in 2014.

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