A Textbook of
SPORTS AND EXERCISE PHYSIOLOGY
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Dedicated to

My parents, wife and son
Human physiology is the science of the mechanical, physical and biochemical functions of human in good health, their organs and the cells of which they are composed. The principal level of focus of physiology is at the level of organs and systems. These organs and systems work in a systematic manner to accomplish all the functions of human body. For example, the air we inhale from the atmosphere to our lungs and oxygen is carried through blood to the tissue where food particles are burned and produced energy is utilized for various activities performed, and the carbon dioxide produced along with energy are also carried out by blood to the lungs from where we exhale it out to the atmosphere. Not only but also in the resting condition, this phenomenon continues even during sleep when the individual is completely at rest physically as well as mentally. Exercise physiology is the study of the functions of human body during various acute and chronic exercise conditions. These effects are significant during both short and high-intensity exercises, as well as with prolonged strenuous exercises performed in endurance sports like marathons, ultramarathons, road bicycle racing, channel swimming, etc.

This book contains fifteen chapters, which represents the various aspects of exercise physiology with special reference given to sports and games. Historical perspective of exercise physiology is the first chapter, as it discusses how the sports physiology has evolved from physiology. The chapter mainly highlights how Harvard Fatigue Laboratory worked on physiology and the importance of studying the physiology of human movement with special interest in the effects of environmental stress. The chapter also depicts the emergence of Scandinavian influence along with contemporary exercise and sports physiology. Finally, it describes the role of exercise physiology in high performance sports.

Chapter 2 represents the basic units of life, i.e. the structure and functions of cell, tissue, organ and the various systems of human body. All the organ systems are described with suitable diagrams. All systems like cardiovascular, respiratory, muscular and endocrine system are represented in Chapter 3, 4, 5 and 6, respectively. Moreover, how cardiovascular and respiratory systems transport nutrients and respiratory gases to the active muscles and waste products away from the body. The emphasis is given to how these systems respond to the effect of maximal and submaximal exercises. These chapters also depict the effects of training with special reference to sports and games.

Chapter 7 represents the basic energy systems that provide the energy needed during rest and at different levels of exercise. Diet, nutrition and supplements for the sports persons are described in Chapter 8. The chapter deals with the use of food supplements to improve athletic performance and also the food for athletes during, before and after competition. Finally, the importance of water balance in the body during exercise in hot and humid conditions are discussed. In Chapters 9 and 10, the discussion is particularly based on three different groups’ of people, i.e. young children, aged and women athletes. The chapter starts with the process of growth and development and their effects on athletic performance. The changes that occur in physical performance due to progress of age and how physical activities can prolong the youthfulness are discussed. Finally, the gender difference and some special physiological concerns of female athletes have been elaborated in this chapter.

The impact of the external environment on physical performance is depicted in Chapter 11. It discusses how the body responds to heat and cold and the impact of low atmospheric pressure, i.e. experience at higher altitudes. The symptoms, causes and recovery from the fatigue are elaborated in Chapter 12. The attention is also paid on three important chapters of this book, which is related to the sports and exercises are listed in Chapters 13, 14 and 15. The process of identification of talent is one
of the most important aspects in modern sports. After identifying the talent, another important factor is grooming and finally to bring them up to the elite level. The process adopted by the Sports Authority of India for their talent hunt program is also discussed elaborately. The focus has been shifted from physiological functions to various anthropometric measurements. All the anthropometric measurements, equipments and the measurement techniques are elaborately discussed in Chapter 14. Finally, the abuse of drug, use of ergogenic aids, substances responsible for improvement of sports performance illegally and what are their consequences, are discussed in details in the concluding chapter of this book.

Exercise and sports physiology is a growing subject. This is an upcoming notion emerging in the field of human physiology. However, application of sports science to achieve excellence in sports is well established. The exercise physiology is introduced in the syllabus of many universities. But books in this area are scanty. However, this book is designed for the student readers, with the goal of making their learning easy and enjoyable. I am sure that the graduate and postgraduate students of physical education, human physiology, sports coaching, sports sciences, coaches, trainers, scientist, etc. will be immensely benefited by this book. The text of this book is written comprehensively but in simple sentences with many examples, tables and diagrams. Care is taken, while writing the text for its practical application by the coaches/trainers.

At the end of each chapter, a summary of the chapter is included which provides the insight of the whole text. Besides summary, at the end of all chapters, the review questions are included to allow the students to test their knowledge about the chapter’s contents. At the end of the book, a thorough index is listed for easy reference. Many of you will read this book only because it is a required text for a required course. But I hope that the information will entice you to continue to study in this relatively new and exciting area. This is not only for those who pursues their career as exercise or sports physiologist but also for anyone who wants to be physically active and lead healthy life.

Swapan Kumar Dey
I would like to extend my gratitude to a number of people for their generous help and assistance. First and the foremost, I would like to thank Sri ASV Prasad, former Executive Director of Sports Authority of India for his inspiration and valuable suggestion. I would further convey my thanks to Regional Director, Sports Authority of India, Eastern Center, Kolkata, West Bengal and Dr G Kishore, Director, Sports Authority of India, Southern Center, Bangaluru, Karnataka for their generous support and encouragement. I am grateful to my esteemed organization, the Sports Authority of India for giving me such an opportunity to work in this field.

I would also be thankful to Dr Parthasarathi Debray, Lecturer, Udaipur College, Tripura and Nabanita Debnath, Former Research Scholar, Department of Physiology, Sports Authority of India, Kolkata, West Bengal for their active cooperation and help to prepare this manuscript. Special thanks is also due to Dr TK Ghosh, Reader, Department of Physiology, City College, Kolkata, West Bengal for his valuable suggestions.

I have no words to express my sincere thanks to Sri Samir Kumar Dey for his generous encouragement to write this book. I thankfully acknowledge Sri Tuhin Subhra Neogi, Scientific Assistant and Triporna Banerjee, Department of Physiology, Sports Authority of India, Kolkata for their active cooperation and help to prepare this manuscript. I am also grateful to my colleagues of the faculty of Sports Sciences, Sports Authority of India, Kolkata, West Bengal for their continuous support.

And finally, I would like to thank my wife Sipra and son Sayan who have taken the entire burden while I am writing, rewriting, editing, and finally proofing this book. Their patience and support can never be compensated. This book would not have come into light without their tireless encouragement and sincere cooperation.
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Historical Perspective of Exercise Physiology

Brief History

The word ‘PHYSIOLOGY’ was first used by the Greeks during 600 BC to describe a philosophical inquiry into the nature of things. Use of this word, to refer to the vital activities of healthy humans, began in 16th century. The ideas of Aristotle and Galen were the first efforts in physiology. Modern physiology took off from the works of William Harvey (1628). Curiosity, medical necessity and economic interest stimulated research on the functional aspects of living things during the 19th century and created a wider definition of physiology. The efforts of Claude Bernard (1813–78, of France), J Muller, J von Liebig and C Ludwig (1801–95 of Germany) and Sir M Foster (of England) had produced the necessary insight for this wider definition. It was implied that concepts of physical sciences are to be used along with experimental methods to investigate the causes and mechanisms of the activities of all living things. As on today, the word ‘PHYSIOLOGY’ means the study of the structural and functional aspects of the living organisms. Or it may be define as Physiology is the study of body function. In physiology, we study how our organ systems, tissues and cells work and how their functions are integrated to regulate our internal environments. In sports, we are restricting the physiological study on human being only.

Although, the ancient Greeks made a fair start at studying the function of the human body, not until the 1500s were any truly significant contributions made to understanding both the structure and function of the human body. A land mark text by Andres Vesalius, entitled Fabrica Humani Corporis (structure of the human body), published in 1543, change the direction of future studies.

Most early attempts at explaining physiology were either incorrect or so vague that they could be considered only speculation. Attempts to explain how a muscle generates force, for example, were usually limited to a description of its change in size and shape.
during action because observations were limited to what could be seen with the eye. From such observations, Hieronymus Fabricius (1574) suggested that a muscle’s contractile power resided in its fibrous tendons, not in its “flesh”.

**Evolution of Exercise Physiology**

The first work on exercise physiology or sports physiology could be traced back to J Barcroft in 1914. He had made a publication on “The respiratory function of the blood”. Later the studies of AV Hill from Oxford University gave the modern look to the subject. In fact the concepts on energy metabolism developed by Hill are still valid. His historical publications in 1927 include “Living machinery” and “Muscular movement in man.” There was a person to introduce the concept of “Steady state of exercise”.

Exercise and sports physiology have evolved from anatomy and physiology and anatomy is the study of an organism’s structure, or morphology. From anatomy we learn the basic structure of various body parts and their interrelationships. Physiology is the study of body functions. As we have mentioned earlier in physiology, we study of how our organ systems, tissues and cells work and how their functions are integrated to regulate our internal environments.

Exercise physiology is the study how our bodies’ structures and functions are altered when we are exposed to acute and chronic bouts to exercise. Sports physiology further applies the concepts of exercise physiology to training the athletes and enhancing the athlete’s sports performance. Thus, sports physiology is derived from exercise physiology. Exercise physiology has unfolded the sequence of events that occur when the human body is trained beyond its ability to adapt and this information guide how to framed and evaluation of systematic training programs to reduce the under or over training and here the sports physiology has applied.

The Emergence of Exercise Physiology

Exercise physiology is relatively new into the world of science. Before the late 19th century, physiologists’ major goal was to gain information of clinical value. The body’s response to exercise received almost no attention. Although the value of regular physical activity was well known in the mid 1800s, the physiology of muscular activity gained little attention until the later part of that century. However, the first textbook on exercise physiology was published in 1889 by Fernand LaGrange entitled “Physiology of Bodily exercise”.

The early attempt to explain the bodies’ response to exercise was, in many ways, limited to a lot of rambling theory and little fact. Although some basic concepts of exercise biochemistry were emerging at that time, LaGrange was quick to admit that many details were still in the formative stages. For example, he stated that “... energy metabolism has become very complicated of late; we may say that it is some what perplexed, and that it is difficult to give in a few words a clear and concise summary of it.”

During the late 1800s, many theories were proposed to explain the source of energy for muscle contraction. Muscles were known to generate much heat during exercise, so some theories suggested that this heat was used directly or indirectly to cause muscle fibers to shorten. After the turn of the century, Walter Fletcher and Sir Frederick Gowland Hopkins observed a close
Historical Perspective of Exercise Physiology

A significant relationship between muscle action and lactate formation. This observation led to the realization that energy for muscle action is derived from the breakdown of muscle glycogen to lactic acid, though the details of this reaction remained obscure. Because the energy demands for muscle action are high, this tissue served as an ideal model to help unravel the synthesis of cellular metabolism. In 1921, Archibald (AV) Hill was awarded the Nobel Prize for his findings on energy metabolism.

Although much of Hill’s research was conducted with isolated frog muscle, he also conducted some of the first physiological studies on runners. Such studies were possible through the technical contributions of John Haldane, who develop the methods and equipment needed to measure oxygen use during exercise. These and other investigators provided the basic framework for our understanding of whole body energy production, which became the focus of considerable research during the middle of this century and is incorporated into computer-based systems used to measure oxygen uptake in exercise physiology laboratories today.

**The Harvard Fatigue Laboratory**

No scientific laboratory has had more impact on the field of exercise physiology than the Harvard Fatigue Laboratory (HFL), founded in 1927. Creation of this laboratory is credited to the insightful planning of world famous biochemist Lawrence J Henderson, who recognized the importance of studying the physiology of human movement with special interest in the effects of environmental stress.

Despite little experience in applied human physiology, Dill’s creative thinking and ability to surround himself with young, talented scientists created an environment that would lay the foundation for modern exercise and environment physiology. For example, HFL personnel examined the physiology of endurance exercise and described the physical requirements for success in events such as distance running.

The Harvard Fatigue Laboratory was an intellectual center that attracted young physiologist from many places. Scholars from 15 countries worked in the HFL between 1927 and its closure in 1947. Most of the physiologist went on to develop their own laboratories and to become noteworthy international figures in exercise physiology. Thus, the HFL planted seeds of intellect around the world that resulted in an explosion of knowledge and interest in this new field.

**The Scandinavian Influence**

Scandinavia is considered the birthplace of exercise physiology. Christensen and Hansen conducted several studies on nutrition and exercise performance while Åstrand examined human performance. Saltin later conducted lots of research and was a leader in muscle metabolism.

Early contacts between D.B. Dill and August Krogh, a Danish Nobel prize winner, led to the coming of three exceptional Danish physiologists to the Harvard fatigue laboratory in 1930s. Krogh encouraged Erik Hohwii-Christensen, Erling Asmussen, and Marius Nielsen to spend time at Harvard studying exercise in the heat and at high altitude. After returning to Scandinavia, each of them established a separate line of research. In 1941, Christensen moved to Stockholm to become the first physiology professor of the College of Physical Education. He introduced Per-Olof Astrand to the field of exercise Physiology. Astrand who conducted numerous studies related to physical fitness and endurance capacity during 1950s and 1960s. Astrand and Christensen were also the mentors of Saltin, one of today’s leading contributors to our understanding of muscle metabolism during exercise.

1. **Danish Influence**: In 1909, the University of Copenhagen endowed the equivalent of a chair in Anatomy, Physiology, and Theory of Gymnastics. The first Docent was Johannes Lindhard (1870–1947). He later teamed with August Krogh, (1874–1949), an eminent scientist, specialized in physiological chemistry and research instrument design and construction to conduct many of the now classic experiment in exercise physiology. Krogh and his wife Marie (1910) had proven through a series of ingenious decisive experiment that diffusion is the mechanism by which...
pulmonary gas exchange occurred—not by the secretion of O₂ from lung tissue into the blood during exercise and exposure to altitude. The three other Danish Physiologists—Asmusen (1907 – 1991), Christenson, and Nielsen conducted pioneering studies in exercise physiology. They published numerous research papers from 1930s to 1970s in this field.

2. **Swedish Influence**: Modern exercise physiology in Sweden can be treated to Per Henrik Ling (1776 – 1839), who in 1813 became the first Director of Stockholm’s Royal Central Institute of Gymnasia. His son, Hjalmar, also had a strong interest in medical gymnastics and physiology & anatomy, in part due to his attendance at lecture by physiologist Claude Bernad in Paris, in 1854. Per-Olf Åstrand is the most famous graduate of the college of Physical Education (1946); in 1952 he presented his thesis to the Karoliniska Institute of Medical School. He taught in the Department of Physiology in college of physical education from 1946 to 1977.

Two Swedish scientists currently at the Karoliniska Institute, Jonas Bergstrom and Erik Hultman, performed important experiment with the needle biopsy procedure (muscle fibre typing) that has provided a new vista from which to study exercise physiology.

3. **Norwegian and Finish Influence**: The new generation of exercise physiologists trained in the late 1940s who analyzed respiratory gases for CO₂ and O₂ in expired air. The method of analysis was developed in 1967 by Norwegian Scientist Per Scholander (1905 – 1980). The another prominent Norwegian researcher was Hermensen (1933 – 1984) from the Institute of Work Physiology whose many contribution are also the guidelines in today’s exercise physiology.

In Finland, Karvonen from the physiology department of the Institute of Occupational Health, Helsinki was best known for a method to predict optimal exercise training heart rate. He also conducted studies dealing with exercise performance and the role of exercise in longevity.

### Contemporary Exercise and Sports Physiology

Many advancement in Exercise physiology must be credited to improvements in technology. For example, in 1960s, development of electronic analyzers to measure respiratory gases made studying energy metabolism much easier and more productive than before. This technology and radio-telemetry used monitor heart rate and body temperature during exercise were developed as a result of the US space program. Until the late 1960s, most exercise physiology studies focused on the whole body’s response to exercise. The majority of investigations were measurements of such variables as oxygen uptake, heart rate, body
temperature and sweat rate. Cellular responses to
exercise received little attention.
Now that we have an understanding of the
historical basis for the discipline of exercise
physiology, from which sports physiology emerged,
we can explore the scope of exercise and sports
physiology.

**Scope of Exercise Physiology**

Exercise physiology is either an academic major
with Bachelor through Doctor of Philosophy
degrees in many colleges and universities across
the United States including some of the states of
India and other countries or an academic
concentration in exercise science with an academic
major in kinesiology, human performance, or one
of several other possible titles. While the academic
major in exercise physiology generally requires
more exercise physiology course work and hands-
on laboratory experiences than the academic
concentration, the significant number of variations
in academic programs has resulted in concern as
to what constitutes the academic core course work
for the emerging exercise physiology profession.
The variations in academic offerings are under
consideration by the (ASEP) particularly in United
States. American Society of Exercise Physiologists.
Accreditation Committee

Many of the exercise physiology courses
encompass classes that concern how exercise,
physical activity and a sedentary lifestyle each
influence the human body. A large component of
this knowledge base is derived from introductory
and advanced classes in exercise physiology.
However, additional courses such as sports
biomechanics, anatomy, kinesiology, exercise
testing, exercise prescription, cardiopulmonary
rehabilitation, biochemistry, electrocardiography,
laboratory procedures, body composition, motor
learning, research design, and statistics are integral
to the knowledge and skill competencies of the
profession of exercise physiology.

Based on an understanding of the information
provided above, exercise physiology is more than
a course topic, or a branch of physiology. Exercise
physiology is an emerging profession that has
application to the fitness, allied health, clinical
settings and athletic performance.

**What is an Exercise Physiologist?**

An exercise physiologist is a person who has
completed at least a bachelor's degree in exercise
physiology (science). As certain individuals
completed graduate degrees in exercise physiology
(science) without an undergraduate degree in the
same field, the title of an exercise physiologist could
also apply to this academic preparation so long as
undergraduate prerequisites for such graduate
degree programs have been completed.

**ASEP Certified Exercise Physiologist (EPC)**

In the United States, ASEP certified exercise
physiologist (EPC) is an exercise physiologist who
has graduated from an ASEP accredited (or
approved) program, and has successfully completed
the ASEP certification examination. However, in
other countries like India, no such graduation
course is existing.

**Job of an EPC trained Person**

The EPC is a broadly trained and competent
professional who can function in fitness, allied
health and wellness, clinical work settings, and
athletics that require the use of exercise for the
purposes of developing and maintaining good
health and fitness, disease diagnosis, physiological
assessment, rehabilitation and athletic performance.
The knowledge and skills of an EPC include, but
are not limited to the:

- knowledge of how human physiology changes
during different types and intensities of exercise
- knowledge of how the human body changes in
structure and function in response to exercise
training and regular physical activity
- knowledge of the most appropriate methods for
fitness and physical performance evaluations,
including body composition, flexibility, muscu-
lar strength and power, maximal oxygen con-
sumption and metabolic thresholds, etc.
- knowledge to provide professional advice on
appropriate modes and intensities of exercise for
specific outcomes
skills to administer graded exercise tests, and monitor blood pressure, heart rate, ratings of perceived exertion and cardiorespiratory responses during exercise

skills to prescribe exercise training

skills to conduct clinical or applied research involving exercise in whatever manner or form.

**Places where EPC should be able to Work**

The potential work competencies of all EPCs are influenced by the level of their terminal degree. The EPC could have either a Bachelor degree, Master degree, or Doctor of Philosophy degree; all with either a major or an emphasis in exercise physiology (science). The employment settings of the EPC become increasingly diverse with the PhD degree that results from first having fulfilled requirements for the BS/ BSc and MS/ MSc degrees.

**Bachelors Degree EPC**

The EPC with at least a Bachelor degree is suitably trained to work in the clinical and fitness industries. ASEP supports the development of a clinical workplace where the EPC is an important component of patient care that develops and performs under the direction of a physician and support nursing staff, the exercise testing or training of patients from a diverse number of clinical specialties such as but not limited to cardiology, nutrition, pulmonology, endocrinology, nephrology, physical therapy, occupational therapy, orthopedics, immunology, pediatrics, geriatrics and hematology. The EPC can function in the use of exercise for the diagnosis, prevention and rehabilitation of diseases, and in the research of diseases or disease processes that can be influenced by exercise. EPCs can also work in the fitness industry, where they can function as a program director, program coordinator, research technician, and exercise technician involved with the assessment of physical fitness parameters and the prescription and supervision of exercise sessions.

**Master Degree EPC**

The progression to the successful completion of a Master degree enables the EPC to become more employable in each of the employment conditions of the Bachelor degree EPC. In addition, the completion of the Master degree reveals the added knowledge, competency and research inquiry skills that would allow the EPC to supervise other EPC employees; direct exercise programs within the clinical and fitness settings, and be more competent in research. For many Master level individuals with the EPC qualification, employment in many teachers colleges/universities are also possible.

**PhD Degree EPC**

The PhD degree EPC has completed the highest academic degree available. With the advanced training, the employment opportunities in teaching and research within universities, research positions within medical schools and hospitals, and research positions within the private business sector (e.g. pharmaceutical companies, nutrition food and beverage companies, and similar companies) are open.

**How does the Exercise Physiologist provide Professional Services?**

In brief, by acknowledging that the exercise physiologist’s primary objective is to serve the public by optimizing human performance. It is by sharing, guiding, counseling and educating clients, for example, as an exercise physiologist in:

**Sports Programs**

- Sports director
- Strength coach
- Director/manager of state and national teams

**College/University Programs**

- Professor
- Researcher
- Administrator

**Community Practice**

- Manage health/wellness programs
- Direct corporate fitness/wellness programs
- Health/fitness club instructor
- Health/fitness director in correctional services and in police organizations

**Clinical Practice**

- Test/supervise cardiopulmonary patients
- Evaluate/supervise special populations
  - Diabetics
  - Obesity
  - Rheumatoid arthritis
Historical Perspective of Exercise Physiology

- Dyslipoproteinemia
- Cystic fibrosis
- Hypertension
- Children with heart disease
- Low functional capacity
- Pregnancy
- Exercise technologies in cardiology suites
- Work hardening
- Occupation rehabilitation
- Government/Military Services
  - Fitness director/manager in military, including
    - Air force
    - Army
  - Careers in military services
- Business
  - Sports management
  - Consultant
- Private Practice
  - Personal health/fitness consultant
  - Sports biomechanics
- Sports Nutrition Programs
  - Exercise nutritionist
  - Exercise counselor
- International Programs/Practice
  - Health/fitness promotion
  - Sports consultant
  - Affiliation with international organizations.

Exercise Physiology in General Population

Study of Exercise Physiology has tremendous implication for improving the health and well-being of general population. This field of specialization helps in identifying the health related problems and their treatment through exercise therapy. Rehabilitation of cardiac patients need specific exercise therapy. Exercise therapy in orthopedic problems, respiratory problems, etc. are also important areas where undergoing the research.

Economic Benefits of Exercise

It is clear that medical expenditure of individuals is increasing with the advent of mechanized society. Man is becoming dependent on technological development and thus, reducing the physical activity for their daily work, which was beneficial to them. Participation in regular physical activity would be useful in making a person fit for the stress of life and thus, provide a cut in medical expenditure.

Role of Exercise Physiology in High Performance Sports

Selection of Talents

Physiological concepts are quite useful in the selection of sports talents. Genetic characters are the major determinants for a person’s success in sports. Presence of these genetic characters, such as muscle fiber composition, body built, and others, could be identified using physiological tests and evaluation. Even the suitability of a person for a specific event could be identified at an early age.

Monitoring Training Schedule

The purpose of training is to develop adaptational changes in the body for tolerating high degree of stress produced due to sporting activities. It is important to evaluate whether these changes are at all taking place in the body. This can be done by studying the physiological status of the athlete at different stages of the training. In case, the expected changes are not found, the training schedule should be modified. In modification of training schedule also knowledge of exercise physiology is helpful. A number of research studies have established the nature of training required to produce certain type of changes in the body. So, evaluation/assessment of various physiological profiles of an athlete indicate the trainer/coaches to formulate the systematic and scientific training program for improvement of sports performance.

Evaluation of Physiological Performance

For the above purposes periodical evaluation of physiological performance is required. Knowledge of physiology also helps in recognizing the effects of pharmacological preparations on the human body. This evaluation will also indicate the strength and weaknesses of an athlete in terms of their physiological qualities.
CURRENT TRENDS IN EXERCISE PHYSIOLOGY

The present day exercise physiology has developed tremendous diversity in its field. The research work is being done at a micro-level, i.e. even to change the genetical characters by modulating genetic information of DNA. The following are the recent research trends in exercise physiology:

1. Muscle metabolism and its modulation through training and other methods. Lactate metabolism, involvement of amino acids and contribution in fatigue, etc.

2. Muscle fiber composition and its manipulation through training and nerve transfer.

3. Effects of pharmacological preparations on performance and metabolism.

Through these research works, many of the conventional beliefs are being proved as simply wrong. Lactic acid was considered as a harmful product in the body. Research has shown that lactic acid production is essential for maintaining the glycolysis process. Even lactic acid can be useful as a fuel for sparing the vital glycogen store during long distance running. Such research works are changing the sports scenario everyday.

PHYSIOLOGY IN INDIA

In India, the subject of human physiology was taught as a pre-clinical part of medical education. Later in the year 1913 Calcutta University gave first MSc degree in non-medical physiology. However, study of physiology related to exercise was started during 1950’s. Specialized teaching in work physiology was started in 1970 and sports physiology during 1981. Netaji Subhas National Institute of Sports has started teaching and research in physiology during the year 1981. However, full fledged Faculty of sports sciences was started from 1983; first as a research wing and then as teaching faculty.

Later in this decade Sports Authority of India established its Scientific Wing as Exercise Physiology in one of the branch with the basic aims to provide the scientific inputs to athletes and coaches, teaching, the subjects to the coaches and the research in this field. Sports physiologists are working in its four major regional center at Kolkata, Bangalore, Patiala and Delhi. Many colleges and number of University in West Bengal are awarding the Bachelor, Master and PhD Degree respectively. Recently, the Punjabi University is also giving Bachelor and Master Degree in Exercise Physiology. Research work in also solving undergoing in DRDO/DIPAS partieularly on Army personal.

SUMMARY

1. Physiology means the study of the structural and functional aspects of the living organisms, or it may be define as Physiology is the study of body function. In physiology, we study how our organ systems, tissues and cells work and how their functions are integrated to regulate our internal environments. Exercise physiology is the study of how our bodies’ structures and functions are altered when we are exposed to acute and chronic bouts to exercise. Sports physiology further applies the concepts of exercise physiology to training the athletes and enhancing the athlete’s sports performance. Thus, sports physiology is derived from exercise physiology.

2. Exercise physiology is a relatively new into the world of science. Before the late 19th century, physiologists’ major goal was to gain information of clinical value. The body’s response to exercise received almost no attention. Although the value of regular physical activity was well known in the mid 1800s, the physiology of muscular activity gained little attention until the later part of that century.

3. The Harvard Fatigue Laboratory was an intellectual center that attracted young physiologist from many places. Scholars from 15 countries worked in the HFL between 1927 and its closure in 1947. Most went on to develop their own laboratories and to become noteworthy international figures in exercise physiology. Thus, the HFL planted seeds of intellect around the world that resulted in an explosion of knowledge and interest in this new field.

4. Early contacts between DB Dill and August Krogh, a Danish Nobel prize winner, led to the coming of three exceptional Danish
physiologists to the Harvard fatigue laboratory in the 1930s. After returning to Scandinavia, each man established a separate line of research. In 1941, Christensen moved to Stockholm to become the first physiology professor of the College of Physical Education. He introduced Per-Olof Åstrand to the field of exercise Physiology. Åstrand who conducted numerous studies related to physical fitness and endurance capacity during the 1950s and 1960s.

5. Modern exercise physiology in Sweden can be treated to Per Henrik Ling who in 1813 becomes the first Director of Stockholm’s Royal Central Institute of Gymnastia. Per-olf Astrand is the most famous graduate of the college of Physical Education (1946). The method of analysis was developed in 1967 by Norwegian Scientist Per Scholander (1905 – 1980). Another prominent Norwegian researcher was Hermensen (1933 – 1984) from the Institute of Work Physiology whose many contributions are the guidelines in today’s exercise physiology.

6. Exercise physiology is either an academic major with Bachelor through Doctor of Philosophy degrees in many colleges and universities across the United States including India and other countries or an academic concentration in exercise science with an academic major in kinesiology, human performance, or one of several other possible titles. Many of the exercise physiology courses encompass classes that concern how exercise, physical activity and a sedentary lifestyle each influence the human body.

7. Physiological concepts are quite useful in the selection of sports talents. The purpose of training is to develop adaptational changes in the body for tolerating high degree of stress produced due to sporting activities. For the above purposes periodical evaluation of physiological performance is required. Knowledge of physiology also helps in recognizing the effects of pharmacological preparations on the human body.

8. The present day exercise physiology has developed tremendous diversity in its field. The research work is being done at a micro-level, i.e. even to change the genetical characters by modulating genetic information of DNA. In India the subject physiology was taught as a preclinical part of medical education. Later in the year 1913 Calcutta University gave first MSc degree in non-medical physiology. However, study of physiology related to exercise was started during 1950’s.

**Review Questions**

1. What do you mean by physiology? Define Exercise physiology? What is Sports physiology?
2. Discuss the evolution of Exercise physiology?
3. Who is the founder of Harvard Fatigue Laboratory? Write some of the areas of research in this laboratory.
4. Write down the emergence of Scandinavian influence in exercise physiology.
5. Who were the physiologists collaborated in describing the muscle fiber characteristics in human?
6. What is an exercise physiologist? Discuss the scope of exercise physiology.
7. How does the exercise physiologist provide professional services? Discuss the role of Exercise Physiology in general population.
8. What is the role of Exercise Physiology in high performance sports?
9. Discuss about the current trends in Exercise Physiology.
10. Write about the development of exercise physiology in India.
SECTION 2

Cell, Tissue, Organ and System

- Cell Structure and Functions
- Classification of Tissue and their Functions
- Organs of Human Body
  - Human Brain
  - Human Lungs
  - Human Heart
  - Human Stomach
  - Human Liver
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- Organ Systems of Human Body and their Functions
  - Skeletal System
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  - Digestive System
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  - Endocrine System
  - Reproductive System

**Cell Structure and Functions**

The smallest unit of protoplasm, capable of carry out independent existence is the cell. The word cell (in Latin- Cella = a store room, a chamber) was first introduced in the biology by Robert Hook (1635–1703). The cell is the basic unit of life. Its study constitutes a separate branch of biological sciences called cytology, which is now referred as cell biology. The cell biology, in fact, deals with the study of cells from morphological, biochemical, physiological, developmental, genetical, pathologiical and evolutionary point of views.

**Definition:** The cell is the structural and functional unit of the living matter and is capable of carrying on the process of life independently. Cells are basically of two types such as—prokaryotic and eukaryotic.

**Prokaryotic cells:** Prokaryotes differ from eukaryotes since they lack a nuclear membrane and a cell nucleus. Prokaryotes also lack most of the intracellular organelles and structures that are seen in eukaryotic cells. There are two kinds of prokaryotes, bacteria and archaea, but these are similar in the overall structures of their cells. Most functions of organelles, such as mitochondria, chloroplasts and the Golgi apparatus, are taken over by the prokaryotic cell’s plasma membrane. Prokaryotic cells have three architectural regions: appendages called flagella and pili — proteins attached to the cell surface; a cell envelope - consisting of a capsule, a cell wall, and a plasma membrane; and a cytoplasmic region that contains the cell genome (DNA) ribosomes and various sorts of inclusions. Other differences include: The plasma membrane (a phospholipid bilayer) separates the interior of the cell from its environment and serves as a filter and communications beacon.

Most prokaryotes have a cell wall [some exceptions are Mycoplasma (bacteria) and Thermoplasma (archaea)]. This wall consists of peptidoglycan in bacteria and acts as an additional barrier against exterior forces. It also prevents the cell from “exploding” (cytolysis) from osmotic pressure against a hypotonic environment. A cell wall is also present in some eukaryotes like plants (cellulose) and fungi, but has a different chemical composition.
A prokaryotic chromosome is usually a circular molecule (an exception is that of the bacterium *Borrelia burgdorferi*, which causes Lyme disease). Even without a real nucleus, the DNA is condensed in a nucleoid. Prokaryotes can carry extrachromosomal DNA elements called plasmids, which are usually circular. Plasmids can carry additional functions, such as antibiotic resistance.

**Eukaryotic cells:** Eukaryotic cells are about 10 times the size of a typical prokaryote and can be as much as 1000 times greater in volume. The major difference between prokaryotes and eukaryotes is that eukaryotic cells contain membrane-bound compartments in which specific metabolic activities take place. Most important among these is the presence of a cell nucleus, a membrane-delineated compartment that houses the eukaryotic cell’s DNA. It is this nucleus that gives the eukaryote its name, which means “true nucleus.” Other differences include: The plasma membrane resembles that of prokaryotes in function, with minor differences in the set up. Cell walls may or may not be present.

The eukaryotic DNA is organized in one or more linear molecules, called chromosomes, which are associated with histone proteins. All chromosomal DNA is stored in the cell nucleus, separated from the cytoplasm by a membrane. Some eukaryotic organelles also contain some DNA.

**The structure of a cell:** In multicellular organs, the cells are not of same size and shape only due to the presence of differentiation of functions. But there are certain structural characteristic features, which are common to them all. Each cell can be broadly divided into two principal units- i) cytoplasm and ii) nucleus.

(i) **Cytoplasm:** Cytoplasm is the protoplasm, which surrounds the nucleus and is bounded peripherally by the cell membrane. The cytoplasm is capable of performing different kinds of work directed by the nucleus. Specialization of cytoplasm for special functions, the appearance as well as the protoplasmic constituents are also changed from cell to cell. Under the microscope, the cytoplasm can be classified into two groups: (a) cytoplasmic inclusions, (b) cytoplasmic organelles.

(a) **Cytoplasmic inclusions:** This is not the living metabolic machineries of the body, but, certain structures are present in the cytoplasm of the cell. These are-
1. Stored foods (carbohydrate, protein and fat)
2. Secretion granules
3. Pigments
4. Crystals etc.

(b) **Cytoplasmic organelles:** Under the electron microscopic studies, certain organelles have got membranous structures. This can be classified as follows-
1. Membranous organelles- (a) plasma membrane, (b) endoplasmic reticulum (E.R.), (c) golgi apparatus, (d) mitochondria, (e) lysosomes
2. Cytoplasmic ribonucleic acid (RNA)- ribosome
3. Centrosomes
4. Various fibrils, filaments and tubules.

![Fig. 2.1: A generalized animal cell showing finer details as observed through an electron microscope](image-url)
Membranous Organelles

a) Plasma membrane: The plasma membrane or plasmalemma or cell membrane is the outermost covering of the cell and is a flexible, responsive and dynamic structure. This membrane isolates the individual cell from its neighbors and takes part in the maintenance of the internal environment by active transport of ions and nutrients. The electron microscope shows that the membrane as a trilaminar structure. This basic trilaminar structure of all cell membrane is generally described as unit membrane. The cell membrane consists of double layer of lipid molecules, which are sandwiched within the two densely stained protein layers. Lipid layer is mostly phospholipid of which the head end contains the water-soluble, positively charged, polar phosphate group i.e., Hydrophilic while the tail end contains the water-insoluble, negatively charged, nonpolar lipid group i.e., Hydrophobic. Thus, the non-polar groups of lipid molecule face each other but the protein molecules that form the inner and outer layers of the unit membrane are adsorbed on the polar groups.

Functions:

i. The main function of plasma membrane is to transport of materials across it.
ii. Helps in the protection of cell.
iii. Excrete waste products.
iv. Takes in food and 
v. Receive stimuli from the outside.

b) Endoplasmic reticulum: These consist of network of tubules, three dimensional and bounded by a thin membrane (80Å in thickness). The elements of the endoplasmic reticulum may connect intermittently with the plasma membrane at one hand and on the other hand with the outer nuclear membrane. Two types of endoplasmic reticulum have been recognized; such as (i) rough surfaced (granular) and (ii) smooth surfaced (agranular) reticulum.

i. Rough surfaced (granular): This reticulum is studded with osmophilic granules—the ribosome lining in rows in contact with the membranes of the endoplasmic reticulum.

ii. Smooth surfaced (agranular): This type of endoplasmic reticulum does not possess osmophilic granules—the ribosomes at the outer border of the membrane.

Functions:

i. Smooth surfaced endoplasmic reticulum is responsible for synthesis of steroid hormone in the interstitial cells of the testes.
ii. In the gastric mucosa, it is concerned with the secretion of hydrochloric acid.
iii. In the skeletal muscle it concerned with the binding of the Ca++ ions and also conducting impulses in the substances of muscle cells.
iv. In the liver cells both types of reticulum are concerned with the synthesis of protein and carbohydrate.

c) Golgi Apparatus: The structure look like a network of fine threads or irregular granular material. It is usually located near the nucleus and in the gland cells found between the nucleus and apex of the cell. Three main structures can be observed in Golgi apparatus under electron microscope. These are: (a) flattened vesicles, (b) secretory vesicles and (c) micro—vesicles. Flattened vesicles are the most prominent vesicles. The secretory vesicles are not normally visible until these are bounded off from the distended peripheral end of flattened vesicles (Fig. 2.2).

Functions:

i. The Golgi complex is mainly concerned with the formation and packaging of material for export from the cell across the plasma membrane by a process of reverse pinocytosis.
ii. This is probably concerned with the synthetic process of the cell, specially—secretions.
iii. Golgi apparatus independently synthesizes polysaccharide part of glucoprotein secretion.

d) Mitochondria: These are relatively solid bodies, granular, rod shaped or filamentous in form and remain scattered throughout the cytoplasm of the cell. They are surrounded by a trilaminar double membrane, the inner one of which remains folded and forms a number of partitions, the cristae mitochondriales. These cristae may be complete, septate or incomplete (Fig 2.31).

Functions:

i. In presence of oxygen, the TCA cycle runs within the mitochondria with the catalyzing help of another set of enzymes—respiratory enzymes. These enzymes present in the mitochondria help in oxidative phosphorylation
and are the site for formation of ATP, which is the high energy—producing substance in the cell.

ii. The mitochondria supply 95 percent of cells energy and are called “power house of the cell”.

Lysosomes: The lysosomes have been discovered and are recognized as a separate cytoplasmic organelle. The size of the lysosomes varies from 0.25 μ to 0.50 μ. These are membranous vesicles having a spherical and bag like structure and are filled with hydrolytic enzymes. The lysosomes are present in all animal cells except in erythrocytes. Under certain conditions, it may digest its own cellular contents and for this reason it is sometimes described dramatically as “Suicide Bag”.

Functions:

i. The general function of the lysosomes is in the intracellular digestion and this reason, it is sometimes described as digestive apparatus of the cell.

ii. Another function is cell necrosis or autolysis. When the cell is damaged, the lysosomal digestive enzymes are released and digest off cellular elements.

iii. Phagocytosis is also one of the remarkable function of lysosomes.

iv. The rupture of lysosome is the stimulus for cell division and alteration of the behavior of
lysosome may be one of the causes of cancerous growth.

**Ribosome:** Ribosomes are ribonucleoprotein in nature and are found scattered throughout the cytoplasm either singly or in groups (polyribosomes or polysomes) and range in size from 100 to 150 Å in diameter. They are so rich in RNA that they may contain as much as 60 percent of total RNA in the entire cell. These ribonucleoproteins are concerned with protein synthesis and their presence gives the membrane a strong basophilia. Cells responsible for the secretion of proteins have an abundance of granular reticulum.

**Functions:**
1. Ribosomes synthesize protein.
2. Ribosomes also synthesize canals of the reticulum work as passageways through which proteins move on to way to Golgi apparatus. So robosomes are called “Protein Factories”.

**Centrosome:** Centrosome consists of another specialized part of clear cytoplasm, the centrosphere, containing in its interior two or more deeply staining particles—the centrioles lying close to the nucleus in the resting cell. The centriole is an empty cylinder, which is 3 to 5 μ long, the compact walls of thin parallel nine tubular structures longitudinally arranged. Each tubule consists, in turn, of three subunits or triplets. A system of radiating lines, made up of microtubules, grows out from each of the two newly formed centrioles, the whole structure is called aster and diverging fibers from the two asters meet at the equator of cell forms the achromatic spindle.

**Function:**
1. Centriole is closely related to spindle formation during mitosis.
2. Centriole controls polarization of spindle fibers.
3. Centrioles serve as foci for the production of new centrioles and basal bodies.

**Nucleus:** The nucleus is generally a round body occupying the center of the cell. Its shape, size, position and number usually differ. The nucleus may contain many lobes. The nucleus is very rich in deoxyribonucleic acid (DNA) while cytoplasm is rich in ribonucleic acid (RNA).

a. **Nuclear membrane:** The nucleus is surrounded by a thin membrane is called nuclear membrane. It is also a unit membrane surrounding the nucleus, there is a lipoprotein nuclear envelope, which is, doubled layer. The membrane having pores, which permits molecules from the nucleus to the cytoplasm.

b. **Nucleolus:** Inside the nucleus there is usually single or 2 to 5 smaller bodies, which are known as nucleolus, lie among nuclear sap. The nucleolus comprises the irregular network or rows of fine granules are called nucleonema. The nucleolus contains still smaller nucleus known as nucleololus.

c. **Chromatin:** The numerous particles of irregular shape but smaller than nucleoli are found in the nucleolus. This material is generally described as chromatin. Chromatin contains different genes, which determine the heredity of the cell.

**Classification of Tissue and Their Functions**

**Definition:** A tissue may be defined as an aggregate of same type of cells combined by sub-serving the same general function independently and united by varying amounts of intracellular substance. Example- blood, bones, cartilage, muscle, nervous tissue etc. The human body is composed of the following elementary tissues:

1. **Epithelial Tissue**

**General character:**
1. It forms a limiting and a lining membrane, the epithelial membrane and as such covers the free surface.
ii. The supporting loose vascular connective tissue, lamina propria which transport metabolic requirements from blood vessels to this layer.

iii. There is generally a basement membrane upon which cells are set.

iv. The cells remain closed together with one type of cementing substance named mucoprotein which containing hyaluronic acid and calcium salts.

**Classification of Epithelial Tissue:** According to shape and layers of cells the epithelial tissue is classified into two types: such as- simple and compound.

a) **Simple Epithelial Tissue:** The various types of simple epithelial tissues which are as follows:

I. Pavement (squamous): It is composed of a single layer of large flat cells placed on a thin basement membrane. It is found in the alveoli, serous membrane, Bowman’s capsule, Henle’s loop, inner lining of heart and blood vessels etc.

Functions:

i. It has got a dialyzing or filtering function.

ii. It helps for the easy passes of liquid and gases through it.

II. Cubical (cuboidal): They are composed of a single layer of cubical cells having same dimensions on each side and placed upon a basement membrane. They are found in the terminal respiratory bronchioles, digestive gland, salivary gland, thyroid, covering of ovary etc.

Functions:

i. It forms a protective layer on the surface.

ii. It often serves some other important functions, such as secretion, storage etc.
III. Columnar (cylindrical): They are composed of a single layer of cells of which the height is more than their breadth, arranged on a basement membrane. It is found in stomach, small and large intestine, alveoli, ducts of many glands etc.

**Functions:**

i. One type of columnar cells named Goblet cells, which is responsible for the secretion of mucus.

ii. It also helps for absorption in small intestine.

IV. Ciliated: These cells are generally columnar in shape but at places may be cubical. The free surface has got hair-like processes on each cell, called cilia or flagella. They are found in the respiratory passages, central nervous system, fallopian tube of ovary, uterus etc.

**Functions:**

i. They maintain the flow of mucus or liquid in one direction.

ii. They help to maintain the circulation of cerebrospinal fluid in the ventricles and central canal of the spinal cord.

V. Glandular: They are generally cubical, short columnar or polyhedral in shape and consist of one layer. They are generally found in mammary gland, sweat gland, thyroid, alveoli etc.

**Function:** They serve the very important function of manufacturing the new substances and pass them out into their respective secretions.

b) **Compound Epithelial Tissue:** Various types of compound epithelial tissues are as following:

i. **Transitional:** It consists of three or four layers of pyriform and polyhedral cells and occupies the intermediate position between the layer of simple and stratified epithelium. They are found in the pelvis of kidney, ureter, urinary bladder, urethra etc.

**Functions:**

i. It prevents reabsorption of the excreted material back to the system.

ii. It also prevents in drawing of water from blood and tissue by the higher osmotic pressure of urine.

II. Stratified squamous cornified: It is composed of many layers of the cells. Usually the superficial layers are horny due to the deposition of keratin. It is found in the skin, hair, nails, horns etc.

**Function:** This type of epithelium is always found in those places, which are constantly exposed to atmosphere, mechanical pressure, friction and injury.

III. Stratified squamous non-cornified: They are composed of spherical layers and the upper layer is not keratinized. It is found in the cornea, mouth, pharynx, esophagus, etc.

**Function:** It affords mechanical protection.

IV. Stratified columnar: It is rare and found only in a few places, covering small areas, e.g. fornix of conjunctiva, some parts of pharynx, epiglottis etc.

V. Stratified columnar ciliated: This also is found only in small areas, e.g. nasal surface of the soft palate, some parts of larynx etc.

2. **Connective Tissue**

**General character:**

i. They are all developed from the mesoderm.

ii. The intercellular substance is prominent and may be considerable in amount. This is a great contrast with the epithelial tissues.

iii. The intercellular ground substance may contain different types of fibrous elements.

**Types:** The following varieties have been described: (1) Areolar tissue, (2) Adipose tissue, (3) White fibrous tissue, (4) Yellow elastic tissue, (5) Reticular tissue, (6) Blood and hemopoietic tissue, (7) Cartilage, (8) Jelly-like tissue, (9) Osseous tissue (bone) and (10) Reticulo-endothelial tissue.
I. Areolar Tissue: It is distributed in the subcutaneous, sub mucous and sub serous tissues, between muscles, vessels and nerves, in the interior of organs binding the different parts. It is composed of fibers and cells. The variety of cells such as- fibroblasts, histiocytes, basophil cells, plasma cells, pigment cells and mast cells etc.

Function:
   i. The proteolytic enzymes present in this tissue which destroy the digestible ingested materials.
   ii. It takes a great part in replacement fibrosis during repair of inflammation.

II. Adipose Tissue: The adipose or loose tissue is characterized by containing free fat inside the fat cells. The cells are generally large, rounded or oval in shape. They are found in fat depots, viz., momentum, subcutaneous tissue, mesentery, sub pericardial tissue, etc.

Functions:
   i. It remains under the surface gives shape to the limbs and body.
   ii. It takes part in the regulation of body temperature.

III. White Fibrous Tissue: It is made up of shining white fibers. They are thin, non-branching and run in bundles. They are found in tendons, ligaments, articular capsule, dura matter, etc.

Functions:
   i. It connects the different tissues and different parts of the body to afford mechanical protection against stretch and pressure.
   ii. It is made up of a protein known as collagen; digestible with pepsin and from this gelatin is easily obtained by boiling with a little hydrochloric acid.

IV. Yellow Elastic Tissue: They are another variety of fibrous tissue. It is thicker, yellowish in color and forms a network. It is found in areolar tissue, hollow viscera, bronchial walls etc.

Functions:
   i. They serve the purpose of a strong elastic rope due to the presence of a protein, elastin, which is responsible for this elastic property.
   ii. In the form of ligaments it holds the connected parts firmly together and at the same time allows considerable latitude of movement.

V. Reticular Tissue: They are similar to areolar tissue, the reticular fibers resemble with white fibrous tissue. They are thinner, freely branching and found in lymph nodes, liver, spleen, bone marrow, etc.

Functions:
   i. They form the basement membrane of many epithelia (e.g., reticulo-endothelial system).
   ii. They form the framework of many organs, supporting their essential cellular elements.
VI. Blood and Hemopoietic Tissue: Blood cells are considered to be the special type of connective tissue. The hemopoietic tissues are mainly of two types such as myeloid and lymphatic tissue. Myeloid tissue produces erythrocytes, leucocytes and platelets. The lymphatic tissue is found in lymph node, spleen, thymus and tonsil.

Functions:
1. They can supply the lymphocytes to the blood and lymph stream.
2. They can serve as a great defense against bacterial infection.

VII. Cartilage: It is a connective tissue which is more or less translucent, firm in texture and to some extent elastic. It is found in bones, external ear, Eustachian tube, epiglottis, larynx, trachea, etc. Cartilage is divided into classes such as- hyaline cartilage, fibro cartilage and elastic cartilage.

Functions:
1. It helps to maintain shape and rigidity of structure combined with certain amount of elasticity.
2. It forms primary medium of bone and maintains flexibility of the substances between bones.

VIII. Osseous Tissue or Bone: This tissue, which constitutes the skeleton, is the hardest of all connective tissue. It is made up of bone cells and intracellular ground substances. There are three type of bone cells such as—osteoblast, osteocyte and osteoclast.

Functions:
1. It performs a mechanical function in forming the skeletal support and shape to the body.
2. It affords protection to the vital organs of the cranial, thoracic cavities, blood vessels, nerves, etc.

IX. Reticulo-endothelial Tissue: This possesses various types connective tissue cells which are widely distributed in the body, the phagocytic cells, which are found in bone marrow, liver capillaries, lymph nodes, etc.

Functions: It is important for defense of body against foreign particles and microorganisms.

Muscular Tissue

General character:
1. Muscular tissue has the ability to contract when excited.
2. The property of conductivity is also well developed in muscular tissue.
3. The energy required for prolonged muscular work is obtained through metabolism of the food and for constant supply of food an efficient blood circulation is required.
4. Muscle fiber is attributed to a muscle cell to its elongated shape, which is adopted for the contractile function.

Figs 2.9A to C: (A) Skeletal muscle, (B) Cardiac muscle and (C) Visceral muscle
Classification of muscular tissue: There are different types of muscular tissue such as: skeletal, cardiac and visceral muscle.

a. **Skeletal Muscle**: The skeletal muscle fibers are multinucleated cylindrical structures having a clear display of longitudinal and cross-striations, i.e. striated and voluntary in nature. These muscles mostly in all instances are attached to osseous tissue (bones).

b. **Cardiac Muscle**: The cardiac muscles fibers are uninucleus, striated, bifurcate, come in contact with that of the neighboring fibers and thus forms a three dimensional network and involuntary in nature. They are found mainly in heart and also present in small amounts in the great vessels ending in or opening from the heart.

c. **Visceral Muscle**: The visceral muscle is also known as the plain, non-striated, smooth involuntary muscle. It is called non-striated because it has got no cross-striations. The contraction of this muscle is not controlled by volition or will. These muscles are present all hollow viscera such as—gastro-intestinal tract, ducts of the glands, blood vessels, respiratory, urogenital and lymphatic systems of the body.

**Functions of the Muscular Tissue**

i. Locomotion of the body, changes in position of the body, development of muscular skills involved in various activities.

ii. Production of body heat and fluid balance is a concomitant contribution of muscle to the whole body.

iii. Muscles help in maintaining the posture of the body.

iv. As muscles are dependent on the circulatory system, they reciprocate by helping to protect blood vessels and assist in maintaining the circulation of body by forceful contraction and relaxation of the heart.

v. As muscles help in respiration, they contribute to the provision for oxygen and eliminate carbon dioxide maintaining the vital acid-base balance of the body.

vi. Muscles are agents of the brain by means of which we maintain our independence and give over expression to our inner thoughts and feelings.

**Nervous Tissue**

General characters:

i. This is a highly specialized tissue for reception, discharge of stimuli and its transmission.

ii. It is made up of nerve cells and their processes, called the nerve fibers.

iii. Receptive processes are known as dendrites or dendrites and the discharging process is known as axon.

iv. A nerve cell body or perikaryon with all its process is called a neuron.

v. Besides these, there are neurological cells, which support the nerve cells.

Classification of nerve fibers: There are two types of nerve fibers (according to the structure) such as: medullated and non-medullated nerve fibers.

a. **Medullated**: It is composed of three elements, i.e. axis cylinder, myelin sheath or medullary sheath and neurolemma. The axis cylinder is central and is the direct continuation of the protoplasm of the nerve cells. It remains covered by a thin tubular sheath the axolemma, which
is possibly the modified surface membrane of
the corresponding nerve cells.
b. Non-medullated: These are composed of two
elements only—the central axis cylinder and the
neurolemma. Non-myelinated nerve fibers differ
from myelinated nerve fibers in great reduction
or absence of the myelin sheath, the fiber being
directly invested with the neurolemma. In the
peripheral nerve trunks in the fibers are grouped
into separate bundles. The individual nerve
fibers are held together by loose connective
tissue, called endoneurium.

Functions:
i. The function of the nerve tissue is mainly
the reception, discharge of stimuli and
transmission.

ii. The function of the medullary sheath is to
insulate the nerve fibers and thus to prevent
the spread of the nerve impulse to the
adjacent fiber.

iii. The other functions are to supply the
nutrition (partly) to the nerve fiber.

iv. To play an essential role in the regeneration
of the damage of peripheral nerves.

ORGANS OF HUMAN BODY

An organ is a group of more commonly two or
more tissues, which is basically functions
independently in some instances, in particular
patterns to form larger functional units.

Human Brain

System: Nervous system

Location: Inside the skull

Physical description: Pale gray, the size of a
small cauliflower and the texture of pate. The
brain is made of many parts, each of which has a
specific function. It can be divided into four
areas: the cerebrum, the diencephalons, the
brainstem and the cerebellum.

i) Cerebrum: The cerebrum is the largest part of
the brain. It is located on top of rest of the brain,
rather like a mushroom cap covering its stalk. It
has a heavily folded grey surface, the pattern of

![Fig. 2.11: Various organs and their location in the human body](image-url)
which is different from one person to the next. Some of the grooves in its surface mark out different functional regions.

The front section of a cerebrum, the frontal lobe, is involved in speech, thought, emotion and skilled movements. Behind this is the parietal lobe which perceives and interprets sensations like touch, temperature and pain. Behind this, at the center back of the cerebrum, is a region called the occipital lobe which detects and interprets visual images. Either side of the cerebrum is the temporal lobes which are involved in hearing and storing memory. The cerebrum is split down the middle into two halves, called hemispheres that communicate with each other.

ii) Cerebellum: Cerebellum is the second largest part of the brain. It is located underneath the back of your cerebrum. It is involved in coordinating the muscles to allow precise movements and control of balance and posture of an individual.

iii) Diencephalon: Diencephalon is located beneath the middle of cerebrum and on top of the brainstem. It contains two important structures called the thalamus and the hypothalamus. Thalamus acts as a relay station for incoming sensory nerve impulses, sending them on to appropriate regions of the brain for processing. It is responsible for letting your brain know what’s happening outside of your body.

The hypothalamus plays a vital role in keeping conditions inside the body constant. It does this by regulating body temperature, thirst and hunger, amongst other things. And by controlling the release of hormones from the nearby pituitary gland.

iv) Brainstem: Brainstem is responsible for regulating many life support mechanisms, such as heart rate, blood pressure, digestion and breathing, etc. It also regulates when the individual sleep and wake reticular system.

**Brain Protection**

Brain is one of the most important organ, but it is made of soft delicate tissue that would be injured by even the slightest pressure. As a result, it is well protected:

- Three tough membranes called meninges surround the brain.
- The space between brain and the meninges is filled with a clear fluid, which cushions your brain, provides it with energy and protects it against infection.
- The skull encases your brain in a bony shell, cerebrospinal fluid and meninges.

**Function:** To control the body and house your mind. Information, in the form of nerve impulses, travels to and from brain along the spinal cord. This allows the brain to monitor and regulate unconscious body processes, such as digestion and breathing and to coordinate most voluntary movements of the body. It is also the site of your consciousness, allowing you to think, learn and create something.

**Human Lungs**

**System:** Respiratory system.

**Location:** In the chest, inside the rib cage.

**Physical description:** Large, rounded, light, spongy, inflatable organs. Lungs are a pair of large sponge-like organs that almost fill the chest cavity. The left lung is slightly smaller than the right lung, to make space for the heart.

When you breathe in, you inhale air in through your nose and mouth and down a tube called the trachea. Trachea divides into two tubes called the primary bronchi. One enters each lung. From there, the bronchi progressively branch into smaller airways, which eventually lead to tiny air sacs called alveoli. This intricate network of airways looks like an upside-down tree.

**Function:** To deliver oxygen to and remove carbon dioxide from your blood the following activity performed by the lungs.

i) **Exchanging gases:** Alveoli are surrounded by minute blood vessels, as this is where gases diffuse from the lungs into blood and from blood into the lungs. Oxygen passes from alveoli into blood and carbon dioxide, which is produced when cells break down nutrients, passes from your blood into alveoli.

The total surface area of alveoli is about the size of a tennis court. However, if you’re not doing vigorous exercise, you only use about
one-twentieth of your lungs’ gas-exchanging surface.

ii) Breathing in and out: Normally an individual breathe in and out about 500 ml of air 16–18 times a minute. The nervous system automatically increases the rate and depth of breathing if the body needs more oxygen, for example, when you’re doing exercise.

Air is forced in and out of the lungs by movements of diaphragm and other breathing muscles. When you breathe in, your breathing muscles contract, pulling your ribs up and out. The space within the chest increases and reduces the air pressure inside lungs. As a result, air flows into the lungs. When you breathe out, the respiratory muscles relax and ribs move down and in. The space within the chest cavity decreases again, the pressure inside the lungs increases, and air flows out.

Human Heart

System: Cardiovascular System.

Location: Between lungs inside the chest cavity.

Physical description: It is made up of cardiac muscle, which only exists in the heart. Unlike other types of muscle, cardiac muscle never gets tired. The heart is divided into four hollow chambers. The upper two chambers are called atria or auricle. They are joined to two lower chambers called ventricles. The ventricle is separated by interventricular septum. These are the pumps of your heart. One-way valves between the chambers keep blood flowing through heart in the right direction.

As blood flows through a valve from one chamber into another the valve closes, preventing blood flowing backwards. As the valves snap shut, they make a thumping, ‘heart beat’ noise.

Function: To pump oxygen-rich blood throughout the body and oxygen-poor (CO₂ reach) blood to the lungs. Blood carries oxygen and many other substances around your body. Oxygen from blood reacts with sugar (carbohydrate) in the cells to make energy. The waste product of this process, carbon dioxide, is carried away from cells in the blood.

Heart is a single organ, but it acts as a double pump. The first pump carries oxygen-poor blood to lungs, where it unloads carbon dioxide and picks up oxygen. It then delivers oxygen-rich blood back to the heart. The second pump delivers oxygen-rich blood to every part of your body. Blood needing more oxygen is sent back to the heart to begin the cycle again. In a day the heart transports all your blood around your body about 1000 times.

The right ventricle pumps blood to the lungs and the left ventricle pumps blood all around the body. The muscular walls of the left ventricle are thicker than those of the right ventricle, making it a much more powerful pump. For this reason, it is easiest to feel your heart beating on the left side of the chest.

Pacemaker

Unlike skeletal muscle cells that need to be stimulated by nerve impulses to contract, cardiac
muscle cells can contract all by themselves. However, if left to their own devices, cardiac muscle cells in different areas of the heart would beat at different rates. Muscle cells in ventricles would beat more slowly than those in atria. Without some kind of unifying function, heart would be an inefficient, uncoordinated pump. So, the heart has a tiny group of cells known as the sinoatrial node (SA-node) that is responsible for coordinating heart beat rate across your heart. It starts each heartbeat and sets the heartbeat pace for the whole heart. Damage to the sinoatrial node can result in a slower heart rate. When this is a problem, an operation is often performed to install an artificial pacemaker, which takes over the role of the sinoatrial node.

**Heart Rate**

Without nervous system control, the heart would beat around 100 times per minute. However, when you are relaxed, your parasympathetic nervous system sets a resting heart beat rate of about 72 beats per minute, (resting heart rate is usually between 72 to 80 beats per minute in women and 66 to 74 beats per minute in men).

During exercise or feel anxious the heart beats more quickly, increasing the flow of oxygenated blood to your muscles. This is triggered by your sympathetic nervous system. The heart rate also increases in response to hormones like adrenalin. On average, the maximum heart rate is 220 beats per minute minus your age. So, a 40-year old would have a maximum heart rate of 180 beats per minute.

**Oxygen Supply to the Heart**

Although the heart is continually filled with blood, this blood doesn’t provide your heart with oxygen. The blood supply that provides oxygen and nutrients to the heart is self is provided by blood vessels that wrap around the outside of the heart.

**Physical description:** A ‘J’-shaped elastic sac which is the widest part of the digestive system.

**Function:** Storing food, breaking food down and mixing it with juices enzyme secreted by the stomach lining

i) **Storage of food:** Stomach has a short-term food-storage facility. This allows consuming a large meal quickly and then digesting it over an extended period of time. When full, the stomach can hold around one liter of chewed up food. Swallowed food is propelled down to the esophagus into the stomach. Food is enclosed in the stomach by two circular muscles, known as sphincters.

ii) **Chemical breakdown:** As soon as food enters into the stomach, the stomach lining releases enzymes that start breaking down proteins in the food. The stomach lining also secretes hydrochloric acid, which creates the ideal conditions for the protein-digesting enzymes to work. The potent hydrochloric acid kills bacteria, protecting your body from harmful microbes which can enter your body through food.

The stomach also protects itself from being digested by its own enzymes, or burnt by the corrosive hydrochloric acid, by secreting sticky, neutralizing mucus that clings to the stomach walls. If this layer becomes damaged in any way it can result in painful and unpleasant stomach ulcers.

iii) **Physical breakdown:** Waves of muscular contraction along with the stomach wall, known as peristalsis, break food down into smaller pieces, mix it with fluids secreted from stomach lining and move it through the stomach. This creates a mixture that resembles thick cream.

iv) **Release of food into small intestine:** When food has been broken down sufficiently, small amounts are squirted out of stomach into your small intestine for further processing. This normally occurs within four hours of eating a meal, but can take six or even more hours if your meal has a high fat content.

**Human Stomach**

**System:** Digestive System.

**Location:** Between a muscular tube called the esophagus and the small intestine in the abdominal part of human body.

**Human Liver**

**System:** Digestive System.

**Location:** Under the diaphragm (abdominal region), more to the right side of the body.
Physical description: Wedge-shaped, spongy organ. The liver is a largest internal organ. A big blood vessel, called the portal vein, carries nutrient-rich blood from the small intestine directly to the liver.

Function: To get rid of toxins, to regulate the blood sugar levels and to produce bile.

i) Chemical processing factory: Hepatic cells make-up about 60 percent of liver tissue. These specialized liver cells carry out more chemical processes than any other group of cells in the body. They change most of the nutrients you consume into forms your body cells can use. They -

i. Convert sugars and store and release them as needed, thereby regulating the blood sugar level.
ii. Breakdown fats and produce cholesterol.
iii. Remove ammonia from the body and produce blood proteins, including blood clotting factors.

Other functions of the hepatic cells are to:

i. Detoxify drugs and alcohol.
ii. Produce bile, which breaks down fats in the food which you eat.

ii) Security guard: A second important group of liver cells are the Kupffer cells. They

i. Remove damaged red blood cells.
ii. Destroy microbes and cell debris.

iii) Essential for life: As the liver fulfills so many vital functions, one would die within 24 hours if it stopped working. A common sign of a damaged liver is jaundice, a yellowness of your eyes and skin. This happens when bilirubin, a yellow breakdown product of your red blood cells, builds up in blood.

Human Kidney

System: Urinary System.

Location: At the bottom of ribcage and towards the back side of the body.

Physical description: Fist sized, dark red and kidney bean-shaped.

Function: To make urine from waste products and excess water found in the blood.

I. Balancing blood: For body to work properly, the conditions inside it, such as water, pH and salt levels, need to be kept constant. Kidneys play a vital role in keeping blood composition constant. They filter your blood to remove excess water and waste products, which are secreted from kidneys as urine.

One quarter of your blood supply passes through kidneys every minute. It enters into the kidney and is distributed to minute filtration units known as nephrons. Each of your kidneys contains more than one million nephrons. The main substances of nephrons filter out of blood are:

- Water.
- Nitrogen-containing compounds like urea that are produced when the body breaks down proteins.
- Salts.
- Acids.
- Alkalis.

Nephrons filter these substances out of blood and then reabsorb some of them back into blood.
This keeps blood composition constant. Excess water and waste products are then secreted as urine. Kidneys vary the amount of a substance that is reabsorbed into the blood or secreted as urine. This determines the volume and composition of urine. For example, when you drink a lot of water, your kidneys produce a lot of urine to stop the water levels in your body getting too high. But, if you don’t drink much, your kidneys only produce a small amount of concentrated urine, keeping as much water as possible in the body. In 24 hours, your kidneys filter around 150 liters of blood and produce roughly 1.5 liters of urine.

i) **Regulating blood pressure:** When the kidney detect that blood pressure is dropping, they secrete an enzyme called rennin from the juxtaglomerular apparatus of the kidney. This enzyme triggers a chain of events that makes your kidneys reabsorb more salt and water, leading to an increase in blood pressure.

ii) **When kidneys dysfunction:** Human being can live healthily with one functioning kidney. However, when about 90 percent of kidney function has been lost, a person can only survive by having dialysis. Dialysis works by using a machine that replicates the blood-cleaning function of healthy kidneys. In the most extreme cases of kidney failure, survival depends on the person receiving a donor organ.

iii) **Effects of exercise formation of urine:** Exercise always reduces urine volume. The initial effects are not due to adrenaline secretion or renal vasoconstriction. Emotional states also produce similar result. It is suggested that both exercise and emotion, acting upon the hypothalamus—pituitary mechanism, increase the secretion of anti-diuretic hormone (ADH) and thus urine output is reduced. After severe exercise the urine volume is further reduced and becomes more acidic in reaction.
ORGAN SYSTEMS OF HUMAN BODY AND THEIR FUNCTIONS

Organ systems are composed of two or more different organs that work together to provide a common function or when several organs functions interrelatedly they form organ system. There are 10 major organ systems in the human body; they are as follows:

Skeletal System

The human skeleton consists of both fused and individual bones supported and supplemented by ligaments, tendons, muscles, and cartilage. It serves as a scaffold which supports organs, anchors muscles, and protects organs such as the brain, lungs, and heart. The longest and heaviest bone in the body is the ‘femur’ and the smallest is the ‘stapes’ bone in the middle ear. In an adult, the skeleton comprises around 20 percent of the total body weight.

Fused bones include those of the pelvis and the cranium. Not all bones are interconnected directly. There are six bones in the middle ear called the ossicles (three on each side) that articulate only with each other. The hyoid bone, which is located in the neck and serves as the point of attachment for the tongue, does not articulate with any other bones in the body, being supported by muscles and ligaments.

Fig. 2.16: Human nervous system (image courtesy of G Huang)

Fig. 2.17: Human respiratory system (image courtesy of G Huang)
**Major Role:** The main role of the skeletal system is to provide support for the body, to protect delicate internal organs and to provide attachment sites for the organs.

**Major Organs:** Bones, cartilage, tendons and ligaments.

**Muscular System**

**Major Role:** The main role of the muscular system is to provide movement. Muscles work in pairs to move limbs and provide the organism with mobility. Muscles also control the movement of materials through some organs, such as the stomach and intestine and the heart and circulatory system.

**Major Organs:** Skeletal muscles and smooth muscles throughout the body.

**Circulatory System**

It is well organized transport system of the body by which the blood being circulated with in a closed system under different pressure gradients, created by the pumping mechanism where heart act as the central pump.

**Major Role:** The main role of the circulatory system is to transport nutrients, gases (such as oxygen and Carbon dioxide), hormones and wastes through the body needs for their proper function.

**Major Organs:** The cardiovascular system includes (i) heart, (ii) arteries, (iii) capillaries and (iv) veins. They all differ in structures as well as in functions. The system is functioning in two ways, i.e. by maintaining an efficient circulation, so that the blood supply to every part of the body in rest and activity may be assured and by maintaining an optimum blood pressure which is essential for capillary exchange.

**Nervous System**

The nervous system is the most important organization which controls and integrates the different bodily functions and likewise maintains a stability of the external environment despite extreme changes in the external environment.

**Major Role:** The main role of the nervous system is to relay electrical impulses through the body.

The nervous system directs behavior and movement and along with the endocrine system, controls physiological processes such as digestion, circulation, etc. The system operates through two main systems: (a) the central or somatic nervous system and (b) the autonomic nervous system.

**Major Organs:** Neuron, receptor, brain, ganglia, etc.

**Respiratory System**

In humans the **respiratory system** consists of the airways, the lungs and the respiratory muscles that mediate the movement of air into and out of the body. Within the alveolar system of the lungs, molecules of oxygen and carbon dioxide are passively exchanged, by diffusion, between the gaseous environment and the blood. Thus, the respiratory system facilitates oxygenation of the blood with a concomitant removal of carbon dioxide and other gaseous metabolic wastes from the circulation. The system also helps to maintain the acid-base balance of the body through the efficient removal of carbon dioxide from the blood.

**Major Role:** The main role of the respiratory system is to provide gas exchange between the blood and the environment. Primarily, oxygen is
Fig. 2.19: Human excretory system (image courtesy of G Huang)

Fig. 2.20: Human endocrine system (image courtesy of G Huang)
absorbed from the atmosphere into the body and carbon dioxide is expelled from the body.

**Major Organs:** Nose, trachea and lungs.

**Digestive System**

**Major Role:** The human digestive system serves the following functions: (a) ingestion of food; (b) digestion of food; (c) secretion of various digestive juices; (d) absorption of water, salts, vitamins and end products of food digestion, etc. for growth and maintenance.

**Major Organs:** The major organs of digestive system are: Mouth, esophagus, stomach, small and large intestines and liver.

**Excretory System**

Excretion is the process by which living organisms get rid of their metabolic waste products. These if retained in the body are toxic. The organs associated with the process of excretion are called excretory organs and the system is called excretory system. Thus, excretion may be defined as the process of removal of nitrogenous waste products like ammonia, urea, uric acid, etc. along with excess of water, salts and pigments out of the body.

**Major Role:** The main role of the excretory system is to filter out cellular wastes, toxins and excess water or nutrients from the circulatory system.

**Major Organs:** Kidneys, ureters, urinary bladder and urethra.

**Endocrine System**

The endocrine system is an integrated system of small organs that involve the release of extracellular signaling molecules known as hormones. The endocrine system is instrumental in regulating metabolism, growth, development and puberty, tissue function and also plays a part in determining various vital functions of the body. The field of medicine that deals with disorders of endocrine glands is endocrinology, a branch of the wider field of internal medicine.
**Major Role:** The Endocrine system is an information signal system much like the nervous system. However, the nervous system uses nerves to conduct information, whereas the endocrine system mainly uses blood vessels as information channels. Glands located in many regions of the body release into the bloodstream specific chemical messengers called hormones. Hormones regulate the many and varied functions of an organism, e.g., growth and development, tissue function and metabolism, as well as sending messages and acting on them.

**Major Organs:** Many glands exist in the body that secrete endocrine hormones. Among these are the hypothalamus, pituitary, thyroid, pancreas, adrenal glands, etc. are important.

**Reproductive System**

The reproductive system is a system of organs within an organism which work together for the purpose of reproduction. Many non-living substances such as fluids, hormones and pheromones are also important accessories to the reproductive system. Unlike most organ systems, the sexes of differentiated species often have significant differences. These differences allow for a combination of genetic material between two individuals, which allows for the possibility of greater genetic fitness of the offspring.

**Major Role:** The main role of the reproductive system is to manufacture cells that allow reproduction. In the male, sperms are created to inseminate egg cells produced in the female.

**Major Organs:** The major organs of the human reproductive system include the external genitalia (penis and vagina) as well as a number of internal organs including the gamete producing gonads (testicles and ovaries). Diseases of the human reproductive system are very common and widespread, particularly communicable sexually transmitted diseases. Female (top): ovaries, oviducts, uterus, vagina and mammary glands. Male (bottom): testes, vas deferens seminal vesicles and penis.

**Summary**

1. The cell is the structural and functional unit of the living matter and is capable of carrying on the process of life independently.

2. Cells are of two types—Prokaryotes and Eukaryotes. Prokaryotes differ from eukaryotes since they lack a nuclear membrane and a cell nucleus. Prokaryotes also lack most of the intracellular organelles and structures that are seen in eukaryotic cells. The major difference between prokaryotes and eukaryotes is that eukaryotic cells contain membrane-bound compartments in which specific metabolic activities take place. Most important among these is the presence of a cell nucleus, a membrane-delineated compartment that houses the eukaryotic cell’s DNA.

3. The plasma membrane or plasmalemma or cell membrane is the outermost covering of the cell and is a flexible, responsive and dynamic structure. The elements of the endoplasmic reticulum may connect intermittently with the plasma membrane at one hand and on the other hand with the outer nuclear membrane. Mitochondria are relatively solid bodies, granular, rod shaped or filamentous in form and remain scattered throughout the cytoplasm of the cell. They are surrounded by a trilaminar double membrane, the inner one of which remains folded and forms a number of partitions, the cristae mitochondriales. Ribosomes are ribonucleoprotein in nature and are also found scattered throughout the cytoplasm either singly or in groups.

4. A tissue may be defined as an aggregate of same type of cells combined by sub-serving the same general function independently and united by varying amounts of intracellular substance. Example—blood, bone, cartilage, muscle, nervous tissue, etc. The organs mainly made up of similar type of tissue combine together and accomplished the same function. Brain, heart, lung, kidney, etc. are some of example of organs.

5. The brain is made of many parts, each of which has a specific function. It can be divided into four areas: the cerebrum, the diencephalon, the brainstem and the cerebellum. The cerebrum is the largest part of the brain. It is located on top of rest of the brain, rather like a mushroom cap covering its stalk. It has a heavily folded gray surface, the pattern of which is different from one person to the next. Some of
the grooves in its surface mark out different functional regions. Cerebellum is the second largest part of the brain. It is located underneath the back of the cerebrum. It is involved in coordinating the muscles to allow precise movements and control of balance and posture of an individual.

6. Human lung is a large, rounded, light, spongy, inflatable organ. Lungs are a pair of large sponge-like organs that almost fill the chest cavity. The left lung is slightly smaller than the right lung, to make space for the heart. To deliver oxygen to and remove carbon dioxide from blood performed by the lungs. Heart is made up of cardiac muscle, which only exists in the heart. Unlike other types of muscle, cardiac muscle never gets fatigue. The heart is divided into four hollow chambers. The upper two chambers are called auricles. They are joined to two lower chambers called ventricles. The ventricle is separated by interventricular septum. These are the pumps of heart. To pump oxygen-rich blood throughout the body and oxygen-poor (CO₂ reach) blood to the lungs. Blood carries oxygen and many other substances around the body. Oxygen from blood reacts with sugar in cells to make energy in the cell. The waste product of this process, carbon dioxide, is carried away from the cell in the blood. Another vital organ is present in human body is stomach. Stomach is a short-term food-storage facility. This allows consuming a large meal quickly and then digesting it over an extended period of time. Two pears shaped, dark brown colored kidney are located in the back of upper abdominal area in the human body. They excrete waste products/metabolites from human body.

7. Organ systems are composed of two or more different organs that work together to provide a common function or when several organs functions interrelatedly, they form organ system. There are ten major organ systems in the human body. They are Skeletal System, muscular system, circulatory system, nervous system, respiratory system, digestive system, excretory system, endocrine system and reproductive system.

**Review Questions**

1. What is the structural and functional unit of life?
2. What are the differences between Prokaryotes and Eukaryotes cell?
3. Write the name of important cytoplasmic organelles of Eukaryotic cell.
4. Why mitochondria are called ‘the power house’ of cell?
5. Define cell. What do you mean by Prokaryotic and Eukaryotic cell?
6. Draw a neat label diagram of Eukaryotic cell. Write down the important functions of cell organelles.
7. Draw a structure of mitochondria and write its various functions.
8. What is tissue? Write the classification with example of various tissues in human body.
9. Discuss the general characteristics and important functions of various tissues in human body.
10. What is neuron? Draw a label diagram of neuron. Differentiate between medullated and non-medullated neuron.
11. Define organ and system. Write down the name of important organs in human body and their location.
12. What are the major roles of various organs of human body? How digestive system, respiratory system and cardiovascular system are related to each other?
CHAPTER 3

Cardiovascular System

- Formed Elements of Blood
- Functions of Blood
- Blood Groups (ABO System)
- Lymphatic System
- Heart and Circulation
- Circulation of Blood through the Heart
- Rhythmicity of Heart Muscle
- Conducting System of Heart Muscle
- Blood Vessels (Artery, Vein and Capillary)
- Cardiac Cycle
- Electrocardiogram (ECG)
- Functional Capacity of Cardiovascular System
  - Cardiac Output
  - Effect of Training on Cardiac Output
  - Stroke Volume
  - Effect of Training on Stroke Volume
  - Heart Rate
  - Steady State Heart Rate
  - Factors Affecting Heart Rate
  - Effects of Training on Heart Rate
  - Blood Pressure
  - Selected Factors Affecting Blood Pressure
  - Effect of Training on Blood Pressure
- The Athlete’s Heart
- Functional Versus Pathological Hypertrophy

FORMED ELEMENTS OF BLOOD

Blood is a mobile connective tissue composed of corpuscles and a colorless fluid, the plasma. The corpuscles are amoeboid in shaped and are of two types. The pro-leukocyte corpuscles are small, but having proportionately large nuclei, which occupy the main space of the cells. The other type of corpuscles, the phagocytes, are large, engulf bacteria, other harmful microbes and foreign bodies.

**Definition:** Blood is an opaque, slightly alkaline, reddish colored, salty taste and viscous fluid connective tissue.

**Blood volume:** In normal condition an adult human (having normal body weight) contains about 5 to 6 liters of blood.

**Reaction of blood:** Blood is slightly alkaline in nature (pH of the blood is 7.4).

**Color of the blood:** Color of human blood is red. It is due to presence of respiratory pigment called hemoglobin.

**Composition of Blood**

Blood is a highly complex fluid which is composed of two parts—a liquid, called the plasma and different types of cells which remain suspended in the plasma. The cells are called the blood corpuscles. The plasma constitutes about 55% and the cells about 45% of the total volume of human blood. The general composition of the whole blood is as follows:

**Hemoglobin:** Hemoglobin is an iron containing red pigment of blood. It is protein in nature and consists of haem (4%) and globin (96%). Haem is an iron containing part, whereas globin is a simple protein.

Hemoglobin is found in red blood cells of all vertebrates. In normal condition 100 ml of human blood contains about 14.5 gm of hemoglobin in adult male (13.5 gm incase of female). When it falls below the normal level the condition is called anemia.
Plasma: Plasma is the pale yellow colored fiberless inter cellular alkaline fluid in which blood cells remains suspended.

The total amount of plasma (55%) of total blood, consists of 92 percent water and 8 percent solid substances. The solid remain dissolved in the plasma and are mainly of two types:

i. Inorganic constituents: The main inorganic constituents are Sodium, potassium, calcium, magnesium, iron, copper etc.

ii. Organic constituents: The main organic constituents are Proteins, Fat, Non-protein nitrogenous substances (NPN) and other organic substances.

Red Blood Corpuscles (RBCS): The red blood cells of human being are non-nucleated, bi-concave, circular disc shaped red colored cells of the blood. The cytoplasm contains respiratory pigment called hemoglobin and hence the cell looks red. The size of each matured red cell is 7.2 µm in diameter and 2.2 µm in peripheral thickness. The total number of RBC is present in adult males blood is about 5 million/mm³ of blood and in adult females it is about 4.5 million/mm³ of blood.

The red blood cells are formed from the red bone marrow and the process of formation of RBC in bone marrow is called erythropoiesis. The average life span of RBC is 120 days. At the old age the red cells broken by the friction of the capillary walls and the broken pieces are removed by the macro phages and other phagocytic cells present in the spleen, liver, etc.

White Blood Corpuscles (WBCS): The white blood cells are large in size, different shaped and nucleated colorless cells of the blood. The diameters of the WBC are bigger than the RBC and range from 8 µm to 18 µm. The cytoplasm of some of cells contains granules of different sizes and some cells do not contain granules. The leucocytes are nucleated cells are in round or kidney shaped or lobed. The number of WBC is 5000 to 8000 per cubic millimeter in adult human blood.

Types of WBC: White blood corpuscles are generally of two types, i.e. 1. Granulocytes and 2. Agranulocytes. Granulocytes again subdivided into different classes and Agranulocytes are of two types, i.e. Lymphocytes and Monocytes.

1. Granulocytes: The granulocytes having granular cytoplasm. They are formed in the bone marrow from the time of birth onwards. Three types of granulocytes are as follows:

a. Neutrophils: Neutrophils is about 10–12 µ in diameter. Neutrophils are granular leucocytes, which contain 2 to 7 lobed nucleus and fine cytoplasmic granules. The youngest cell has a single nucleus. The number of lobes increases with the degree of maturity of the cell. Due to presence of lobes in the nuclei these cells are called polymorphs. The number of neutrophil is 60 to 70 percent of total leucocytes. The average life span of neutrophil is 10 to 14 days.
b. Eosinophils: Eosinophils is about 10–12 µ in diameter. Eosinophils are granular leucocytes, which contain 2 to 3 lobed nucleus and coarse cytoplasmic granules. The number of eosinophil is 2 to 4 percent of total leucocytes. They are amoeboid but not phagocytic. The average life span of eosinophil is 8 to 12 days.

c. Basophils: The size of Basophil is about 8 to 10 µ. Basophils are also granular leucocytes, which contain bilobed kidney shaped nucleus and coarse cytoplasmic granules. The number of basophil is 0 to 1.0 percent of total leucocytes. They are actively amoeboid but are less so than the other varities of granulocytes. The average life span of basophil is 12 to 15 days.

2. Agranulocytes:
   a. Lymphocytes: Lymphocytes are non-granular and mononuclear leucocytes. According to the size they are of two types—small and large lymphocyte (7.5 µ and 12 µ respectively). The number of lymphocytes is 25 percent of total leucocytes. The average life span of lymphocyte is about 3 days.

   b. Monocytes: Monocytes are the biggest cell in the blood. They are non-granular, mononuclear leucocytes containing frosted cytoplasm. About 16 to 18 µ in diameter, the nucleus is round or oval when cells are young. The number of monocytes is 2 to 5 percent of total leucocytes. The average life span of lymphocyte is 2 to 4 days.

Functions of WBC:
   i. Protection: Neutrophil and lymphocytes protect the body against the invasion of germs and bacteria by their phagocytic nature (phagocytosis).
   ii. Synthesis: Basophils produce heparin (anticoagulant) and some WBC produce trephons (chemical substance for nutrition and growth of tissue).
   iii. Anti-allergy: The granulocytes, specially the eosinophil cells are very rich in histamine. They defend against allergic conditions in which histamine like bodies are produced in excess.
   iv. Repair: Lymphocytes may be converted into fibroblasts in an area of inflammation and thus help the process of repair.

Platelets or Trombocytes: The platelets are small colorless non-nucleated, oval or disc shaped cytoplasmic fragments found in the circulating mammalian blood. A matured platelet is a non-nucleated round or oval biconcave cytoplasmic disk. It measures about 2 to 5 µm in diameter. The adult human blood contains about 2.5 to 4.5 lacs of platelet per cubic millimeter of blood. It formed from the megakaryocytic cell of the bone marrow. The life span of platelets is 5 to 9 days.

Functions of Blood:
   i. Transport of nutrients: Digested food, vitamins and water are transported from alimentary canal to different parts of the body.
   ii. Transport of respiratory gases: Blood carries oxygen from lung to the tissues and carbon dioxide from tissues to the lungs.
   iii. Transport hormones: Blood carries hormones from the endocrine glands to the target organs for their effects.
   iv. Drainage of waste products: It carries metabolic wastes like urea, uric acid, hippuric acid, etc to the excretory organs (e.g., kidneys) for excretion.
   v. Maintenance of water balance: Blood helps to maintain the normal water content of the body.
   vi. Maintenance of acid base balance: Blood contains hemoglobin buffer, bicarbonate buffer, phosphate buffer, etc. which regulate the acid base balance of the body.
vii. Maintenance of body temperature: It regulates body temperature in warm-blooded animals. It transports heat from the deeper parts of the body to the surface for dissipation.

viii. Protective function: The leucocytes kill the germs and bacteria by their phagocytic activity. Gamma globulin of plasma protein forms antibody, which also protects the body.

ix. Acts as protein reserve: The plasma protein acts as protein reserve because during emergency tissue proteins are synthesized from plasma protein.

x. Regulates blood loss: The platelets in presence of fibrinogen and prothrombin helps to clot the blood (coagulation) and thus, prevents excess bleeding.

**Blood Coagulation:** The process by which the shedding of blood from the ruptured blood vessels (wound) loses its fluidity in a few minutes and sets into a semisolid jelly-like mass is known as coagulation. Normal coagulation time is measured according to the method of Lee and White; it is 6 to 17 minutes in glass tube and 19 to 60 minutes in siliconized tube. Normal average bleeding time in 3.25 minutes, the range being 2–5 minutes. It is usually determined by Duk’s method.

The phenomenon of coagulation is of enormous physiological importance. Its purpose is to stop haemorrhage. When bleeding occurs, the shed blood coagulates and the bleeding vessels become plugged off by the clot. The retraction of the clot compresses the ruptured vessels further and in this way bleeding stopped.

**Mechanism:**

**Normally in circulating blood:**
Platelets + Prothrombin + Fibrinogen → No clot.

**After shedding of blood:**

i. Broken platelets or damage tissues clotting factors → Thromboplastin

ii. Prothrombin thromboplastin Ca → Thrombin

iii. Fibrinogen thrombin Fibrin (clot)

**Function of blood coagulation:**

i. It stops hemorrhage.

ii. It blocks the entry of germs through wounds.

iii. It helps in maintaining normal blood volume.

**Serum:** It is the pale straw colored fluid formed from the plasma after clotting blood. It contains only serum albumin.

**BLOOD GROUPS (ABO SYSTEM)**

Intravenous administration of blood to help replenish excess blood loss due to hemorrhage or otherwise, is known as blood transfusion. It is a most effective therapeutic tool when applied properly. Karl Landstainer in 1900 AD was first realized the importance of blood group. Because it is necessary for blood transfusion, identifying a person according to the blood group and find defects in blood group. According to Landstainer, that human being can be divided into several groups. The salient facts about blood grouping are summarized below:

**Agglutinogen:** Some substance, which present on the surface of the red blood cells and responsible for the clumping of red blood cells is called agglutinogen.

There are two types of agglutinogen-

*Agglutinogen A and Agglutinogen B*

**Agglutinin:** The plasma (serum) contains two types of protein substances called agglutinin. Types: a or anti-A Agglutinin and b or anti-B Agglutinin

The human beings may be put in to four different groups according to the nature of the agglutinogen possessed by their corpuscles. These groups are called O, A, B and AB. The main four groups are as follows:

<table>
<thead>
<tr>
<th>Blood groups</th>
<th>Agglutinogen (In RBC)</th>
<th>Agglutinin (In serum)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A (42%)</td>
<td>A</td>
<td>(\beta)</td>
</tr>
<tr>
<td>B (9%)</td>
<td>(\alpha)</td>
<td>(\beta)</td>
</tr>
<tr>
<td>AB (3%)</td>
<td>A and B</td>
<td>Nil</td>
</tr>
<tr>
<td>O (46%)</td>
<td>Nil</td>
<td>(\alpha) and (\beta)</td>
</tr>
</tbody>
</table>

**Transfusion of Blood:** The process by which the blood of one-person flows through a sterilized tube and needle into the veins of the patient is termed as blood transfusion. People who give their blood for transfusion is called donor and who receives blood transfusion is called recipient.
The reactions between red cells and plasma of the four different blood groups are summarized in the following table.

<table>
<thead>
<tr>
<th>Donor’s group corpuscles Agglutinogens</th>
<th>Recipient-groups and serum Agglutinins</th>
</tr>
</thead>
<tbody>
<tr>
<td>AB o</td>
<td>B α</td>
</tr>
<tr>
<td>B</td>
<td>A O</td>
</tr>
</tbody>
</table>

‘+’ means agglutination, and ‘−’ means no agglutination.

Universal Donor: A man posses O-group blood can give blood to all without any reaction (agglutination) due to absence of agglutinogen but can receive blood only from a person having the ‘O’ group. This group of people is called Universal Donor.

Universal Recipient: A man posses AB-group can take blood from all without any reaction (agglutination) due to absence of agglutinin but can donate blood to the person only having the blood group ‘AB’. This group of people is called Universal Recipient.

Rh-factor: The another important agglutinogen has been demonstrated in human RBC by Landstainer and Wiener (1940). It is the agglutinogen of the Rhesus monkey and is present in 85 percent of white people. Amongst Indians the proportion is even larger, about 95 percent or more. There is no corresponding agglutinin in the human plasma. There are six Rh agglutinogens – C, c, s D, d, E, e. Among these ‘D’ and ‘d’ are the commonest. Group D and Dd (collectively called D group) will be Rh positive (Rh+ve) and d will be rh negative (rh−ve). As ‘D’ is Mendelian dominant and ‘d’ is recessive. The clinical importance of Rh factor is immense. If Rh−ve blood be transfused to a Rh+ve patient, an Anti-Rh factor will develop in the patients blood in about 12 days. If a second transfusion of same blood be given to such a patient after this period, haemoagglutination of the donor’s corpuscles will take place.

Immunity: The ability of an organism to resist the harmful effect of a foreign protein body and fight against disease is called immunity.

Function of immunity: It is concerned with detection, elimination of foreign substances, microbial organisms, dead tissues and fight against abnormal or mutant cells (cancer cells).

The microbial proteins or foreign particles entering the body are called antigens and the immune bodies against these antigens are called antibodies.

AIDS (Acquired Immune Deficiency Syndrome): This is a very fatal disease, which is caused by the infection of Human Immunodeficiency Virus (HIV). It is transmitted through blood transfusion, contaminated needles and sexual contact. The virus attacks cells of immune system and ultimately causes death of an individual.

M and N factors: Besides the ABO system, other supplementary agglutinogen have been identified. They are known as M and N factors. This will provide three other independent groups M, N and MN. These groups are of no importance for blood transfusion but have got medicolegal importance.

Lymphatic System

A system of vessels and nodes accessory to the blood vascular system, conveying lymph is called lymphatic system. It consists of lymph, lymphatic vessels and lymphatic glands.

Lymph: The alkaline clear, pale yellow color watery modified tissue fluid found in the lymphatic vessels is called lymph.

Function of lymph:

i. It transports nutrients and oxygen and supply them to tissue cells where the blood cannot reach directly.

ii. It absorbs fat from the intestine.

iii. Lymph drains away excess tissue fluid and metabolites.

iv. Lymphocyte and monocyte present in the lymph protect the body against foreign particles.

Spleen: It is the largest lymphatic organ in human body. It is an irregular somewhat flattened and elongated body of dark red color. It is located along the posterior margin of stomach in the abdominal.
HEART AND CIRCULATION

**Definition:** The hollow muscular pumping organ ever pulsatile and responsible for the circulation of blood in the body is called the heart.

**Location:** It lies in the central portion of the chest cavity, in between the two lungs, directly behind the sternum and above the diaphragm. It remains slightly tilted to the left side of the body.

**Structure:** The human heart is a conical hollow organ and made up of cardiac muscle. It remains covered by a protective double walled membranous sac called **pericardium.** The adult heart measures about 12 cm from base (upper broadest part) to the apex (lower conical part), 8 to 9 cm transversely at the broadest part and 6 cm anteroposteriorly. Its weight varies from 230 to 330 gm.

**Internal Structure:**

i. The human heart is divided by a longitudinal partition (septum) into the right and the left halves. Each half is further divided into two.

ii. Chambers: The upper two chambers of two halves are called **atria or auricle** and the lower two thick walled chambers are called **ventricles.** Thus, the whole heart consists of four chambers viz. a right atrium, a right ventricle, a left atrium and a left ventricle. The septum present in between the atria is called the **intra-atrial septum** and between the ventricles is called the **intraventricular septum.**

iii. The opening between the atria (auricles) and the ventricles of both sides are called the **atrioventricular apertures.** They are guarded by **atrioventricular valves (A-V valves).** Valve is the structure, which allows the blood to flow in one direction only and prevents back flow of blood, e.g. from atria to ventricles. The A-V valves present between the right atrium and the right ventricle, has three leaflets and are called the **tricuspid valves.** The left A-V valves have two leaflets and are called the **bicuspid valves or mitral valves.**

iv. The right atrium receives two great veins (vena cava) called the **superior and inferior vena cava.** The left atrium receives four **pulmonary veins** (a pair arising from each lung).
v. The pulmonary artery and the aorta, the great artery, originate from the right and the left ventricles respectively. The bases of these arteries are guarded by the valves commonly known as semilunar valves. The valves at the base of pulmonary artery and aorta are called pulmonary valves and aortic valves respectively.

Circulation of Blood through the Heart

William Harvey first demonstrated Circulation of blood in human. Circulation of blood through the heart is unidirectional due to the presence of valves in the several openings of the heart. Due to continuous contraction (systole) and relaxation (diastole) of the heart, the blood circulates within it as well as throughout the body.

Steps of circulation:

i. At first the venous blood (less O₂ and more CO₂ containing blood) from the upper and lower part of the body comes through the superior vena cava and inferior vena cava respectively into the right atrium.

ii. From the right atrium the venous blood passes into the right ventricle by opening the tricuspid valves which is located at the junction of right auricle and ventricle.

iii. After contraction of the right ventricle, expels the blood into the lungs through the pulmonary artery for oxygenation.

iv. In the lungs the venous blood (less oxygenated blood) is converted into arterial blood (oxygenated blood) after taking oxygen from the lungs alveoli and giving up CO₂.

v. This arterial blood returns from the lungs through the four pulmonary veins into the left atrium.

vi. From left atrium the oxygenated (arterial) blood passes into the left ventricle by opening the bicuspid or mitral valves present at the left atrio-ventricular opening.

vii. At last during ventricular contraction the arterial blood from the left ventricle passes into the aorta by opening the aortic valves present at the base of the aorta.

viii. The aorta divides and subdivides to form a network of extremely fine vessels called capillaries within the tissues present in different parts of the body. The arterial blood supplies oxygen and nutrition to the tissues and takes carbon dioxide from the tissues.

ix. In this way the arterial blood is converted to venous blood once again which returns to the heart through the veins.
Internal view of human heart

**Fig. 3.6:** Internal view of human heart and its circulatory path

Conduction of impulse through heart

**Fig. 3.7:** Conduction of impulse through heart
Circular Path of Blood Circulation within Heart and Body

Vena cava → Right Atrium → Right Ventricle → Pulmonary Artery → Lungs → Pulmonary Veins → Left Atrium → Left Ventricle → Aorta → Capillaries

RHYTHMICITY OF HEART MUSCLE

In a healthy adult person, the heart beats, i.e. contraction and relaxation of the heart take place rhythmically 70 to 80 times (72 times in average for adult man) per minute.

Rhythmicity is the characteristic property of the heart muscle. It depends on the rate of initiation of cardiac impulse from the Sino-atrial node (SA node). SA node initiates impulse 70 to 80 times per minute and similarly the heart also beats 70 to 80 times per minute. Thus, SA node is known as Pacemaker.

The other junctional tissues present within the heart are atrio-ventricular node (A-V node), Bundle of His and Purkinje fibers. These tissues have the ability to initiate and to conduct the cardiac impulse.

CONDUCTING SYSTEM OF HEART MUSCLE

a) Conduction over atrial muscle: Cardiac impulse originated at the SA node is transmitted over both the atria (Fig. 3.7). The spread of electrical impulse through the SA node is very slow (0.05 m/sec.) but the same through the junctional tissues that connect the node to the atrial musculature or to the AV node is higher (1m/sec.).

b) Conduction over AV node: The SA node sends impulses along certain pathways, causing the atria to contract when the electrical signal reaches them. The impulse then arrives at another node, called the atrio-ventricular node, or AV node. There is also a considerable delay of 0.07 to 0.1 sec. in transmission of impulse in the AV node before excitation spread over the ventricle. This AV nodal delay allows the atrial systole to complete before the ventricle is excited. This delay is observed maximally at the junctional region between the atrium and atrio-ventricular node. The conduction velocity of impulse at this region in about 0.05m/sec

c) Conduction over bundle of His: Beyond the atrio-ventricular region, the impulse is transmitted along the bundle branch at a higher velocity (4 to 5m/sec.). The impulse from the bundle of His passes quickly through the right and left bundle branches and ultimately reaches the Purkinje fibers and ventricular muscle fibers as well.

d) Conduction through purkinje system: The impulse after passing through the right and the left bundle branches, passes into the Purkinje fibers and also its multiple ramifications with in the sub-endocardial surfaces of both the ventricles.

![Fig. 3.8: Path of cardiac impulse in the heart.](image-url)

![Fig. 3.8(A and B): Cross section of (A) Artery and (B) Vein](image-url)
c) **Conduction through ventricular muscle:** In human, the mid portion of the intraventricular septum is activated normally in a left to right direction, so the depolarization of the ventricular muscle begins at the left side of the septum. After mid-septal activation from the left to the right direction the impulse comes down the septum to the apex of the heart and next portion of the myocardium.

### Blood Vessels (Artery, Vein and Capillary)

a) **Arteries:**
   i. Arteries carry blood away from heart.
   ii. All arteries except pulmonary artery carry oxygenated blood. Pulmonary arteries carry deoxygenated blood from right ventricle to lung.
   iii. Arteries have thick, elastic and muscular walls.
   iv. Arteries branch into small arteries, arterioles and end in capillaries.
   v. Arteries are distributing vessels. These carry blood from heart to various body organs.
   vi. Arteries are deeply situated in the body.
   vii. These carry blood under pressure and blood flows with jerks.
   viii. Their lumen is without valves.

**Structure of artery:** The wall of an artery consists of three layers or tunics such as:

i. **Tunica adventitia:** It is the outermost layer. It is made up of white fibrous connective tissue that carries small vessels and nerves to nourish the arterial wall.
ii. **Tunica media:** It is the middle thick coat. It is formed of smooth muscle fibers and connective tissue.
iii. **Tunica intima:** It is the inner thin and delicate lining of endothelial cells.

b) **Veins:**
   i. A vein carries blood towards heart.
   ii. All the veins except pulmonary veins carry deoxygenated blood.
   iii. Veins have thin, less muscular walls.
   iv. Small veins are called venules. These are formed from capillaries and join to form veins.
   v. Veins are collecting vessels. These collect blood from various parts of the body and empty in the heart.
   vi. Veins are superficially situated and can be seen from the surface of skin.
   vii. Blood flows smoothly through them.
   viii. The veins have internal valves to prevent back flow of blood.

The wall of veins like arteries is composed of the same layers, but (a) the muscle layer is thin, (b) have a wide lumen, (c) their tunica adventitia is without nerve fibers, (d) veins collapse when cut.

c) **Capillaries:** Capillaries are microscopic vessels that carry blood from arterioles to small veins or venules. The wall of capillaries is formed of a single layer of endothelial cells. These lie in contact with the body tissues. These supply food and oxygen to the tissue cells and remove injurious wastes. The leucocytes squeeze out through the capillary walls into the surrounding tissue to attack the invading bacteria. The phenomenon of squeezing out of leucocytes is called diapedesis.

**Sinusoids:** Sinusoids and sinusoidal capillaries are not true capillaries and they have got relatively large calibre (30 μ) with irregula and tortuous walls. Continuous endothelial lining is absent. There is also some incomplete lining of Phageytic cells. Due to absence of basal lamina, the blood gets direct contact with the tissue cells.

**Heart sounds:** Each heartbeat is accompanied by two heart sounds. With a stethoscope, these sounds are heard as ‘lubb’ and ‘dup’.

i. The lubb (first sound) sound is lower and lasts longer. It is produced by the contraction...
of ventricular muscles and the vibrations set up by the closure of tricuspid and bicuspid valves. It is also called systolic sound.

ii. The dub sound (second sound) is caused by the closure of semilunar valves of the aorta and pulmonary trunk. It is diastolic sound.

It is short and sharp.

iii. Heart murmur is the abnormal sound produced either by incomplete closing of valves (valvular in sufficiency) or by their narrowing (stenosis).

**Pulse rate:** The beating of the heart is also felt in the arteries as regular jerks, called pulse. Each ventricular systole starts a new pulse. It produces as a wave of expansion throughout the arteries disappearing in the capillaries. The pulse rate is same as the heart rate.

Pulse can be felt wherever an artery lies near the surface, such as radial artery at the wrist; temporal artery in front of ear; common carotid artery in the neck, facial artery on the corners of mouth, the brachial artery at the bend of elbow and popliteal artery in the leg near ankle bone.

**The Heart Beat**

Working of heart includes rhythmic contraction and relaxations of auricles and ventricles. The contraction phase is called systole and the relaxation phase is diastole. A heart beat includes one systole and one diastole. The auricles and ventricles do not contract simultaneously. The heart beat is completed in following stages:

**Stages 1:** Simultaneous contraction of both the auricles pushing the blood into ventricles, which are in relaxing, phase.

**Stages 2:** Simultaneous contraction of ventricles forcing blood into the aorta and the pulmonary trunks. Along with it the auricles start relaxing.

**Stages 3:** In this stage both ventricles and auricles are relaxed as in diastole. This stage is called the general pause or joint diastole. During this stage blood enters into the auricles from the great veins (superior and inferior vena cava). At the end of this phase the next heartbeat starts with the contraction of auricles.

The normal heart beats of young adult is about 70 to 74 times a minute. This is also called heart rate or pulse rate. At each heart beat ventricles pump about 70 ml bloods. This volume is termed as stroke volume. It means heart pumps about 72 x 70 ml or 5040 ml blood per minute (5 liters approximately). This is called cardiac output (CO). This can be calculated by:

$$ CO = HR \times SV $$

(Cardiac output) (Heart rate) (Stroke volume)

**CARDIAC CYCLE**

**Definition:** Changes that occur in the heart during one beat are repeated in the same order in the next beat. This cyclical repetition of the various changes in heart, from beat to beat is called cardiac cycle.

**Cardiac cycle time:** This is the time required for one complete cardiac cycle. The normal time is 0.8 sec. Every event in the cycle will be repeated at the interval of 0.8 sec.

**Various events in the cardiac cycle:** In the cardiac cycle there are four main events such as Atrial systole, Atrial diastole, Ventricular systole and Ventricular diastole.

**The sequence of events in cardiac cycle:**

i. The atrial systole is the first event (0.1 sec). It initiates the cardiac cycle, because the pacemaker SA node is situated here. Due to higher atrial pressure, the first half of atrial systole is stronger than that of the last half.

ii. After systole, the atrial diastole begins (0.7 sec). This atrial systole and atrial diastole follow each other and constitute the atrial cycle (0.8 sec).

iii. Just after the atrial systole, the ventricular systole (0.3 sec) begins and is immediately followed by its diastole (0.5 sec). These two events repeat alternately and make-up the ventricular cycle (0.8 sec).

iv. At the onset of ventricular systole, the AV valves close producing the first sound. The semilunar valves open a little later. The interval between the closing of the AV valves and opening of the semilunar valves is called the isometric contraction period (0.05 sec). During this period ventricles contract as closed cavities and intraventricular pressure steeply rises.

v. After this phase, comes the ejection period—when blood is pumped out of the ventricles.
The first part of this period, when the outflow is very rapid, is called the maximum ejection period (0.11 sec). The second part, when the rate of flow slows down, is known as the reduced ejection period (0.14 sec). Here, ventricular systole ends and diastole begins.

vi. At the beginning of ventricular diastole, the semilunar valves close producing the second sound. There is a brief interval between the beginning of diastole and the closure of the semilunar valves—known as the protodiastolic period (0.04 sec). So that, second sound occurs actually after this period. The AV valves open a little after the closing of the semilunar valves. The interval between these two is called the isometric relaxation period (0.08).

vii. During these periods ventricles relax as closed cavities and intraventricular pressure steeply falls. At the end of this period, the intraventricular pressure goes below that of the atria and the AV valves open. Arterial blood rushes into the ventricles producing the third sound.

viii. Third sound begins the first part of filling which is very rapid and as known as the first rapid filling phase (0.113 sec). The maximum filling takes place during this brief period.

ix. The intermediates part of filling is very slow and as known as diastasis or slow inflow phase (0.16 sec).

x. The last part of diastole corresponds with the atrial systole. Due to active contraction of the atria, filling becomes very rapid.

xi. The last rapid filling phase (0.1 sec) is responsible for the last part of ventricular filling. Due to rapid rush of blood, another sound is produced is called fourth sound of heart.

xii. Ventricular diastole ends and systole commences again. In this way the cardiac cycle continues.

**Electrocardiogram (ECG)**

A graphic record of the electrical variations produced during a heart beat or cardiac cycle is called electrocardiogram. The electrical variations include depolarization and repolarization of the auricle and ventricle bringing about their contraction and relaxation. The instrument used to observe the working of heart is called as

![Fig. 3.10: Sequence of events during cardiac cycle](image_url)
A Textbook of Sports and Exercise Physiology

Electrocardiograph. Electric current produced by the heart is picked up by the electrodes attached in different positions on the body, viz. two arms, left leg and chest passed through a galvanometer fitted in the electrocardiograph.

Electrocardiographic leads used both clinically and experimentally. When the connections are made between two parts of the body, then this specific arrangement of each pair of connections is designated as lead. The different leads that are conventionally used are (i) standard limb leads (I, II and III) (ii) Chest leads (v1, v2, v3, v4, v5, v6) and (iii) Augmented unipolar limb leads (avR, avL and avF).

A typical electrocardiogram of a healthy person shows five waves (fig. 3.10). These are called P, Q, R, S and T. The P, R and T waves which are above the base line of ECG are known as positive waves. The Q and S waves that lie below the base line are called negative waves. The Q, R and S waves are sharp whereas P and T waves are blunt. The part of the base line between any two deflections is called interval.

In the above figure, P wave indicates the impulse of contraction generated by SA node which causes atrial depolarization. The interval PQ represents atrial contraction. This takes 0.1 second. QRS wave indicates spread of impulse of contraction from AV node to the wall of ventricles causing ventricular depolarization. The RS and QRS complex and ST interval shows contraction of ventricles. T wave indicates the relaxation of ventricles. The QRST complex takes about 0.3 seconds. So, ECG is formed of both depolarization and repolarization of the heart muscle.

Any abnormality in the working of heart changes the wave pattern of ECG. A cardiologist or physician can find out the defect in the heart by examining the ECG. Thus, ECG is of immense diagnostic value in cardiac diseases.

Often electrocardiograms are obtained during exercise. These are valuable diagnostic tests. As exercise intensity increases, the heart must beat faster and work harder to deliver more blood to active muscles for adequate energy. If the heart is diseased, an indication may show upon the

![Fig. 3.11: Normal electrographic patterns](image)

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electrocardiogram as the heart increases its rate of work. Exercise ECGs have also been invaluable tools for research in exercise physiology because they provide a convenient method for tracking cardiac changes during acute and chronic exercise.

**FUNCTIONAL CAPACITY OF CARDIOVASCULAR SYSTEM**

**Cardiac Output**

*Definition:* Cardiac output is defined as the volume of blood pumped by the heart in one minute. This is generally expressed as liters per minute and milliliters per minute. It is the product of heart rate and stroke volume (the amount of blood pumped with each beat of the heart). For example, if heart rate equals 72 beats/min and stroke volume equals 70 ml of blood, then cardiac output is equal to 5,040 ml per minute or 5.04 liters per minute (72 × 70).

Cardiac output can also be calculated from the amount of oxygen consumed per minute and the amount of oxygen taken up by the blood as it flows through the lungs. These relationships are expressed by the *Fick Principle*, which is as follows:

\[
\text{Cardiac output} = \frac{\text{Oxygen consumed per minute by the body (ml/min)} \times 100}{\text{Arterial O}_2\text{ content} - \text{venous O}_2\text{ content (ml O}_2\text{ per 100 ml of blood)}}
\]

For example, if the oxygen content of the venous blood entering the lungs is 16 volume percent (16ml of oxygen per 100ml of blood), that of the arterial blood leaving the lungs is 20 volumes percent (20ml of oxygen per 100ml of blood) and the oxygen consumption of the body 200ml per minute, the amount of oxygen used per minute equals the amount of oxygen taken up by the lungs per minute. From the above data we can see that each 100 ml of blood flowing through the lungs picks-up 4 ml of oxygen. And since the total amount of oxygen absorbed into the blood from the lungs each minute is 200 ml, a total of fifty 100 ml portions of blood must flow through the lungs each minute to absorb this amount of oxygen. Thus, the cardiac output is:

\[
\frac{200}{20 - 16} \times 100 = 5,000 \text{ ml.}
\]

**Factors affecting cardiac output:**

i. Metabolic rate: The output is directly proportional to the metabolic rate.

ii. Body surface area: The output is also directly proportional to the body surface area.

iii. Body weight: Like metabolic rate the output is directly proportional to the body weight.

iv. Muscular Exercise: In many exercise the output may be increased 6-10 times the normal value.

v. Posture: The minute volume is greater in the recumbent posture than in standing, because gravity retards venous return in the later.

![Fig. 3.12: Cardiac output in trained and untrained person during exercise](image)
Cardiac output during rest: During rest in the supine position, the normal cardiac output in adults is approximately 5 liters per minute. This is generally achieved with a heart rate of 70 beats per minute for the untrained person and 45 beats per minute for endurance trained person. Since the trained person’s cardiac output at rest is also about 5 liters, then the decrease in heart rate must be offset by an increase in stroke volume if the cardiac output is to remain normal.

\[
\text{Rest cardiac output} = \text{heart rate} \times \text{stroke volume}
\]

- Untrained, 5000 ml = 70 beats per minute \times 71.4 ml
- Trained, 5000 ml = 45 beats per minute \times 111.1 ml

Substituting the heart rate value in the cardiac output formula, the calculated stroke volume for the untrained person would be around 71.4 ml of blood per beat, whereas the stroke volume for the trained person would be about 111.1 ml per beat.

In this connection it should be mentioned that since blood generally pools in the lower portions of the body under the influence of the gravity when assuming a sitting or standing position, this results in a drop in venous return to the heart and thus, a 1 to 2 liters per minute reduction in cardiac output is occurred. Since the heart rate is usually decreased, it is generally believed that this reduction is due entirely to a decrease in stroke volume.

Cardiac output during sub-maximal exercise: During sub maximal exercise (40 to 60 percent of maximal capacity), cardiac output in trained athletes may be increased to 40 liters per minute, whereas untrained subjects may attain outputs of about 20 liters per minute. At this level of work, it is known that this 5 to 7 folds increase in cardiac output is due to increase in both heart rate and stroke volume.

At levels beyond 40 to 60 percent of maximum, increase in cardiac output are mainly functions of heart rate increases.

\[
\text{Exercise cardiac output} = \text{heart rate} \times \text{stroke volume}
\]

- Untrained 20,000 ml = 200 beats per minute \times 100 ml
- Trained 40,000 ml = 200 beats per minutes \times 200 ml

Cardiac output during maximal exercise: During maximum exercise, it should be emphasized that since heart rate in strenuous exercise increase approximately the same in both athletes and non-athletes, the greater changes in cardiac output attained by the trained athletes is due to their greater ability for increasing the stroke volume of the heart.

The heart rate values in the cardiac output formula, the calculated stroke volume for the untrained person would be around 100 ml of blood per beat, whereas the stroke volume for trained person would be approximately 200 ml per beat.

Effect of Training on Cardiac Output

The stroke volume and heart rate are the two components of cardiac output and stroke volume is increase and heart rate is decrease due to training. When at rest or during submaximal exercise at standardized work rates, cardiac output does not change much following endurance training. However, cardiac output increases considerably at maximum rates of work. This results primarily from the increase in maximal stroke volume because \(HR_{max}\) Maximume heart rate changes little, if any.

Stroke Volume

During systole, a certain volume of blood is ejected from the left ventricle. This amount is the stroke volume (SV) of the heart, or the volume of blood
pumped per stroke (contraction). For example, consider the amount of blood in the ventricle before and after contraction. At the end of diastole, just before contraction, the ventricle has completing filling. The volume of blood it now contains is called the end diastolic volume (EDV). At the end of systole, just after contraction, the ventricle has completed its ejection phase. The volume of blood remaining in the ventricle is called the end systolic volume (EVS). Stroke volume is the volume of blood that was ejected, and is merely the difference between the amounts originally there and the amount remaining in the ventricle after contraction. So, stroke volume is simply the difference between the EDV and ESV. The normal value of the stroke volume of sedentary young adult is about 70 ml.

\[
\text{EDV (100 ml.)} - \text{ESV (30 ml.)} = \text{SV (70 ml)}
\]

**Stroke volume in exercise:** The stroke volume increases with increasing rates of work but only up to exercise intensities between 40 percent and 60 percent of maximal capacity. When the body is in upright position, stroke volume almost doubles from resting to maximal values. For example, in active but untrained individuals, it increases from about 50 to 60 ml at rest to 100 to 120 ml at maximal exercise. In highly trained endurance athletes, stroke volume can increase from 80 to 110 ml at rest to 160 to 200 ml at maximal exercise. During supine exercise, such as swimming, stroke volume also increases, but usually by only about 20 to 40 percent, not nearly as much as in an upright position.

**Effect of Training on Stroke Volume**

Endurance training increases the stroke volume. Stroke volume at rest is substantially higher after an endurance training program than it is before training. This training induce increase is also seen during both standardized sub maximal exercise and maximal exercise.

After training, the left ventricle fills more completely during diastole than it does in an untrained heart. It is well known that the blood plasma volume increases with training, which means more blood is available to enter the ventricle, causing an increased end-diastolic volume (EDV). More blood entering the ventricle increases the stretching of the ventricular walls.

It is also well known that the posterior and septal walls of the left ventricle hypertrophy occur with endurance training. Increased ventricular muscle mass can cause more forceful contraction. This increase contractility would cause the end – systolic volume (ESV) to decrease because more blood would be forced out of the heart during the more powerful contractions, leaving less blood in the left ventricle after systole.

Increased contractility coupled with the increased elastic recoil that results from greater diastolic filling increase the ejection fraction in the trained heart. More blood enters the left ventricle, and a greater percentage of what enters is forced out with each contraction, so stroke volume is increased.

**Heart Rate**

The cardiac muscle has got the unique property of rhythmicity, i.e. rhythmic contraction and relaxation. The impulse causes the rhythmic contraction and relaxation of heart muscle is originate with in the heart muscle itself, in the right atrium known as the S-A node and as a result blood circulates throughout the body from the heart and return from the body to the heart respectively. This rhythmic contraction and relaxation of the heart is

![Changing in stroke volume with increasing rate of work](image-url)
known as heart rate. Normally in human sedentary adult male’s heart rate is 72 beats per minute.

It is one of the simplest and most informative state of the cardiovascular parameters. It involves simply taking the subject’s pulse, usually at the radial or carotid site.

Resting heart rate: Resting heart rate averages 60 to 80 beats per minute. In middle aged, unconditioned, sedentary individuals the resting heart rate can exceed ever 100 beats per minute. In highly conditioned endurance trained athletes, resting heart rates in the range of 36 to 40 beats per minute have been reported. The resting heart rates typically decrease with the age. It is also affected by environmental factors; for example, it increases with extremes in temperature and altitude.

Measuring the resting heart rate of a person is completely mentally and physically in resting condition i.e. lying on the bed in the early morning.

Maximum heart rate: The maximum heart rate (HR$_{max}$) is the highest heart rate value one achieves in an all-out effort to the point of exhaustion. This is a highly reliable that remains constant from day-to-day and changes only slightly from year to year. Estimates of maximum heart rate can be made based on age because maximum heart rate shows a slight but steady decrease of about 1 beat per year beginning at 10 to 15 years of age. An individual’s maximum heart rate can be calculated by the following equation:

$$HR_{max} = 220 - \text{age in years}$$

Subtracting the age from 220 provides an approximation of average maximum heart rate. However, this is only estimation—individual values may vary considerably from this average value.

Steady State Heart Rate

When the rate of work is held constant at submaximal levels of exercise, heart rate increases fairly rapidly until it reaches a plateau. This plateau is the steady state heart rate, and it is the optimal heart rate for meeting the circulatory demands at that specific rate of work. For each subsequent increase in intensity, heart rate will reach in new steady state value within 1 to 2 minutes. However, the more intense the exercise, the longer it takes to achieve this steady state value.

The concept of steady state heart rate forms the basis for several tests that have been developed to estimate physical fitness. In one such test, individuals are placed on an exercise device, such as a cycle ergometer and are exercised at two or three standardized rates of work. Those in better physical condition, based on their cardiorespiratory endurance capacity, will have lower steady state heart rates at a given rate of work than those who are less fit. Thus, steady state heart rate is a valid predictor of cardio-respiratory efficiency—a lower rate reflects a more efficient heart.

Factors affecting Heart Rate

Various factors have influenced the resting heart rate such as posture, age, sex, emotions, environment, exercise, etc.

i. **Posture:** The body position has a definite effect upon the heart rate. Generally the rate is lowest in the recumbent followed by the sitting and standing. It appears that the typical response from the recumbent to the standing position is an increase of around 10 to 12 beats per minute. This is due to the influence of gravity, which lowers the volume of blood returning to the heart when one goes from a reclining position to a sitting or standing position.
ii. Age: The resting heart rate drops progressively from birth to adolescence but in old age it again increases slightly. Also maximum heart rate decreases gradually with the advancement of age.

iii. Sex: The adult female’s resting heart rate is some 5 to 10 beats per minute faster than that of the adult male under any given situation. The average resting heart rate is approximately 76–78 beats per minute for adult females. The male has a greater heart volume than the female. This heart size difference is apparently the cause for the 5 to 10 beats per minute faster resting heart rate in women. Although the maximum heart rate in both men and women at a given age is very similar, the heart rate of women during submaximal work has been found to be considerably higher.

iv. Emotion: Emotional stress accelerates the resting as well as the exercise heart rate. Although an increased heart rate is most easily observed during rest in people as an anticipatory reaction, emotion may also result in an excessive cardio respiratory adjustment during light exercise. On the other hand, emotion probably has little effect on the maximal heart rate as well.

v. Environmental factors: The influence of environmental factors on heart functions is that a high temperature and altitude may greatly increase the heart rate. In addition, for any standard temperature and workload, the increase in heart rate will be significantly greater if the air is still and the humidity is high.

vi. Body size: In the animal in general, it seems to be a biological rule that the heart rate varies inversely with the size of the species. For example, the Canary has a rate of approximately 1000 beats per minute, whereas that an elephant is about 25 beats per minute. However, no consistent relationship between size and the heart rate in adult humans has been demonstrated. Ingestion of food the resting heart rate is higher while digestive process is in progress than in the post absorptive state. This is also true in exercise. A given exercise load elicits a greater heart rate after a meal, one of many reasons that melted against heavy exercise immediately after a meal.

vii. Body temperature: With increase in body temperature above normal, the heart rate increases. Conversely with decrease in temperature, the rate slows until a temperature of about 26°C is reached.

viii. Smoking: It has been observe that smoking even one cigarette significantly increases the resting heart rate, in either the sitting or the standing position.

Heart rate response to exercise: The heart rate response is directly proportional and linear to the intensity of exercise. As intensity of exercise increases, the heart rate will continue to increase until exercise reaches at maximal intensity. Based on the intensity of exercise in two different work load, i.e. submaximal and maximal, heart rate response is vary.

Heart rate response to submaximal exercise: The heart rate response is linear at the beginning of the exercise (at certain work load) and if the intensity of exercise is not been increased further the heart rate also will not be increased. If the intensity of exercise is submaximal the heart rate become steady though the exercise being continued for longer period.

Heart rate response to maximal exercise: At maximal intensity, the heart rates will plateau, indicating that
the individual is reaching his or her maximal level. Above this the individual will not able to continue the exercise or activity.

Effects of Training on Heart Rate

Resting heart rate: The heart rate at rest decreases markedly as a result of endurance training. The sedentary individual with an initial resting heart rate of 80 beats per minute, the heart rate will decrease by approximately 1 beat per minute each week for the first few weeks of training. So after 10 weeks of moderate endurance training, the resting heart rate should drop from 80 to 70 beats per minute. Highly conditioned endurance athletes often have resting heart rates of average 42 to 45 beats per minute, and some have even values lower than 36 to 38 beats per minute has been reported.

Sub maximal heart rate: During submaximal exercise, greater aerobic conditioning results in proportionally lower heart rate at a specified rate of work. The figure shows that the heart rate of an individual exercising on a treadmill both in before and after training. At each specified work rate, indicated by the speed at which the subject is walking and running, the post training heart rate is lower than the heart rate before training. Following a six months endurance training program of moderate intensity, heart rate decreases of 20 to 40 beats per minute are common at a standardized submaximal rate of work.

These decreases indicate that the heart becomes more efficient through training. In carrying out its necessary functions, a conditioned heart performs less work than an unconditioned heart.

Maximum heart rate (HR$_{max}$): At maximal rate of exercise, HR$_{max}$ usually remains relatively unchanged following endurance training. However, the untrained people who have maximum heart rate more than 180 beats/min. HR$_{max}$ slightly reduced following training.

During exercise, the heart rate combines with the stroke volume to provide an appropriate cardiac output for the rate of work performed. At maximal or near maximal rate of work, the body might adjust the heart rate to provide the optimal combination of heart rate and stroke volume to maximize the cardiac output. If the heart is too fast, diastole, the period of ventricular filling is reduced and the stroke volume might be compromised. For example, if your HR$_{max}$ is 180 beats per minute, your heart beats three times per second. Each cardiac cycle thus, last for only 0.33 seconds. Diastole is as short as 0.150 seconds or less. This allows very little time for your ventricle to fill. As a consequence, your stroke volume could decrease.

However, if your heart rate slows, the ventricles would have longer to fill. Perhaps this is why highly trained endurance athletes tend to have lower HR$_{max}$ values – their hearts have adapted to training by drastically increasing their stroke volumes so lower HR$_{max}$ values can provide optimum cardiac output.

Fig. 3.17: Changes in submaximal heart rate with endurance training
**Recovery heart rate**: During exercise the heart rate must increase to meet the demands of active muscles. When the exercise bout is finished, the heart does not initially return to its resting level. Instead, it remains elevated for a while, slowly returning to its resting level. The time it takes for heart rate to return to its resting rate is called the heart rate recovery period. Following a period of training the heart rate returns to its resting level much more quickly after exercise than it does prior to training.

The heart rate recovery period is shortened by endurance training; this measurement can be used as an index of cardiorespiratory fitness. In general, a more fit person recover faster after a standardized rate of work than a less fit person.

**Blood Pressure**

**Blood Pressure**: It is a lateral pressure exerted by blood on the walls of blood vessels while flowing through, is known as blood pressure.

**Measurement of Blood Pressure**

Blood pressure may be determined either by indirect (Sphygmomanometer) or direct (catheter) methods. The indirect method is the more common one used in physical education and exercise physiology. In this method commonly the pressure of the brachial artery is measured. The instrument used is known as Sphygmomanometer. There are three methods such as oscillatory, palpatory and ausculatory. Four terms are in common uses which are as follows:

- **Systolic pressure**: Maximum pressure on blood vessels during contraction (systole) of heart. The normal value of systolic pressure is: 110 to 120 mm Hg for young adult.
- **Diastolic pressure**: Minimum pressure on blood vessels during relaxation (diastole) of heart. The normal value of diastolic pressure is: 70 to 80 mm Hg for young adult.
- **Pulse pressure**: It is the difference between systolic and diastolic pressure. The normal value of pulse pressure is 40 mm Hg.
- **Mean pressure**: It is the average value of total systolic and diastolic pressure. The normal value of mean pressure is 90 to 100 mm Hg.

**Exercise Responses**: During exercise, blood pressure increases linearly as a result of an increase in cardiac output. The exercise affects on systolic blood pressure is much more than diastolic or mean pressure. This is due to the fact that during exercise the resistance to blood flow is decreased. The decreased resistance is the result of vasodilatation taking place in the arterioles or the working
muscles. For example, resistance to blood flow can be determined by using the following formula:

\[
\text{Resistance} = \frac{\text{Mean arterial pressure}}{\text{Cardiac output}}
\]

**Rest Conditions**

Cardiac output = 4.5 liters/min  
Mean arterial pressure = 93 mm Hg  
Resistance to blood flow = 20.67 mm Hg/liter/min

**Exercise Conditions**

Cardiac output = 35 liters/min  
Mean arterial pressure = 126 mm Hg  
Resistance to blood flow = 3.60 mm Hg/liter/min

On the other hand, more blood will be able to move from the arteries into the muscle capillaries with only slight changes in diastolic pressure. There is an over 5-fold decrease in resistance between rest and exercise. This is definitely an advantage during exercise. Changes in mean pressure will also be minimized since any mean arterial pressure increases (with increasing cardiac output) will be offset with the decreased resistance.

The type of exercise influences the increase in blood pressure. For example, typical changes during an isometric handgrip contraction (where resistance is equal to 30 percent of the maximum voluntary force) and an exhausting progressive treadmill exercise (dynamic). The tremendous increase in blood pressure compared to heart rate response as well as the parallel increase in both systolic and diastolic pressures during isometric work. Some researchers have found small increases in heart rate, cardiac output, blood pressure and muscle blood flow to a steady state when isometric contractions are less than 15 percent of the maximum force. Generally, exercises of this type can be maintained indefinitely. However, when isometric contractions are greater than 15 percent of maximum the blood pressure increase accordingly.

All of the above variables increase continuously until fatigue occurs. Isometric exercise causes a much greater increase in blood pressure than did the dynamic exercise. Apparently, the body tolerates the increased blood flow of dynamic, rhythmic type activities such as running or jogging better than increased blood pressure by isometric exercise. It is commonly believed among exercise physiologists that the increase in pressure from isometric type exercise is brought about, in part, by a nerve reflex originating in the exercising muscles and also by an increase in intrathoracic pressure (caused from making an expiratory effort against a closed glottis and thus, restricting venous return to the heart).

It is interesting to note that when works dynamically with small muscles such as the arms instead of the legs, a greater than normal increase in blood pressure is also generally observed. This

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**Fig. 3.19:** Blood pressure response to both leg and arm cycling at the same absolute rate of O₂ consumption  
(Adapted from Wilmore and Costill, 1994)
is especially true when one works with the arms such as in snow shoveling or digging or in work above the waist such as in painting or doing carpenter-type work. Because of the uncommonly high blood pressure produced by isometric or dynamic arm work performed above the waist as in snow-shoveling or digging, this type of work is not recommended for older people and people with cardiovascular disease.

Selected Factors Affecting Blood Pressure
There are several factors that affect blood pressure besides exercise and training such as age, sex, emotion, posture etc.

a. **Age:** There is an influence of age on the systolic and diastolic blood pressure. Normally, the blood pressure increases gradually throughout life. The normal systolic blood pressure for adults in western industrialized societies is around 140 mm Hg, while the diastolic pressure is around 90 mm Hg. The average blood pressure tends to be lower in underdeveloped countries than in western societies. On an individual basis, it should be pointed out that pressure values above 140 mm Hg do not necessarily indicate an abnormal state of high blood pressure since the physiological range of normal for some individuals may occasionally reach into the range of abnormal for the total population. In fact, in older people, systolic values of 160 to 170 mm Hg are accepted as normal if there is not much symptom reported.

b. **Sex:** Both the systolic and diastolic blood pressure values in women prior to menopause tend to be about 5 to 10 mm Hg lower than that of the male. However, after menopause the female values are generally found to be slightly higher than their counter parts of similar age.

c. **Emotion:** It is well known that emotional state such as excitement, fear and anxiety increase the arterial blood pressure. In fact, the slightest emotional involvement may cause falsely high results in blood pressure determinations.

d. **Posture:** As is the case of the heart rate, the blood pressure is also affected by posture. When a reclining subject stands up, the hydrostatic pressure increase demands greater arterial pressure and the response by the cardiovascular system generally overshoots the mark until the arterial pressure is usually 10 or 15 mm Hg higher.

It should be pointed out that in changing from supine to erect posture; there is a momentary fall in blood pressure caused by the diminished venous return. But this is normally overcome very quickly.

c. **Diurnal variation:** Blood pressure tends to rise from a low point during sleep to high point (15-20 mm Hg higher) after the evening meal.

d. **Ingestion of food:** After a large meal, there is normally a considerable rise in systolic pressure and sometimes a fall in diastolic pressure.

g. **Obesity:** The systolic pressure is usually high in obese person. In most of the over weight person, the blood pressure is found to be high.

Effect of Training on Blood Pressure
Following endurance training, arterial blood pressure changes very little during standardized submaximal exercise or at maximal work rates. But resting blood pressure is generally lowered in people who are borderline or moderately hypertensive before training. This reduction occurs in both systolic and diastolic blood pressure.

Although resistance type exercise can cause large increases in both systolic and diastolic blood pressure during lifting of heavy weight, chronic exposure to these high pressures does not result in elevations of resting blood pressure. Hypertension is not common in high level weight lifter, or in strength and power athletes. In fact, the cardiovascular system can respond to resistance training by lowering resting blood pressure.

It should be mentioned that training also affects blood pressure. For example prolonged work in the untrained subject leads to a progressive fall in systolic pressure, which indicates nearing exhaustion. At the same time, training retards this phenomenon so that heavy work can be continued for a much longer period of time without a great deal of change in an individual blood pressure. Endurance type of training also improves the blood pressure recovery process following exercise: the better trained the individual, the sooner blood pressure returns to the pre-exercise level.
THE ATHLETE’S HEART

A modest increase in size or hypertrophy is a fundamental adjustment of the healthy heart to regular exercise training. There is greater synthesis of cellular protein as the individual muscle fibers thicken and the contractile elements within each fiber increase in number. This increase in size with training is transient and heart size returns to pre-training levels when training intensity decreases.

The ultrasonic technique of echocardiography has evaluated the structural characteristics of the hearts of athletes and determined if different patterns of cardiac hypertrophy and enlargement are associated with different types of physical conditioning. The structural characteristics of the heart of apparently healthy athletes differ considerably from those of untrained individuals. Also, the pattern of these differences is related to the nature of the exercise conditioning.

For example, the left ventricular volume is 181 ml and mass is 308 gm for the swimmers, and 160 ml and 302 gm for the runner. The non-athletic controls averaged 101 ml for ventricular volume and 211 gm for ventricular mass. Despite their large internal ventricular dimensions, ventricular wall thickness is normal for the endurance athletes.

The athletes involved in resistance exercise training such as weight lifters, shot putters and wrestlers, who are regularly subjected to acute episodes of elevated arterial pressure caused by training-type exercise, have normal ventricular volume but the ventricular wall is thickened. Undoubtedly, this represents compensation for the added workload resistance training imposes on the left ventricle.

The consequences of these apparent differences in training response on long-term cardiovascular health are unknown. However, there is no compelling scientific evidence that arduous exercise training can harm a normal heart.

FUNCTIONAL VERSUS PATHOLOGICAL HYPERTROPHY

Cardiac hypertrophy in response to chronic pathologic states such as hypertension has sometimes been confused with the moderate compensatory growth of the myocardium and enlargement of the left ventricular cavity with endurance training. Although the stress of exercise requires that myocardial fibers generate increased tension, which is a critical requirement for initiating compensatory hypertrophy, the application of this overload differs considerably from that of the chronic pressure overload imposed by vascular disease. During exercise training, the myocardial overload is only temporary, so there is a ‘recuperative’ time during non-exercise periods. Also if compensatory heart growth does occur with training, it is not accompanied by a dilution and weakening of the left ventricle, a frequent response.

<table>
<thead>
<tr>
<th>Dimension</th>
<th>College Runner (n = 15)</th>
<th>College Swimmers (n = 15)</th>
<th>World Class Runners (n = 10)</th>
<th>College Wrestlers (n = 12)</th>
<th>World Class Shot Putters (n = 4)</th>
<th>Normal (n = 16)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVID</td>
<td>54</td>
<td>51</td>
<td>48-59</td>
<td>48</td>
<td>43-52</td>
<td>46</td>
</tr>
<tr>
<td>LVV, ml</td>
<td>160</td>
<td>181</td>
<td>154</td>
<td>110</td>
<td>122</td>
<td>101</td>
</tr>
<tr>
<td>SV, ml</td>
<td>116</td>
<td>113</td>
<td>113</td>
<td>75</td>
<td>68</td>
<td>—</td>
</tr>
<tr>
<td>LV wall, mm</td>
<td>11.3</td>
<td>10.6</td>
<td>10.8</td>
<td>13.7</td>
<td>13.8</td>
<td>10.3</td>
</tr>
<tr>
<td>Septum, mm</td>
<td>10.9</td>
<td>10.7</td>
<td>10.9</td>
<td>13.0</td>
<td>13.5</td>
<td>10.3</td>
</tr>
<tr>
<td>LV mass, gm</td>
<td>302</td>
<td>308</td>
<td>283</td>
<td>330</td>
<td>348</td>
<td>211</td>
</tr>
</tbody>
</table>

LVID: left ventricular internal dimension
LVV: left ventricular volume
SV: stroke volume
LV wall: left ventricular wall
LV mass: left ventricular mass
to chronic hypertension. Whereas the hearts of elite athletes are usually larger than the hearts of their untrained counterparts, heart size is generally within the upper range of normal limits in relation to various measures of body size or to the increase in end-diastolic volume. The ‘athlete’s heart’ is not a dysfunctional organ. To the contrary for the endurance athlete, its functional capacity is superior in terms of stroke volume and maximum cardiac output.

**Summary**

1. Blood is an opaque, slightly alkaline, reddish colored, salty taste, and viscous fluid connective tissue. Hemoglobin is found in red blood cells of all vertebrates. In normal condition 100 ml of human blood contains about 14.5 gm of hemoglobin in adult male. The red blood cells of human being are non-nucleated, bi-concave, circular disc shaped red colored cells of the blood. The white blood cells are large in size, different shaped and nucleated colorless cells of the blood. The platelets are small colorless non-nucleated, oval or disk shaped cytoplasmic fragments found in the circulating mammalian blood.

2. Blood coagulation is the process by which the shedding of blood from the reputed blood vessels (wound) loses its fluidity in a few minutes and sets into a semisolid jelly-like mass is known as coagulation. Normal coagulation time is measured according to the method of Lee and White; it is 6 to 17 minutes in glass tube and 19 to 60 minutes in siliconized tube.

3. Karl Landstainer in 1900 AD was first realized the importance of blood group. Because it is necessary for blood transfusion, identifying a person according to the blood group and find defects in blood group. According to Landstainer, that human being can be divided into several groups. The human beings may be put into four different groups according to the nature of the agglutinogen possessed by their corpuscles. These groups are called O, A, B and AB.

4. The hollow muscular pumping organ ever pulsatile and responsible for the circulation of blood in the body is called the heart. It lies in the central portion of the chest cavity, in between the two lungs, directly behind the sternum and above the diaphragm. It remains slightly titled to the left side of the body. The human heart is a conical hollow organ and made up of cardiac muscle. It remains covered by a protective double walled membranous sac called **pericardium**. The human heart is having four chamber—they are right atrium, left atrium, right ventricle and left ventricle.

5. Circulation of blood through the heart is unidirectional due to the presence of **valves** in the several openings of the heart. Due to continuous contraction (**systole**) and relaxation (**diastole**) of the heart, the blood circulates within it as well as throughout the body. Rhythmicity is the characteristic property of the heart muscle. It depends on the rate of initiation of cardiac impulse from the **Sino-atrial node** (SA node). SA node initiates impulse 70 to 80 times per minute and similarly the heart also beats 70 to 80 times per minute. Thus SA node is known as **Pacemaker**.

6. Changes that occur in the heart during one beat are repeated in the same order in the next beat. This cyclical repetition of the various changes in heart, from beat to beat is called cardiac cycle. The time required for one complete cardiac cycle in normal condition is **0.8 sec**. Every event in the cycle will be repeated at the interval of 0.8 sec. In the cardiac cycle there are four main events such as- Atrial systole, Atrial diastole, Ventricular systole and Ventricular diastole.

7. A graphic record of the electrical variations produced during a heart beat or cardiac cycle is called electrocardiogram. The electrical variations include depolarization and repolarization of the auricle and ventricle bringing about their contraction and relaxation. A typical electrocardiogram of a healthy person shows five waves. These are called P, Q, R, S and T. The P, R and T waves which are above the base line of ECG are known as positive waves. The Q and S waves
that lie below the base line are called negative waves.

8. Cardiac output is defined as the volume of blood pumped by the heart in one minute. This is generally expressed liters per minute and milliliters per minute. It is the product of heart rate and stroke volume (the amount of blood pumped with each beat of the heart). The normal value of the cardiac output is about 5 liter per minute. During sub maximal exercise (40 to 60 percent of maximal capacity), cardiac output in trained athletes may be increased to 40 liters per minute, whereas untrained subjects may attain outputs of about 20 liters per minute. At this level of work, it is known that this 5 to 7 folds increase in cardiac output is due to increase in both heart rate and stroke volume.

9. During systole, a certain volume of blood is ejected from the left ventricle. This amount is the stroke volume (SV) of the heart, or the volume of blood pumped per stroke (contraction). For example, consider the amount of blood in the ventricle before and after contraction. stroke volume increases with increasing rates of work but only up to exercise intensities between 40 and 60 percent of maximal capacity.

10. This rhythmic contraction and relaxation of the heart is known as heart rate. Normally in human sedentary adult male's heart rate is 72 beats per minute. Resting heart rate averages 70 to 80 beats per minute. The maximum heart rate (HRmax) is the highest heart rate value one achieves in an all-out effort to the point of exhaustion. An individual’s maximum heart rate can be calculated by the following equation: \( HR_{max} = 220 - \text{age in years}\).

11. When the rate of work is held constant at submaximal levels of exercise, heart rate increases fairly rapidly until it reaches a plateau. This plateau is the steady state heart rate and it is the optimal heart rate for meeting the circulatory demands at that specific rate of work.

12. At maximal rate of exercise, HRmax usually remains relatively unchanged following endurance training. However, the untrained people who have maximum heart rate more than 180 beats/min. HRmax slightly reduced following training. When the exercise bout is finished, the heart does not initially return to its resting level. Instead, it remains elevated for a while, slowly returning to its resting rate. The time its take for heart rate to return to its resting rate is called the heart rate recovery period.

13. Blood pressure is a lateral pressure exerted by blood on the walls of blood vessels while flowing through, is known as blood pressure. During exercise, blood pressure increases linearly as a result of an increase in cardiac output. The exercise affects on systolic blood is pressure much more than diastolic or mean pressure. This is due to the fact that during exercise the resistance to blood flow is decreased. The decreased resistance is the result of vasodilatation taking place in the arterioles or the working muscles.

14. Following endurance training, arterial blood pressure changes very little during standardized submaximal exercise or at maximal work rates. But resting blood pressure is generally lowered in people who are border line or moderately hypertensive before training. This reduction occurs in both systolic and diastolic blood pressure.

**Review Questions**

1. What stimulates the wave of contraction in atria and which the blood moves?
2. Draw a simple diagram of the internal structure of human heart to show the veins entering it. Label the veins and chambers.
3. What are the difference between arteries and veins?
4. What is blood? Describe its components.
5. Write down various function of human blood.
6. What is electrocardiogram? Write about its significance.
7. Describe the structure of human heart.
8. How does the blood flow through the heart during the different phases of the cardiac cycle?
9. Make a line sketch to show blood circulation in human body.
10. What is the difference between systole and diastole, and how does this relate to systolic blood pressure and diastolic blood pressure?

11. Describe how heart rate, stroke volume, cardiac output and blood pressure respond to sub-maximal and maximal exercise.

12. How do you determine the maximum heart rate? What are alternative methods using indirect estimates?

13. Differentiate between muscular endurance and cardiovascular endurance.

14. Define blood group. What do you mean by universal donor and universal recipient?

15. What are the factors affecting heart rate? Discuss about the steady state heart rate.

16. What are the effects of training on the following? (a) Resting heart rate, (b) Sub-maximal heart rate, (c) Maximal Heart rate and (d) Recovery heart rate.

17. What is ‘Athletes heart’? Discuss about the Functional versus pathological hypertrophy.
Respiratory system: The respiratory system formed by the organs which are concerned for the exchange of gases between the environment and living organism is called respiratory system. The transportation of gases involves four different processes. These are as follows:

i. Breathing is the movement of air into and out of the lungs.

ii. Diffusion is the exchange of gases between the lungs and the blood.

iii. Transport of gases (O$_2$ and CO$_2$) through blood and

iv. Exchange of gases from blood to active tissue and vice versa.

The first two processes are referred to as external respiration because they involve moving gases from outside the body into the lungs and then the blood. Once the gases are in the blood they must travel to the tissues, the fourth step of respiration occurs. This gas exchange between the blood and the tissues where food particles are burned and produce energy is called internal respiration.

STRUCTURE OF HUMAN RESPIRATORY ORGAN (LUNGS)

The respiratory system consists of the following structures:

1. The air conducting passage: The air passage mainly consists of nostril, nasal cavity, bronchi and bronchioles. The air from the atmosphere enters through the nostril, which open through the internal areas into the pharynx. From the pharynx the air passes through the cartilaginous chamber known as larynx.

This structure is also associated with the production of sound. The main air conducting passage is known as trachea. It is about 12 cm long and 15 to 18 mm diameter cartilaginous and membranous windpipe. It begins from the lower part of the larynx and ends by separating into right and left bronchi. The right bronchus is little larger than the left. Within lung the right bronchus is divided into three bronchioles, whereas the left bronchus is divided into two bronchioles. These bronchioles are subdivided into a number of respiratory terminal bronchioles.
1. Schematic representation of airway passage:
   Nostril → Trachea → Primary bronchus → Secondary bronchus → Tertiary bronchioles → Terminal Bronchiole → Atrium → Alveolus.

2. Lungs: Lungs are essential organs for respiration. The lungs are paired, spongy structures situated in the thoracic cavity just above the diaphragm and lateral to the heart. The right lung consists of three lobes and the left lung is divided into two lobes. The membrane, which covers the lungs, is called pleura. There are two such membranes covering (double layered) of lungs, the outer membrane is called parietal pleura and an inner membrane is called visceral pleura. Space i.e., pleural cavity between these membranes is filled with a fluid called pleural fluid.

   The pleural fluid resists friction of the membrane during breathing. The membranes show elasticity. In the lungs the bronchioles terminate into the blind sacs known as alveoli. Alveolus is the physiological unit of the lung. They are lined by a thin layer of squamous epithelium cell. Each alveolus is surrounded by a network of capillaries. Oxygen and carbon dioxide readily diffuse through the thin and moist walls of the alveoli.

3. Associated respiratory organs: The thorax, which contains the lungs, is bounded by the rib cage. On the lower side of the thorax lungs are attached with a dome-shaped sheet of skeletal muscle, the diaphragm. In between the ribs intercostal muscles are present. They are of two types: external intercostal and internal intercostal muscle. These muscles are called muscles of respiration.

   Functions of respiration: The following functions are performed by the respiratory system:
   i. Transfer of O₂ from the alveoli to the venous blood and CO₂ from venous blood to the lungs.
   ii. Maintain the arterial pressure of CO₂ at 40 mm Hg which is essential for many vital functions of the body.
iii. It regulate the acid-base balance (PH of blood) of the body.
iv. Excretion of certain volatile of gasses from the body.
v. Pumping action.

**PHASES OF RESPIRATION (EXTERNAL AND INTERNAL RESPIRATION)**

a. **External respiration:** External respiration is the physical process of interchange of gases between the organism and its environment. Oxygen enters into the body of the organism from the surrounding by diffusion while carbon dioxide diffuses out from the organism to the environment around. Various respiratory organs like trachea, bronchi, lungs, etc. take part for this purpose.

b. **Internal respiration:** Internal respiration is the physical process of interchange of gases between the body fluid and tissue cells. In this phase O₂ enters into the tissue cells by diffusion and is utilized for oxidative process in the cells. This is also called tissue oxidation or tissue respiration. It produces CO₂, which diffuses out from the tissue cells.

**PULMONARY VENTILATION**

Pulmonary ventilation is the process by which air is moved into and out of the lungs.

*Inspiration:* It is an active process through which the diaphragm and the external intercostal muscles increase the dimension and thus, the volume of the thoracic cage and this decrease the pressure in the lungs and draws air in.

*Expiration:* Normal expiration is a passive process. The inspiratory muscles relax and the elastic tissue of the lungs recoils returning the thoracic cage to its smaller, normal dimension. This increases the pressure in the lungs and forces air out.

**Respiratory Quotient or RQ**

Definition: It is the ratio of the volume of carbon dioxide evolved to the volume of oxygen taken in during the process of respiration. Therefore, RQ is represented as CO₂/O₂. It depends on the nature, amount of O₂ present in the respiratory substrate and extent of which the respiratory substrate is broken down. Example-RQ of carbohydrate is 1 whereas in fat is 0.7.

**THE RESPIRATORY MUSCLES**

The respiratory muscles play an important role in respiration. Two types of muscles are involved during respiration such as muscles of inspiration and muscles of expiration.

*Muscles Used in Inspiration*

a. **Diaphragm:** The diaphragm is believed to be the most important single muscle of inspiration. During resting conditions, it makes a dome over the liver and stomach. When it contracts, it causes the bottom of the thorax to
flattens (increases the vertical dimension of the thorax). In other words, the domed area is lowered.

b. External intercostal muscles: During strenuous work when heavy breathing is required, the external intercostal muscles are brought into play much more extensively. They raise the ribs and sternum in order to create additional enlargement of the anteroposterior and lateral diameters of the chest cavity.

c. Scalene: During exercise, the large volume of inspired air is also aided by contraction of other inspiratory muscle. For instance, contraction of the scalene muscle helps to raise the first two ribs.

d. Sterno-Clavicular: The other inspiratory muscle involves during exercise and contraction of the sterno-clavicular muscle helps to raise the sternum.

e. Extensor muscles: It should also be pointed out that the extensor muscles of the back and neck help to facilitate inspiratory breathing during vigorous work.

f. Trapezius: The large trapezius muscle also helps to facilitate inspiratory breathing during vigorous work.

**Muscles Used in Expiration**

During normal resting conditions, expiration is primarily due to the elastic recoil of the inspiratory muscles (diaphragm and external intercostals muscles) as they return to their resting positions. This means that during normal resting conditions, expiration plays a passive role while inspiration plays more active role. However, during exercise, expiration is aided by contraction of the expiratory muscles, thus making it an active process.

a. **Abdominal muscles:** The abdominals are the most important muscles of expiration during heavy work. When these muscles contract, they not only flex the trunk, but they also press down the lower ribs. These, in turn, help raise the pressure inside the abdomen.

b. **Diaphragm:** The diaphragm is then forced upward into the thoracic region, thus reducing the overall size of the thoracic area and aiding in expiration.

c. **Internal intercostal muscles:** In addition to the abdominals, the internal intercostal muscles are also brought into play during heavy breathing. During the contraction of above muscles they will pull the ribs down, thus bringing them closure to each other. Hence, the combined action of the abdominals and the internal intercostal muscles aid in reducing the size of the thorax and thus facilitate the role of expiration.

**MECHANISM OF BREATHING**

a. **Intra-alveolar pressure:** The respiratory muscles cause pulmonary ventilation by alternatively compressing and distending the lungs, which in turn causes the pressure in the alveoli to raise and fall. During inspiration the intra-alveolar pressure become slightly negative with respect to atmospheric pressure (-3 mm Hg) and causes air to flow inside. During expiration the intra-alveolar pressure rises and causes air to flow out. During maximum inspiratory and expiratory effort the intra-alveolar pressure reduced.

---

**Major Respiratory Muscles Involved during Rest and Exercise**

<table>
<thead>
<tr>
<th>Respiratory phase</th>
<th>Muscle acting during rest</th>
<th>Action</th>
<th>Muscle acting during exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Inspiration</strong></td>
<td>i. Diaphragm</td>
<td>i. Flattens</td>
<td>i. Diaphragm</td>
</tr>
<tr>
<td></td>
<td>ii. External Intercostals</td>
<td>ii. Raises Ribs</td>
<td>ii. External Intercostals</td>
</tr>
<tr>
<td></td>
<td>i. Flattens</td>
<td>iii. Elevates first and second ribs</td>
<td>iii. Scaleni</td>
</tr>
<tr>
<td></td>
<td>i. Flattens</td>
<td>iv. Elevates Sternum</td>
<td>iv. Sterno-Clavicular</td>
</tr>
<tr>
<td><strong>Expiration</strong></td>
<td>None</td>
<td>i. Lowers Ribs</td>
<td>i. Internal Intercostals</td>
</tr>
<tr>
<td></td>
<td>None</td>
<td>ii. Depresses Lower Ribs and Forces Diaphragm into Thorax</td>
<td>ii. Abdominal</td>
</tr>
</tbody>
</table>
(-80 mm Hg) and increased (100 mm Hg) respectively.

b. *Fluid pressure in the intra-pleural cavity:* The normal pressure of the fluid in the intra-pleural space is between –10 and –12 mm Hg. This negative pressure acts as a force to hold the visceral pleura of the lungs tightly against the partial pleura of the chest wall. Due to enlargement and shortening of the chest cavity this negative pressure causes the lungs also to large and short.

The cause of the very negative intra-pleural fluid pressure is the continual tendency of the pleural capillaries to absorb fluid from the intra-pleural space. This is particularly true of the visceral pleural capillaries, because these are part of the pulmonary circulatory system and have a very low capillary pressure (about 7 mm Hg) that causes rapid absorption of fluid.

c. *Recoil tendency of the lungs and inter-pulmonary pressure:* The lungs have a continual tendency to collapse and therefore, to recoil away from the chest wall. This tendency is caused by two different factors:

i. Elastic fiber-constantly stretched and shortened.

ii. Surface tension: Due to intra-molecular attraction of the surface layer of the fluid of alveoli; they have got a tendency to collapse.

This collapsing force of the millions of alveoli produces a summated effect resulting in tendency of the whole lung to recoil away from the chest wall.

d. *The diaphragm and the abdominal muscles:* Inspiration is an active process by which the air is taken from the atmosphere into the lungs. Normal inspiration is caused principally by the contraction of diaphragm. This muscle is belled shaped so that contraction of any of its muscle fibers pulls it downward to cause inspiration.

Expiration is the passive process by which the air is given out from lungs to the atmosphere. During expiration the relaxation of intercostal and diaphragm muscles take place. The diaphragm and the thoracic wall return to its former position.

c. *Muscles that raise and lowers the chest cage:* Three different group of muscles cause inspiration by elevating the entire chest cage. The sternocleidomastoid muscles lift upward on the sternum; the anterior serrati lift many of the ribs; and the scalene lift the 1st two ribs.

To cause expiration, the abdominal recti, in addition to helping to compress the abdominal contents upward against the diaphragm, also pull downwards the lower ribs, thereby decreasing the anteroposterior (AP) diameters of the chest. Thus, these muscles act as muscles of expiration both by
depressing the ribs cage and by compressing the abdominal contents upward.

The ribs during expiration are angled downward and the external intercostals are stretched in a forward and downward direction. As they contract they pull the upper ribs forward in relation to the lower ribs and this causes leverage on the ribs to rise then upward. Conversely, in the inspiratory position, the internal intercostal muscles are stressed and their contraction pulls the upper ribs backward in relation to the lower ribs. This causes leverage in the opposite direction and lowers the chest cage.

**FUNCTIONAL ASPECT OF RESPIRATION**

i. *Gaseous exchange:* Process of respiration supplies oxygen from the atmosphere to the tissue and removes carbon dioxide from the body to the atmosphere.

ii. *Excretory functions:* In addition to the carbon dioxide some volatile metabolic waste products such as acetone, ammonia, oils, alcohol, water vapor, etc. are also excreted through expiration.

iii. *Regulates water balance:* During expiration a large amount of water (about 700 ml) is lost as water vapor.

iv. *Regulates acid-base balance:* The normal pH of the body is 7.4. Any change of pH alters the rate and depth of respiration.

v. *Regulates body temperature:* Certain amount of heat is lost during expiration.

vi. *Controls metabolic functions:* For metabolism oxygen is the most important constituent, which is taken during inspiration.

**LUNG VOLUME AND CAPACITIES**

The various lung volume that reflect one’s ability to increase the depth of breathing. To obtain these measurements, the subject breathes through Expirograph, which is shown below (Fig. 4.6). Lung volumes are basically divided into static and dynamic lung volumes.

a. *Static lung volume:* The bell of Spirometer shown in the figure (4.6) falls and subsequently rises as air is inhaled and exhaled to record the ventilatory volume and breathing rate. The volume of air moved during either the Inspiratory or Expiratory phase of each breathe is termed Tidal Volume. Such volumes are called static lung volume.

b. *Dynamic lung volume:* In appraising the adequacy of pulmonary ventilation, the important consideration is the ability to sustain high levels of air flow rather than the quantity of air moved in a single breathes which is termed as dynamic lung volume.

**Factors affecting Lung Volumes and Capacities**

Several factors affect lung volumes and capacities, some that can be controlled and some that can not. These factors include: age gender height body surface area, smoking, altitude etc.

A person who is born and lives at sea level will have a smaller lung capacity than a person who spends their life at a high altitude. This is because the atmosphere is less dense at higher altitude and therefore, the same volume of air contains fewer molecules of all gases, including oxygen.

In response to higher altitude, the body’s diffusing capacity increases in order to be able to
process more air. When someone from sea level travels up to the higher parts of the earth (e.g. the Andes, Mexico City, Tibet and the Himalayas) they will often develop a condition called altitude sickness because their lungs cannot process enough oxygen for their body’s needs.

On the other hand cigarette smoking is inversely related to the lung volumes and capacity along with the flow rates. Cigarette smoke containing more than 200 chemical identified and the carbon monoxide has got the most deleterious effects on the respiratory system.

Taller height and larger surface area of the body are linearly related with lung volumes and capacities. As male have reported more lung volumes and capacities as compared to female because of their body height and bigger surface area.

Measurement of lung function: Pulmonary ventilation constitute the first part of respiratory process and ventilation has the dimensions of both volume and time. Ventilation occurs as a result of a pressure difference between the alveolar and oral ends of the airways. This ventilation can be measured with a machine called ‘siprometers’ which consists of an inverted belljar over a double-walled chamber with water in between two wall and the details expirograph arise given below:

<table>
<thead>
<tr>
<th>Factors</th>
<th>Larger volumes</th>
<th>Smaller volumes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td>Males have greater lung volumes and capacities.</td>
<td>Females have smaller lung volumes and capacities.</td>
</tr>
<tr>
<td>Age</td>
<td>Age up to maturity increase lung volumes and capacity linearily.</td>
<td>As increase in age (after 40 yrs. of age) lung functions decreases.</td>
</tr>
<tr>
<td>Body height</td>
<td>Body height has got linear relation with lung volumes and capacities</td>
<td>Shorter people have got smaller lung volumes and capacities.</td>
</tr>
<tr>
<td>Body surface area</td>
<td>Body surface area has also got linear relationship with lung volumes and capacities, i.e. the people have bigger surface area have larger volumes.</td>
<td>People having smaller body surface area have smaller lung volumes and capacities.</td>
</tr>
<tr>
<td>Smoking habit</td>
<td>Non-smokers show higher values of lung volumes and capacities.</td>
<td>Smoking has got deleterious effects on lungs. So, smokers have lower lung volumes and capacities.</td>
</tr>
<tr>
<td>Lung disease</td>
<td>The people do not suffering from any respiratory disease having bigger lung volumes and capacities.</td>
<td>Respiratory diseases like asthma, emphysema and bronchitis reduces the lung volumes and capacities.</td>
</tr>
<tr>
<td>Sports training</td>
<td>Physical training improves the lung volumes and capacities, e.g. athletes</td>
<td>Sedentary nature of life does not improve lung function parameters, e.g. non-athletes</td>
</tr>
<tr>
<td>High altitude</td>
<td>People living at high altitudes have bigger lung volumes and capacities.</td>
<td>People living at low altitudes have less lung volumes and capacities as compare to people live at high altitude.</td>
</tr>
</tbody>
</table>
along with the pen recorder and produce graph from which different parameters are calculated.

**LUNG FUNCTION**

The abbreviations of lung volumes and lung capacities those are commonly used in the Spirometric measurements along with average values of men and women are given in the table below:

<table>
<thead>
<tr>
<th>Volume Type</th>
<th>Average Value (Women)</th>
<th>Average Value (Men)</th>
</tr>
</thead>
<tbody>
<tr>
<td>IC (Inspiratory Capacity)</td>
<td>4,000 ml</td>
<td>4,300 ml</td>
</tr>
<tr>
<td>VC (Vital Capacity)</td>
<td>4,000 ml</td>
<td>4,300 ml</td>
</tr>
<tr>
<td>FEV&lt;sub&gt;1&lt;/sub&gt; ( Forced Expiratory Volume in 1 second)</td>
<td>3,200 ml</td>
<td>3,500 ml</td>
</tr>
<tr>
<td>FEV&lt;sub&gt;2&lt;/sub&gt; ( Forced Expiratory Volume in 2 seconds)</td>
<td>2,800 ml</td>
<td>3,100 ml</td>
</tr>
</tbody>
</table>

**TRANSPORT OF RESPIRATORY GASES**

Gasses in our body are dissolved in fluids, such as plasma. They are dissolve in liquids in proportion to their partial pressure depending also on their solubilities in the specific fluids and on the temperature. But the solubility and the temperature in the blood is constant. However, the most critical factor for gas exchange between the alveoli and the blood is the pressure gradient.

**Transport of Oxygen**

The oxygen content of the arterial and venous blood and relevant data are given in the following table:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial blood</td>
<td>O&lt;sub&gt;2&lt;/sub&gt; content: 19.3, Oxy-Hb: 19, O&lt;sub&gt;2&lt;/sub&gt; in Sol: 0.3, PO&lt;sub&gt;2&lt;/sub&gt;: 100 mm Hg</td>
</tr>
<tr>
<td>Mixed venous blood</td>
<td>O&lt;sub&gt;2&lt;/sub&gt; content: 14.2, Oxy-Hb: 14, O&lt;sub&gt;2&lt;/sub&gt; in Sol: 0.2, PO&lt;sub&gt;2&lt;/sub&gt;: 40 mm Hg</td>
</tr>
</tbody>
</table>

About 98 percent of oxygen is present in the blood in chemical combination with hemoglobin and only 0.3 ml in arterial blood (about 2%) is in physical solution in water of the plasma. However, this small quantity in physical solution is responsible for tension of oxygen in the plasma. It is the oxygen tension in the blood, which is responsible for transfer of this gas from alveolar air to the venous blood across the alveolo-capillary membrane and also for transfer of this gas from arterial blood to...
<table>
<thead>
<tr>
<th>Symbols</th>
<th>Term used</th>
<th>Definitions</th>
<th>Average values (ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>VT</td>
<td>Tidal volume</td>
<td>Volume of gas inspired or expired during each respiratory cycle.</td>
<td>600</td>
</tr>
<tr>
<td>VC</td>
<td>Vital capacity</td>
<td>Maximal amount of gas that can be expelled from the lungs following a maximal inspiration.</td>
<td>4800</td>
</tr>
<tr>
<td>IC</td>
<td>Inspiratory capacity</td>
<td>Maximal amount of gas that can be inspired from resting expiratory level.</td>
<td>3600</td>
</tr>
<tr>
<td>IRV</td>
<td>Inspiratory reserve volume</td>
<td>Maximal volume of gas that can be inspired from end tidal inspiration.</td>
<td>3000</td>
</tr>
<tr>
<td>ERV</td>
<td>Expiratory reserve volume</td>
<td>Maximal volume of gas that can be expired from resting expiratory level.</td>
<td>1200</td>
</tr>
<tr>
<td>FVC</td>
<td>Forced vital capacity</td>
<td>Volume of gas expired after full inspiration, expiration being as rapid and complete as possible.</td>
<td>4800</td>
</tr>
<tr>
<td>FEV₁</td>
<td>Forced expiratory volume (1 sec)</td>
<td>This is the volume of air that can be exhaled out within first 1 seconds of maximum expiration.</td>
<td>4500</td>
</tr>
<tr>
<td>FEV₁%</td>
<td>Percentage of forced expiratory volume (1 sec)</td>
<td>This is the ratio between FEV₁ and FVC and expressed in percentage.</td>
<td>90–95%</td>
</tr>
<tr>
<td>FEV₉</td>
<td>Forced expiratory volume, qualified by time interval (sec)</td>
<td>Volume of gas expired over given time interval during the performance of a forced vital capacity.</td>
<td>2.5–3.0 sec.</td>
</tr>
<tr>
<td>PEF₂₅₋₇₅%</td>
<td>Forced mid expiratory flow</td>
<td>The volume of air per unit of time exhaled during the middle half of the expired volume of the forced expiratory spirogram.</td>
<td>295 L/min 275 L/min</td>
</tr>
<tr>
<td>MVV₁</td>
<td>Maximum voluntary ventilation (uncontrolled)</td>
<td>Volume of air which a subject can breathe with voluntary maximal ventilatory effort per unit of time.</td>
<td>120–130 L/min 110–120 L/min</td>
</tr>
<tr>
<td>PEFR</td>
<td>Peak expiratory flow rate</td>
<td>This is the maximal flow, which can be sustained for a period of 10 milliseconds during a forced expiration starting from total lung capacity.</td>
<td>450 L/min 350 L/min</td>
</tr>
</tbody>
</table>

Tissue fluid across the capillary membrane. Further, it is the tension of oxygen in plasma, which controls the amount of oxy-hemoglobin present in the blood.

Hemoglobin is remarkable in that it can combine with oxygen forming a loose chemical compound known as oxyhemoglobin, which differs from oxide of hemoglobin known as methemoglobin in the fact that the O₂ in methemoglobin is firmly fixed with the hemoglobin molecule.

The amount of O₂ of the hemoglobin will combine with forming oxyhemoglobin is dependent upon the tension of oxygen in the medium where hemoglobin is placed. If the O₂ tension is high a large amount of oxyhemoglobin will be formed whereas on a medium of low O₂ tension the oxyhemoglobin will breakdown liberating O₂, itself being converted into reduced hemoglobin.

O₂ capacity: One gram of hemoglobin when fully saturated will combine with 1.34 ml of O₂. Since
the blood contains approximately 15 grams of Hb per 100 ml—the oxygen content of the blood when fully saturated will be about $15 \times 1.34 = 20$ ml. This is called O₂ capacity of the blood. Naturally the O₂ capacity depends upon the amount of hemoglobin in blood.

**O₂ content:** It is usually determined by Van Slyke’s manometric apparatus in which 1 ml of blood is treated anaerobically with potassium ferricyanide solution in the chamber of the apparatus. The pressure exerted by the liberated oxygen after absorption of CO₂ on a manometer attached with the apparatus is noted and is used for calculation of the oxygen content.

If the blood is first agitated with air so that the Hb gets fully saturated with O₂ the result will indicate O₂ capacity. Percentage saturation of Hb with O₂ = (O₂ content $\times 100$)/O₂ capacity.

**Oxygen exchange in the lungs:** The tension of O₂ in the alveolar air is 100 mm Hg and that of the dissolved O₂ in the plasma of the mixed venous blood is only 40 mm Hg.

Due to the tension gradient, O₂ diffuses rapidly from the alveolar air to the mixed venous blood increasing the quantity and also tension of O₂ in the plasma of the mixed venous blood is only 40 mm Hg.

The oxygenated blood leaves the pulmonary capillaries in tension equilibrium with alveolar air, that is, with O₂ tension of 100 mm Hg and with hemoglobin 98 percent saturated with oxygen.

1. The gaseous composition of blood is different in different veins of the body. O₂ content of blood of veins draining metabolically active tissues (e.g. muscles, heart) is low whereas the O₂ content of blood of veins from skin and brain is rather high. The term mixed venous blood indicates sample of venous blood from the right heart or pulmonary artery.

2. The actual gas exchange in the lungs take place between the gases dissolved in alveolar fluid and plasma water.

**Oxygen transport in the tissues:** Oxygen tension in the tissue fluid is low and is about 40 mm Hg during ‘rest’. The arterial blood enters the tissue capillaries with an oxygen tension of 100 mm Hg and with hemoglobin 98 percent saturated with oxygen. So the O₂ diffuses from the plasma to the tissue fluid due to tension gradient between the two fluids. As O₂ diffuses the amount of O₂ in solution decreases and the tension of O₂ in the arterial blood falls. This results in desaturation of hemoglobin, which gives oxygen firstly to the plasma from where it goes to the tissue space—the guiding force being the tension gradient. The reaction is very rapid and so the blood leaves the tissues with oxygen in tension equilibrium with tissue fluid that is 40 mm Hg and consequent 75 percent saturation of hemoglobin with oxygen.

**Carbon Dioxide Transport**

Carbon dioxide is carried in blood (i) in physical solution and (ii) as chemical compounds.

i) **Carbon dioxide in physical solution:** Roughly about 5 percent of CO₂ is carried in physical solution in blood. In solution, CO₂ exist mostly as H₂CO₃ according to the following equation—

$$CO₂ + H₂O \leftrightarrow H₂CO₃ \leftrightarrow H^+ + HCO₂⁻$$

Since the reaction is reversible it is clear that H₂CO₃ always remains in equilibrium with small amount of molecular O₂ gas, which is responsible for CO₂ tension of blood.

The ingress of CO₂ into the blood in the tissue capillaries is not accompanied by any significant in H⁺ concentration because of the effective ‘buffering’ action of the blood and of hemoglobin in particular. The buffering property of hemoglobin is due to imidaxole group of histidine linked with haem, which contains an ionizable H⁺ ion. On oxygenation, histidine parts with its H⁺ ion thus act as an acid. On reduction the H⁺ ion is mostly linked with N and thus acts as a weaker acid. Reduced Hb, therefore, is potentially H⁺ ion acceptor. 1mMol of reduced Hb can accept 0.7 mMol of H⁺ ion without any change of blood reaction. It has been suggested that the H⁺ of the –NH₂ radical of haem linked valine is also oxylabile like that of histidine and thus confers upon hemoglobin its remarkable buffering property. This explains why venous blood with
reduced Hb contains more CO₂ than arterial blood at a given PCO₂ and the dissociation curve of CO₂ for venous blood is situated at higher level.

i) CO₂ as bicarbonate: It is clear that more than 80 percent of the CO₂ is carried as bicarbonate in the blood and that the major fraction of the carbonate is present in plasma. However, most of the bicarbonate found in plasma are primarily formed within the RBC and then shifted to plasma.

1. CO₂ transport in the tissues: Tension of CO₂ in the tissue fluid is 46 mm Hg (at rest) and that in arterial end of the capillary is 40 mm Hg, CO₂ therefore, diffuses from the tissue space to the capillary due to tension gradient.

The H⁺ ion and HCO₃⁻ ion formed are disposed off as follows:

- \( H^+ + NaPr \leftrightarrow Na^+ + HPr \)
- \( Na^+ + HCO₃^- \leftrightarrow NaHCO₃ \)
- \( CO₂ + H₂O \leftrightarrow H₂CO₃ \leftrightarrow H^+ + HCO₃^- \) (Carbamino)
- \( H^+ + Na₂HPO₄ \leftrightarrow NaH₂PO₄ + Na^+ \)
- \( Na^+ + HCO₃^- \leftrightarrow NaHCO₃ \)

When equilibrium is established the plasma changes may be summarized:

- Some free molecular CO₂ in solution, which is responsible for CO₂ tension.
- Some CO₂ in solution in H₂O as H₂CO₃
- Some CO₂ buffered as Na₂CO₃
- Some CO₂ as carbaminoprotein

Since formation of HCO₃⁻ is accompanied by formation of almost equivalent quantities of NaHCO₃, the ratio H₂CO₃/NaHCO₃ is but little distributed and the pH of the blood remains almost unaffected.

2. In the RBC: CO₂ diffuses rapidly and the reaction CO₂ + H₂O \( \leftrightarrow \) H₂CO₃ \( \leftrightarrow \) H⁺ + HCO₃⁻ takes place 13,000 times quicker than in plasma because of the presence of the specific enzyme carbonic anhydrase.

3. Carriage of CO₂ as carbamino compound: CO₂ combine directly with free amino group of the globin and forms carbamino compounds which may be represented as follows:

- \( Hb-NH_2 + CO₂ \leftrightarrow Hb-NHCOO^- + H^+ \)

This occurs:

- independent of carbonic anhydrase and therefore, the reaction is not inhibited by cyanides.
- CO₂ is not initially changed to H₂CO₃
- At a very rapid rate in the tissues where hemoglobin is desaturated.

Carbamino compounds are also formed by direct union of CO₂ with plasma proteins but since the concentration of hemoglobin within the RBC
is high a larger fraction of carbamino compounds are carried within the RBC than in the plasma.

Further an increase in PCO₂ does not increase the formation of carbamino compounds since increase PCO₂ means increased H⁺ concentration that leads to formation Hb NH₃⁺. This does not react with CO₂ directly.

Oxygenation of hemoglobin inhibits carbamino compound formation because oxy-hemoglobin by virtue of its greater acidity liberates H⁺, which blocks carbamino compound formation. Reduced Hb, on the other hand, favors formation of carbamino compound.

In the tissues oxy-Hb is also being converted simultaneously to reduce Hb, which is a weaker acid in comparison with the former and has got a weaker hold on the base (K) available within the RBC. If oxy-Hb is represented symbolically by HbO₂ its ‘salt’ with potassium may be symbolized as KḥbO₂. With the ingress of CO₂ and reduction oxy-Hb the following changes occur-

\[
\text{CO}_2 + \text{H}_2\text{O} \leftrightarrow \text{H}^+ + \text{HCO}_3^- + \text{KḥbO}_2 \leftrightarrow \text{HHb} + \text{KHCO}_3 + \text{O}_2 \text{ (liberated)}
\]

The KHCO₃, of course, ionizes to K⁺ + HCO₃⁻. Large quantities of carbamino-hemoglobin are also formed which ionizes liberating H⁺, which are neutralized by imidazole group of histidine and β-amino group of valine of the globin polypeptide chains.

As already explained the HCO₃⁻ from within the RBC is exchanged for Cl⁻ of the plasma according to the law of Donan's equilibrium (Chloride shift). The chloride shift mechanism prevents accumulation of large amount of HCO₃⁻ within the RBC and thus, prevents shifting of its pH to the alkaline side.

**In the lungs:** CO₂ diffuses from plasma (PcO₂ = 46 mm Hg) to the alveolar air (PcO₂ = 40 mm Hg)

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**Fig. 4.9:** Schematic representation of the steps of chloride shift

**Fig. 4.10:** Schematic representation of interrelation between carriage of O₂ and CO₂
and so the H$_2$CO$_3$ breaks down liberating further amount of CO$_2$.

Simultaneously oxy-hemoglobin is formed which being a stronger acid snatches off K within the RBC from its combination with acid ions and form KhbO$_2$. Reversed chloride shift occurs and the HCO$_3^-$ion enters the RBC and is broken down liberating further amount of CO$_2$, which passes from RBC to plasma and then to the lungs.

\[
\text{KHHb + HCO}_3^- + \text{O}_2 \leftrightarrow \text{H}_2\text{CO}_3 + \text{KhbO}_2 \\
\text{H}_2\text{CO}_3 \xrightleftharpoons{\text{CA}} \text{H}_2\text{O} + \text{CO}_2
\]

Oxygenation of hemoglobin also breaks down carbamino compound liberating further quantity of CO$_2$.

**FACTORS INFLUENCING OXYGEN DELIVERY AND UPTAKE**

The rates of oxygen delivery and uptake depend on three major variables:

i. The oxygen content of blood  
ii. The amount of blood flow  
iii. The local conditions

At beginning of the exercise, each of these variables must be adjusted to ensure increased oxygen delivery to our active muscles. Under the normal circumstances hemoglobin is 98 percent saturated with oxygen. Any reduction in the blood’s normal oxygen carrying capacity would hinder oxygen delivery and reduced cellular uptake of oxygen.

An exercise causes increased blood flow through the muscles. As more blood carries oxygen through the muscles, less oxygen must be removed from each 100 ml of blood. Thus, increased blood flow improves oxygen delivery and uptake.

Many local changes in the muscle during exercise affect oxygen delivery and uptake. For example, muscle activity increases muscle acidity because of lactate production. Also, muscle temperature and carbon dioxide concentration both increase because of increased metabolism. All of these oxygen unloading from the hemoglobin molecule, facilitating oxygen delivery and uptake by the muscles.

During maximal exercise, however, when we push our bodies to the limit, changes in any of these areas can impair oxygen delivery and restrict our abilities to meet oxidative demands.

**SECOND WIND**

During the first minute of exercise, the load may appear very strenuous. One may experience dyspnea, but this distress eventually subsides and one experiences a second wind. The factors eliciting the distress may be an accumulation of metabolites in the activated muscles and in the blood because the oxygen transport is inadequate to satisfy the requirement.

So, a phenomenon usually associated with ventilation is called “second wind”. It is generally characterized by a sudden transition from a rather ill-defined feeling of distress or fatigue during the early portion of prolonged exercise to a more comfortable, less stressful feeling later in the exercise. This apparent distress is—dyspnea, rapid and shallow breathing, chest pain, headache and pain in various muscles. It is generally found at different times during exercise in between 2 and 18 min during a 20 min treadmill run.

However by what mechanism this changed environment is brought to consciousness is not known. During heavy exercise there is actually a hypoventilation at the commencement of the activity caused by a time lag in the chemical regulation of the respiration. The issue then becomes a matter of a length/tension in appropriateness in the intercostal muscles. When the second wind occurs, the respiration is increased and adjusted according to the requirement.

**STITCH PHENOMENON**

It appears that the respiratory muscles are forced to work anaerobically during the initial phases of the exercise if there is a time lag in the redistribution of blood. A *stitch* in the site can then develop. This is an exercise-related transient abdominal pain, which probably is caused by diaphragmatic *ischemia* and stress on the visceral ligaments. It is most common in untrained persons and is particularly to occur if heavy exercise is performed shortly after a large meal, when the circulatory adjustment at the commencement of exercise is slower. As the blood supply to the respiratory muscles is
improved, the pain disappears. This theory is not entirely satisfactory. The stitch is more common when running than in cycling and swimming. An alternative trigger of this stitch could be a mechanical stimulation of pain receptors in the abdominal region. A bouncing effect on the abdominal organs is certainly evident in jogging and running. However, it is puzzling that this type of problem does not follow a strict and reproducible course. It was previously believed that the pain was caused by an emptying of the blood depots in the spleen and the contraction-taking place in the spleen. In humans, the spleen serves no such depot function, however, furthermore, persons who have had their spleen removed can still experience such pain. Well-trained athletes who have warmed up adequately before a muscular effort seldom experience such pain.

**Pulmonary Ventilation**

The pulmonary ventilation refers to the movement of air into and out of the lungs. The amount of air ventilated per minute is the product of the frequency ($f$) of breathing and the amount of air moved per breath, i.e. tidal volume. $VE = TV \times f$. In a 65 kg man, $VE$ at rest is around 7.0 liters/min. with a tidal volume of 0.45 liters and a frequency of 16 per minute. During maximal exercise, the ventilation may reach to 120 to 175 liters per minute, with a frequency of 40 to 50 and a tidal volume of approximately 3 to 5.5 liters.

In well-trained athlete, it may go over 200 liters/min which is more than 30 times the resting value. Such an increase is brought about by a tremendous increase in the breathing rate and in the tidal volume.

**VENTILATORY RESPONSE DURING REST AND EXERCISE**

*At rest:*
Normal range of $VE = $ Tidal volume $\times$ Breathing frequency
4 to 15 l/min. = 400 – 600 ml $\times$ 10 – 23 breaths/min.

*During Exercise:*
70 to 135 l/min = 2000–3000 ml $\times$ 35–45 breaths/min.

**Ventilatory Responses to Sub-maximal and Maximal Exercise**

*Changes before exercise:* In anticipation of the ensuing exercise bout stimulate the cerebral cortex

![Fig. 4.11: The ventilatory response to light, moderate, and heavy exercise](image-url)
of the brain which results in ventilation to be increased before the exercise bout.

**Changes during exercise:** Pulmonary ventilation is increased very rapidly with in only a few seconds of start of exercise. This is related to stimulation arising from the joints resulting from the movements generated by the working muscles. In sub-maximal exercise, the rapid rise in ventilation soon stops and is replaced by a slower rise to reach a steady state value. The arterial pressure of oxygen and carbon dioxide are maintained relatively constant during this type of exercise. The ventilation in maximal exercise continues to increase until the exercise is terminated. These changes are thought to be stimulated by chemical stimuli mainly from the CO\textsubscript{2} in the blood produced during exercise. During prolonged exercise in a hot and humid environment, ventilation ‘drifts’ upward due to influence of rising body temperature on the respiratory control center.

**Changes during recovery:** A sudden decrease in pulmonary ventilation immediately after cessation of the exercise is due to the stop of motor activity from the muscles and various joints. The gradual decrease in pulmonary ventilation towards resting condition is probably related to the decrease in chemical influence resulting in the muscular tissue. Greater the intensity of effort the longer it takes for ventilation to return to its resting level.

**Ventilatory Equivalent:** It is the ratio between the volume of air ventilated and the amount of O\textsubscript{2} consumed by the tissues indicates breathing economy. This ratio is referred to as the ventilatory equivalent for oxygen (VE/VO\textsubscript{2}). In this resting condition the value can range from 23 to 28 liter of air Per Liter of O\textsubscript{2} consumed. The value change very little during mild exercise but at near maximum exercise the value can be more than 30 liter of air per liter of O\textsubscript{2} consumed. The value remain constant over a wide range of exercise levels and it only change at near maximal exercise.

**Respiratory Regulation of Acid-Base Balance**

The intense muscular activity often results in the production and accumulation of lactate and H\textsuperscript{+}. This can impair energy metabolism and reduce muscle contractile force. Although the body’s regulation of acid base balance involves more than control of respiration, and the respiratory system plays a crucial role in rapid adjustment of the body’s acid-base status during and immediately after exercise.

Whenever H\textsuperscript{+} levels start to rise, the inspiratory center responds by increasing respiration. Removing carbon dioxide is an essential means for reducing the H\textsuperscript{+} concentrations. Carbon dioxide is primarily transported bound to bicarbonate. Once it reaches the lungs, carbon dioxide is formed again and exhaled.

Wherever H\textsuperscript{+} levels begins to rise, whether from carbon dioxide or lactate accumulation, bicarbonate ion can buffer the H\textsuperscript{+} to prevent acidosis.

**Summary**

i. The system formed by the organs which are concerned for the exchange of gases between the environment and living organism is called respiratory system. The transportation of gases involves four different processes. These are as follows:

  - Breathing is the movement of air into and out of the lungs.
  - Diffusion is the exchange of gases between the lungs and the blood.
  - Transport of gases (O\textsubscript{2} and CO\textsubscript{2}) through blood and
  - Exchange of gases from blood to active tissue and vice versa.

The first two processes are referred to as external respiration because they involve moving gases from out side the body into the lungs and then the blood. Once the gases are in the blood they must travel to the tissues, the fourth step of respiration occurs. This gas exchange between the blood and the tissues is called internal respiration.

ii. External respiration is the physical process of interchange of gases between the organism and its environment. Internal respiration is the physical process of interchange of gases between the body fluid and tissue cells. Pulmonary ventilation is the process by which
air is moved into and out of the lungs. The respiratory muscles play an important role in respiration. Two types of muscles are involved during respiration such as- muscle used for inspiration and muscle used for expiration.

iii. The respiratory muscles cause pulmonary ventilation by alternatively compressing and distending the lungs, which in turn causes the pressure in the alveoli to raise and fall. During expiration the intra-alveolar pressure rises and causes air to flow out. The intra pleural pressure and recoil tendency of the lungs are also takes part in the breathing process. The diaphragm, abdominal muscle and intercostals muscle have got vital role in the process of breathing.

iv. Functions of respiration are gaseous exchange, excretory functions, regulate water balance, regulates body temperature; regulate acid-base balance and controls metabolic functions.

v. The various lung volumes that reflect one’s ability to increase the depth of breathing. To obtain these measurements, the subject breathes through expirograph; The lung volumes are basically divided into static and dynamic lung volumes. Tidal volume is called static lung volume. The quantity of air moved in a single breathes which is termed as dynamic lung volume.

vi. The lung functions are studied by Spirometer/Expirograph machine. The subject breathe through mouth piece and Spiro metric bell moves up and down along with the pen recorder and produce graph from which different parameters are calculated. Tidal volume ($V_T$), vital capacity (VC), inspiratory capacity (IC), inspiratory reserve volume (IRV), expiratory reserve volume (ERV), forced vital capacity (FVC), forced expiratory volume (1 sec) ($FEV_1$), Percentage of forced expiratory volume (1 sec) ($FEV_1\%$), forced expiratory time (FET), maximum voluntary ventilation (MVV), peak expiratory flow rate (PEFR) etc. can be calculated.

vii. The oxygen tension in the blood, which is responsible for transfer of this gas from alveolar air to the venous blood across the alveolo-capillary membrane and also for transfer of this gas from arterial blood to tissue fluid across the capillary membrane. Further, it is the tension of oxygen in plasma, which controls the amount of oxy-hemoglobin present in the blood. Carbon dioxide is carried in blood (i) in physical solution and (ii) as chemical compounds. Tension of $CO_2$ in the tissue fluid is 46 mm Hg. (at rest) and that in arterial end of the capillary is 40 mm Hg. CO$_2$, therefore, diffuses from the tissue space to the capillary due to tension gradient.

viii. During the first minute of exercise, the load may appear very strenuous. One may experience dyspnea, but this distress eventually subsides, and one experiences a second wind. It appears that the respiratory muscles are forced to work anaerobically during the initial phases of the exercise if there is a time lag in the redistribution of blood. A stitch in the side can then develop. This is an exercise-related transient abdominal pain, which probably is caused by diaphragmatic ischemia and stress on the visceral ligaments.

ix. The pulmonary ventilation refers to the movement of air into and out of the lungs. Pulmonary ventilation is increase very rapidly within only a few seconds of start of exercise. This is related to stimulation arising from the joints resulting from the movements generated by the working muscles. In sub-maximal exercise, the rapid rise in ventilation soon stops and is replaced by a slower rise tend to reach a steady state value. The gradual decrease in pulmonary ventilation towards resting condition is probably related to the decrease in chemical influence resulting in the muscular tissue.

**Review Questions**

1. Describe the anatomical structures involved in pulmonary ventilation.
2. Why vigorous exercise sometimes results in muscular pain?
3. How does hemoglobin help in the transports of oxygen from lung to tissue?
4. Write the role of diaphragm and intercostals muscles in the breathing process?
5. How is carbon dioxide taken up from tissue and transported to lungs?
6. How is oxygen transported in the blood and released in the tissue?
7. What is the partial pressure? How does it help in gaseous exchange during respiration?
8. Explain the terms: (a) Tidal volume; (b) Vital capacity and (c) residual volume in relation to respiration.
9. Describe the respiratory system of men.
10. Describe the mechanism of breathing in man.
11. How does the exchange of gases occur in respiration between blood and alveolar air?
12. Distinguish between: (a) External and Internal respiration; (b) Inspiratory muscle and expiratory muscles; (c) Trachea and bronchioles; (d) Alveolar air and inspired air.
13. Define Ventilatory break point and anaerobic threshold. What is the importance in sports training?
14. What do you mean by external and internal respiration? Discuss the role of muscle action during forceful inhalation.
15. Distinguish between lung volumes and capacities. What are the factors affecting lung volumes and capacities?
16. Write notes on Second wind and Stitch phenomenon.
17. Discuss about the Ventilatory response during rest and exercise.
Muscular System

Introduction: Muscles are classified according to their structure and functions. Structurally muscles are classified into two categories, i.e. (I) Striated, (II) Non-Striated. Striated muscles are subdivided into two: (a) Skeletal, (b) Cardiac. On the other hand, muscles are classified into two categories depending on their functions. They are the (i) voluntary (Skeletal muscle) and (ii) involuntary muscles (Cardiac and Smooth muscles).

i. Voluntary
   ii. Involuntary

Properties of Muscles

The basic properties of muscles are as follows:

i. Extensibility and Elasticity: Muscle extends when stretched. When the tension is released, it goes back to its original length. But this elastic return is a little slower. Muscle does not immediately come back to its original position. It takes a little longer time. This is called extension remainder.

ii. Excitability: It is an important property of muscle. With an adequate stimulus, muscles are excitable. The stimulus may be mechanical, thermal, chemical or electrical. For facilities of accurate adjustment, electrical stimulus is used in laboratory experiments.

iii. Contractility: It is another important property of muscle. With excited, the muscle contracts. This is immediately followed by relaxation. A single induction shock will produce a single contraction (twitch). If the stimulus be strong, it may cause stronger contraction.
### Classification of Muscle

#### A. Difference between skeletal and smooth muscles:

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Skeletal</th>
<th>Smooth</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1. Histology</strong></td>
<td>i. Size and Shape</td>
<td>Cylindrical; 1–40 mm long, 10–100 µ in diameter</td>
</tr>
<tr>
<td></td>
<td>ii. Striations</td>
<td>Transverse, longitudinal</td>
</tr>
<tr>
<td></td>
<td>iii. Nucleus</td>
<td>Multiple, just under the sarcolemma</td>
</tr>
<tr>
<td></td>
<td>iv. Sarcotubular System</td>
<td>Present, T-systems are at junctions of A-I band. Terminal cisterna is prominent</td>
</tr>
<tr>
<td></td>
<td>v. Cell-to-cell conduction</td>
<td>Nil</td>
</tr>
<tr>
<td><strong>2. Properties</strong></td>
<td>i. Rhythmicity</td>
<td>Nil</td>
</tr>
<tr>
<td></td>
<td>ii. Conductivity</td>
<td>Very fast</td>
</tr>
<tr>
<td></td>
<td>iii. Contractility</td>
<td>Simple muscle curve with characteristic features</td>
</tr>
<tr>
<td></td>
<td>iv. Refractory period</td>
<td>Short-within latent period</td>
</tr>
<tr>
<td></td>
<td>a. Tetanus</td>
<td>Possible</td>
</tr>
<tr>
<td></td>
<td>b. Fatigue</td>
<td>Possible</td>
</tr>
<tr>
<td></td>
<td>v. Tonicity</td>
<td>Tone depends on nerves</td>
</tr>
<tr>
<td><strong>3. Composition</strong></td>
<td>i. Protein</td>
<td>Maximum</td>
</tr>
<tr>
<td></td>
<td>ii. Glycogen</td>
<td>Less</td>
</tr>
<tr>
<td></td>
<td>iii. Carnosine</td>
<td>Maximum</td>
</tr>
<tr>
<td><strong>4. Metabolism</strong></td>
<td>Blood supply and O₂ consumption</td>
<td>Moderate</td>
</tr>
<tr>
<td><strong>5. Distribution</strong></td>
<td>Skeletal (Biceps, Triceps etc.)</td>
<td>Hollow viscera, capsules, skin, etc.</td>
</tr>
<tr>
<td><strong>6. Control</strong></td>
<td>Under the will, so voluntary</td>
<td>Not so, involuntary</td>
</tr>
<tr>
<td><strong>7. Nerve Supply</strong></td>
<td>Somatic with special nerve endings</td>
<td>Autonomic with ganglia and free nerve terminals</td>
</tr>
</tbody>
</table>

#### B. Difference between skeletal and cardiac muscles:

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Skeletal</th>
<th>Cardiac</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1. Histology</strong></td>
<td>i. Size and Shape</td>
<td>Cylindrical; 1–40 mm long, 10–100 µ in diameter</td>
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<tr>
<td></td>
<td>ii. Nucleus</td>
<td>Multiple, just under the sarcolemma</td>
</tr>
<tr>
<td></td>
<td>iii. Sarcotubular System</td>
<td>Present, T-systems are at junctions of A-I band. Terminal cisterna is prominent</td>
</tr>
</tbody>
</table>

Contd...
### Muscular System

#### Characteristics

<table>
<thead>
<tr>
<th>iv. Branch</th>
<th>Nil</th>
<th>Multiple branches in all directions—three dimensional networks without cytoplasmic continuity</th>
</tr>
</thead>
<tbody>
<tr>
<td>v. Cell-to-cell</td>
<td>Nil</td>
<td>Through specialized areas of intercalated disc</td>
</tr>
<tr>
<td>conduction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>i. Rhythmicity</td>
<td>Nil</td>
<td>Present and characteristic</td>
</tr>
<tr>
<td>ii. Conductivity</td>
<td>Very fast</td>
<td>Slower, different in different parts</td>
</tr>
<tr>
<td>iii. All-or-none law</td>
<td>True for single fiber</td>
<td>True for the whole heart, because of functional syncytium</td>
</tr>
<tr>
<td>iv. Contractility</td>
<td>Simple muscle curve with characteristic features</td>
<td>Characteristic muscle curve contraction is longer than relaxation.</td>
</tr>
<tr>
<td>v. Refractory period</td>
<td>Short-within latent period</td>
<td>Longest, whole contraction period is absolute refractory</td>
</tr>
<tr>
<td>a. Tetanus</td>
<td>Possible</td>
<td>Possible</td>
</tr>
<tr>
<td>b. Fatigue</td>
<td>Possible</td>
<td>Impossible</td>
</tr>
<tr>
<td>vi. Tonicity</td>
<td>Tone depends on nerves</td>
<td>Independent of nerve</td>
</tr>
</tbody>
</table>

#### Properties

<table>
<thead>
<tr>
<th>i. Protein</th>
<th>Maximum</th>
<th>Less</th>
</tr>
</thead>
<tbody>
<tr>
<td>ii. Glycogen</td>
<td>Less</td>
<td>More</td>
</tr>
<tr>
<td>iii. Carnosine</td>
<td>Maximum</td>
<td>Less</td>
</tr>
<tr>
<td>iv. Fats</td>
<td>Mostly neutral fats</td>
<td>More phosphatids and cholesterol than in others.</td>
</tr>
<tr>
<td>v. Inorganic</td>
<td>Na/K-1/5.</td>
<td>Na/K-1/2, i.e. more Na.</td>
</tr>
</tbody>
</table>

#### Action of ions

<table>
<thead>
<tr>
<th>i. Sodium</th>
<th>Excitation</th>
<th>Initiates and maintains heartbeat.</th>
</tr>
</thead>
<tbody>
<tr>
<td>ii. Calcium</td>
<td>Present mostly in sarcoplasmic reticulum and stimulates ATP-ase activity during muscular contraction.</td>
<td>Increases strength of contraction and duration of systole</td>
</tr>
<tr>
<td>iii. Potassium</td>
<td>Reduces excitability and hastens fatigue.</td>
<td>Inhibits contraction and produces relaxation</td>
</tr>
</tbody>
</table>

#### Metabolism

<table>
<thead>
<tr>
<th>i. Carbohydrates</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Lactic acid</td>
<td>Oxidized less easily than glucose and often incomplete.</td>
<td>Completely and more readily than glucose</td>
</tr>
<tr>
<td>b. Glycogen</td>
<td>Reduced in starvation and Diabetes Mellitus</td>
<td>Increased</td>
</tr>
<tr>
<td>ii. Blood supply and O₂ consumption</td>
<td>Moderate</td>
<td>High</td>
</tr>
</tbody>
</table>

#### Distribution

<table>
<thead>
<tr>
<th>Skeletal (Biceps, Triceps etc.)</th>
<th>Only in heart</th>
</tr>
</thead>
<tbody>
<tr>
<td>O₂ consumption</td>
<td>High</td>
</tr>
</tbody>
</table>

#### Control

<table>
<thead>
<tr>
<th>Under the will, so voluntary</th>
<th>Not so, involuntary</th>
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#### Nerve Supply

<table>
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<tr>
<th>Somatic with special nerve endings.</th>
<th>Autonomic with ganglia and free nerve terminals</th>
</tr>
</thead>
</table>
SKELETAL MUSCLE

There are more than 600 muscles in the body, which together account for about 40 percent of a person’s body weight.

Most skeletal muscles have names that describe some feature of the muscle. Often several criteria are combined into one name. Associating the muscle’s characteristics with its name will help to learn and remember them. The following are some terms relating to muscle features that are used in naming muscles.

- **Size**: vastus (huge); maximus (large); longus (long); minimus (small); brevis (short).
- **Shape**: deltoid (triangular); rhomboid (like a rhombus with equal and parallel sides); latissimus (wide); teres (round); trapezius (like a trapezoid, a four-sided figure with two sides parallel).
- **Direction of fibers**: rectus (straight); transverse (across); oblique (diagonally); orbicularis (circular).
- **Location**: pectoralis (chest); gluteus (buttock or rump); brachii (arm); supra- (above); infra- (below); sub- (under or beneath); lateralis (lateral).
- **Number of origins**: biceps (two heads); triceps (three heads); quadriceps (four heads).
- **Origin and insertion**: sternocleidomastoideus (origin on the sternum and clavicle, insertion on the mastoid process); brachioradialis (origin on the brachium or arm, insertion on the radius).

- **Action**: abductor (to abduct a structure); adductor (to adduct a structure); flexor (to flex a structure); extensor (to extend a structure); elevator (to lift or elevate a structure); masseter (a chewer).

Listed below are some significant and obvious muscles arranged in groups according to location and/or function.

1. Muscles of the Head and Neck
2. Muscles of the Trunk
3. Muscles of the Upper Extremity
4. Muscles of the Lower Extremity

Muscles of the Head and Neck

Humans have well-developed muscles in the face that permit a large variety of facial expressions. Because the muscles are used to show surprise, disgust, anger, fear and other emotions, they are an important means of nonverbal communication. Muscles of facial expression include frontalis, orbicularis oris, orbicularis oculi, buccinator and zygomaticus. These muscles of facial expression are identified in the illustration below.

There are four pairs of muscles that are responsible for chewing movements or mastication. All of these muscles connect to the mandible and they are some of the strongest muscles in the body.
Muscles of the Trunk

The muscles of the trunk include those that move the vertebral column, the muscles that form the thoracic and abdominal walls, and those that cover the pelvic outlet.

The erector spinae group of muscles on each side of the vertebral column is a large muscle mass that extends from the sacrum to the skull. These muscles are primarily responsible for extending the vertebral column to maintain erect posture. The deep back muscles occupy the space between the spinous and transverse processes of adjacent vertebrae.

The muscles of the thoracic wall are involved primarily in the process of breathing. The intercostal muscles are located in spaces between the ribs. They contract during forced expiration. External intercostal muscles contract to elevate the ribs during the inspiration phase of breathing. The diaphragm is a dome-shaped muscle that forms a partition between the thorax and the abdomen. It has three openings in it for structures that have to pass from the thorax to the abdomen.

The abdomen, unlike the thorax and pelvis, has no bony reinforcements or protection. The wall consists entirely of four muscle pairs, arranged in layers and the fascia that envelopes them. The abdominal wall muscles are identified in the illustration below.

The pelvic outlet is formed by two muscular sheets and their associated fascia.

Muscles of the Upper Extremity

The muscles of the upper extremity include those that attach the scapula to the thorax and generally move the scapula, those that attach the humerus to the scapula and generally move the arm, and those that are located in the arm or forearm that move the forearm, wrist and hand. The illustration below shows some of the muscles of the upper extremity.

Muscles that move the shoulder and arm include the trapezius and serratus anterior. The pectoralis major, latissimus dorsi, deltoid, and rotator cuff muscles connect to the humerus and move the arm. The muscles that move the forearm are located along the humerus, which include the triceps brachii, biceps brachii, brachialis and brachioradialis. The twenty or more muscles that cause most wrist, hand and finger movements are located along the forearm.

Muscles of the Lower Extremity

The muscles that move the thigh have their origins on some part of the pelvic girdle and their insertions on the femur. The largest muscle mass belongs to the posterior group, the gluteal muscles, which, as a group, abduct the thigh. The iliopsoas, an anterior muscle, flexes the thigh. The muscles in the medial compartment adduct the thigh.
The illustration above shows some of the muscles of the lower extremity. Muscles that move the leg are located in the thigh region. The quadriceps femoris muscle group straightens the leg at the knee. The hamstrings are antagonists to the quadriceps femoris muscle group, which are used to flex the leg at the knee. The muscles located in the leg that move the ankle and foot are divided into anterior, posterior, and lateral compartments. The tibialis anterior, which dorsiflexes the foot, is antagonistic to the gastrocnemius and soleus muscles, which plantar flex the foot.

**STRUCTURE OF SKELETAL MUSCLE**

A skeletal muscle consists of about 40 percent of the total body weight. As the name implies, they are attached to the bony skeleton and are responsible for limb movement, etc. The units of skeletal muscle are long cylindrical muscle fibers, which vary in length from 1–40 mm, and in diameter from 10–100 microns (1 micron is equal to 0.001 mm). Although the number of muscle fibers per muscle may vary somewhat depending upon the size and function of each muscle, it appears that by the time an embryo reaches a fetus stage of between 4 and 5 months of age, the actual number of muscle fibers per muscle is already established. Thus, the capability for an individual obtaining muscularity is apparently set genetically at the time of conception.

Each muscle fiber is composed of many smaller units called myofibrils, which lie parallel in the sarcoplasm muscle cell fluid similar to the intracellular fluid of other cells. Myofibrils are the contractile elements of the muscles, and they range from 1–2 microns in diameter. Each muscle fiber is surrounded by a thin elastic noncellular membrane called the sarcolemma. The muscle fibers are contained in bundles by sheaths of connective tissue (epimysium). The various connective tissue sheaths spread throughout the whole muscle and they are structurally blended...
with the tendons, by which the muscle is connected to the bones of the particular joint it moves.

Under the electron microscope, the myofibrils appear as alternate light (I band) and dark (A band) areas. Each myofibril consists of fine protein threads (myofilaments) called actin and myosin. The actin filament contains two important proteins called troponin and tropomyosin, while the myosin filaments contain small protein projections called cross bridges. Tropomyosin is a long, narrow molecule located on the surface of the actin filament with its ends fixed firmly in globular molecules of troponin. Along with the actin filaments the cross bridges play an important part in the contraction mechanism, as will be seen subsequently. The light bands (I) arise where there is only one protein, whereas the dark bands (A) arise where both actin and myosin strands are found.

The area in the center of each A band is a less dense region called the H zone. Each I band is halved by a dark line called the Z line or Z membrane. The Z lines lend stability to the entire structure. They may also play a major part in the
relaying of nerve impulses from the sarcolemma to the myofibrils. Thus, every myofibril is made up of units that encompass all elements between two Z lines. Each unit is called a sarcomere and is approximately 2 microns in length. The sarcomeres repeat themselves in a specific pattern in each myofibril.

**THE CONTRACTILE PROCESS: SLIDING FILAMENT THEORY**

Research utilizing the electron microscope has led scientist to hypothesize the currently accepted “sliding filament” theory of muscular contraction. During contraction the actin or thin filament is believed to slide over the myosin or thick filament toward the center of the sarcomere, thus shortening the muscle. The exact element involved in causing the sliding action of the filament during contraction has not been completely elucidates. It has been postulated that there is an interaction or “hook-up” between the two by way of “cross bridges” which allows the actin filaments to slide over the myosin filaments. This “hook-up” between actin and myosin forms a protein complex called actomyosin \((A + M \rightarrow AM)\). There are three different phases of muscular contraction which are as follows:

**A. Rest:** During rest, it is believed that the myosin cross bridges remain extended as a result of the electrostatic forces that exist at both ends of the bridges and both charges are negative. The high-energy ionized compound molecule called adenosine tri-phosphate- \((ATP)\), which is present at the end of the cross bridge with a negative charge along with a fixed negative charged at the base of the cross bridge. The two ends repel each other, thus allowing the bridges to stay extended. Because the active sites on the actin protein filaments also possess a negative charge, the cross bridges do not hook up to them during rest. During this period, this arrangement is referred to as an uncharged ATP cross bridge complex. It is generally believed that during rest, in the absence of calcium \((Ca^{++})\), these electrostatic charges in all of the aforementioned examples are due to the specific structure of actin, tropomyosin and troponin which, in turn, prevents the myosin cross bridge from interacting with actin.

**B. Contraction:** The following steps appear to be the normal process by which the bridges connect to the actin filaments so that contraction may occur. When the action potential (or stimulus delivered by a motor nerve fiber or electric shock) signal passes through the T system, \(Ca^{++}\) is released from the sarcoplasmic reticulum near the transverse

![Fig. 5.7: Electron microscopic structure of skeletal muscle fibers](image-url)
tubules in the form of free Ca++. The Ca++ diffuses to the active sites on the actin and myosin filaments and by some reaction involving tropomyosin, the Ca++ is quickly taken up by the troponin molecules on the actin filaments. It is believed that the chemical reactions involved in this process create electrostatic forces between the myosin ATP cross bridges and the actin protein filaments which cause them to attract each other and form actomyosin and thereby promote the sliding process. In other words, because Ca++, with two positive charges, are attracted to the negative charges of the myosin ATP cross bridges and the active sides on the actin protein filaments, an electrostatic bond between actin and myosin is developed (thus, the uncharged ATP cross bridge complex is transformed into a charged ATP cross bridge complex) and is thus responsible for the formation of actomyosin complex.

At the same time, the two positive charges of the Ca++ neutralized the two negative charges of the myosin ATP cross bridges and the actin filaments. At this point, the cross bridges which are now “hooked” up to the actin or thin filaments collapsed and pulls the actin filaments towards the middle of the sarcomere, thus causing the muscle to shorten. It has been postulated a single bridge hooks onto an active site, pulls the thin filaments a short distance to word the center of the sarcomere, releases it and then hooks onto the next active site. Thus, the actin filaments slides along over the myosin filaments by a “ratchet” mechanism, the actin molecule first locking with one cross bridge, then with the next (Fig. 5.9). Obviously, tension is developed during this movement since each actin filament is connected to the Z line. It should be pointed out that any one myosin cross bridge may “hook-up” and “break” with the actin filaments a hundred or more times during a period of one second. At the same time, however, within any one sarcomere the amount of tension buildup and the shortening that takes place is generally somewhat small. On the other hand it should be remembered that a high level of tension and shortening of intact muscle is due to several hundred sarcomeres contracting simultaneously.

Although the provision of energy for muscular contraction is a complex process, it would appear that at each cross bridge site where the protein of the two filaments are in contact, myosin acts as an enzyme (called myosin ATP-ase) to split a phosphate group from ATP and ADP and thus provide the energy for contraction. Once ATP is replenished and the negative charges are restored, the cross bridges are re-extended and the whole processes is repeated with new active sites on the thin protein filaments. Apparently, muscle contraction cannot take place (such as during rest) without this chemical bond between this actin and myosin. That is neither protein by itself is contractile. Neither the myosin nor the actin filaments decrease in length during normal contraction.

C. Relaxation: Following the cessation of the nervous impulses over the motor nerve innervating the muscle, the sarcoplasmic reticulum removes Ca++ from the troponin molecules on the actin filaments and stores it in the outer vesicles (Fig. 5.10). Once Ca++ has been withdrawn from the troponin, the actin filament is now no longer active. In addition, the myosins ATP cross bridge complexes are also now no longer able to form an electrostatic bond with the active sites of the actin filament. Since the myosin ATPase activity is now turned off, no more molecules of ATP can be split for energy. Thus, by way of elastic recoil, the muscle filaments return to and remain in a relaxed state.
Energy for muscular Contraction

The energy for muscular contraction is provided by the splitting of ATP (adenosine tri-phosphate) to ADP (adenosine di-phosphate) and phosphoric acid (ATP ↔ ADP + Pi + energy for contraction). Thus, when one molecule of ATP splits to form one molecule of ADP and a molecule of phosphoric acid, approximately 8000 calories of energy released. It appears that it is this energy which creates the force between myosin and actin filaments that cause muscle contraction.

After ATP has been broken down into ADP and phosphoric acid, it is essential that these substances be reconverted into ATP for reuse at a later period. Hence, this is done by the metabolism of carbohydrates, fats and proteins. For example, the metabolism of one molecule of glucose with oxygen to form carbon dioxide and water releases 680,000 calories of energy. The energy from this reaction is used to synthesize 38 molecule of ATP. Following contraction, the increase metabolism in a given muscle fiber restocks the chemical stores that are consumed during the contractile process.

**Basic Steps of Muscle Contraction Process According to the Sliding Filament Theory**

I. Rest:
   a. No nerve impulse
   b. Myosin uncharged ATP cross bridges remain extended
   c. Ca²⁺ remains store in sarcoplasmic reticulum
   d. Specific structure of actin, tropomyosin, and troponin prevents myosin and actin from interacting

II. Contraction:
   a. Nerve impulse to muscle results in an action potential
b. Action potential causes Ca++ to be released from sarcoplasmic reticulum

c. Ca++ diffuses to active sites on actin and myosin where it is taken up by troponin

d. Myosin ATP cross bridges becomes charged

e. Actin and myosin interact to form actomyosin

\( A + M \rightarrow AM \)

f. Myosin ATPase splits a phosphate group from ATP and ADP

g. Energy for muscle contraction is provided from the splitting of phosphate from ATP and ADP

\( ATP \rightarrow ADP + Pi + \text{energy} \) → myosin ATPase

h. Muscle contracts by the actin sliding over the myosin

III. Relaxation:

a. Nerve impulse stops

b. Sarcoplasmic reticulum removes Ca++ from troponin

c. Myosin ATP cross bridge becomes uncharged

d. Actomyosin dissociates into actin and myosin

e. ATP is resynthesized

f. Muscle is now back to its resting state

**Types of Muscle Action**

Muscle movement can generally be categorized into four different types of actions:

- Concentric (Isotonic)
- Static (Isometric)
- Eccentric
- Isokinetic

In many activities, such as running and jumping, all three types of actions may occur in the execution of a smooth, coordinated movement.

**Concentric (Isotonic) action:** A muscle’s principal action, shortening, is referred to as a concentric action. Human are most familiar with this type of action. Concentric actions are considered dynamic action.

All lifting exercises require Isotonic contractions. This happens when the muscle shortens as it contracts. An example of isotonic contraction can be seen when we flex the biceps muscle. Stand with one arm straight and the palm of the hand facing up. Roughly measure the length from the start of the biceps muscle to the point where it meets the shoulder. Now curl the hand towards the shoulder, the biceps muscle shortens as it contracts. When you reach the end point take another rough measurement of the biceps again, it will be much shorter.

Another example is the triceps muscle (opposite of biceps). Do the same experiments again this time measure the triceps and start at the curled position. The triceps shortens as the arm straightens.

**Other examples are as follows:**

- Lifting objects above the head - front shoulder (anterior deltoid) shortens
- Lifting object up from lying position - chest muscle shortens
- Lifting body up from squat position - quadriceps muscle shortens as legs extend

![Fig. 5.11: Concentric Isotonic contraction](image-url)
A Textbook of Sports and Exercise Physiology

- doing a sit up
- throwing a ball
- swinging a bat

In fact, Isotonic contractions are the most common, many exercises and activities involve this type of contraction.

**Static (Isometric) action:** Muscle can also act without moving. When this happens, the muscle generates force but its length remains unchanged (static). This is called a static action. This has also been termed as isometric action. This occurs, for example, when you try to lift an object that is heavier than the force generated by your muscle, or when you support the weight of an object by holding it steadily with your elbow flexed. In both cases, you fill your muscle tense but they can not move the weight so they do not shorten. In this action the myosin cross bridge formed and recycled, producing force, but the external force is too great for the actin filaments to be moved. They remain in their normal position, so shortening cannot occur.

Some bodybuilders makeup their own exercises using Isometric contraction in order to develop strength. An example is when someone attempts to curl one arm upwards but is held by using equal resistance from the other arm.

- attempting to lift an immovable object
- holding a weight at arm’s length
- some wrestling movements

The above examples are advanced forms of exercise and should not be attempted when losing weight.

**Eccentric action:** Muscles may even exert force while lengthening. This movement is an eccentric action. In this case joint movement occurs and this is also a dynamic action. An example of this is the

![Fig. 5.12: Isometric (Static) contraction](image)

![Fig. 5.13: Eccentric contraction](image)
action of the biceps brachii when your elbow is extended to lower a heavy weight. In this case, the actin filaments are pulled further away from the center of the sarcomere, essentially stretching it.

An example is when someone manages to pull your arm straight while at the same time you are try to keep the arm locked in one position. In other words, the load is too heavy.

Other examples are as follows:
- running downhill
- walking downstairs
- landing on the ground from a jump

This type of contraction is not always recommended.

Isokinetic

Similar to the Isotonic contraction, the Isokinetic contraction causes the muscle to shorten as it gains tension. The difference is Isokinetic requires a constant speed over the entire range of motion, therefore, this type of contraction require special equipment to exercise properly. An example is an arm stroke when swimming, the even resistance from the water offers a constant speed at maximal contractions.

Muscle Fiber

All motor units function in a similar manner although they may have different contractile and metabolic characteristics. Some motor units are more suited for aerobic metabolism, whereas others are more appropriate for anaerobic activity. Two distinct fiber types have been identified and classified by their contractile and metabolic characteristics. These have been termed slow-twitch and fast-twitch fibers, also referred to as type I and type II fibers, respectively. These fibers possess certain distinguishable characteristics that make them suited either for prolonged, low to moderate intensity activity (slow-twitch fibers) or short-duration, high-intensity activity (fast twitch fibers).

For many years, muscle fiber classification was limited to these two classifications (type I and type II), with type II fibers being further subdivided into two distinct divisions: IIA and IIB. Type IIA fibers have a well-developed capacity for both aerobic and anaerobic metabolism and are commonly termed fast oxidative glycolytic. Type IIB fibers possess the greatest anaerobic capability and are termed fast glycolytic. In the last decade, with the improvement of muscle-staining techniques, additional subtypes within each fiber type have been reported by various scientists at beginning of nineties. Subtypes of type I oxidative fibers have been labeled type I and type Ic. Type Ic fibers are thought to have less oxidative capacity than type I fibers. There have also been five different fiber subtypes identified for type II fibers. These fibers, IIc, IIac, IIA, IIab and IIB, represent a continuum of aerobic and anaerobic characteristics.

It is believed that genetics largely determine muscle fiber type distribution and that it is set at birth or early in life. The average individual (man or woman) has an equal proportion of both slow and fast twitch fibers. The composition of muscle fiber types (percentage of type I to type II) is consistent among the major muscle groups in the body.

On the average, most muscles are composed of roughly 50 percent. Type I fibers and 25 percent Type IIA fibers. The remaining 25 percent are mostly Type IIB, with Type IIc fibers making up only 1 percent to 3 percent of the muscles.

In elite athletes, the predominance of a particular fiber type appears to correspond to the metabolic requirements of their respective sport.

![Fig. 5.14: Percentage of Type I fiber in distance runners, middle distance runners and sprinters](image)
Characteristics of Type I and Type II Muscle Fibers (Type IIa and Type IIb):

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Type I (Slow Twitch)</th>
<th>Type IIa (Fast Twitch)</th>
<th>Type IIb (Fast Twitch)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Force production</td>
<td>Low</td>
<td>Intermediate</td>
<td>High</td>
</tr>
<tr>
<td>Contraction speed</td>
<td>Slow</td>
<td>Fast</td>
<td>Fast</td>
</tr>
<tr>
<td>Myofibrillar ATPase activity</td>
<td>Low</td>
<td>High</td>
<td>High</td>
</tr>
<tr>
<td>Fatigue resistance</td>
<td>High</td>
<td>Moderate</td>
<td>Low</td>
</tr>
<tr>
<td>Glycolytic capacity</td>
<td>Low</td>
<td>High</td>
<td>High</td>
</tr>
<tr>
<td>Oxidative capacity</td>
<td>High</td>
<td>Medium</td>
<td>Low</td>
</tr>
<tr>
<td>Capillary density</td>
<td>High</td>
<td>Intermediate</td>
<td>Low</td>
</tr>
<tr>
<td>Mitochondrial density</td>
<td>High</td>
<td>Intermediate</td>
<td>Low</td>
</tr>
<tr>
<td>Myoglobin content</td>
<td>High</td>
<td>Intermediate</td>
<td>Low</td>
</tr>
<tr>
<td>Endurance capability</td>
<td>High</td>
<td>Moderate</td>
<td>Low</td>
</tr>
<tr>
<td>Glycogen storage capability</td>
<td>No difference</td>
<td>No difference</td>
<td>No difference</td>
</tr>
</tbody>
</table>

Endurance athletes have a large percentage of type I fibers, whereas highly anaerobic athletes (e.g., sprinters) have a predominance of type II fibers. Elite endurance athletes may have 90 percent of their skeletal muscle made up of type I fibers, providing a large advantage for success in aerobic performance. Similarly, athletes with exceptional explosive power and speed would have a predominance of type II fibers.

**Fiber Type Conversions**

The proportion of type I to type II muscle fibers appear to be genetically determined and their expression set early in life. A number of studies have examined whether conditioning programs can alter the proportion of type I to type II muscle fibers. Some studies have suggested that aerobic training may be able to increase the percentage of type I fibers, while others have reported increases in type II fiber proportion after sprint training. However, the overwhelming majority of investigations have been unable to see any alterations in fiber-type composition as a consequence of conditioning programs. It is generally believed that only fiber type transformations within a fiber type can be accomplished through training.

Scientists have reported that high intensity resistance training appears to be a potent stimulus in causing a transformation of the type IIb to type IIa fiber subtype. Most of the type IIb fibers have been reported to be converted to type IIa fibers after 20 weeks of resistance training. This is similar to the type II fiber conversions previously thought to be associated with aerobic exercise training. Kraemer, Patton, and their colleagues (1995) have also demonstrated that skeletal muscle fiber subtype transformations from IIb to IIa in subjects performing high-intensity resistance training and in subjects performing a combined high-intensity resistance training and endurance training program. Subjects who were performing only endurance exercises also tended to increase the proportion of type IIa fibers but significantly elevated their type IIc fibers. This would be expected considering that the type IIc fibers are the most oxidative of the type II subtypes.

Fiber subtype transformations appear to occur rapidly (within 2 weeks) during participation in physical conditioning programs. These adaptations, however, may be transient. During periods of inactivity or detraining, a transformation of fast-twitch fiber subtypes from type IIa back to type IIb is observed. A return to training will result in a fiber-type transformation back to its trained state in a relatively shorter period of time. These studies highlight the dynamic nature of skeletal fiber transformations.

**Effect of Concurrent Training on Muscle Growth and Muscle Fiber Characteristics**

Many endurance athletes are concerned that the inclusion of a resistance training program would cause physiological changes to the muscle that would be detrimental to endurance performance (e.g. muscle hypertrophy, decrease in mitochondrial volume and capillary density). In some studies examining this particular question,
no significant alterations in muscle size or muscle fiber composition were reported in endurance-trained subjects performing resistance exercise for the first time. It should be noted, however, that these studies were 10 to 12 weeks in duration. It is generally understood that muscle hypertrophy occurs approximately 6 to 8 weeks after the initiation of a resistance-training program in previously untrained individuals. It is possible that the time course for muscle adaptations in individuals with limited resistance training experiences may be longer when concurrently performing another mode of training. Based on available evidence, it appears that 10 to 12 weeks of resistance training added to the exercise program of endurance-trained subjects do not cause any significant changes to muscle fiber composition or to fiber cross-sectional area. The effect of training durations exceeding 3 months is not known. However, by manipulating acute program variables, the coach and athlete can specifically focus on the physiological adaptations that are beneficial to the endurance athlete.

The effect of concurrent training on muscle adaptations in other population groups may be different. In previously untrained subjects, 6 weeks of resistance training resulted in a significant increase in the muscle fiber area of both type I and type II fibers in a group that performed only strength training. These muscles continued to hypertrophy even after 12 weeks of training. In comparison, a group of subjects performing both endurance and resistance training showed no significant changes in muscle fiber area after 6-weeks of training. However, after 12 weeks of training, significant increases were observed in the muscle fiber area of type II fibers only. This study, although showing significant increases in muscle fiber area, did suggest that combining both endurance and strength training might suppress some of the adaptations observed when performing only strength training. Figure below demonstrated that significant increases in both type I and type II fibers were seen in both strength and combined strength and endurance trained groups. A significant reduction in the type I fiber was seen in a group of subject performing only endurance training. This indicated that benefit of resistance training for the endurance trained individuals.

Subject who performed only strength training or performed both strength and endurance training so similar type II fiber subtype transformations (IIa ← IIb) after training in contrast, subjects who performed only endurance training had a die to muscle fiber subtype transformations from fibers that were more glycolytic to fiber subtypes that were more oxidative in nature (IIc ← IIa ← IIb). In other words, the inclusion of a high intensity resistance-training program appears to reduce the magnitude of the type II fiber transformation to more oxidative fibers.

**Fig. 5.15:** Comparison of effects of strength training, endurance training and concurrent training on muscle fiber type
**Muscle Fiber Distribution and Sports Performance**

The human skeletal muscles have a mixture of both slow (ST) and fast (FT) twitch fibers. While the range of fiber mixture is relatively wide (ranging from 40–87% for FT and 13–60% for ST), biopsy research has shown that there are specific muscles that are regarded as having predominantly either ST or FT fibers. For instance, the soleus, semitendinosus, vastus lateralis, rectus abdominis, and rectus femoris are just a few that contain a large percentage of ST fibers (Type I). At the same time, the brachii, deltoid, gastrocnemious and latissimus dorsi are a few that have high levels of FT (Type II) fibers.

Investigations have also shown a rather interesting relationship between specific fiber types and certain athletic abilities. For example, world-class sprinters are characterized by high percentage (up to around 74%) of FT fibers in their leg muscles, while at the same time, world-class distance runners possess a much higher level of ST fibers (Type I). At the same time, the brachii, deltoid, gastrocnemious and latissimus dorsi are a few that have high levels of FT fibers.

While it would appear that muscle fiber composition may play an important part in determining championship performance, an important question that needs to be answered is what effect does training have on an individual’s inherited muscle fiber population. Various studies have shown that only the capacities and size of the fibers increase as the result of training and not the number or twitch contraction times. In other words, with endurance training, both fiber types become better suited for producing ATP for aerobic work, while at the same time, fiber types can still be clearly identified and separated on the basis of twitch contraction times since FT fibers remain FT fibers and are not changed into ST fibers. While training may cause an increase in size and capacities, in short, it does not bring about changes in the proportion of ST and FT fibers in a muscle. The only way that a ST fiber can be functionally changed to FT fibers or vice versa would be to actually remove the original motor nerve from one fiber type and transplant it to the other fiber or vice versa.

However, an individual who has a high percentage of FT fiber in the leg muscles may be a world-class sprinter but not necessarily. While muscle fiber composition is apparently important, there are many other factors that enter into making championship performance. Factors such as dedication, motivation, training, skill, body build, coaching and in some cases, plain old luck that may actually overshadow an apparent physiological advantage such as fiber type. This is especially true at the lower levels of competition, whereas at the national and international levels, inheritance of fiber types probably plays a more important role in determining championship performance.

**Skeletal Muscle Adaptation**

Human body has immense capacity to adapt itself with wide variation in load and environmental conditions. Muscles show adaptative changes due to short-term and long-term training. Natures of these changes depend on the type of training undertaken.

Skeletal muscle is dynamic in its response to training and adapt to a wide range of functional demands. When skeletal muscle is forced to work intensities exceeding to 60 to 70 percent of its maximal force generating capacity, adaptations occur that may result in an increasing muscle size and strength.

The initial increases seen in muscle strength after resistance training have been attributed primarily to the neurological adaptation further increases in skeletal muscle strength appear to be the result of a growth in muscle size. An increase in the size of pre-existing muscle fiber (hypertrophy) or an increase in the number of fiber within the muscle (hyperplasia) may result in skeletal muscle growth.

**Endurance Training**

Blood supply: Number of capillaries per muscle fiber increase with endurance training. The quantity
of blood flow is also increases. In untrained persons exercise does not make open all the capillaries existed in the muscle. In short-term training these capillaries opens up to meet the requirement. In long-term adaptation the capillaries branch out and increase the density.

**Chemical Changes in the Muscle**

Endurance training leads to the changes in type IIb fibers in favor of type IIa fiber, so the number of type IIa fiber increases. Glycogen content of the muscle cell increases. No change or slight increases in myoglobin content of the muscle has been indicated.

Number of mitochondria in the muscle cell increase with endurance training. This increase in turn increases the amount of oxidative enzymes. Fatty acid oxidation is increased with endurance training which delays the critical accumulation of lactic acid.

**Strength Training**

Strength training is mainly aimed at increasing the muscle mass. However, no short-term physiological adaptation is possible even if there is increase in muscle strength. It is a common experience that the muscle bulk increases with strength training. This increase in muscle bulk is not due to the increase in the number of muscle fibers. The mean cross-sectional area of the existing fibers increase considerably and this particular increase is mainly among the fast twitch fibers. This is called as the ‘Hypertrophy of the muscle’. Also hypertrophy is more in male than in female. The myofibrils also split longitudinally in two or more myofibrils in the same muscle fiber.

In long-term strength training the percentage of type IIa fibers increase by changing the characteristics of Type IIb fibers. Number of capillaries per muscle fiber does not change; a capillary per unit of muscle area is markedly reduced in lifters. Body builder show comparatively higher number of capillaries.

Number of mitochondria reduces and enzymes related to anaerobic energy production increases. Amount of stored ATP and CP increases. Decrease in myoglobin content was found in many strength trained athletes.

**Speed Training**

Basic speed is not a trainable element. Speed of running is normally increased through strength execution. Data regarding changes due to speed training is only limited. The store of ATP and CP may increase and there may be more dependence on anaerobic glycolysis.

**Muscle Hypertrophy**

Increase in muscle size is generally seen after 6 to 8 weeks of heavy resistance training. However, some evidence suggests that muscle growth may occur even earlier. Increases in muscle size have been attributed to increases in the cross sectional area of existing muscle fiber. This process of fiber growth appears to be related to the increase synthesis of contractile proteins (actin and myosin filaments) and to the increase formation of sarcomere within the fiber. The synthesis of these filaments, which constitute the contractile element of muscle fiber, may be related to the repeated trauma to the fibers from high intensity resistance training. During recovery from the cellular damage caused by such training, an over compensation of proteins synthesis may occur, resulting in the noted anabolic effects.

Muscles hypertrophy in both type I and type II fibers after resistance training programs. However, the type II fiber appears to undergo a great relative hypertrophy. Since both type I and type II fibers are recruited during maximal contraction the greater hypertrophy seen in the type II fiber may be related to the greater activation high threshold units than normally activated during daily activity.

Increases in fiber size do not appear to be accompanied by increases in mitochondrial number or in the capillary to fiber ratio. This lowering of the mitochondrial and capillary volume density in the fibers may not hinder strength or power performance, but it may have important implications for endurance capability in those muscles. This change might alter the oxygen kinetics within the muscle by delaying transport of oxygen from the vasculature to the exercising muscle. It is noted that endurance training decreases fiber size while causing increases to both mitochondrial and capillary density, thus potentially improving the aerobic capacity of the muscle. In
contrast the sarcoplasmic reticulum and transverse tubule volume density increases in proportion to the change in myofibrillar volume, thus maintaining or improving contraction capabilities of the muscle.

**Muscle Hyperplasia**

It has been generally known that muscle fiber number is fixed from birth and that skeletal muscle growth is a result of hypertrophy of existing muscle fibers. However, a number of studies have suggested that high intensity resistance training may cause muscle hyperplasia.

For animals, muscle hyperplasia may be an important compensatory mechanism for combating muscle overload. It has been reported that bodybuilders had a greater number of muscle fibers than trained control subjects. This suggested that the greater number of fibers seen in the bodybuilders was attributable to years of high intensity resistance training.

If muscle hyperplasia does occur, it is thought to be either through the development of new fibers from satellite cells. Satellite cells (located between the basement membrane and the plasma membrane) are thought to proliferate and grow to a myoblast and eventually myotubes that may develop into the new muscle fibers. The myotube that may also fuse with existing muscle fibers and remain incomplete along its length, leading to the wrong impression of a split fiber. With longitudinal splitting, a hypertrophied muscle fiber that has reached some predetermined maximal ceiling of growth is thought to split into two or more smaller daughter cells through a process of lateral budding.

There does not appear to be any convincing support for the occurrence of muscle hyperplasia in humans. However, conflicting results still make this issue controversial and its potential appealing. If hyperplasia does exist, it most probably occurs in a small portion of type II fibers when they reach their predetermined genetic growth limit.

**SUMMARY**

1. Structurally muscles are classified into two classification, i.e. (I) Striated (II) Non-Striated. Again Striated muscles are subdivided into two- (a) Skeletal (b) Cardiac. On the other hand muscles are classified into two categories depending on their functions. They are the (i) voluntary (Skeletal muscle) and (ii) involuntary muscles (Cardiac and Smooth muscles).

2. There are more than 600 muscles in the body, which together account for about 40 percent of a person’s body weight. Humans have well-developed muscles in the face that permit a large variety of facial expressions. The muscles of the trunk include those that move the vertebral column, the muscles that form the thoracic and abdominal walls, and those that cover the pelvic outlet. The muscles of the upper extremity include those that move the arm, the muscles that form the torso, the muscles that move the arm, and those that are located in the arm or forearm that move the forearm, wrist and hand.

3. The muscles that move the thigh have their origins on some part of the pelvic girdle and their insertions on the femur. The largest muscle mass belongs to the posterior group, the gluteal muscles, which, as a group, abduct the thigh. The iliopsoas, an anterior muscle, flexes the thigh. The muscles in the medial compartment adduct the thigh.

4. The units of skeletal muscle are long cylindrical muscle fibers, which vary in length from 1 to 40 mm, and in diameter from 10 to 100 microns (1 micron is equal to 0.001 mm). Under the electron microscope, the myofibrils appear as alternate light (I band) and dark (A band) areas. Each myofibril consists of fine protein threads (myofilaments) called actin and myosin.

5. During contraction the actin or thin filament is believed to slide over the myosin or thick filament toward the center of the sarcomere, thus shortening the muscle. The exact element involved in causing the sliding action of the filament during contraction has not been completely elucidates. It has been postulated that there is an interaction or “hook-up” between the two by way of “cross bridges” which allows the actin filaments to slide over the myosin filaments.
6. The energy for muscular contraction is provided by the splitting of ATP to ADP and phosphoric acid ($\text{ATP} \leftrightarrow \text{ADP} + \text{Pi} + \text{energy}$ for contraction). Thus, when one molecule of ATP splits to from one molecule of ADP and a molecule of phosphoric acid, approximately 8000 calories of energy released. It appears that it is this energy that creates that force between myosin and actin filaments that cause muscle contraction.

7. Muscle movement can generally be categorized into three types of actions: Concentric (Isotonic), Static (Isometric), Eccentric and Isokinetic. In many activities, such as running and jumping, all three types of actions may occur in the execution of a smooth, coordinated movement.

8. Concentric (Isotonic) Action: A muscle’s principal action, shortening, is referred to as a concentric action. Human are most familiar with this type of action. Muscle can also act without moving. When this happens, the muscle generates force but its length remains unchanged (static). This is called a static action. Muscles may even exert force while lengthening. This movement is an eccentric action. In this case joint movement occurs and this is also a dynamic action. Similar to the isotonic contraction, the Isokinetic contraction causes the muscle to shorten as it gains tension. The difference is Isokinetic requires a constant speed over the entire range of motion, therefore, this type of contraction require special equipment to exercise properly.

9. Two distinct muscular fiber types have been identified and classified by their contractile and metabolic characteristics. These have been termed slow-twitch and fast-twitch fibers, also referred to as type I and type II fiber, respectively. These fibers possess certain distinguishable characteristics that make them suited either for prolonged, low to moderate intensity activity (slow-twitch fibers) or short-duration, high-intensity activity (fast twitch fibers). The proportion of type I to type II muscle fibers appear to be genetically determined and their expression set early in life. A number of studies have examined whether conditioning programs can alter the proportion of type I to type II muscle fibers.

10. The human skeletal muscles have a mixture of slow (ST) and fast (FT) twitch fibers. While the range of fiber mixture is relatively wide (ranging from 40 to 87% for FT and 13 to 60% for ST), biopsy research has shown that there are specific muscles that are regarded as having predominantly either ST or FT fibers. World-class sprinters are characterized by high percentage (up to around 74%) of FT fibers in their leg muscles, while at the same time, world-class distance runners possess a much higher level of ST fibers (up to around 74%) in their leg muscles than normal proportion for untrained subjects.

11. Skeletal muscle is dynamic in its response to training and adapt to a wide range of functional demands. When skeletal muscle is forced to work intensities exceeding to 60 to 70 percent of its maximal force generating capacity, adaptations occur that may result in an increasing muscle size and strength. Number of capillaries per muscle fiber increase with endurance training. The quantity of blood flow also increases. In untrained persons exercise does not make open all the capillaries existed in the muscle. In short-term training these capillaries opens up to meet the requirement. In long-term adaptation the capillaries branches out and increase the density.

12. Muscles hypertrophy in both type I and type II fibers after resistance programs. However, the type II fiber appears to undergo a great relative hypertrophy. Since both type I and type II fibers are recruited during maximal contraction the greater hypertrophy seen in the type II fiber may be related to the greater activation high threshold units than normally activated during daily activity.

**Review Questions**

1. Classified the muscle according to their structure and functions. Write down basic properties of muscle.
2. What are the similarity and difference among skeletal muscle, cardiac muscle and smooth muscle?
3. Write down some groups of muscle of Head and Neck, Lower extremity, Muscles of trunk, and Upper extremity.

4. Draw a neat label diagram of the ultrastructure of skeletal muscle and describe each component.

5. Discuss the basic steps of muscular contraction according to the ‘Sliding Filament theory’.

6. Describe the different types of muscle actions with suitable diagram and example.

7. What are the basic characteristics of slow and fast twitch muscle fibers?

8. What is the role of genetics in determining the proportions of muscle fiber types and the potential for success in selected sports activities?

9. What are the effects of training on conversion of muscle fiber typing? Discuss with suitable example.

10. Write down about the relationship of muscle fiber distribution and athletic performance.

11. What is the pattern of muscle fiber recruitment during (a) high jump; (b) running a 10 km race, and (c) running a marathon?

12. Differentiate and give examples of concentric, static, and eccentric actions.

13. What are the effects of aerobic and anaerobic training of muscle fiber?

14. Write brief notes on (i) muscle hypertrophy and (ii) muscle hyperplasia.
A hormone (from Greek ὁρμή - “impetus”) is a chemical released by one or more cells that affects cells in other parts of the organism. Only a small amount of hormone is required to alter cell metabolism. It is essentially a chemical messenger that transports a signal from one cell to another. All multicellular organisms produce hormones; plant hormones are also called phytohormones. Hormones in animals are often transported in the blood. Cells respond to a hormone when they express a specific receptor for that hormone. The hormone binds to the receptor protein, resulting in the activation of a signal transduction mechanism that ultimately leads to cell type-specific responses.

Endocrine hormone molecules are secreted (released) directly into the bloodstream, while exocrine hormones (or ectohormones) are secreted directly into a duct, and from the duct they either flow into the bloodstream or they flow from cell to cell by diffusion in a process known as paracrine signalling.

**Definition of Hormone:** Hormones are chemical substances that circulate in the blood and interact with organs in the body to help combat various stresses. The primary role of hormones is to help combat various stresses and to maintain internal equilibrium (homeostasis). Most hormones are synthesized in endocrine glands located throughout the body. Upon stimulation, the glands secrete their hormones into the surrounding extracellular space. The hormones then diffuse into the circulatory system and are transported to their respective target areas to perform their designated function. It can exert their influence on specific target tissues or cells through the unique interaction between a hormone and the receptors, on the cell membrane or within the cell, that are specific to that hormone.

**Overview of Endocrine System**

The endocrine system is a group of glands that work together and secrete many types of different hormones that regulate the body functions. The field of study that deals with disorders of endocrine glands is endocrinology, a branch of the
wider field of internal medicine. The endocrine system is an information signal system much like the nervous system. Hormones regulate many functions of an organism, including mood, growth and development, tissue function and metabolism.

Hormones influence the rate of specific cellular reactions by changing the rate of protein synthesis or enzyme activity and by including secretion of other hormones. In addition, hormone can facilitate or inhibit uptake of substance by cells. For example, insulin facilitates the uptake of glucose into the cell, and epinephrine inhibits glucose uptake to increase its concentration in the circulation.

Hormones can stimulate the production of enzymes or activate inactive enzymes. They can also combine with an enzyme to alter its space (allosteric modulation), which will cause either an increase or decrease in the effectiveness of the enzyme.

Generally hormones are of two types: they are (i) steroid hormones and (ii) non-steroid hormones. Steroid hormones have a chemical structure similar to cholesterol, and most are derived from it. For this reason, they are lipid soluble and non steroid hormones are not lipid soluble.
# Endocrine System

## Endocrine Glands: Their Secretion and Functions

Table 6.1: Endocrine glands, their secretions, functions, control factors, effects of hypo and hyper secretion and the effects of exercise on hormone output

<table>
<thead>
<tr>
<th>Host Gland</th>
<th>Hormone</th>
<th>Hormone effects</th>
<th>Control of hormone secretion</th>
<th>Effects of hypo and hyper secretion</th>
<th>Exercise effects on hormone secretion</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Hypothalamus</td>
<td>Thryotropin Releasing Factor (TRF)</td>
<td>Stimulates anterior pituitary to secrete TSH</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Corticotropin Releasing Factor (CRF)</td>
<td>Stimulates anterior pituitary to secrete ACTH</td>
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<td></td>
<td>Somatostatin</td>
<td>Inhibits secretion of GH from anterior pituitary</td>
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<tr>
<td></td>
<td>FSH Releasing Factor (FSHRF)</td>
<td>Stimulates anterior pituitary to secrete FSH</td>
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<tr>
<td></td>
<td>LH Releasing Factor (LHRF)</td>
<td>Stimulates anterior pituitary to secrete LH</td>
<td></td>
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<tr>
<td></td>
<td>Prolactin Releasing Factor (PRF)</td>
<td>Stimulates anterior pituitary to secrete hormone prolactin</td>
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<tr>
<td></td>
<td>Growth Hormone Releasing Factor (GHRF)</td>
<td>Controls the release of growth hormone from anterior pituitary</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>A. Anterior Pituitary</td>
<td>Thyroid Stimulating Hormone (TSH)</td>
<td>Stimulates production and release of thyroxine from thyroid gland</td>
<td>Hypothalamic TSH-releasing factor; thyroxine</td>
<td>Hyper: (i) Gigantism in children (ii) Acromegaly in adults</td>
<td>Increases With increasing exercise</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Hypo: (i) Cretinism in children: stunted growth, mental retardation (ii) Myxoedema in adults: low BMR, constipation, dry skin, puffy eyes, edema, lethargy</td>
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<thead>
<tr>
<th>Host Gland</th>
<th>Hormone</th>
<th>Hormone effects</th>
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<th>Exercise effects on hormone secretion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adrenocorticotrophic Hormone (ACTH)</td>
<td>Stimulates production and release of cortisol, aldosterone and adrenal hormones</td>
<td>Hypothalamic ACTH-releasing factor, cortisol</td>
<td>Hypo: Rarely seen</td>
<td>Hyper: Cushing’s disease</td>
<td>Unknown</td>
</tr>
<tr>
<td>Lutinizing Hormone (LH)</td>
<td>LH stimulates for the production of progesterone by ovaries.</td>
<td>Hypothalamic LH releasing factor; Female progesterone; Hypothalamic FSH releasing factor; female estrogen and Male testosterone.</td>
<td>Hypo: failure of sexual maturation</td>
<td>Hyper: none</td>
<td>No change</td>
</tr>
<tr>
<td>Follicle Stimulating Hormone (FSH)</td>
<td>FSH stimulates for the production of estrogen by ovaries and testosterone by male testes</td>
<td>Hypothalamic FSH releasing factor; female estrogen and Male testosterone.</td>
<td>Hypo: failure of sexual maturation</td>
<td>Hyper: none</td>
<td>No change</td>
</tr>
<tr>
<td>Prolactin (PRL)</td>
<td>(i) Inhibits testosterone (ii) Mobilizes fatty acids</td>
<td>Hypothalamic PRL-inhibiting factor</td>
<td>Hypo: Poor milk production in nursing women</td>
<td>Increases with increasing exercise</td>
<td>Hyper: (i) Cessation of menses in females (ii) Importance in males galactorrhea,</td>
</tr>
<tr>
<td>Endorphins</td>
<td>(i) Blocks pain (ii) Promotes euphoria (iii) Affects on feeding (iv) Affects on female menstrual cycle</td>
<td>Stress-physical/emotional (may be intensity related)</td>
<td>Unknown</td>
<td>Increases with long duration exercise</td>
<td></td>
</tr>
<tr>
<td>Host Gland</td>
<td>Hormone</td>
<td>Hormone effects</td>
<td>Control of hormone secretion</td>
<td>Effects of hypo and hyper secretion</td>
<td>Exercise effects on hormone secretion</td>
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<tr>
<td>B. Posterior Pituitary</td>
<td>Oxytocin</td>
<td>(i) Stimulates contraction of uterine muscles</td>
<td>Neural impulse from the hypothalamus</td>
<td>Not known</td>
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<td></td>
<td></td>
<td>(ii) Stimulates the release of milk from mammary gland</td>
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<td></td>
<td>Antidiuretic Hormone (ADH) or Vasopressin</td>
<td>(i) Stimulates water reabsorption by distal and collecting tubules</td>
<td>Neural impulse from the hypothalamus</td>
<td></td>
<td>ADH promotes water conservation by increasing water permeability of the kidney and collecting ducts</td>
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<td></td>
<td></td>
<td>(ii) Reduces water loss in urine</td>
<td></td>
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<td></td>
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<tr>
<td>3. Thyroid Gland</td>
<td>Thyroxin</td>
<td>(i) Increases cell metabolism and energy production</td>
<td>Increased or decreased water excretion</td>
<td></td>
<td>Increase the plasma thyroxine level in maximum exercise but remain same during submaximal exercise</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(ii) Regulates growth and sexual maturity</td>
<td></td>
<td></td>
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<td></td>
<td>Calcitonin</td>
<td>Decreases calcium ion concentration in blood</td>
<td>Hypo: Causes simple goitre</td>
<td></td>
<td>Not known</td>
</tr>
<tr>
<td>4. Parathyroid Gland</td>
<td>Parathyroid Hormone (PTH)</td>
<td>(i) Increases blood calcium and lowers blood potassium</td>
<td>Hypo: i) Causes nerve and muscle abnormality ii) Tetanus like condition</td>
<td></td>
<td>Increased PTH level</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(ii) Accelerates excretion of phosphate</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>5. Pancreas (Islets of Langerhans)</td>
<td>Insulin</td>
<td>(i) Controls metabolism and transport of glucose (sugar and fats) into cell</td>
<td>Hypo: Causes diabetes mellitus</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(ii) Promotes protein synthesis</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Glucagon</td>
<td>(i) Controls breakdown of glycogen in liver</td>
<td>Gradual rise during the period of aerobic exercises</td>
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<tr>
<td></td>
<td></td>
<td>(ii) Increase lipid metabolism</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Host Gland</td>
<td>Hormone</td>
<td>Hormone effects</td>
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<tr>
<td>6. Adrenal Gland</td>
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<tr>
<td>A. Adrenal Cortex</td>
<td>Glucocorticoids (Cortisol and Corticosterone)</td>
<td>(i) Accelerates tissue protein mobilization and gluconeogenesis from proteins. (ii) Raises blood glucose level (iii) Promotes fat utilization (iv) Helps to overcome norepinephrine’s vasoconstricting effect.</td>
<td>Hypo: Causes loss of gluconeogenesis and more oxidation of glucose</td>
<td>Hyper: Causes impaired carbohydrate metabolism</td>
<td>Increase beging of the exercise and decreased gradually during the prolonged exercise</td>
</tr>
<tr>
<td></td>
<td>Mineralocorticoids (aldosterone)</td>
<td>(i) Increases sodium retention (ii) Increases potassium elimination (iii) Increases water retention</td>
<td>Hypo: Causes loss of sodium ions</td>
<td>Hyper: Leads to impaired salt balance</td>
<td>Increased</td>
</tr>
<tr>
<td></td>
<td>Androgens (Androsterone and others similar to testosterone)</td>
<td>In women it causes such masculine traits as beard, deep voice and regression of certain female reproductive organs.</td>
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</tr>
<tr>
<td>B. Adrenal Medulla</td>
<td>Epinephrine (Adrenaline)</td>
<td>(i) Increases blood pressure, blood sugar and heart rate (ii) Inhibits gastrointestinal tract secretion (iii) Hastens blood coagulation (iv) Decreases glycogen in liver</td>
<td>Exercise, changes in body position psychological stress</td>
<td></td>
<td>Increased during prolonged exercise</td>
</tr>
<tr>
<td></td>
<td>Norepinephrine (Noradrenaline)</td>
<td>i) Influences vasoconstriction ii) Resembles action</td>
<td></td>
<td></td>
<td>Increased during prolonged exercise</td>
</tr>
</tbody>
</table>
REGULATION OF HORMONE SECRETION

The hypothalamus, like the rest of the brain, consists of interconnecting nerve cells (neurons) that are nourished by a rich supply of blood. To understand hypothalamic function it is necessary to define the various forms of neurosecretion. First, there is neurotransmission, which occurs throughout the brain and is the process by which one nerve cell communicates with another via a synapse, a small gap between the ends (nerve terminals) of neurons. Nerve terminals are often called pre-synaptic or postsynaptic in reference to the direction in which an impulse is traveling, with the pre-synaptic neuron transmitting an impulse to the postsynaptic neuron. Transmission of an electrical impulse requires the secretion of a chemical substance that diffuses across the synapse from the pre-synaptic membrane of one neuron to the postsynaptic membrane of another neuron. The chemical substance that is secreted is called a neurotransmitter. The process of synthesis and secretion of neurotransmitters is similar to that of protein hormone synthesis, with the exception that the neurotransmitters are contained within neurosecretory granules that are produced in the cell body and migrate through the axon (a projection of the neuron) to the nerve terminal, from which they are discharged into the synaptic space.

There are four classic neurotransmitters: epinephrine, nor-epinephrine, serotonin and acetylcholine. A large number of additional neurotransmitters have been discovered, of which an important group is the neuropeptides. The neuropeptides function not only as neurotransmitters but also as neuromodulators. As neuromodulators, they do not act directly as neurotransmitters but rather increase or decrease the action of neurotransmitters. Well known examples are the opioids (e.g. enkephalins), so named because they are endogenous (produced in the human body) peptides (short chains of amino acids) with a strong affinity for the receptors that bind opiate drugs, such as morphine and heroin.

The brain and indeed the entire central nervous system consist of an interconnected network of neurons. The secretion of specific neurotransmitters and neuropeptides lends an organized, directed function to the overall system. The connection of the hypothalamus to many other regions of the brain, including the cerebral cortex, allows intellectual and functional signals, as well as external signals, including physical and emotional stresses, to be funneled into the hypothalamus to the endocrine system. From the endocrine system these signals are able to exert their effects throughout the body.

The hypothalamus produces and secretes not only neurotransmitters and neuropeptides but also several neurohormones that alter anterior pituitary gland function and two hormones, vasopressin and oxytocin, that act on distant target organs. The neurons that produce and secrete neurohormones are true endocrine cells in that they produce hormones that are incorporated into secretory granules that are then carried through the axons and stored in nerve terminals located in the median eminence or posterior pituitary gland. In response to neural stimuli, the contents of the secretory granules are extruded from the nerve terminals into a capillary network. In the case of hormones that affect pituitary function, the contents of the secretory granules are carried through the hypothyseal-portal circulation and are delivered directly into the anterior pituitary gland.

These hypothalamic neurohormones are known as releasing hormones because their major function is to stimulate the secretion of hormones originating in the anterior pituitary gland. They consist of simple peptides ranging in size from only three amino acids (thyrotropin-releasing hormone) to 44 amino acids (growth hormone-releasing hormone). One hypothalamic hormone, somatostatin, has an inhibitory action, primarily inhibiting the secretion of growth hormone, although it can also inhibit the secretion of other hormones. The neurotransmitter dopamine, produced in the hypothalamus, also has an inhibitory action, inhibiting the secretion of the anterior pituitary hormone prolactin. The cell bodies of the neurons that produce these neurohormones are not evenly distributed throughout the hypothalamus. Instead, they are grouped together in paired clusters of cell bodies known as nuclei.
A classic model for neurohormonal activity is the posterior lobe of the pituitary gland (neurohypophysis). Its secretory products, vasopressin and oxytocin, are produced and packaged into neurosecretory granules in specific groups of nerve cells in the hypothalamus (the supra-optic nuclei and the Para ventricular nuclei). The granules are carried through the axons that extend through the infundibular stalk and end in and form the posterior lobe of the pituitary gland. In response to nerve signals, the secretory granules are extruded into a capillary network that feeds directly into the general circulation.

**FIGHT-OR-FLIGHT RESPONSE**

The “fight-or-flight response”, also called the “fight-or-flight-or-freeze response”, the “fright, fight or flight response”, “hyperarousal” or the “acute stress response”, was first described by Walter Cannon in 1929. His theory states that animals react to threats with a general discharge of the sympathetic nervous system, priming the animal for fighting or fleeing. This response was later recognized as the first stage of a general adaptation syndrome that regulates stress responses among vertebrates and other organisms.

**Biology of the Stress Response**

Normally, when a person is in a serene, unstimulated state, the “firing” of neurons in the locus coeruleus is minimal. A novel stimulus (which could include a perception of danger or an environmental stressor such as elevated sound levels or over-illumination), once perceived, is relayed from the sensory cortex of the brain through the hypothalamus to the brainstem.

That route of signaling increases the rate of noradrenergic activity in the locus coeruleus, and the person becomes alert and attentive to the environment. Similarly, an abundance of catecholamines at neuroreceptor sites facilitates reliance on spontaneous or intuitive behaviors often related to combat or escape.

If a stimulus is perceived as a threat, a more intense and prolonged discharge of the locus coeruleus activates the sympathetic division of the autonomic nervous system. This activation is associated with specific physiological actions in the system, both directly and indirectly through the release of epinephrine (adrenaline) and to a lesser extent norepinephrine from the medulla of the adrenal glands. The release is triggered by acetylcholine released from preganglionic sympathetic nerves. The other major factor in the acute stress response is the hypothalamic-pituitary-adrenal axis.

**Physiology of the Stress Response**

These catecholamine hormones facilitate immediate physical reactions associated with a preparation for violent muscular action. These include the following:

- Acceleration of heart and lung action
- Paling or flushing, or alternating between both
- Inhibition of stomach and upper-intestinal action (digestion slows down or stops)
- General effect on the sphincters of the body
- Constriction of blood vessels in many parts of the body
- Liberation of nutrients (particularly fat and glucose) for muscular action
- Dilation of blood vessels for muscles
- Inhibition of the lacrimal gland (responsible for tear production) and salivation
- Dilation of pupil (mydriasis)
- Relaxation of bladder
- Evacuation of colon
- Inhibition of erection
- Auditory exclusion (loss of hearing)
- Tunnel vision (loss of peripheral vision)
- Acceleration of instantaneous reflexes
- Shaking.

**Psychology of the Stress Response**

A typical example of the stress response is a grazing zebra, calmly maintaining homeostasis. If the zebra sees a lion closing in for the kill, the stress response is activated. The escape requires intense muscular effort, supported by all of the body’s systems. The sympathetic nervous system’s activation provides for these needs. A similar example involving fight is of a cat about to be attacked by a dog. The cat shows accelerated heartbeat, piloerection (hair standing on end, normally for conservation of
heat), and pupil dilation, all signs of sympathetic arousal.

Though Cannon, who first proposed the idea of fight-or-flight, provided considerable evidence of these responses in various animals, it subsequently became apparent that his theory of response was too simplistic. Animals respond to threats in many complex ways. Rats, for instance, try to escape when threatened, but will fight when cornered. Some animals stand perfectly still so that predators will not see them. Many animals freeze or play dead when touched in the hope that the predator will lose interest.

Others have more exotic self-protection methods. Some species of fish change color swiftly, to camouflage themselves. These responses are triggered by the sympathetic nervous system, but in order to fit the model of fight or flight, the idea of flight must be broadened to include escaping capture in either a physical way or in a sensory way. Thus, flight can be disappearing to another location or just disappearing in place, and often both fight and flight are combined in a given situation.

The fight or flight actions also have polarity - the individual can fight or fly against or away from something that is threatening, such as a hungry lion, or fight or fly for or towards something that is needed, such as the safety of the shore of a raging river.

A threat from another animal does not always result in immediate fight or flight. There may be a period of heightened awareness, during which each animal interprets behavioral signals from the other. Signs such as pallor, piloerection, immobility, sounds, and body language communicate the status and intentions of each animal. There may be a sort of negotiation, after which fight or flight may ensue, but which might also result in playing, mating, or nothing at all. An example of this is kittens playing: each kitten shows the signs of sympathetic arousal, but they never inflict real damage.

**Behavioral Manifestations of Fight-or-Flight**

In prehistoric times when the fight or flight response evolved, fight was manifested in aggressive, combative behavior and flight was manifested by fleeing potentially threatening situations, such as being confronted by a predator. In current times, these responses persist, but fight and flight responses have assumed a wider range of behaviors. For example, the fight response may be manifested in angry, argumentative behavior, and the flight response may be manifested through social withdrawal, substance abuse and even television viewing.

Males and females tend to deal with stressful situations differently. Males are more likely to respond to an emergency situation with aggression (fight), while females are more likely to flee (flight), turn to others for help, or attempt to defuse the situation – ‘tend and befriend’. During stressful times, a mother is especially likely to show protective responses toward her offspring and affiliate with others for shared social responses to threat.

**Negative Effects of the Stress Response in Humans**

The stress response halts or slows down various processes such as sexual responses and digestive systems to focus on the stressor situation and typically causes negative effects like, constipation, anorexia, erectile dysfunction, difficulty urinating, and difficulty maintaining sexual arousal. These are functions which are controlled by the parasympathetic nervous system and therefore, suppressed by sympathetic arousal.

Prolonged stress responses may result in chronic suppression of the immune system, leaving the body open to infections, however there is a short boost of the immune system shortly after the fight or flight response has been activated. This is due to an ancient need to fight the infections in a wound that one may have received during interaction with a predator.

Stress responses are sometimes a result of mental disorders such as post-traumatic stress disorder, in which the individual shows a stress response when remembering a past trauma and panic disorder, in which the stress response is activated by the catastrophic misinterpretations of bodily sensations.

For hormones to function properly, their secretion rate must be precisely controlled. A signal needs to be received that triggers the necessary steps for hormone secretion. The initial step is the detection of an actual or threatened homeostatic
imbalance. This imbalance must be able to activate a secretory apparatus (e.g., the endocrine gland), resulting in hormone secretion. The circulating hormones interact with its target organ or tissue and exert its effect. Once the hormonal effect has occurred, the hormonal signal has to be turned off and the hormone needs to be removed from the circulation. Finally, the secretory apparatus must replenish the hormone in its secretory cells.

The secretion of most hormones is regulated by negative feedback, meaning that some consequence of the hormone secretion acts directly or indirectly on the secretory apparatus to inhibit further secretion. This type of secretory mechanism is self-limiting. Positive feedback mechanisms are rare in endocrine regulation. During this type of regulation, some consequence of the hormonal secretion causes an augmented secretory drive. Rather than being self-limiting, the stimulus for triggering hormonal secretion becomes stronger. An example of positive feedback is the release of oxytocin from the posterior pituitary gland caused by dilation of the uterine cervix during childbirth. The oxytocin causes a greater dilation that in turn creates a greater stimulus for further oxytocin release.

**CHANGES IN CIRCULATING HORMONAL CONCENTRATIONS**

Increases in the concentration of hormones can be attributed to a number of different physiological mechanisms. Exercise or other physical or psychological stresses appear to be potent stimulators in elevating the secretory patterns of hormones. Fluid volume shifts, changes in clearance rates, and venous pooling of blood are additional mechanisms that may increase circulating concentration of hormones. Regardless of the mechanism, there is an increased potential for interaction with the receptor of the target tissue, leading to the desired cellular response.

Receptors are found in all types of cells within the body and each hormone reacts with its specific receptor. The interaction of the hormone to its receptor has been called the lock and key therapy. The receptor is the lock and the hormone is the key. There is some cross reactivity, meaning that there may be more than one hormone that can bind with the receptor. When this occurs, the resulting biological actions are different from those induced by the primary hormone.

It is the hormone-receptor complex that results in a message being delivered to the cell muscles for the cell nucleus for either inhibition or facilitation of protein synthesis. The number of receptors available for interactions with circulating hormones is considered another mechanism in initiating cellular action. Hormonal receptors are dynamic in that they also respond to physiological demand. They may increase in number to meet the demand of a rise in the circulating concentration of hormones. Such an increase in receptor number is termed up-regulation. Similarly, the number of receptors can be decreased if adaptation is no longer possible or to prevent an
Endocrine System

over-response by persistently increasing hormone levels. This adaptation is called down-regulation. This type of control on the part of the receptor is as dramatic as the changes in hormonal secretory patterns.

**HORMONE AND EXERCISE**

Exercise has been shown to be a potent stimulus to the endocrine system. The hormonal responses to an acute exercise session suggest that hormones may be involved in the recovery and remodeling processes that occur after exercise. The exercise stimulus has an important role in the hormonal secretion pattern. Variables such as intensity of exercise, volume of exercise, rest intervals, choice of exercise, and recovery status of the muscle appear to influence the hormonal response.

The mechanisms of hormonal interaction with the remodeling of muscle tissue are based on several factors. The acute increase in hormonal concentration caused by the exercise stimulus allows for a greater interaction between the hormone and its receptors. Since the adaptations to exercise (particularly resistance exercise) are anabolic in nature, the recovery mechanisms involve tissue repair and remodeling. In instances when training intensity or volume exceeds an individual’s ability to recover, a possible situation of overtraining or overwork can occur, resulting in a greater catabolic effect. The hormonal response will either repair or remodel muscle tissue or perhaps impede this process.

The hormonal mechanisms may respond differently between trained and untrained individuals. Furthermore, some hormonal mechanisms may not be operational in both males and females (e.g., testosterone). In addition, the effect of program design, genetic predisposition, fitness level, training experience and adaptation potential all seem to affect the endocrine mechanism for maintaining hormone secretion.

**Testosterone**

Testosterone is an androgen, a steroid hormone that has masculinizing effects. It is also anabolic because of its role in the maintenance and growth of muscle and bone tissue. Most of the circulating testosterone is produced in the testes, while small amounts are produced in the adrenal glands. The physiological roles of testosterone are as follows:

i. Increase in protein synthesis, resulting in muscle growth

ii. Development and maturation of male sex organs

iii. Development of secondary sexual characteristics:
   a. Increase in body hair
   b. Development of masculine voice
   c. Development of male pattern baldness
   d. Development of libido
   e. Control of spermatogenesis
   f. Aggressive behavior

iv. Interaction in secretion of sebaceous glands, contributing to acne

v. Possible role in glycogen synthesis

**Acute exercise response:** A single training session of resistance exercise has been demonstrated to significantly increase the peripheral concentration of testosterone above resting levels in males. However, this may also depend on length of training experience. For example, the male weightlifters with more than two years of training experience had a significantly greater testosterone response to an exercise session than less than two years of lifting experience. Exercise response patterns of testosterone also appear to be related to the design of the exercise program. Testosterone concentration is significantly higher when rest periods between sets are reduced and intensity of exercise is reduced.

There appears to be a biphasic response of testosterone to an acute bout of aerobic exercise, which depends upon the duration of exercise. Testosterone levels will continue to elevate as exercise is prolonged and then begin to decline toward baseline levels before exercise is completed.

An increase in testosterone concentrations during an acute bout of anaerobic exercise also depends on the duration of exercise. For example, after intermittent anaerobic exercise in male runners testosterone concentrations increases significantly.

**Long-term response to exercise:** It has been noted that high resting concentrations of
testosterone may enhance or facilitate the building of lean tissue. This has primarily been the reason for the widespread use of anabolic steroids by power athletes and bodybuilders. For example, after two years of resistance training, elite weightlifters are able to significantly increase their resting testosterone concentrations while also improving their strength. It is possible that changes in resting testosterone concentrations may be a reflection of an advanced strategy to increase force capability in subjects who have little potential for change in muscle hypertrophy.

Low resting levels of testosterone are frequently observed in endurance-trained athletes. The depressed level of testosterone seen in these athletes may be insufficient to stimulate skeletal muscle growth and also make it difficult to counteract the catabolic effects of glucocorticoids on skeletal muscle.

**Growth Hormone**

Growth hormone (GH) is a polypeptide hormone secreted from the anterior pituitary gland. Its secretion and releases are controlled by neurotransmitters of the central nervous system. Physiological stimuli such as sleep, diet and stress (including exercise) can all stimulate a GH response. The actions of GH are mediated to a certain extent by secondary hormone known as insulin-like growth factors (IGF). The basic physiological actions of GH are as follows:

i. Increase in protein synthesis

ii. Increase in amino acid transport across cell membrane

iii. Growth and development of bones

iv. Reduction of glucose utilization

v. Decrease in glycogen synthesis

vi. Increase in utilization of fatty acids

vii. Increase in lipolysis

viii. Metabolic sparing of glucose and amino acids

ix. Collagen synthesis

x. Stimulation of cartilage growth

**Acute response to exercise:** The acute GH response to a resistance exercise session is related to specific component variables of the training program. Both volume and intensity of training appear to be important factors in eliciting a GH response. When more moderate exercise intensity is employed (10 RM), a significant increase in GH is observed. This increase is significantly greater than that seen after a resistance-training program of higher intensity (5 RM).

The volume of training also appears to a potent stimulus in the GH response to exercise. The greater fatigue (greater blood lactate concentrations) observed in the high-volume training program most likely contributed to the elevated GH response.

The GH response to resistance exercise in females appears to be sensitive to changes in acute program variables (e.g. rest, intensity, volume of training).

Elevations in GH concentrations are typically reported during aerobic exercise and these elevations are positively related to both the duration and intensity of exercise. It has been suggested that exercise above the lactate threshold needs to be for a minimum duration, but the blood lactate levels cannot predict the amplitude and duration of the GH response.

**Long-term adaptations to exercise:** Significant increases in the GH to exercise have been seen after resistance training programs. However, resistance training does not appear to alter resting GH concentrations. A year of exercise training at exercise intensity above the lactate threshold has been shown to amplify the pulsatile release of GH at rest. A reduction in the GH response to exercise may occur within 3 weeks of training. However, these changes may be related to a lower relative intensity used for the post exercise period. When trained subjects exercise at the same relative intensity, according for improvements in performance, a greater GH response to the exercise stimulus is occur.

**Optimizing hGH Therapy with Diet and Exercise**

The importance of diet and exercise in optimizing growth hormone levels applies to those who select growth hormone therapies as well as those who don’t. Irregular insulin levels and lack of exercise are known to contribute to accelerated symptoms of aging like heart disease, obesity and diabetes.
The diet and exercise recommendations for boosting hGH help to control the endogenous factors of disease and aging as well as enhancing the effectiveness of hGH therapies.

Many athletes who train heavily are known to maintain youthful levels of growth hormone right in their 50’s, 60’s and 70’s. There are several reports of increased strength, stamina, and muscle mass with young athletes, but there is not enough data to draw any definitive conclusions or make recommendations for this application. However, test results with athletes whose initial IGF-1 were near optimal contributed to the conclusions that GH will not become over-stimulated in people who have close to optimal GH levels. Even the 8 percent increases in IGF-1 that have been observed with athletes on Symbiotropin can make a tremendous difference in the competitive edge of a body builder or track field athlete.

Growth hormone levels increase significantly when insulin levels are low, about four hours after a meal. It is at this point that the fat burning potential of GH tends to be at its daytime peak. But the largest burst of GH is released during the early hours of sleep—hence, the evening eating habits are crucial to maximizing this night-time secretion. By avoiding food during the last four hours before bedtime we may enhance circadian growth hormone release, and fat burning potential.

Exercise is a potent growth hormone stimulator. Many athletes choose to use natural secretagogues before a work-out, no matter what time of day, in order to enhance the bump in GH that comes from exercises like high-intensity running and weight lifting. For those of us who are not athletes, increasing growth hormone levels with injections or secretagogues often leads to the improved strength and energy that it takes from exercises like high-intensity running and weight lifting. For those of us who are not athletes, increasing growth hormone levels with injections or secretagogues often leads to the improved strength and energy that it takes from exercises like high-intensity running and weight lifting.

Growth hormone significantly improves body composition

Depending on a person’s initial body composition and exercise habits, losing fat and gaining muscle with GH therapy may not result in weight loss, but it will result in better measurements. When we increase muscle mass, we increase our overall metabolism thereby contributing to our ability to burn more body fat and calories. Supporting the growth of muscle tissue with diet and exercise is an integral part of optimizing GH therapy. It has because found the best results clinically when patients eat fruit or other healthy carbohydrates about an hour after taking Symbiotropin.

The best carbohydrates to use as a replacement are those that will breakdown to glucose more slowly. We may refer to a glycemic index for specific values of certain foods, but as a rule consume complex carbohydrates combined with fiber and good fats.

Optimum GH Enhancing Routine

Certain supplements, like chromium picolinate, have been shown to have a regulatory effect on insulin thereby enhancing GH release. The following products may be used to enhance any GH protocol, but they may be especially important for diabetics or hypoglycemic, who may have a resistance to GH therapy.

Chromium Picolinate: A trace mineral that helps insulin to fit into the cell better thereby overcoming insulin resistance and lowering circulating insulin and blood sugar. These processes are important in reducing fat storage, burning existing fat and promoting muscle growth. Research on chromium picolinate shows that it is consistently effective at doing all of the above, even in sedentary individuals. Suggested dose: 200 to 600 mcg/day.

Vanadium: A trace mineral that has an insulin mimicking effect. Assists in utilization of blood glucose and subsequent management of insulin. Clinical experience shows that the vanadyl complex is better absorbed and can be used in lower doses than other, more poorly absorbed forms like vanadyl sulfate. Recommended dose: 200 to 500 mcg/day.

Gymnema Sylvestre: This herb is used widely in India to treat blood sugar abnormalities. It contains a molecule that is so similar to glucose that it sits on sugar receptors, which helps to inhibit the
release of glucose into the bloodstream and control insulin response. It is used as an aid in weight control and an inhibitor of sugar cravings, when applied directly to the tongue it will block the ability to taste anything sweet. Suggested use: standardized extract, 500 to 1000 mg before meals.

**Proteusterone**: This herbal complex has a large part of its action in the support of liver, pancreatic, and adrenal function in ways that help to manage insulin and blood sugar. Clinical experience has shown an improved response in Symbiotropin therapy for diabetics who use Proteusterone at night. As an endocrine supplement, this product is designed to enhance any individual hormone therapy, and is used by many athletes to improve muscle growth and stamina. Suggested use: 1 to 3 tablets/day.

Studies show that there are specific exercises that are particularly effective at stimulating GH release. It is important to point out that any exercise will help to enhance the effects of growth hormone. The following exercises are used specifically help to increase GH release and have a rate of effectiveness that, for the most part, is proportionate to the intensity of the exercise.

All weight-training exercises are effective promoters of GH release, but those that involve the use of high-resistance and major muscle groups tend to be the most effective. Applying maximum effort to fewer repetitions of squats, leg presses, dead lifts, overhead presses, bench presses, standing curls and leg curls will optimize the results. In addition, go to MLC for one repetition of each of these exercises, no more than once a week to create an additional boost of GH release. If you are not experienced with weight training, go to your physician to determine your physical condition and

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![Fig. 6.4: Schematic diagram of the role of GH for improvements of body composition](image1)

![Fig. 6.5: Optimum GH enhancing routine](image2)
work with a qualified personal trainer who can teach you proper technique.

**Insulin**

Insulin is a protein hormone secreted by the β-cells of the islets of Langerhans within the pancreas. The basic physiological actions of the hormone are as follows:

1. Regulate glucose metabolism in all tissues except the brain.
2. Facilitate an increase in the rate of glucose uptake into both muscles tissue and fat cells. Glucose that is not used is converted into glycogen. If glycogen stores are full, excess carbohydrates are stored as triglycerides in adipose tissue.
3. Increase the rate of amino acid uptake by skeletal muscle and other tissue.
4. Role in muscle remodeling may become more prevalent when functioning to decrease the rate of protein degradation within muscle tissue.
5. Provide the muscle with sufficient nutrients to stimulate muscle growth.

**Exercise Response:** Exercise appears to decrease the circulating concentrations of insulin. This is likely the result of the inhibitory effect of catecholamines on the β-cells of the pancreas. The reduction of insulin appears to be a function of the duration of exercise. As exercise duration lengthens, a greater decrease in insulin concentrations is seen. Insulin levels also decrease during both mild and moderate exercise intensities. A single bout of exercise enhances insulin sensitivity and skeletal muscle responsiveness to glucose uptake in exercise muscles. Thus, exercise-induced increased insulin sensitivity of glucose uptake serves to replenish depleted glycogen stores during the post exercise meal.

**Effect of Training:** Training appears to increase the sensitivity to insulin of both the skeletal muscle and the liver. Thus, less insulin is required to regulate blood glucose in trained individuals. Trained individuals also appear to have a less pronounced insulin reduction during exercise than untrained individuals.

**Insulin and Glucagon:** These two hormones respond to the same stimuli but exert opposite actions relative to the mobilization of liver glucose and adipose tissue free fatty acid (FFA). In fact, it is the ratio of glucagon to insulin that provides control over the mobilization of this fuel. Insulin to be the primary hormone involved in the uptake and storage of glucose and FFA, and glucagon to cause the mobilization of those fuel from storage, as well as increase gluconeogenesis.

Insulin is directly involved in the uptake of glucose into tissue, and that glucose uptake by muscle can increase seven- to twenty fold during exercise. Insulin concentration decrease during exercise of increasing intensity; this of course is an appropriate response. If exercise were associated with an increase in insulin, the plasma glucose would be taken up into all tissues (including adipose tissue) at a faster rate, leading to an immediate hypoglycemia. The lower insulin

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Table 6.2: Dentecity of exercise and HGH secretion of different mode of exercise.

<table>
<thead>
<tr>
<th>Exercise</th>
<th>Intensity</th>
<th>HGH secretion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Running (Women)</td>
<td>High</td>
<td>266% increase in trough level</td>
</tr>
<tr>
<td></td>
<td></td>
<td>75% increase in daily secretion</td>
</tr>
<tr>
<td>Running (Men)</td>
<td>Moderate</td>
<td>0% - Moderate</td>
</tr>
<tr>
<td>Stationary Bike (both sexes)</td>
<td>High</td>
<td>166% increase</td>
</tr>
<tr>
<td>Stationary Bike (both sexes)</td>
<td>Moderate</td>
<td>166% increase</td>
</tr>
<tr>
<td>Weight Training (both sexes)</td>
<td>85% MLC*</td>
<td>400% increase</td>
</tr>
<tr>
<td>Weight Training (both sexes)</td>
<td>70% MLC*</td>
<td>300% increase</td>
</tr>
<tr>
<td>Weight Training (both sexes)</td>
<td>Moderate - High</td>
<td>Immediate and Sustained increase</td>
</tr>
<tr>
<td>Treadmill (both sexes)</td>
<td>High</td>
<td>Increased GH Pulse</td>
</tr>
</tbody>
</table>

*MLC = Maximal Lift Capacity, the maximum amount of weight able to be lifted once.
concentration during exercise favors the mobilization of glucose from the liver and FFA from adipose tissue, both of which are necessary to maintain the plasma glucose concentration.

With plasma insulin decrease with long term exercise, it should be no surprise that the plasma glucagons concentration increases. This increase in plasma glucagons favors the mobilization of FFA from adipose tissue and glucose from the liver, as well as an increase in gluconeogenesis. Overall, the reciprocal responses of insulin and glucagons favor the maintenance of the plasma glucose concentration at a time when the muscle is using plasma glucose at a high rate. An endurance training program the glucagons response to a fixed exercise task is diminished to the point that there is no increase during exercise. In effect, endurance training allows the plasma glucose concentration to be maintained with little or no change in insulin and glucagons. This is related in part to an increase in glucagons sensitivity in the liver, a decrease in glucose uptake by muscle, and an increase in the muscle’s use of fat as a fuel.

These findings raise several questions. If the plasma glucose concentration is relatively constant during exercise, and the plasma glucose concentration is a primary stimulus for insulin and glucagons secretion, what cause the insulin secretion to decrease and glucagons secretion to increase? There is no question that changes in the plasma glucose concentration provide an important level of control over the secretion of glucagon and insulin. However, when the plasma glucose concentration is relatively constant, the sympathetic nervous system can modify the secretion of insulin and glucagon. Epinephrine and norepinephrine stimulate the adrenergic receptors on the beta cells of the pancreas to decrease insulin secretion during exercise when the plasma glucose concentration is normal. Further epinephrine (E) and norepinephrine (NE) stimulate β-adrenergic receptors on the alpha cells of the pancreas to increase glucagon secretion when the plasma glucose concentration is normal. Endurance training decrease the sympathetic nervous system response to a fixed exercise bout, resulting in less stimulation of adrenergic receptors on the pancreas, and less change in insulin and glucagons secretion.

The observation that plasma insulin decreases with prolonged submaximal exercise raises another questions. How can exercising muscle take up glucose seven to twenty times faster than at rest if the insulin concentration is decreasing. Glucose delivery is the product of muscle blood flow and the blood glucose concentration. Therefore, during exercise more glucose and insulin are delivered to muscle than at rest, and because the muscle is using glucose at a higher rate, a gradient for its facilitated diffusion is created.

**Cortisol**

Cortisol, a steroid hormone synthesized and released from the adrenal cortex of the adrenal gland, is the primary glucocorticoid hormone found in humans. Its synthesis is stimulated by adrenocorticotrophic hormone (ACTH), which is secreted by the anterior pituitary. The physiological functions of Cortisol are as following:

i. Conversion of amino acids to carbohydrates
ii. Increase in proteolytic enzymes
iii. Inhibition of protein synthesis
iv. Increase in protein degradation in muscle
v. Stimulation of gluconeogenesis
vi. Increase in blood glucose concentrations
vii. Facilitation of lipolysis

**Exercise response:** The acute response of cortisol to a resistance training session appears to be related to the volume of training. When volume of training increases, a significant increase in cortisol is observed. The elevated cortisol levels in elite weight lifters may reflect an ability of these athletes to push them maximally during each training session.

**Response to training:** Prolonged aerobic exercise appears to be a potent stimulator of the adrenocortical system. Increases in cortisol appear to be proportional to the intensity of exercise. However, when exercise is greater than 70 percent of VO₂ max the cortisol level increases consistently. Significant increases in cortisol concentrations also occur during short bout (1 min) of exercise as long as the exercise is performed at maximal intensity. Training appears to lower the cortisol response during prolonged endurance exercise. These changes appear to reflect a better maintenance of blood glucose levels in these individuals.
Catecholamines

The catecholamines (epinephrine, norepinephrine, dopamine) are secreted by the adrenal medulla of the adrenal gland and are controlled entirely by sympathetic nervous input. Catecholamines are stimulated by hypoglycemia, physical or physiological trauma, circulatory failure, stress, exercise, illness, hypoxia, cold exposure, etc. The direct and indirect actions of catecholamines on muscle function are as follows:

1. Increase in force production
2. Increase in contraction rate
3. Increase in blood pressure
4. Increase in energy availability
5. Augmentation of secretion rates of other hormones

Exercise response: Catecholamines concentration appears to be elevated during both endurance and resistance training exercises. Short-duration sprints (several seconds) of maximal intensity appear sufficient to elevate both epinephrine and norepinephrine concentrations.

During exercise at sub maximal intensities, there may be a different catecholamines response that appears to depend on the duration of exercise. During such exercise, increases in norepinephrine concentrations are seen within 15 min without any increase in epinephrine. As exercise duration increases, epinephrine concentrations may then increase above resting level. This most likely reflects a greater need for substrate mobilization during exercise of longer duration.

Response to training: Training does not appear to alter resting catecholamines concentrations. However, trained individuals do appear to have a greater capacity to secrete epinephrine and perhaps norepinephrine as well.

Epinephrine and Norepinephrine: Epinephrine and Norepinephrine are also involved in the mobilization of glucose from the liver, FFA from adipose tissue, and may interfere with the uptake of glucose by tissue. Although plasma NE can increase ten- to twenty fold during exercise and can achieve a plasma concentration that can exert a physiological effect, the primary means by which NE acts is when released from sympathetic neurons onto the surface of the tissue under consideration. The plasma level of NE is usually taken as an index of overall sympathetic nerve activity, but there is evidence that muscle sympathetic nerve activity during exercise may be a better indicator than that of plasma NE. Epinephrine, released from the adrenal medulla, is viewed as the primary catecholamine in the mobilization of glucose from the liver.

Plasma E and NE to increase linearly with duration of exercise. These changes are related to cardiovascular adjustments to exercise, as well as to the mobilization of fuel. These responses favor the mobilization of glucose and FFA to maintain the plasma glucose concentration. While it is sometimes difficult to separate the effect of E from NE, E seems to be more responsive to changes in the plasma glucose concentration. A low plasma glucose concentration stimulates a receptor in the hypothalamus to increase E secretion while having only a modest effect on plasma NE. In contrast, when the blood pressure is changed, as during an increased heat load, the primary catecholamine involved is Norepinephrine. Epinephrine binds to β-adrenergic receptors on the liver and stimulates the breakdown of liver glycogen to form glucose for release into the plasma. For example, when arm exercise is added to existing leg exercise, the adrenal medulla secretes a large amount of E. This causes the liver to release more glucose than muscles are using, and the blood glucose concentration actually increases. If β-adrenergic receptors are blocked with propranolol (a β-adrenergic receptor blocking drug), the plasma glucose concentration is more difficult to maintain during exercise, especially if the subject has fasted. In addition, since the propranolol blocks β-adrenergic receptors on adipose tissue cells, less FFA are released and the muscles have to rely more on the limited carbohydrate supply for fuel.

Endurance training causes a very rapid decrease in the plasma E and NE responses to a fixed exercise bout. Within three weeks the concentration of both catecholamines is greatly reduced. Paralleling this rapid decrease in E and NE with endurance exercise training is a reduction in glucose mobilization. In spite of this, the plasma glucose concentration is maintained because there is also a reduction in
glucose uptake by muscle at the same fixed workload following endurance training. Interestingly, during a very stressful event, a trained individual has a greater capacity to secrete E than an untrained individual. In addition, when exercise is performed at the same relative workload (%VO2 max) after training, the plasma NE concentration is higher. This suggests that physical training, which stimulates the sympathetic nervous system on a regular basis, increases its capacity to respond to extreme challenges.

**Thyroid Hormone**

The thyroid gland secretes three hormones. Calcitonin is secreted by parafollicular cells and is involved with the regulation of calcium balance. The thyroid gland also secretes thyroxine (T4) and triidothyronine (T3). These hormones are made up of both iodide and the amino acids tyrosine. Secretion of these hormones is stimulated by thyroid stimulating hormone (TSH) released by the anterior pituitary gland. The primary functions of the thyroid hormones are as follows:

i. Increase the basal metabolic rate (BMR)
ii. Potentiate the glucose uptake caused by insulin
iii. Increase in liver glycogen depletion during over secretion of thyroid hormone.
iv. Stimulates all aspects of lipid metabolism.
v. High level of thyroid hormone has a catabolic effect on skeletal muscle.

**Exercise response:** The response of both T3 and T4 to acute exercise has been reported to increase from resting levels at varying intensities. However, the influence of acute exercise may not be detectable until several days after the exercise session.

**Response to training:** During prolonged periods of high intensity training (specially resistance training), decreases in resting levels of T3 and T4 has been reported. However, the thyroid hormone acts as a possible hormone indicator of overtraining.

**Thyroxin:** The discussion of substrate mobilization during exercise must include thyroxin, a hormone

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**Fig. 6.6:** Schematic diagram of ADH regulate the water balance in the body
whose concentration doesn’t change dramatically from resting to the exercise state. The thyroid hormones $T_3$ and $T_4$ are important in establishing the overall metabolic rate, and in allowing other hormones to exert their full effect (permissive hormone). Thyroxin accomplishes this latter function by influencing either the number of receptors on the surface of a cell (for other hormones to interact with), or the affinity of the receptor for the hormones. For example, without thyroxin, epinephrine has little effect on the mobilization of free fatty acid from adipose tissue. During exercise there is an increase in “free” thyroxin due to change in the binding characteristics of the transport protein. $T_3$ and $T_4$ are removed from the plasma by tissues during exercise at a greater rate than at rest. In turn, TSH secretion from the anterior pituitary is increased to stimulate the secretion of $T_3$ and $T_4$ from the thyroid gland to maintain the plasma level. A low thyroxin (hypothyroid) state would interfere with the ability of other hormones to mobilize fuel for exercise.

**Antidiuretic Hormone (ADH):** The two primary hormones involved into regulation of fluid balance are aldosterone and ADH. When plasma volumes decreased, the enzyme renin from kidney converts angiotensinogen into angiotensin I. Which later become angiotensin II, which increase peripheral resistance, raising the blood pressure. Aldosterone release and Na$^+$ reabsorption start in the kidney which cause water retention these increasing the plasma volumes. ADH is released in response to increase plasma osmolality, the hypothalamus triggers ADH release. ADH meets on the kidneys promoting water conservation and blood osmolality decreases.

**SUMMARY**

1. The endocrine system is a group of glands that work together and secrete many types of different hormones that regulate the body. Hormones regulate many functions of an organism, including mood, growth and development, tissue function and metabolism. Hormones are chemical substances that circulate in the blood and interact with organs in the body to help combat various stresses. Hormones influence the rate of specific cellular reactions by changing the rate of protein synthesis or enzyme activity and by including secretion of other hormones.

2. The hypothalamus produces and secretes not only neurotransmitters and neuropeptides but also several neurohormones that alter anterior pituitary gland function and two hormones, vasopressin and oxytocin, that act on distant target organs. The neurons that produce and secrete neurohormones are true endocrine cells in that they produce hormones that are incorporated into secretory granules that are then carried through the axons and stored in nerve terminals located in the median eminence or posterior pituitary gland. These hypothalamic neurohormones are known as releasing hormones because their major function is to stimulate the secretion of hormones originating in the anterior pituitary gland and control the functions of endocrine system.

3. Walter Cannon in 1929 states that animals react to threats with a general discharge of the sympathetic nervous system, priming the animal for fighting or fleeing. This response was later recognized as the first stage of a general adaptation syndrome that regulates stress responses among vertebrates and other organisms. The catecholamine hormones facilitate immediate physical reactions associated with a preparation for violent muscular action.

4. Increases in the concentration of hormones can be attributed to a number of different physiological mechanisms. Exercise or other physical or psychological stresses appear to be potent stimulators in elevating the secretary patterns of hormones. Fluid volume shifts, changes in clearance rates, and venous pooling of blood are additional mechanisms that may increase circulating concentration of hormones. Regardless of the mechanism, there is an increased potential for interaction with the receptor of the target tissue, leading to the desired cellular response.
5. Exercise has been shown to be a potent stimulus to the endocrine system. The hormonal responses to an acute exercise session suggest that hormones may be involved in the recovery and remodeling processes that occur after exercise. The exercise stimulus has an important role in the hormonal secretion pattern. Variables such as intensity of exercise, volume of exercise, rest intervals, choice of exercise, and recovery status of the muscle appear to influence the hormonal response.

6. Testosterone is an androgen, a steroid hormone that has masculinizing effects. It is also anabolic because of its role in the maintenance and growth of muscle and bone tissue. A single training session of resistance exercise has been demonstrated to significantly increase the peripheral concentration of testosterone above resting levels in males. Long-term response to exercise has been noted that high resting concentrations of testosterone may enhance or facilitate the building of lean tissue. This has primarily been the reason for the widespread use of anabolic steroids by power athletes and bodybuilders.

7. Growth hormone (GH) is a polypeptide hormone secreted from the anterior pituitary gland. Its secretion and release are controlled by neurotransmitters of the central nervous system. The acute GH response to a resistance exercise session is related to specific component variables of the training program. The volume of training also appears to be a potent stimulus in the GH response to exercise. The greater fatigue (greater blood lactate concentrations) observed in the high-volume training program most likely contributed to the elevated GH response. The GH response to resistance exercise in females appears to be sensitive to changes in acute program variables (e.g. rest, intensity, volume of training). A year of exercise training at exercise intensity above the lactate threshold has been shown to amplify the pulsatile release of GH at rest. A reduction in the GH response to exercise may occur within 3 weeks of training.

8. Exercise is a potent growth hormone stimulator. Many athletes choose to use natural secretagogues before a work-out, no matter what time of day, in order to enhance the bump in GH that comes from exercises like high-intensity running and weight lifting. For those of us who are not athletic, increasing growth hormone levels with injections or secretagogues often leads to the improved strength and energy that it takes to increase the intensity, and subsequent GH release, of the exercises that we’re already doing. It’s a two way street, growth hormone enhances exercise and exercise enhances growth hormone.

9. Insulin is a protein hormone secreted by the β-cells of the islets of Langerhans within the pancreas. Exercise appears to decrease the circulating concentrations of insulin. This is likely the result of the inhibitory effect of catecholamines on the β-cells of the pancreas. The reduction of insulin appears to be a function of the duration of exercise. Training appears to increase the sensitivity to insulin of both the skeletal muscle and the liver. Trained individuals also appear to have a less pronounced insulin reduction during exercise than untrained individuals.

10. Catecholamines concentration appears to be elevated during both endurance and resistance training exercises. Short-duration sprints of maximal intensity appear sufficient to elevate both epinephrine and norepinephrine concentrations. Training does not appear to alter resting catecholamines concentrations. However, trained individuals do appear to have a greater capacity to secrete epinephrine and perhaps norepinephrine as well.

**REVIEW QUESTIONS**

1. Write the name of different glands and their locations in the human body.

2. What is an endocrine gland, and what are the functions of hormones? Briefly outline the major endocrine glands, their hormones and the specific action of these hormones.
3. Discuss in brief the major role of hypothalamus in regulation of hormone secretion.

4. What is a hormone? What do you mean by endocrine gland? Which hormone deficiency can cause diabetes insipidus?

5. Name the hormones secreted by the follicles of thyroid. Give two symptoms of hypothyroidism and name the disease.

6. What do you mean by stress hormone? Discuss the psycho-physiological response of stress hormone.

7. What is the role of testosterone in athletic performance? Discuss with suitable example.

8. ‘Growth Hormone Significantly Improves Body Composition’ Discuss with suitable example.

9. What is catecholamine? Discuss the response of various exercise and training on these hormones.

10. Which glands are known as gland of emergency? How adrenal medulla and sympathetic nervous system function as closely integrated system?

11. What are the seven principal hormones produced by the anterior pituitary? What function does it serve?

12. What hormones are produced when the body’s blood glucose level drop below normal? How do these hormones act to return the level to normal? What hormone is produced when the body’s blood glucose levels become elevated? How does this hormone act to return the level to normal?

13. Describe the hormonal regulation of metabolism during exercise. What hormones are involved, and how do they influence the availability of carbohydrates and fats for energy?
Energy Metabolism

Introduction to Energy Metabolism

The human body must be continually supplied with chemical energy to perform its many complex functions. Energy derived from the oxidation of food is not released suddenly at some kindling temperature because the body, unlike a mechanical engine, cannot use heat energy. If this is the fact, the body fluids would actually boil and our tissue would burst into flames. Rather, the chemical energy trapped within the bounds of carbohydrates, lipids and proteins is extracted in relatively small quantities during complex, enzymatically-controlled reactions that occur in the relatively cool, watery medium of the cell. This process reduces the loss of energy as heat and provides for much greater efficiency in energy transformations. This enables the body to make direct use of chemical energy.

Food materials are transformed into their simple and soluble form by digestion prior to the entry from the intestine. After entering into the blood they undergo a series of biochemical reactions stream place. The reactions which are included into the process of synthesis of larger protoplasmic...
molecules from lower ones for building up tissues are collectively known as\textit{ anabolism}, and those reactions which are included in the process of breakdown of larger protoplasmic molecules to smaller ones for the energy are collectively called\textit{ catabolism}. Thus, the term \textit{metabolism} of a food substance is meant by a series of specific biochemical reactions occurring with in the living organism from the time of its incorporation into the cell and of which some are concerned with tissue building system and other which tissue breakdown unit are termed as anabolism and catabolism respectively.

\textbf{Energy:} Energy is usually defined as the capacity to perform work. Normally there are six forms of energy such as mechanical, heat, light, chemical, electrical and nuclear energy. Each can readily be converted from one form to another. For example a person exercising or playing basketball is converting chemical energy (food stuffs) to heat and mechanical energy. Thus, it can be seen that in every human movement, whether it be contraction of the viscera within the body or the throwing of a baseball, the energy require to perform it is originated from food.

\begin{equation}
\text{Energy input} = \text{Energy output}
\end{equation}

\text{(Chemical energy of food) = (heat energy + work energy + stored chemical energy)}

\textit{Work:} The work is defined as a product of force times the distance for which this force acts. This may be expressed in the following equation:

\begin{equation}
\text{Work} = \text{force} \times \text{distance}
\end{equation}

Thus, lifting 5 pounds to a height of 5 feet will constitute 25 foot-pounds of work.

\textit{Power:} The term power is used to represent work in a unit of time. It consists of strength and speed and may be stated as:

\begin{equation}
\text{Power} = \text{work/time, or} \\
\text{Power} = (\text{force} \times \text{distance})/\text{time}
\end{equation}

In the above example, if the 5-pound weight were raised 5 feet in 1 second, power would be expressed as:

\begin{equation}
\text{Power} = \frac{5 \text{ lb} \times 5 \text{ ft}}{1 \text{ sec}} = 25 \text{ ft-lb/sec}
\end{equation}

\textbf{Efficiency:} Efficiency is defined as a percent of total energy used. Researches have generally utilized two distinct methods for computing it. The first and simplest is gross efficiency, in which the total energy used is divided into the total measurable work. It is written as:

\begin{equation}
\text{Gross efficiency (percent)} = \frac{\text{external work output}}{\text{total energy used for work}} \times 100
\end{equation}

The second method is called net efficiency. Since at any given time of the day a certain amount of the energy used up by the body is being used merely to maintain life (basal requirements), the calculated gross efficiency does not fairly represent the efficiency of the working muscles that are being appraised. In order to determine the net efficiency of the working body, the basal requirements should be subtracted from the total energy cost. Thus, the net efficiency is calculated with the following equation:

\begin{equation}
\text{Net efficiency (percent)} = \frac{\text{external work output}}{(\text{total energy used} - \text{basal requirements})} \times 100
\end{equation}

\textbf{Basal Metabolic Rate (BMR)}

The BMR may be defined as the amount of heat given out by a subject who, though, lying in a state of maximum physical and mental rest under comfortable conditions of temperature, pressure and humidity (12–18 hours after meals). Basal metabolic rate is usually expressed as the heat production per square meter of body surface per hour.

\textit{Normal Value:} In adult male normal BMR is about 40 cal/sq meter of body surface per hour and in the adult female is about 37 calories.

\textit{Factors affecting BMR:} The following factors which affecting BMR:

i. \textit{Age:} The BMR of children is much higher than the adults, i.e. with advancement of age, the BMR falls.

ii. \textit{Sex:} The BMR of the male is slightly higher than the female.

iii. \textit{Body Surface Area (BSA):} The BMR is directly proportional to the body surface area.
Larger the surface area greater will be the heat loss.

iv. Climate: In cold climates the BMR is high and in tropical climates the BMR is proportionately low.

v. Training: Trained athletes have a slightly higher BMR than a sedentary one.

vi. Diet: Prolong under nutrition lowers the BMR.

vii. Hormones: Some hormones secreted by adrenal, thyroids and anterior pituitary gland in circulating level, increase BMR.

viii. Pregnancy: The BMR of the pregnant women after 180 days of gestation rises.

ix. Body temperature: The BMR increases about 12 percent with the rise of 1°C of body temperature.

x. Drugs: Some drugs like Caffeine, Benzedrine, etc. increases the BMR.

**RESPIRATORY QUOTIENT (RQ)**

It is the ratio of the volume of CO₂ produced by the volume of O₂ consumed during a given time. Normal RQ of a healthy human adult it is 0.85 for a mixed diet.

**Factors affecting RQ**

i. Role of diet: Incase of carbohydrate diet the RQ is unity. Because in carbohydrate diet the volume of CO₂ produced is same as the volume of O₂ consumed. Incase of fats the RQ will be lowest and is about 0.7; because fat is an O₂ poor compound. Incase of proteins the RQ is about 0.8.

ii. Interconversion in the body: When carbohydrates are converted into fats in the body, RQ will rise and when fat is converted into carbohydrate just the reverse effects will be produced and RQ will fall.

iii. Acidosis and alkalosis: During acidosis CO₂ out put is greater than O₂ consumption so the RQ rises. The RQ falls due to the reverse condition of acidosis, i.e. CO₂ output is lesser than O₂ consumption.

iv. Rise of body temperature: It may increase RQ as in acidosis.

v. Starvation: RQ will fall than the normal.

vi. Exercise: With moderate exercise the RQ remains same. But in the maximum exercise lactic acid produces acidosis and as a result RQ rises. However, during recovery RQ falls because less production of CO₂.

**METS (Metabolic Equivalents):** This expression is used to describe the energy cost of work. One MET represents the net energy cost during rest (approximately 0.25 liters of oxygen or 1.25 kcal); two METS corresponds to two times the resting value; three METS is three times the resting value, etc. in some situations where work and/ or power is difficult to measure (such as in isometric work) and where direct measurements of the metabolic energy cost is not only time consuming, but requires rather expensive equipment, the MET concept undoubtedly has some practical value and will more than likely be used extensively in future metabolic investigations.

**SOURCES OF ENERGY**

**ATP-CP SYSTEM**

The energy set free during the breakdown of food is not utilized directly by the muscle cells. Instead, it is used by the body to build another, more complex powerhouse chemical compound known as ATP (adenosine triphosphate). ATP is found in the cytoplasm of cells and it consists of a adenosine component and three “high energy” phosphate groups. During the breakdown of ATP, one of these phosphate bonds is spilt or removed from the rest of the molecule, and approximately 8,000 calories (8 kcal) of energy are set free and free phosphate (Pi) plus adenosine diphosphate (ADP) are formed. It is known to us that the breakdown of ATP represents the immediate source of energy of the contractile process of the actin and myosin protein filaments of the myofibril.

In addition to ATP, creatine phosphate (CP or phosphoryl creatine) is another important chemical, which provides stored energy. Although CP also contains the high-energy phosphate bond, it cannot be used directly by the cells as a source of energy. Instead, it is used to resynthesize ATP from ADP. During the breakdown of CP when its
phosphate group is removed, a large amount of energy is set free and creatine plus free phosphate are formed. This energy is immediately available to reform ATP. For example, when ATP is broken down during heavy work, it is continuously regenerated from ADP and Pi by the energy set free during the breakdown of the stored CP; CP, like ATP, is in short supply in the muscle and must be resynthesized continuously.

It is interesting to note that the only means by which CP can be regenerated from Pi and creatine is from the energy set free by the breakdown of ATP. Thus, there are two ultimate sources of energy for the resynthesis of the phosphagens (ATP and CP): (i) breakdown of food, and (ii) glycolysis, the breakdown of glycogen resulting in the formation of lactic acid. The second of these processes is reversible: with an input of energy from food combustion, lactic acid is reconstituted to glycogen.

ATP and CP are not dependent on oxygen nor on a series of reactions and for this reason, they are extremely important not only during muscular work involving powerful quick starts of football players, high jumpers, sprinters and basketball players, but also in events that require only a few seconds to complete such as sprinting up a flight of stairs.

AERobic AND ANAEROBic Metabolism (Carbohydrate Breakdown)

The major purpose of aerobic and anaerobic metabolism is to provide energy for the body’s cells. Anaerobic metabolism uses carbohydrates (glucose and glycogen) exclusively for the manufacture of ATP, whereas aerobic metabolism can use all three foodstuffs (carbohydrates, fats and proteins) for its fuel.

**Anaerobic Metabolism**

Carbohydrates are broken down into glucose (in absence of O₂) where it is transported by the blood and stored in the muscles and liver in the form of glycogen. It is generally believed that somewhere between 350 to 450 grams of glycogen are stored in the human body. While each glucose molecule is made up of 6 carbon atoms, glycogen molecules are merely clusters of glucose sugar molecules that are linked to each other in chain-like structures. The actual process of breaking down glycogen involves the removal of a glucose molecule from the chain-like structure one at a time. This process is called glycogenolysis, while breakdown of glycogen or glucose to pyruvic acid (anaerobic) which is further oxidized to CO₂ and H₂O through TCA cycle is called glycolysis.

**Steps of anaerobic metabolism**: Anaerobic metabolism (in the absence of oxygen) involves a series of chemical reactions staring with the 6-carbon glucose molecule being broken down partially from glycogen into two 3-carbon molecules of pyruvic or lactic acid. This process is referred to as anaerobic glycolysis. Anaerobic glycolysis takes place entirely in the sarcoplasm/cytoplasm of the cell. Since all the enzymes that catalyze these reactions are located in this area.

**Reaction Sequence of Glycolysis**

i. Muscles, brain, kidneys and erythrocytes collect glucose from the blood and glycolyze that glucose directly. In such cases, hexokinase phosphorylates glucose to glucose 6-phosphate with the help of ATP. It involves the expenditure of one high-energy phosphate bond of ATP per glucose molecule changed (Fig. 7.2).

ii. Phosphohexose isomerase isomerizes glucose 6-phosphate to fructose 6-phosphate.

iii. Phosphofructokinase phosphorylates fructose 6-phosphate to fructose 1,6-diphosphate with the help of ATP and Mg²⁺.
iv. Aldolase catalyzes an aldol cleavage of fructose 1, 6-diphosphate into two triose phosphates, viz., dihydroxyacetone phosphate and glyceraldehydes 3-phosphate.

v. Phosphotriose isomerase an aldose-ketose isomerase, isomerizes dihydroxyacetone phosphate to glyceraldehydes 3-phosphate.

vi. Glyceraldehyde 3-phosphate dehydrogenase catalyzes the oxidation and phosphorylation of each glyceraldehyde 3-phosphate molecule to 1, 3-diphosphoglycerate, which bears a high energy C1-acyl phosphate group. NAD$^+$ is used as the electron-acceptor for the oxidation and is thereby reduced to NADH. This is the only oxidative reaction in glycolysis.

vii. Phosphoglycerate kinase transfers the high energy C1-phosphate group from 1, 3-diphosphoglycerate to Mg$^{2+}$-ADP, produc-

Fig. 7.2: Schematic representation of path of glycolysis (anaerobic pathway)
Energy Metabolism

vi. Mg$^{2+}$-ATP and 3-phosphoglycerate. Thus, two high-energy phosphate bonds are produced at this step from two molecules of 1, 3-diphosphoglycerate formed from each glucose molecule.

viii. Phosphoglyceromutase next isomerizes 3-phosphoglycerate to 2-phosphoglycerate. The enzyme requires Mg$^{2+}$ and a trace of 2, 3-diphosphoglycerate for its activity.

ix. Enolase catalyzes the dehydration of 2-phosphoglycerate to phosphoenolpyruvate (PEP) in presence of Mg$^{2+}$.

x. Pyruvate kinase, a tetrameric protein transfers the high-energy phosphate group from phosphoenolpyruvate to pyruvate and ADP in presence of Mg$^{2+}$. Thus, two high-energy phosphate bonds are produced at this step from two molecules of PEP formed from each glucose molecule.

xi. To maintain the availability of NAD$^+$ for glycolysis, cytoplasmic NADH produced during the action of glyceraldehyde 3-phosphate dehydrogenase must be speedily reoxidized.

The primary purpose of anaerobic metabolism is to provide energy for the body and that glucose is used exclusively for the production of ATP. For every single molecule that undergoes glycolysis results in a net production of 2 ATP molecules. While this represents only approximately 5% of the total number of ATP molecules that can be produced when the same amount of glucose is breakdown by aerobic metabolism (36 molecules of ATP). But anaerobic metabolism is tremendously important to us, especially during high-intensity type work. For example, when the body is unable to supply oxygen to the cell in sufficient quantity such as in underwater swimming or as in sprinting a 220-yd run, anaerobic metabolism is able to furnish energy for muscular contraction. At the same time, while anaerobic metabolism may function in the total absence of oxygen, this process is inefficient when compared to aerobic metabolism since it results in the accumulation of lactic acid. Because there is a finite limit as to the amount of lactic acid that can be tolerated in the human body, this process can continue for only a short period of time before muscular fatigue sets in.

Aerobic Metabolism

When oxygen is available in sufficient quantities, aerobic metabolism provides energy for the body’s cell. While the breakdown of glucose from glycogen starts in the same manner as anaerobic glycolysis, the pyruvic acid molecules (under aerobic conditions) are not converted to lactic acid, but instead, they diffuse from the sarcoplasm fluid across the mitochondrial membrane to the inside of the mitochondria where a series of chemical reactions take place to form carbon dioxide and water with the production of ATP’s taking place simultaneously.

When oxygen supply is plentiful and the muscles are not under heavy stress (such as in exhaustive-type anaerobic work), a glucose molecule is completely broken down to carbon dioxide and water with 36 molecules of ATP being produced in addition to those found in the anaerobic glycolysis is produced aerobically.

There are two important series of chemical reactions that play a vital role in aerobic metabolism: namely, the Krebs cycle and the electron transport system.

The Krebs cycle or Citric Acid cycle or Tricarboxylic Acid cycle (TCA cycle): The Krebs cycle is the final common path for the aerobic oxidation of the end products of glycolysis, β-oxidation and catabolism of many amino acids. This cycle operates in the mitochondrial matrix and is highly operative in cardiac muscle, liver and in the brain also, but not in erythrocytes as devoid of mitochondria.

Reaction Sequence of Krebs Cycle

In this cycle, an acetyl (C$_2$) unit from acetyl-CoA condenses with oxaloacetate (C$_4$) to form citrate (C$_6$), which is systematically oxidized, and decarboxylated by mitochondrial enzymes to regenerate oxaloacetate finally.

There are four steps of oxidation in the cycle, each yielding high-energy phosphate bonds through oxidative phosphorylation. Another step of this cycle forms one high-energy phosphate bond through substrate-level phosphorylation.

i. Citrate synthetase, a homodimeric (α$_2$) enzyme, condenses acetyl-CoA with oxaloacetate by aldol condensation. The
thioester bond in citryl-CoA is immediately hydrolyzed to release citrate and coenzyme A.

ii. Aconitase, a homodimeric (α₂) enzyme, isomerizes citrate to isocitrate. It first dehydrates, citrate to cis-aconitate by removing H and OH groups from its carbons derived respectively from the methylene-C and the ketonyl-C of oxaloacetate.

iii. Next the same enzyme Aconitase hydrates cis-aconitate to isocitrate by adding H and OH groups in a reverse order to those carbons.

iv. Isocitrate dehydrogenase, a heterotetrameric (α₂βγ) enzyme, changes isocitrate to α-ketoglutarate (C₅) in mitochondria. First it oxidizes isocitrate to oxalosuccinate, using NAD⁺ as the acceptor of a hydride (H⁺) ion from isocitrate. This is the first oxidative step of the TCA cycle; the NADH produced is reoxidized to NAD⁺ by the mitochondrial electron transport chain, generating three high-energy phosphate bonds through oxidative phosphorylation.

Fig. 7.3: Schematic representation of TCA cycle (Krebs cycle)
v. α-ketoglutarate dehydrogenase, converts α-ketoglutarate to succinyl CoA; this is the second oxidative step of the TCA cycle.

vi. Succinate thiokinase converts the succinyl CoA to Succinate and Coenzyme-A and utilize the energy released in this reaction. Thus a high-energy phosphate bond is produced at this step.

vii. Succinate dehydrogenase oxidized Succinate to fumarate. This reaction leads to the formation of two high-energy phosphate bonds through oxidative phosphorylation.

viii. Fumarase converts fumarate to malate.

ix. Malate dehydrogenase oxidized malate to oxaloacetate and this is the fourth-oxidative step of the TCA cycle. The mitochondrial electron transport chain oxidizes NADH produced in this reaction, yielding 3 high-energy phosphate bonds through oxidative phosphorylation. Formation of oxaloacetate complete one turn of TCA cycle.

**The electron transport system:** In mitochondria the electron transport system that is the respiratory chain where a chain of oxidative enzymes and coenzymes to combine oxygen that we breathe with the electrons and hydrogen ions that are released in Krebs cycle to form water and ATP. It is generally believe that the key to aerobic metabolism is the ability of flavoprotein and cytochromes to receive the electron and hydrogen ions from the Krebs cycle and then to pass them along the electron transport system to combine eventually with the oxygen that we breathe. In so doing, the majority of the energy is produced in this system. In fact, for each molecule of glucose entering the metabolic system, 34 molecules of ATP are produced via the electron transport system. Two ATP molecules are released from both the TCA cycle and the anaerobic glycolysis for a net gain of 38 molecules.

The energy production is much more efficient when glucose is broken down through aerobic metabolism then by way of the anaerobic system. In fact, 19 times more ATP is produced per glucose molecule aerobically then by anaerobic glycolysis (38 ATP = aerobic; 2 ATP = anaerobic). This helps to explain why a marathon runner can run so much longer (at higher efficiency level) at submaximal and steady state speed than at maximal speed.

**FAT METABOLISM**

The oxidation of fats can account for most of the ATP production during certain types of exercise.

The majority of fat (lipid) that is consumed by man is stored in the body as triglycerides. A four-part molecule made up of three molecules of fatty acids and one molecule of glycerol. It should be pointed out that this discussion will be restricted to the fatty acid molecule since most of ATP production from fats come from the fatty acid molecules that are split off (by an enzyme called lipase) from the triglycerides molecules of the adipose tissue. Each fatty acid molecule is composed of 16 or 18 long chains of carbon atoms with hydrogen atoms attached to them.

One fatty acid molecule splits off from the triglycerides molecule of the adipose tissue, it diffuses over into the blood where it is transported into the muscles and eventually undergoes a chemical transformation called beta-oxidation in which the fatty acid molecule is degraded to acetyl coenzyme and the acetyl coenzyme enters the Krebs cycle in the mitochondria to produce ATP.

However, for the normal person, since approximately 25 to 30 percent of the calories in the diet comes from fats. Thus it is important for energy source. Fat also appears to have the advantage over carbohydrates in terms of energy stored per unit of weight. For example, a gram of fat produces energy as 9.0 kcal/gm whereas a gram of carbohydrate produces 4.0 kcal/gm.

Another advantage of fat is that the human body is able to store in the cells of adipose tissue large quantities of fat, which can be called for later use, especially during a long duration type of exercise, i.e. longer endurance type activities such as marathon running, or long physical labor.

**PROTEIN METABOLISM**

Protein makes only slight contributions to ATP production during exercise. In fact, the breakdown of protein supplies only about 5 to 15 percent of the body’s total energy. Thus, consuming a large
amount of protein prior to an event or contest from the viewpoint of energy metabolism is supported. While proteins are not a common source of fuel, they are, however an important part of the diet. They are used primarily for construction of new body tissue cells.

Each protein molecule contains complex chains of carbon, oxygen, hydrogen and nitrogen atoms with amino acids being the basic unit. In fact, 20 different amino acids have been determined to be present in proteins. Several of these amino acids such as alanine, serine and cysteine can be broken down to pyruvic acids and thus enter into the Krebs cycle for oxidation and subsequent production of ATP. In addition, within the Krebs cycle other amino acids can be converted into molecules for final oxidation and production of ATP. While it is obvious that proteins do have the potential for ATP production, they, unlike carbohydrates and fats, are not considered to be a primary source of energy. As a source of fuel, they are used only under extreme circumstances with their major contribution being that of building and repairing body tissue.
ENERGY METABOLISM DURING REST,
EXERCISE AND RECOVERY

Rest: As oxygen consumption during rest remains relatively constant and is adequate to supply the required ATP and because blood lactic acid level remains within the normal range, it is apparent that metabolism of the resting conditions is aerobic. In fact, the aerobic breakdown of fats and glucose supplies all the ATP required by the body on the resting conditions. Fats and the remaining one-third contribute approximately two-thirds of the food source by the glucose.

Exercise: Both aerobic (in presence of oxygen) and anaerobic (in absence of oxygen) pathways contribute a certain amounts of energy during maximal exercise of various durations, and it is somewhat difficult to determine the major energy source in activities lasting from 2 to 4 minutes such as middle distance events. During this time, the aerobic and anaerobic energy sources are of equal importance. On the other hand at one extreme, maximal exercises of short duration are supplied via anaerobic metabolism, whereas exercises that can be performed for relatively long period of time such as marathon running are supplied primarily by aerobic metabolism. So the pathways of energy release will be divided into three categories, which are as follows:

a) Very short duration, high intensity exercises: If the duration of exercise is very short (within 15 seconds) and the intensity/workload is maximum, the energy will be liberating to breaking down the store ATP, CP are already store in the skeletal muscle tissues. For example: 100 m sprint, jumping, throwing, lifting weights, etc are the examples of such energy dependent event/game.

b) Short duration, high intensity exercises: Exercise in this category (duration- more than 15 sec. to 2 or 3 minutes) energy liberates mainly from the food fuel, i.e. carbohydrate and other two constituents-fats and proteins contributes very negligible

Fig. 7.5: The approximate percentage of contribution of aerobic and anaerobic energy sources in selected track events. (Shaded areas represent events in which aerobic and anaerobic systems are of nearly equal importance)

Table 7.1: General characteristics of the three systems by which ATP is formed

<table>
<thead>
<tr>
<th>System</th>
<th>Food or Chemical Fuel</th>
<th>O₂ required</th>
<th>Speed</th>
<th>Relative ATP Production</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anaerobic</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>i. ATP-PC system</td>
<td>Phosphocreatine</td>
<td>No</td>
<td>Fastest</td>
<td>Few; limited</td>
</tr>
<tr>
<td>ii. Lactic acid system</td>
<td>Glycogen (glucose)</td>
<td>No</td>
<td>Fast</td>
<td>Few; limited</td>
</tr>
<tr>
<td>Aerobic</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>i. Oxygen system</td>
<td>Glycogen, fats, proteins</td>
<td>Yes</td>
<td>Slow</td>
<td>Many; unlimited</td>
</tr>
</tbody>
</table>
amount. Carbohydrates breakdown (anaerobic glycolysis) in absence of oxygen produce ATP with the by-product of lactic acid. This category include sprinting 200 meter, 400 meter dashes, the 800 meter run and other events in which the required rate of work can be maintained only up to two or possibly 3 minutes.

c) Long continues sub-maximal exercise: Any exercise that can be maintained for relatively longer period of time (more than 2 or 3 minutes to few hours) should be included under this category. The nature of the activity should be long continuous (longer duration) and low intensity (sub-maximal workload). In this category the major foodstuffs are carbohydrates and fats. The major source of ATP is supplied by the aerobic system. The lactic acid and ATP-PC systems also contribute, but only at the beginning of the exercise, before oxygen consumption reaches a new steady state level. Once oxygen consumption reaches a new steady state level it is sufficient to supply all of the ATP energy required for the exercise. For this reason blood lactic acid does not accumulate to very high levels.

In prolonged activities of very low intensity, such as walking, playing golf and certain industrial tasks, lactic acid does not accumulate above the normal resting level. This is so because the phosphagen system alone is sufficient to supply the additional ATP energy needed prior to reaching a steady state of oxygen consumption. In these cases, fatigue can be delayed even up to 6 hours or more.

Many exercise activities require a blend of both anaerobic and aerobic metabolism. For example, in the 1500-meter run, the anaerobic systems supply the major portion of ATP during the sprint at both the start and finish of the race, with the oxygen system predominating during the middle,
or steady state, period of the run. This information is useful when developing training programs.

**Recovery:** The primary purpose of the metabolic pathway during the recovery period following exercise is to repay the energy stores that were used up during the exercise period. This is accomplished solely by the aerobic pathways.

**Oxygen Debt**

An oxygen debt is defined as all post exercise oxygen consumption above the basal oxygen consumption level. This means that the oxygen taken in during recovery over and above that which would have normally been consumed for the same period of time during rest is used to provide energy for repaying the energy stores that were used up during exercise.

Since there is generally a lag in the circulatory-respiratory systems during the transitional period of rest and exercise, a small oxygen debt can occur even in light exercise for which a steady state (when the rate of work is such that the metabolic demand can be met aerobically) can be attained. Ordinarily, this oxygen debt is paid off quickly during the recovery period. During high-intensity work (anaerobic) in which a steady state cannot be achieved, the oxygen debt will continue to rise until work ceases (Fig. 7.8).

The duration of the exercise is generally limited by the individual’s ability to tolerate a large oxygen debt. Research has illustrated that in maximal, all out work the anaerobic metabolism furnishes most of the energy needs, the length of work is restricted to approximately 30 seconds while recovery period may last as long as 90 minutes. The untrained sedentary person will usually stop work when an oxygen debt of about 10 liters has been reached, whereas with endurance training the debt capacity may be increased to somewhere around 17 to 18 liters. Apparently, the highly trained athlete is able to tolerate a much larger oxygen debt than the untrained. In determining the oxygen debt, two measurements are required: (i)
the resting oxygen consumption and the oxygen consumed during the recovery period. For example, let us assume that we found during a 5-minute rest period the subject consumed 2,000 ml of oxygen or a resting rate of 400 ml per minute; and during a 20-minute recovery period following an exercise bout he consumed 10,000 ml of oxygen. From the recovery oxygen (10,000 ml), we subtract that amount which would have been used if the subject were resting for that particular time period; or 10,000 ml - (20 minutes × 400 ml), which equals 2,000 ml (oxygen debt).

**A lactic acid and lactacid debts:** Although the oxygen debt is generally attributed to the cost of oxidation and reconversion of lactic acid (the by-product of anaerobic glycolysis), no very firm correlation of the debt volume and lactic acid content has been found. In fact early research indicated that an oxygen debt of approximately 2.4 liters could be accumulated without significant increases in blood lactate concentration. Because these findings have been supported by other investigators working in this area, the oxygen debt has been generally accepted as having two components: alactacid, for which no significant lactate increment is found and lactacid, which is represented by proportional increments in blood lactate. In addition, researches have also found a tremendous difference in the repayment of these two oxygen debt components. The alactacid debt accounts for the fast component of the recovery curve and it is repaid at a rate approximately 30 times faster than the lactacid debt (the slow component of recovery).

The alactacid debt is generally attributed to the restoration of the ATP and CP stores in the muscles that were depleted during exercise, whereas the lactacid debt is more associated with the metabolic cost of converting lactic acid build-up back to energy. More specially, it is believed to be the direct cost of converting some of the lactic acid (approximately three-quarters) by way of the Krebs cycle and electron transport system to carbon dioxide and water with the production of ATP. Also, it is believed to be the direct cost of converting a small amount (about 10 percent) of lactic acid back to glucose in the liver and released into the blood stream as blood glucose. The remaining portion of lactic acid is unaccounted for.

As stated earlier the lactacid oxygen debt is repaid at a rate approximately 30 times slower than the lactacid debt. This is because it takes a longer period of time to convert lactic acid back into glucose than it does to restore ATP and CP.

Evidence is rather clear that lactic acid can be removed from the blood and converted into glucose and/or carbon dioxide and water at a faster rate following exercise if an individual performs light work such as walking or slow jogging or cycling rather than just sitting down and doing nothing. By keeping active during the recovery period, this not only allows for a faster conversion of lactic acid by the liver, but the heart as well as the active skeletal muscles are also using some of the lactic acid as a source of energy. Because more of the lactic acid is used as fuel under these conditions, the lactacid oxygen debt is reduced to about 1 to 2 liters in size. These findings support the general practice by most athletes of moving around by way of walking or jogging in between their events or matches rather than resting during recovery. This procedure obviously allows them to recover more quickly and be better preparing for their next event or match.

**Excess Post Exercise Oxygen (O₂) Consumption (EPOC)**

The oxygen debt is also called excess post exercise O₂ consumption (EPOC) is the oxygen consumption above rest following exercise. Several factors contribute to the EPOC. First, some of the O₂ consumed early in the recovery period is used to resyntheses of stored PC in the muscle and replace O₂ stores in both muscle and blood. Other factors that contribute to the “slow” portion of the EPOC include an elevated body temperature, O₂ required to convert lactic acid to glucose and elevated blood levels of epinephrine and nor-epinephrine.

**Replenishment of Energy Stores during Recovery**

It is recalled that there are two sources of energy that are depleted to various extends during exercise: (i) anaerobic glycolysis (the phosphagens ATP and PC stored in the muscle cells) and (ii) the glycogen stored in large amount in muscles as well as in the liver which serves as an important source of fuel during most exercise activities.
Restoration of ATP, PC and alactacid O₂ debt: Most of the muscular stores of ATP and PC that were depleted during exercise are rapidly restored during the first 3 to 5 minutes of the recovery period. The ATP energy required for this process is supplied mainly by the aerobic system through the oxygen consumed during the alactacid debt. The repayment of the alactacid debt is also rapid, requiring only a few minutes. The maximum size of the alactacid component ranges between 2 and 3 liter of oxygen, although much higher values have been recorded in trained athletes.

Restoration of muscle and liver glycogen: Restoration of muscle and liver glycogen stores depleted during exercise is depend upon the type of exercise performed (continuous versus intermittent) and may require several days for completion during which time dietary intake of carbohydrate is necessary. Following continuous exhausting exercise, muscle glycogen restoration is 60 percent completed in 10 hours of recovery and is fully completed within 46 hours approximately. Following intermittent, exhausting exercise, restoration of muscle glycogen is 53 percent completed in 5 hours and is fully completed in 24 hours. Only small amount of muscle and liver glycogen are restored within the immediate recovery (1–2 hours) period following maximal exercise of either type. The ATP for muscle and liver glycogen restoration comes from the aerobic system, but does not involved, to a great extend, the oxygen consumed during the lactacid debt component.

Production of Lactic Acid

ATP is generated through three energy systems which are as follows:
- ATP-PCr System
- The glycolytic system
- The oxidative system

The glycolytic system involves the process of glycolysis. Carbohydrates are broken down into glucose where it is transported by the blood and stored in the muscles and liver in the form of glycogen. It is generally believed that somewhere between 350 to 450 grams of glycogen are stored in the human body. While each glucose molecule is made up of 6 carbon atoms, glycogen molecules...
are merely clusters of glucose sugar molecules that are linked to each other in chain-like structures. The actual process of breaking down glycogen involves the removal of a glucose molecule from the chain-like structure one at a time. This process is called glycogenolysis.

Glycolysis (stated earlier) ultimately produces pyruvic acid. This process does not require oxygen, but the use of oxygen determines the fate of pyruvic acid formed by glycolysis. In this context, when it referred to the glycolytic system it is referring to the process of glycolysis as it occurs without the involvement of oxygen. In this case the pyruvic acid is converted to lactic acid.

Another major limitation of anaerobic glycolysis is that it causes an accumulation of lactic acid in the muscle and body fluids. In all out sprint events lasting up to 2 minutes the demands of the glycolytic system are high, and muscle lactic acid levels can increase from a resting value of about 1 mmol/kg of muscle to more than 25 mmol/kg. This acidification of muscle fibers inhibits further glycogen breakdown because it impairs glycolytic enzyme function. In addition, the acid decreases

---

**Table 7.2:** Minimum and maximum period suggested for recovery process after maximum exercise

<table>
<thead>
<tr>
<th>Recovery Process</th>
<th>Suggested Recovery Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Restoration of muscle phosphagen stores (ATP + PC)</td>
<td>Minimum: 2 min, Maximum: 5 min</td>
</tr>
<tr>
<td>Repayment of the lactacid O₂ debt component</td>
<td>Minimum: 10 hours (continuous), Maximum: 46 hours</td>
</tr>
<tr>
<td>Muscle glycogen resynthesis</td>
<td>Minimum: 5 hours (intermittent), Maximum: 24 hours</td>
</tr>
<tr>
<td>Liver glycogen replenishment</td>
<td>Unknown, Minimum: 12–24 hours</td>
</tr>
<tr>
<td>Removal of lactic acid from blood and muscles</td>
<td>Minimum: 30 min (active-recovery), Maximum: 1 hour</td>
</tr>
<tr>
<td>Repayment of lactacid O₂ debt component</td>
<td>Minimum: 30 min (rest-recovery), Maximum: 1 hour</td>
</tr>
<tr>
<td>Restoration of O₂ stores</td>
<td>Minimum: 10–15 sec, Maximum: 1 hour</td>
</tr>
</tbody>
</table>
the fibers calcium binding capacity and thus may impede muscle contraction, which ultimately leads to the fatigue.

**Removal of Lactic Acid from Blood and Muscle**

The lactic acid accumulated in blood and muscle during exercise is removed during the recovery period. The speed of lactic acid removal depends on whether one rests during recovery (rest-recovery) or performance light exercise (30–50% VO₂ max) during recovery (exercise-recovery). Lactic acid is removed faster during exercise-recovery. The fate of the lactic acid removed is: (a) conversion to glucose and/or glycogen, (b) conversion to protein, and (c) oxidation to CO₂ and H₂O by the aerobic system. The major fate is oxidation, which occurs mainly in skeletal muscle but also occurs in heart, kidney, liver, and brain tissues. Although at least part of the oxygen and ATP required for removal of lactic acid probably comes from the lactic acid oxygen debt component, no quantitative relationship between the two has been determined. The maximal size of the lactacid debt is usually between 5 and 10 liters of oxygen.

**Anaerobic Threshold**

A disproportionate increase in ventilation without increasing oxygen consumption led to early speculation that the ventilatory break point might be related to the threshold (the point at which the blood lactate begins to accumulate above resting levels during a graded exercise test). Ventilatory break point reflects an increase in the volume of CO₂ produced per minute. The increased VCO₂ was brought to result from excess CO₂ being released from bi-carbonate buffering lactic acid. Wasserman and McIlroy coined the term anaerobic threshold to describe the above phenomenon because they assumed the sudden increase in CO₂ reflected a shift toward

![Fig. 7.11: Removal of lactic acid from blood at rest recovery (passive) and at exercise recovery (active)](image-url)
more anaerobic metabolism. They used the increase in respiratory exchange ratio (RER) as the marker of anaerobic threshold and believed that this was a good non-invasive alternative to blood sampling for detecting the onset of anaerobic metabolism.

Over the years, this concept is refined considerably. The most accurate technique for identifying anaerobic threshold now appears to involve monitoring both the ventilatory equivalent for oxygen (VE/VO₂) and the ventilatory equivalent for CO₂ (VE/VCO₂), which is the ratio of the amount of air breathed to the amount of CO₂ produced. The most specific criteria for estimating anaerobic threshold is a systematic increase in VE/VO₂ without a concomitant increase in VE/VCO₂. The ventilatory equivalent for CO₂ remains relatively constant, indicating that ventilation matches the body’s need to remove CO₂. The increase in VE/VO₂ indicates that the increase in ventilation to remove CO₂ is disproportionate to the body’s need to provide O₂.

Anaerobic threshold has been used as a non-invasive estimate of lactate threshold and under most conditions the two occur at the same point at time during at incremental exercise bout, or at the same percentage of maximal oxygen uptake. However, there are exceptions. For example, people with McArdle’s disease are incapable of increasing blood lactate and H⁺ levels during exercise due to a lack of muscle phosphorylase. They demonstrate a clear anaerobic threshold during exercise of increasing intensity even though blood lactate concentration remains at resting levels. Depleting the glycogen stores prior to exercise also alters the relationship between anaerobic threshold and lactate threshold.

**Ventilatory Threshold**

As exercise intensity increases towards maximum, at some point ventilation increases disproportionately as compare to oxygen consumption. This point is called the ventilatory break point. When the work rate exceeds 55 to 70 percent of one’s VO₂ max, oxygen delivery to the muscles can no longer support the oxygen requirements of oxidation. To compensate, more energy is derived from anaerobic glycolysis. This results in increased lactic acid production and accumulation. This lactic acid combines with sodium bicarbonate and forms sodium lactate, water and CO₂. As we know the increase in CO₂ stimulates chemoreceptors that signal the inspiratory center to increase ventilation. Thus, the ventilatory break point reflects the respiratory response to increase CO₂ levels and ventilation increases dramatically beyond the ventilatory break point.

**Lactate Threshold**

Lactate threshold is the point at which blood lactate begins to rapidly accumulate above resting level during exercise. The onset of blood lactate accumulation (OBLA) is a standard value set at either 2 or 4 mmol lactate/liter of oxygen and is used as a common reference point. Generally, individuals having higher lactate threshold or OBLA values, expressed as a percent of their VO₂ max, are capable of the best endurance performance.

*Importance in high performance sports:* The concept of an anaerobic threshold is a very attractive one because it offers a method of identifying the exercise intensity at which anaerobic metabolism makes a significant contribution to the provision of ATP. It is well established that...
endurance training increases the exercise intensity at which there is a significant rise in blood lactic acid concentration. This improvement in aerobic capacity is a consequence of a training-induced increase in the number of capillaries surrounding the type I and type II fibers along with the increase number of mitochondria. These changes should be detectable as changes in the anaerobic threshold whether it is measured as a lactate or the ventilatory threshold.

The anaerobic threshold concept also offers a submaximal method of assessing responses to training and also a way of describing the aerobic capacity of an individual in terms of percent of VO₂ max. This particular definition of aerobic fitness allows individuals with different VO₂ max values to be compared because the anaerobic threshold response to endurance training and is independent to VO₂ max.

The anaerobic threshold is also appealing because it may be more sensitive to training induced adaptation then VO₂ max alone. This is especially useful for assessing the adaptation to training of well-trained individuals who often show little additional improvement in VO₂ max with further training, but significant improvements in endurance capacity.

### Aerobic and Anaerobic Training Methods

General principles of training such as specificity and overload apply to all physical conditioning programs. In other words, for any training program to be effective, it must develop not only the specific energy systems involved, but it must also develop the specific muscle or muscle group as well as the specific movement patterns involved in the activity. In addition, it is well established that in order for any training program to be successful, it should follow the progressive overload principle. In the development of aerobic and anaerobic endurance, this means that as the person becomes endurance trained, any additional gains in endurance will only be experienced if he or she accomplished more work during each of the training sessions than can be normally accomplished.

Once it has been established which specific energy systems is involved in training as well as the specific movement patterns and muscle or muscle groups involved, selecting the proper training method that will bring about the desired changes is the next logical step. Table 7.3 illustrates some of the more popular training regimes. Within this table the approximate development percentage

![Fig. 7.13: The relationship between exercise intensity and blood lactate accumulation](image-url)
of the three energy systems for each of the various training programs.

The aerobic endurance can be developed by several methods such as jogging, continuous slow and fast running or swimming, it is generally agreed among coaches and exercise physiologists that interval training is probably the most popular aerobic and aerobic type training program used nowadays.

As any coach or sports scientist knows, one of the primary objectives of a training program is to obtain the greatest possible workload with the smallest physiological strain. The fact that this can best be achieved through the methods of interval training is well supported on heart rate and blood lactate. One of the advantages that interval training has is that it is very flexible and can be adapted for developing aerobic endurance, but also anaerobic endurance as well as the ATP-PC system (or all three systems equally as well).

The interval training method is based upon the overload principle. In order for the overload principle to be successful for interval training over a period of time, the intensity of the workouts must also be progressively increased as endurance is gained. In interval training, the intensity of the workouts can be adjusted by the manipulation of 5 different variables of which they are:
1. Rate and distance,
2. Number of repetitions during each workout,
3. Time of rest interval between the work intervals, 4. Type of activity during rest interval, and
5. Number of training periods per week.

It has been suggested that in order for optimal changes in the effectiveness of the oxygen transport system to be accomplished through interval training, the work bouts should be between 3 and 5 minutes in length, with light activity or short rest intervals between the bouts. The level of conditioning of the individual and the purpose of the training should determine the number of training periods per week, the number of repetitions during each workout, the intensity of each repetition, and the amount of rest between each bout.

In training for endurance, heart rate should be used as the criterion for determining the optimal training intensity. For example, heart rate during interval training should be kept at a rate between 60 and 90 percent of maximum during each of the 3 to 5 minute work periods (depends on training age). For healthy, sedentary people, the lower figure (60%) is sufficient for increase in endurance to be noticed, while a trained athlete should use the upper level (90%). The following equation can be used for both males and females with a relatively high degree of accuracy:

\[
\text{Maximal heart rate} = 220 - \text{age}
\]

Normally, the rest interval between exercise intervals should be equal to or less than the time of the actual work bouts. Also, it may be determined by the recovery heart rate. Generally,

<table>
<thead>
<tr>
<th>Training Method</th>
<th>Development (Percent)</th>
<th>Speed (ATP-CP)</th>
<th>Aerobic System (Oxygen)</th>
<th>Anaerobic System (Lactic Acid)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Repetitions of sprints</td>
<td>90</td>
<td>4</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>Continuous slow running</td>
<td>2</td>
<td>93</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Continuous fast running</td>
<td>2</td>
<td>90</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>Slow interval training</td>
<td>10</td>
<td>60</td>
<td>30</td>
<td></td>
</tr>
<tr>
<td>Fast interval training</td>
<td>30</td>
<td>20</td>
<td>50</td>
<td></td>
</tr>
<tr>
<td>Repetition running</td>
<td>10</td>
<td>40</td>
<td>50</td>
<td></td>
</tr>
<tr>
<td>Speed play (fartlek)</td>
<td>20</td>
<td>40</td>
<td>50</td>
<td></td>
</tr>
<tr>
<td>Interval sprinting</td>
<td>20</td>
<td>70</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Acceleration sprinting</td>
<td>90</td>
<td>5</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Hollow sprints</td>
<td>85</td>
<td>5</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Jogging</td>
<td>—</td>
<td>100</td>
<td>—</td>
<td></td>
</tr>
</tbody>
</table>

Table 7.3: Several Training Methods and the Approximate Development Percentage of the various Energy Systems
when the heart rate reaches 120 beats/min, the individual starts the next exercise bout.

The level of conditioning of the person and the purpose of the training should determine the number of training periods per week. For example, the competitive endurance athlete will need to train between 5 and 7 days per week, whereas the team sports athlete who is supplementing has regular training program with anaerobic endurance training can benefit from working out 2 to 4 times per week. At the same time, the sedentary person who is training for general endurance fitness can also benefit by working out between 2 and 4 days per week. Once the training benefits have been developed, they can be expected to be retained for several months with a weekly maintenance-training program of only one workout.

Repetitions of Sprints: This type of training involves several repetitions of sprints over distances between 60 and 220 yards at absolute maximum speed. Because the heartbeats so fast (around 200 beats/min or higher) during this type of training, a heart expansion stimulates does not normally take place since the heart does not fill to its maximum during the diastolic or resting period. As a result, an increased stroke volume of the heart is not generally produced. Instead, the primary effect of sprint training is the development of the ATP-CP energy system.

Continuous Slow Running: Continuous slow running is a form of training that develops almost totally aerobic endurance. This type of running is also known as LSD (long, slow distance). It involves running over long distances (somewhere between 3 and 20 miles and even further) at slow speeds (7 minute miles and slower). The amount of distance covered in this type of training is generally determined by the individual’s competitive distance. For instance, a 6-miler might run between 12 and 18 miles, while a miler might run between 3 and 5 miles. This type of training is performed at a relatively low intensity (about 60 to 80 percent of maximum heart rate), and is generally considered to be the best method for developing stroke volume and Capillarization. This type of training is being used; the speed by which it takes to bring the heart rate up to between 60 and 80 percent of maximum heart rate will depend upon the ability of the individual athlete. For example, a 7-minute mile pace might be the appropriate speed for an inexperienced college miler, while at the same time; a 6-minute mile pace might be adequate for a world-class marathon runner.

Continuous Fast Running: While this type of training is more intense than the slow continuous running, it also develops mainly aerobic endurance, the distance covered in this type of training are often in excess of the competitive distance; however, they are usually not as long as those performed in the slow continuous running. For instance, a 6-miler, instead, of running 12 to 18 miles as under continuous slow running training might run 8 to 10 miles at a steady, but faster pace, while a miler might run 1 1/2 to 2 ½ miles, and repeat the distance 2 to 3 times, alternately walking and jogging for 5 minutes after each run. This type of training is performed at a relatively high intensity (about 85 to 90 percent of maximum heart rate).

Slow Interval Training: This type of training causes the heart to beat approximately 170 to 180 times/min during the work phase, and develops mostly aerobic endurance. It is generally restricted to distances up to 880 yards. These would include repetitions of either 110, 220, 440, and 880 yards. The speed by which this type of training is carried out is somewhat faster than in continuous fast running training, but at the same time, slower than the athlete’s normal competitive speed. An example for an athlete who is capable of running the mile in 4 minutes might be as follows: running twenty 220-yd intervals in a time of 33 seconds each with each run followed by jogging 110 yards in 30 to 45 seconds each. Complete recovery is usually not experienced by the athlete during the between runs. Generally, when the recovery heart rate reaches 120 beats/min, the athlete starts the next work bout.

Fast Interval Training: During the work or “effort” phase of the fast interval training, the heart beats in excess of 180 beats/min. It develops primarily anaerobic endurance or speed. It is usually restricted to distances between 110 and 440 yards. An example for a 4-minute miler might be as follows: running several 440 yard intervals in 56 to 59 sec each, with each run followed by
jogging 440 yards in 2 to 3 minutes each. Again, like the slow interval training, complete recovery is not normally witnessed by the athlete during between runs. Fast interval training is generally not undertaken until a good overall background of aerobic or general endurance has been developed.

Repetition Running: When compare to interval training, repetition running generally involves longer distances with a more heart rate recovery (by way of walking) following each run. When using this type of training the speed determines whether an aerobic and anaerobic training benefit takes place. For instance, when the repetitions are run at speeds near racing conditions anaerobic conditions are developed. On the other hand, when the pace is slower than the racing speed, aerobic endurances usually develop. When repetitions of running beyond competitive distance are undertaken, the speed by which they are run should be significantly slower than the racing speed. At the same time, when repetition running reaches competitive speed, the distance of the fast run should not suppress half the racing distance for which the individual is running.

Speed Play: This type of training ("farlek") involves informal fast and slow-running (as compared to the formal fast-slow running in interval training) alternately at various speeds and distance over unmarked terrains such as golf courses, forests, country roads, etc. All of the aforementioned types of training may be combined in various ways in speed play, when carried out properly, this type of training should develop not only aerobic and anaerobic endurance, but also speed.

Interval Sprinting: This form of training consists of training for 50 yards and jogging for 60 yards after each for distances up to 3 miles. In other words, for each 440 yards, the athlete would combine four 50-yd sprints with four 60-yd jogs. Because of early fatigue (generally after the first several points), this type of training not only keeps the athlete from running at his or her maximal sprint speed, but it also causes the athlete to gradually extend his or her recovery jogging time. Therefore, the major training effect is primarily aerobic endurance.

Acceleration sprinting: This type of training develops almost exclusively speed and strength. It involves 50 to 110 yards of jogging, followed by 50 to 110 yards of fast striding, and finally 50 to 110 yards of sprinting. Following a recovery (via walking) distance of 50 to 110 yards, the procedure should be repeated. As it points out, this type of training is excellent when running outside in cold weather, since the athlete, instead of suddenly reaching his or her top sprint speed, gradually obtains it and therefore, avoids the possibility of muscle injury as so often occurs in conditions of low atmospheric temperatures.

Hollow Sprints: Hollow sprints involves sprinting, jogging, walking and for recovery prior to the next repetition. This type of training, provided adequate recovery takes place during the walking phase, mainly develops speed and muscular strength.

Jogging: Jogging has gained a tremendous amount of popularity in recent years, especially with older adults who are not training for competitive purpose, but instead, to lose a few pounds of fat and for health reasons. For example, it is well known that jogging is one of the best ways of improving the cardiovascular system for fighting coronary heart disease. Jogging, especially recreational jogging, generally consist of long, slow running.

**Adaptation to Aerobic Training**

Systematic endurance training results the adaptations in the following:

i. In muscle: (a) Muscle fiber type, (b) Capillary supply, (c) Myoglobin content, (d) Mitochondrial function and oxidative enzyme.

ii. Effecting energy source/release: (a) Carbohydrate and (b) Fat.

iii. Cardio-vascular adaptation: (a) Heart size, (b) stroke volume, (c) Heart rate, (d) Cardiac out put, (e) Blood flow, (f) Blood pressure and (g) Blood volume.

iv. Respiratory adaptation: (a) Lung volume, (b) Respiratory rate, (c) Pulmonary ventilation and (d) Pulmonary diffusion.

**Maximal Oxygen Consumption—The \( \text{VO}_2 \text{ Max} \)**

High maximal oxygen consumption (\( \text{VO}_2 \text{ max} \)) is one of the hallmark characteristics of great
endurance performers in running, cycling, rowing and cross-country skiing, so it must be pretty important.

VO₂ max is the maximum volume of oxygen that the body can consume during intense, whole-body exercise, while breathing air at sea level. This volume is expressed as a rate, either liters per minute (L/min) or milliliters per kg bodyweight per minute (ml/kg/min). Because oxygen consumption is linearly related to energy expenditure, when we measure oxygen consumption, we are indirectly measuring an individual’s maximal capacity to do work aerobically.

To rephrase, we might start by asking “what are the physiological determinants of VO₂ max?” Every cell consumes oxygen in order to convert food energy to usable ATP for cellular work. However, it is muscle that has the greatest range in oxygen consumption. At rest, muscle uses little energy. However, muscle cells that are contracting have high demands for ATP. So it follows that they will consume more oxygen during exercise. The sum total of billions of cells throughout the body consuming oxygen and generating carbon dioxide, can be measured at the breath using a combination of ventilation volume-measuring and O₂/CO₂-sensing equipment. The figure above summarizes this process of moving O₂ to the muscle and delivering CO₂ back to the lungs. So, if we measure a greater consumption of oxygen during exercise, we know that the working muscle is working at a higher intensity. To receive this oxygen and use it to make ATP for muscle contraction, our muscle fibers are absolutely dependent on two things: (1) an external delivery system to bring oxygen from the atmosphere to the working muscle cells and (2) mitochondria to carry-out the process of aerobic energy transfer. Endurance athletes are characterized by both a very good cardiovascular system and well developed oxidative capacity in their skeletal muscles. We need a big and efficient pump to deliver oxygen rich blood to the muscles, and we need mitochondria-rich muscles to use the oxygen and support high rates of exercise. Which variable is the limiting factor in VO₂ max, oxygen delivery or oxygen utilization.

**In the Well-trained, Oxygen Delivery Limits VO₂ Max**

Several experiments of different types support the concept that, in trained individuals, it is oxygen delivery, not oxygen utilization that limits VO₂ max. By performing exercise with one leg and directly measuring muscle oxygen consumption of a small mass of muscle (using arterial
catheterization) it has been shown that the capacity of skeletal muscle to use oxygen exceeds the heart’s capacity for delivery. Thus, although the average male has about 30 to 35 kg of muscle, only a portion of this muscle can be well perfused with blood at any one time. The heart can’t deliver a high blood flow to all skeletal muscle, and still maintain adequate blood pressure. This limitation is analogous to the water pressure in our house. If we turn all the faucets on while trying to take a shower, the shower pressure will be inadequate because there is not enough driving pressure. Without getting into deep on the hemodynamics, it seems that blood pressure is a centrally controlled variable; the body will not “open the valves” to more muscle than can be perfused without compromising central pressure and blood flow to the brain. The bigger the pumping capacity of the heart, the more muscle can be perfused while maintaining all-important blood pressure. 

As further evidence for a delivery limitation, long-term endurance training can result in a 300 percent increase in muscle oxidative capacity, but only about a 15 to 25 percent increase in VO₂ max. VO₂ max can be altered artificially by changing the oxygen concentration in the air. VO₂ max also increases in previously untrained subjects before a change in skeletal muscle aerobic capacity occurs. All of these observations demonstrate that VO₂ max can be dissociated from skeletal muscle characteristics.

Stroke volume, in contrast, is linearly related to VO₂ max. Training results in an increase in stroke volume and therefore, an increase in maximal cardiac output. Greater capacity for oxygen delivery is the result. More muscle can be supplied with oxygen simultaneously while still maintaining necessary blood pressure levels.

**In the Untrained, Skeletal Muscle Capacity can be Limiting**

Heart performance dictates VO₂ max, it is important to explain the contributing, or accepting, role of muscle oxidative capacity. Oxygen consumption = Cardiac output x arterial-venous oxygen difference (a-v O₂ diff). As the oxygen rich blood passes through the capillary network of a working skeletal muscle, oxygen diffuses out of the capillaries and to the mitochondria (following the concentration gradient). The higher the oxygen consumption rate by the mitochondria, the greater the oxygen extraction, and the higher the a-v O₂ difference at any given blood flow rate. Delivery is the limiting factor because even the best-trained muscle cannot use oxygen that isn’t delivered. But, if the blood is delivered in to muscles that are poorly trained for endurance, VO₂ max will be lower despite a high delivery capacity. When we perform VO₂ max tests on untrained persons, we often see that they stop at a time point in the test when their VO₂ max seems to still be on the way up. The problem is that they just do not have the aerobic capacity in their working muscles and become fatigued locally prior to fully exploiting their cardiovascular capacity. In contrast, when we test athletes, they will usually show a ideal flattening (leveling off) out of VO₂ despite increasing intensity towards the end of the test. Heart rate peaks out, VO₂ maxes out, and even though some of the best trained individual can hold out at VO₂ max for several minutes, and changes at the muscular level that inhibit muscular force production and bring on exhaustion.

**How VO₂ Max is Measured?**

In order to determine an athlete’s true maximal aerobic capacity, exercise conditions must be created that maximally stress the blood delivery capacity of the heart.

A physical test that meets this requirement must:
- Employ at least 50 percent of the total muscle mass. Activities which meet this requirement include running, cycling, rowing, etc. The most common laboratory, method is the treadmill running test. A motorized treadmill with variable speed and variable inclination is employed.
- Be independent of strength, speed, body size, and skill. The exception to this rule is specialized tests for swimmers, rowers, skaters, etc.
- Be of sufficient duration for cardiovascular responses to be maximized. Generally, maximal tests using continuous graded
exercise protocols are completed in 6 to 12 minutes.

Be performed by someone who is highly motivated! VO₂ max tests are tough, but they don’t last too long.

If we use a treadmill test as an example, here is what will happen. After a medical examination and after being attached to an ECG machine to monitor cardiac electrical activity, the subject might start the test by walking on the treadmill at low speed and zero percent grades. If subject’s fitness level is quite high, the test might be initiated at a certain running speed. Then, depending on the exact protocol, speed or inclination (or both) of the treadmill will increase at a regular intervals (30 sec to 2 minutes). While running, the subject will be breathing through a two-way valve system (open circuit). Air will come in from the room, but will be expired through sensors that measure both volume and oxygen concentration in the expired air. Using these values and some calculation, oxygen uptake will be calculated by a computer at each stage. With each increase in speed or incline, more muscle mass will be employed at a greater intensity. Oxygen consumption will increase inclination with increasing the workload. However, at some point, an increase in intensity will not result in an “appropriate” increase in oxygen consumption. Ideally, the oxygen consumption will completely flatten out despite ever-increasing workload. This is the true indication of achieving VO₂ max along with other criteria i.e. RQ = 1 & maximum heart rate is around 180 b/min.

The value is given by the test administrator will be in one of two forms. The first is called absolute VO₂ max. This value will be in liters/min and will probably be between 3.0 and 6.0 liters/min if the subject is a man and between 2.5 and 4.5 l/min if she is a woman. This absolute value does not take account differences in body size, so a second way of expressing of VO₂ max is common. This is called the relative VO₂ max. It will be expressed in milliliters per min per kg bodyweight (ml/min/kg). So, if absolute VO₂ max was 4.0 liters/min and body weighed is 75 kg, then the relative VO₂ max would be 4000 divided by 75, or 53.3 ml/min/kg. In general, absolute VO₂ max favors the large endurance athlete, while relative VO₂ tends to be higher in smaller athletes.

For comparison, the average maximal oxygen consumption of an untrained male in his mid 30s is about 40 to 45 ml/min/kg, and decreases with age. The same person who undergoes a regular endurance exercise program might increase to 50 to 55 ml/min/kg. A champion male master runner age 50 will probably have a value of over 60 ml/min/kg. An Olympic champion 10,000-meter runner will probably have a VO₂ max over 80 ml/min/kg. The underlying physiology is the same, however specific differences result in lower population values for VO₂ max in untrained,
trained and champion females when compared to men at a similar relative capacity.

**VO₂ Max as a Predictor of Performance**

In elite athletes, VO₂ max is not a good predictor of performance. The winner of a marathon race for example, cannot be predicted from maximal oxygen uptake. Perhaps more significant than VO₂ max is the speed at which an athlete can run, bike or swim at VO₂ max. Two athletes may have the same level of aerobic power but one may reach their VO₂ max at a running speed of 20 km/hr and the other at 22 km/hr.

While a high VO₂ max may be a prerequisite for performance in endurance events at the highest level, other markers such as lactate threshold are more predictive of performance. Again, the speed at lactate threshold is more significant than the actual value itself. So, VO₂ max as an athlete’s aerobic potential and the lactate threshold as the marker for how much of that potential they are tapping.

**Role of Heredity**

There is a significant genetic component to most of the underlying physical qualities that limit just how “Citius, altius, fortius” we can be with training. VO₂ max is no exception. The reality is that if an adult male with a natural, untrained VO₂ max of 45 ml/min/kg trains optimally for 5 years, they might see their VO₂ max climb to around 60 to 65 ml/min/kg. This is a huge improvement. Yet, the best runners have a VO₂ max of 75 to 85 ml/kg so our hard training normal person is still going to come up way short against the likes of these people. If they were to stop training for a year, their VO₂ max might fall to about where the average person’s topped out after years of optimal training. The bottom line is that Olympic champions are born with unique genetic potential that is transformed into performance capacity with years of hard training. Scientific studies focusing on the genetics of exercise adaptation have also demonstrated that not only is our starting point genetically determined, but our adaptability to training (how much we improve) is also quite variable and genetically influenced. While the typical person will show a substantial increasing in VO₂ max with 6 months of exercise, carefully controlled research studies have shown that a small percentage of people will hardly show an increase in VO₂ max at all.

**VO₂ Max in Athletes and Non Athletes**

VO₂ max varies greatly between individuals and even between elite athletes that compete in the same sport. The table below (Table 7.4) lists normative data for VO₂ max in various population groups.

Heredity plays a major role in a person’s VO₂ max and heredity can account for up to 25 to 50 percent of the variance observed between individuals. The highest ever recorded VO₂ max is 94 ml/kg/min in men and 77 ml/kg/min in women. Both were cross-country skiers. Untrained girls and women typically have a maximal oxygen uptake 20 to 25 percent lower than untrained men. However, when comparing elite athletes, the gap tends to close to about 10%. Taking it step further, if VO₂ max is adjusted to account for fat free mass in elite male and female athletes, the differences disappear in some studies. Cureton and Collins suggest that sex-specific essential fat stores account for the majority of metabolic differences in running between men and women.

**Effects of Aging on VO₂ Max**

VO₂ max decreases with age. The average rate of decline is generally accepted to be about 1% per year or 10% per decade after the age of 25. One large cross sectional study found the average decrease was 0.46 ml/kg/min per year in men (1.2%) and 0.54 ml/kg/min in women (1.7%). However, this deterioration is not necessarily due to the aging process. In some cases the decrease may be purely a reflection of increased body weight with no change in absolute values for ventilation of oxygen. VO₂ max is usually expressed relative to body weight. If this increases, as tends to happen with age and aerobic fitness stays the same then VO₂ max measured in ml/kg/min will decrease. Usually, the decline in age-related VO₂ max can be accounted for by a reduction in maximum heart rate, maximal stoke volume and maximal A-V difference, i.e. the difference between oxygen concentration in arterial blood and venus blood.
Energy Metabolism

Vigorous training at a younger age does not seem to prevent the fall in VO2 max if training is ceased altogether. Elite athletes have been shown to decline by 43 percent from ages 23 to 50 (from 70 ml/kg/min to 40 ml/kg/min) when they stop training after their career is over. In some cases, the relative decline is greater than for the average population - as much as 15 percent per decade or 1.5 percent per year. However, in comparison, master athletes who continue to keep fit only show a decrease of 5 to 6 percent per decade or 0.5 to 0.6 percent per year. When they maintain the same relative intensity of training, a decrease of only 3.6 percent over 25 years has been reported and most of that was attributable to a small increase in bodyweight. It seems that training can slow the rate of decline in VO2 max but becomes less effective after the age of about 50.

Table 7.4: Maximal oxygen uptake (ml/kg/min) in various population groups

<table>
<thead>
<tr>
<th>Age</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non Athletes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10–19*</td>
<td>45–50</td>
<td>38–46</td>
</tr>
<tr>
<td>20–29*</td>
<td>45–52</td>
<td>40–48</td>
</tr>
<tr>
<td>30–39*</td>
<td>43–48</td>
<td>38–45</td>
</tr>
<tr>
<td>40–49*</td>
<td>40–45</td>
<td>35–40</td>
</tr>
<tr>
<td>50–59*</td>
<td>38–40</td>
<td>30–36</td>
</tr>
<tr>
<td>60–69*</td>
<td>35–38</td>
<td>28–30</td>
</tr>
<tr>
<td>70–79*</td>
<td>30–35</td>
<td>25–27</td>
</tr>
<tr>
<td>Athletes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseball/softball</td>
<td>18–32</td>
<td>52–57</td>
</tr>
<tr>
<td>Basketball</td>
<td>18–30*</td>
<td>48–60</td>
</tr>
<tr>
<td>Bicycling</td>
<td>18–26</td>
<td>62–74</td>
</tr>
<tr>
<td>Canoeing</td>
<td>22–28</td>
<td>55–67</td>
</tr>
<tr>
<td>Football</td>
<td>20–36</td>
<td>42–60</td>
</tr>
<tr>
<td>Gymnastics</td>
<td>18–22</td>
<td>41–52</td>
</tr>
<tr>
<td>Ice Hockey</td>
<td>10–30</td>
<td>50–63</td>
</tr>
<tr>
<td>Jockey</td>
<td>20–40</td>
<td>50–60</td>
</tr>
<tr>
<td>Orienteering</td>
<td>20–60</td>
<td>47–53</td>
</tr>
<tr>
<td>Racquetball</td>
<td>20–35</td>
<td>55–62</td>
</tr>
<tr>
<td>Rowing</td>
<td>20–35</td>
<td>60–72</td>
</tr>
<tr>
<td>Skiing, alpine</td>
<td>18–30</td>
<td>57–68</td>
</tr>
<tr>
<td>Skiing, nordic</td>
<td>20–28</td>
<td>65–94</td>
</tr>
<tr>
<td>Ski jumping</td>
<td>18–24</td>
<td>58–63</td>
</tr>
<tr>
<td>Soccer</td>
<td>22–28*</td>
<td>54–64</td>
</tr>
<tr>
<td>Speed skating</td>
<td>18–24</td>
<td>56–73</td>
</tr>
<tr>
<td>Swimming</td>
<td>10–25</td>
<td>50–70</td>
</tr>
<tr>
<td>Track and field, discuss</td>
<td>22–30</td>
<td>42–55</td>
</tr>
<tr>
<td>Track and field, running</td>
<td>18–39*</td>
<td>60–85</td>
</tr>
<tr>
<td>Track and field, shot put</td>
<td>22–30</td>
<td>40–60</td>
</tr>
<tr>
<td>Volleyball</td>
<td>18–22*</td>
<td>47–52</td>
</tr>
<tr>
<td>Weightlifting</td>
<td>20–30</td>
<td>38–52</td>
</tr>
<tr>
<td>Wrestling</td>
<td>20–30</td>
<td>52–65</td>
</tr>
</tbody>
</table>

Adopted from Wilmore and Costil (2005) (3), “*”–Study done by author.

Determining VO2 Max

VO2 max can be determined through a number of physical evaluations. These tests can be direct or
Direct testing requires sophisticated equipment to measure the volume and gas concentrations of inspired and expired air. There are many protocols used on treadmills, cycle ergometers and other exercise equipment to measure VO\(_2\) max directly. The direct measurement of VO\(_2\) max can be done in the laboratory as well as in the field. One of the most common protocols is the Bruce protocol often used for testing VO\(_2\) max in athletes or for signs of coronary heart disease in high-risk individuals.

Indirect testing is much more widely used by scientists/coaches as it requires little or no expensive equipment. There are many indirect tests used to estimate VO\(_2\) max. Some are more reliable and accurate than others but none are as accurate as direct testing. Examples include the multistage shuttle run (bleep test), 12 minute walk test, 1.5 mile run, etc. which are as follows:

**Cooper 12 Minute run and walk test**

**Purpose:** To test aerobic fitness (the ability of the body to use oxygen to power it while running).

**Equipment required:** Flat oval or running track, marking cones, recording sheets, stopwatch.

**Description/procedure:** Place markers at set intervals around the track to aid in measuring the completed distance. Participants run for 12 minutes, and the total distance covered is recorded. Walking is allowed, though the participants must be encouraged to push themselves as hard as they can.

**Table 7.5: Normative Value for adult males**

<table>
<thead>
<tr>
<th>Rating</th>
<th>Distance (meters)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Excellent</td>
<td>&gt; 2700 m</td>
</tr>
<tr>
<td>Good</td>
<td>2300–2700 m</td>
</tr>
<tr>
<td>Average</td>
<td>1900–2300 m</td>
</tr>
<tr>
<td>Below average</td>
<td>1500–1900 m</td>
</tr>
<tr>
<td>Poor</td>
<td>&lt; 1500 m</td>
</tr>
</tbody>
</table>

**Scoring:** There are several equations that can be used to estimate VO\(_2\) max (in ml/kg/min) from the distance score (a formula for either kms or miles):

\[
VO_2\text{max} = (35.97 \times \text{miles}) - 11.29 \\
VO_2\text{max} = (22.351 \times \text{kilometers}) - 11.288
\]

The above table also gives general guidelines for interpreting the results of this test for adults.

- **Target population:** This test can be modified to be suitable for most populations. For those who are unfit or unable to run, there are similar walking tests that can be performed.
- **Validity:** Cooper (1968) reported a correlation of 0.90 between VO\(_2\) max and the distance covered in a 12 min walk-run.
- **Reliability:** The reliability of this test would depend on practice, pacing strategies and motivation level. There should be good reliability if these issues are addressed.
- **Advantages:** Large groups can be tested at once, and it is a very cheap and simple test to perform.
- **Disadvantages:** Practice and pacing is required, and performance on this test can be affected greatly by motivation.
- **Variations/modifications:** The test can also be conducted by running on a treadmill for 12 minutes, set to level 1 (1 percent) incline to mimic outdoor running. There are many variations of the walk/run test.

**Queens College Step Test**

**Purpose:** This step test provides a measure of cardio-respiratory endurance fitness.

**Equipment required:** 16.25 inches or 41.3 cm step (box/stool), stopwatch, metronome, heart rate monitor (optional).

**Description:** The athlete steps up and down on the platform at a rate of 22 steps per minute for females and at 24 steps per minute for males, for a total period of 3 minutes. The athlete immediately stops on completion of the exercise test, and the heart beats are counted for 15 seconds from 5 to 20 seconds of recovery.

**Scoring:** An estimation of VO\(_2\) max can be calculated from the test results, using the formula below:

- Men: \( VO_2\text{max} (\text{ml/kg/min}) = 111.33 - 0.42 \times \text{heart rate (bpm)} \)
- Women: \( VO_2\text{max} (\text{ml/kg/min}) = 65.81 - 0.1847 \times \text{heart rate (bpm)} \)
Reliability: Test re-test reliability for recovery heart rate has been measured as $r = 0.92$.

Validity: Correlation between recovery heart rate and $\text{VO}_2\text{max}$ has been measured as $r = -0.75$.

Advantages: Minimal equipment and costs involved, little time required and can be self-administered.

Disadvantages: Biomechanical characteristics vary between individuals (e.g. taller people are at an advantage). Also, apparently the data was formulated from treadmill running, therefore, their assumption is that stepping and treadmill running have the same oxygen cost.

Harvard Step Test

The Harvard Step Test is a test of aerobic fitness, developed by Brouha et al. (1943) in the Harvard Fatigue Laboratories during World War-II. The features of this test are that it is simple to conduct and requires minimal equipment. There are many other variations of step tests too.

Equipment required: Step or platform 20 inches/50.8 cm high (18” for Indian male), stopwatch, metronome or cadence tape.

Description/procedure: The athlete steps up and down on the platform at a rate of 30 steps per minute (every two seconds) for 5 minutes or until exhaustion. Exhaustion is defined as when the athlete cannot maintain the stepping rate for 15 seconds. The athlete immediately sits down on completion of the test, and the total number of heart beats is counted between 1 to 1.5 minutes after finishing. This is the only measure required if using the short form of the test. If the long form of the test is being conducted, there is an additional heart rate measures at between 2 to 2.5 minutes, and between 3 to 3.5 minutes.

Scoring: The Fitness Index score is determined by the following equations. For example, if the total test time was 300 seconds (if completed the whole 5 minutes), and the number of heart beats between 1–1.5 minutes is 90, between 2–2.5 it is 80 and between 3–3.5 it is 70, then the long form Fitness Index score would be: $(100 \times 300)/(240 \times 2) = 62.5$. Note: you are using the total number of heart beats in the 30 second period, not the rate (beats per minute) during that time.

<table>
<thead>
<tr>
<th>Rating</th>
<th>Fitness index (long form)</th>
</tr>
</thead>
<tbody>
<tr>
<td>excellent</td>
<td>&gt; 90</td>
</tr>
<tr>
<td>good</td>
<td>80–89</td>
</tr>
<tr>
<td>high average</td>
<td>65–79</td>
</tr>
<tr>
<td>low average</td>
<td>55–64</td>
</tr>
<tr>
<td>poor</td>
<td>&lt; 55</td>
</tr>
</tbody>
</table>

Fitness Index (short form) = $(100 \times \text{test duration in seconds})$ divided by $(5.5 \times \text{pulse count between 1 and 1.5 minutes})$.

Fitness Index (long form) = $(100 \times \text{test duration in seconds})$ divided by $(2 \times \text{sum of heart beats in the recovery periods})$.

Validity: Correlation to $\text{VO}_2\text{max}$ has been reported as between 0.6 to 0.8 in numerous studies.

Advantages: This test requires minimal equipment and costs, and can be self-administered.

Disadvantages: Biomechanical characteristics vary between individuals. For example, considering that the step height is standard, taller people are at an advantage as it will take less energy to step up onto the step box. Body weight has also been shown to be a factor. Testing large groups with this test will be time consuming.

Comments: The Harvard Step Test was developed by Brouha et al. (1943) in the Harvard Fatigue Laboratory during World War-II. Some sources suggest a 40 cm high bench, which is not the standard and original bench height. Since the original description of this test, there have been variations in the test procedure such as reducing the bench height for female subjects in some research studies.

Beep Test (Multistage Fitness Test, 20m Shuttle run, Bleep Test)

The multistage fitness test is a commonly used maximal running aerobic fitness test. It is also known as the 20 meter shuttle run test, beep or bleep test among others. The details of the tests are as follows:

Equipment required: Flat, non-slip surface, marking cones, 20m measuring tape, CD or pre-recorded audio tape, recording sheets.

Description: This test involves continuous running between two lines 20m apart in time to
recorded beeps. For this reason the tests is also often called the ‘beep’ or ‘bleep’ test. The test subjects stand behind one of the lines facing the second line, and begin running when instructed by the CD or tape. The speed at the start is quite slow. The subject continues running between the two lines, turning when signaled by the recorded beeps. After about one minute, a sound indicates an increase in speed and the beeps will be closer together. This continues each minute (level). If the line is not reached in time for each beep, the subject must run to the line turn and try to catch up with the pace within 2 more ‘beeps’. Also, if the line is reached before the beep sounds, the subject must wait until the beep sounds. The test is stopped if the subject fails to reach the line (within 2 meters) for two consecutive ends. There are several versions of the test, but one commonly used version has an initial running velocity of 8.5 km/hr, which increases by 0.5 km/hr each minute.

**Scoring:** The athletes score is the level and number of shuttles (20 m) reached before they are unable to keep up with the recording. This score can be converted to a VO₂ max equivalent score using this calculator/chart.

**Target population:** This test is suitable for sports person and school groups, but not for populations in which a maximal exercise test would be contraindicated.

**Validity:** There are published VO₂ max score equivalents for each level reached. The correlation to actual VO₂ max scores is high.

**Reliability:** Reliability would depend on how strictly the test is run and the practice allowed for the subjects.

**Advantages:** Large groups can perform this test all at once for minimal costs. Also, the test continues to maximum effort unlike many other tests of endurance capacity.

**Disadvantages:** Practice and motivation levels can influence the score attained, and the scoring can be subjective. As the test is often conducted outside, the environmental conditions can affect the results.

**Other considerations:**
- As the audio-tapes may stretch overtime, the tapes need to be calibrated which involves timing a one-minute interval and making adjustment to the distance between markers. The recording is also available on compact disc, which does not require such a stringent calibration, but should also be checked occasionally.
- This test goes by many names, though you need to be careful as the different names also may signify that these are different versions of the test. Therefore, you need to be wary when comparing results or comparing to norms.

![Fig. 7.16: Dimension of bleep test protocol](image)
This test is a maximal test, which requires a reasonable level of fitness. It is not recommended for recreational athletes or people with health problems, injuries or low fitness levels.

**PRT 1.5 Mile Run Test**

This test forms part of the Navy Physical Readiness Test (PRT), performed by US Navy personnel. An alternative to this test is the 500 yard swim test.

**Purpose:** This test measures aerobic fitness and leg muscles endurance.

**Equipment required:** 1.5 mile flat and hard running course, stopwatch

**Description/procedure:** The aim of this test is to complete the 1.5 mile course in the shortest possible time. At the start, all subjects line up behind the starting line. On the command ‘go,’ the clock will start, and the subject will begin running at their own pace. Although walking is authorized, however it is strongly discouraged. A cool down walk should be performed at the completion of the test.

**Scoring:** The total time to complete the course is to be recorded.

**Comments:** It is permitted to pace a participant (such as running ahead of, along side of, or behind) during the run, as long as there is no physical contact with the runner and it does not physically hinder other people taking the test. Cheering or calling out the elapsed time is also permitted. During the 1.5-mile run, it is critical to have some type of medical support in place or a medical emergency plan. Dangerous climate conditions such as hot/humid weather should be avoided and water or other fluids should be made available upon completion of the assessment.

**SUMMARY**

1. Energy is usually defined as the capacity to perform work. Normally there are six forms of energy such as mechanical, heat, light, chemical, electrical and nuclear. Each can readily be converted from one form to another. Food materials are transformed into their simple and soluble form by digestion prior to the entry from the intestine. After entering into the blood they undergo a series of biochemical reactions takes place. The reactions which are included into the process of synthesis of larger protoplasmic molecules from lower ones for building up tissues are collectively known as **anabolism**, and those reactions which are included in the process of breakdown of larger protoplasmic molecules to smaller ones for the energy are collectively called **catabolism**. Thus, the term **metabolism** of a food substance is meant by a series of specific biochemical reactions occurring with in the living organism from the time of its incorporation into the cell and of which some are concerned with tissue system and other which tissue breakdown unit are termed as anabolism and catabolism respectively.

2. The BMR may be defining as the amount of heat given out by a subject who, though, lying in a state of maximum physical and mental rest under comfortable conditions of temperature, pressure and humidity. Respiratory quotient (RQ) is the ratio of the volume of CO₂ produced by the volume of O₂ consumed during a given time. Normal RQ of a healthy human adult it is 0.85 for a mixed diet. METS (Metabolic Equivalents) expression is used to describe the energy cost of work. One MET represents the net energy cost during rest; two METS corresponds to two times the resting value; three METS is three times the resting value, etc.

3. The energy set free during the breakdown of food is not utilized directly by the muscle cells. Instead, it is used by the body to build another, more complex powerhouse chemical compound known as ATP. In addition to ATP, creatine phosphate (CP or phosphoryl creatine) is another important chemical, which provides stored energy. ATP and CP are not dependent on oxygen nor on a series of reactions and for this reason, they are extremely important not only during muscular work involving powerful quick starts of football players, high jumpers, sprinters and basketball players, but also in events that require only a few seconds to complete such as sprinting up a flight of stairs.
4. The major purpose of aerobic and anaerobic metabolism is to provide energy for the body's cells. Anaerobic (in absence of O_2) metabolism uses carbohydrates (glucose and glycogen) exclusively for the manufacture of ATP, whereas aerobic (in presence of O_2) metabolism can use all three foodstuffs (carbohydrates, fats and proteins) for its fuel. The primary purpose of anaerobic metabolism is to provide energy for the body and that glucose is used exclusively for the production of ATP. For every single molecule that undergoes glycolysis results in a net production of 2 ATP molecules. When oxygen supply is plentiful and the muscles are not under heavy stress (such as in exhaustive-type anaerobic work), a glucose molecule is completely broken down to carbon dioxide and water with 36 molecules of ATP being produced in addition to those found in the anaerobic glycolysis is produced aerobically.

5. As oxygen consumption during rest remains relatively constant and is adequate to supply the required ATP, and because blood lactic acid level remains within the normal range, it is apparent that metabolism of the resting conditions is aerobic. In fact, the aerobic breakdown of fats and glucose supplies all the ATP required by the body on the resting conditions. Both aerobic (in presence of oxygen) and anaerobic (in absence of oxygen) pathways contribute a certain amounts of energy during maximal exercise of various durations, and it is somewhat difficult to determine the major energy source in activities lasting from 2 to 4 minutes such as middle distance events. During this time, the aerobic and anaerobic energy sources are of equal importance. On the other hand at one extreme, maximal exercises of short duration are supplied via anaerobic metabolism, whereas exercises that can be performed for relatively long period of time such as marathon running are supplied primarily by aerobic metabolism.

6. Oxygen debt is defined as all post exercise oxygen consumption above the basal oxygen consumption level. This means that the oxygen taken in during recovery over and above that which would have normally been consumed for the same period of time during rest is used to provide energy for repaying the energy stores that were used up during exercise. Oxygen debt has been generally accepted as having two components: alactacid, for which no significant lactate increment is found, and lactacid, which is represented by proportional increments in blood lactate. The lactacid debt is generally attributed to the restoration of the ATP and CP stores in the muscles that were depleted during exercise, whereas the lactacid debt is more associated with the metabolic cost of converting lactic acid build-up back to energy.

7. Anaerobic threshold has been used as a non-invasive estimate of lactate threshold, and under most conditions the two occur at the same point at time during at incremental exercise bout, or at the same percentage of maximal oxygen uptake. As exercise intensity increases towards maximum, at some point ventilation increases disproportionately as compare to oxygen consumption. This point is called the ventilatory break point. Lactate threshold is the point at which blood lactate begins to rapidly accumulate above resting level during exercise. The onset of blood lactate accumulation (OBLA) is a standard value set at either 2 or 4 mmol lactate/liter of oxygen and is used as a common reference point.

8. The concept of an anaerobic threshold is a very attractive one because it offers a method of identifying the exercise intensity at which anaerobic metabolism makes a significant contribution to the provision of ATP. The anaerobic threshold concept also offers a submaximal method of assessing responses to training and also a way of describing the aerobic capacity of an individual in terms of percent of VO_2 max. The anaerobic threshold is also appealing because it may be more sensitive to training induced adaptation then VO_2 max alone.

9. VO_2 max is the maximum volume of oxygen that by the body can consume during intense,
whole-body exercise, while breathing air at sea level. By oxygen consumption we are indirectly measuring an individual’s maximal capacity to do work aerobically. If we measure a greater consumption of oxygen during exercise, we know that the working muscle is working at a higher intensity. To receive this oxygen and use it to make ATP for muscle contraction, our muscle fibers are absolutely dependent on two things: (1) an external delivery system to bring oxygen from the atmosphere to the working muscle cells, and (2) mitochondria to carry-out the process of aerobic energy transfer. Endurance athletes are characterized by both a very good cardiovascular system, and well developed oxidative capacity in their skeletal muscles.

10. In order to determine an athlete’s true maximal aerobic capacity, exercise conditions must be created that maximally stress the blood delivery capacity of the heart. A physical test that meets this requirement must employ at least 50 percent of the total muscle mass. Activities which meet this requirement include running, cycling, rowing, etc. The most common laboratory method is the treadmill running test. A motorized treadmill with variable speed and variable inclination is employed. High VO$_2$ max may be a prerequisite for performance in endurance events at the highest level, other markers such as lactate threshold are more predictive of performance. Again, the speed at lactate threshold is more significant than the actual value itself.

11. VO$_2$ max varies greatly between individuals and even between elite athletes that compete in the same sport. Heredity plays a major role in a person’s VO$_2$ max and heredity can account for up to 25 to 50 percent of the variance observed between individuals.

12. VO$_2$ max decreases with age. The average rate of decline is generally accepted to be about 1 percent per year or 10 percent per decade after the age of 25. Usually, the decline in age-related VO$_2$ max can be accounted for by a reduction in maximum heart rate, maximal stoke volume and maximal a-v O$_2$ difference, i.e. the difference between oxygen concentration in arterial blood and venous blood.

**Review Questions**

1. Define RQ. What is its significance?
2. What is metabolism? Define the term ‘Anabolism’ and ‘Catabolism’.
3. Why does anaerobic respiration produce less energy than aerobic respiration?
4. Why can red muscle fibers work for longer periods continuously? While white muscle fibers get easily fatigue?
5. Give schematic representation of an overall view of aerobic metabolism.
6. When and where does anaerobic respiration occur in men?
7. What is the other name of Krebs cycle? Where does it occur? Write its importance.
9. What is glycolysis? Explain the major steps of glycolysis. Where does this process occur in a cell?
10. What role does oxygen play in the process of aerobic metabolism?
11. What is the respiratory exchange ratio? Explain how it is used to determine the oxidation of carbohydrate and fat.
12. Define VO$_2$ max? What are the various field methods by which you can predict VO$_2$ max of an individual? Discuss the procedure of ‘Multistage Physical Fitness Test’.
13. What do you mean by anaerobic threshold? How can we use measurements of oxygen consumption to estimate one’s exercise efficiency?
14. Why do athletes with high VO$_2$ max values perform better in endurance events than those with lower values?
15. Give examples of interval training sessions that might be used to develop the ATP-PCr, glycolytic, and oxidative system for a runner.
All living organisms require food. The food like carbohydrates, proteins, and fats, minerals, vitamins, and water are required for various life processes. All these essential substances are collectively called nutrients. The process that involves ingestion and digestion of food materials and after that absorption and finally assimilation of absorbed food is known as nutrition.

The basic understanding of nutrition and its effects on health, weight control, and physical performance is essential for all people. An athlete’s performance may be improved with good, sound nutrition, while at the same time; it may deteriorate with poor nutritional practices.

**Definition:** “Nutrition is the combination of processes by which the living organism receives and utilizes the materials necessary for the maintenance of its functions and for the growth and the renewal of its components.”

**Importance of Nutrition**

i. To promote growth, repairing wear and tear of the damaged tissues and to gain energy in order to control the different metabolic processes are the main functions of nutrition.

ii. The potential energy stored within the food is transformed into usable energy, which is used for different physiological functions like movement, locomotion, excretion, reproduction, etc.

iii. The disease-resistant power of the living body is developed through nutrition.

iv. The future energy is produced from stored food matters (glycogen and fat) during shortage of food.

v. Nutrition plays a special role in production of heat energy in the body to meet the caloric demand of an individual.

**Nutrients:** The organic and inorganic materials, which the living organism collects from the nature to perform all the fundamental activities of the body, are called nutrients.

All nutrients that are collected by the living organisms from their surroundings are not considered as food. Nutrients do not require digestion. The essential substances like minerals, vitamins, and water are collectively called nutrients.
Food

*Definition:* By taking those edible substances, due to which growth, nutrition, yield of energy and building new tissue of the living body take place, are considered as food.

**CLASSIFICATION OF FOODS**

Food is divided into two types according to their functions such as- (i) primary or nutritive food, (ii) secondary or protective food.

**CLASSIFICATION AND FUNCTIONS OF CARBOHYDRATE, FAT AND PROTEIN**

*Primary or nutritive food:* The food substances which help in energy production, body growth, repair of wear and tear of damaged tissue, etc. are called primary or nutritive food. Carbohydrate, protein and fat are the components of nutritive food.

*a. Carbohydrates:* Carbohydrate is defined as a neutral organic compound made up of carbon, hydrogen and oxygen; the last two elements remain in the same proportion as in water (2:1).

Classification of carbohydrates: According to sugar unit carbohydrates are of the following types:

1) **Monosaccharides:** These carbohydrates contain only a single 6-carbon sugar molecule like glucose, fructose, galactose, etc.

   The most common or simplest monosaccharide is glucose, which can be oxidized and used directly by the body for energy or it may be broken down by the digestive system and converted into glycogen (a polysaccharide) and stored in the muscle and liver for later use.

   Once the storage capacity for glycogen has been reached in the muscles and liver, the excess glucose is converted into fat and stored in the fatty (adipose) tissue of the body. This means, therefore, that even if a person is on a high carbohydrate and low fat diet, it is still possible, if excessive amounts of calories are consumed for that person to increase his or her body fat level.

   In addition to glucose, there are two other monosaccharides fructose and galactose (same chemical make-up like glucose). In the final products of carbohydrates digestion, glucose makes up about 80 percent, while fructose and galactose make up approximately 10 percent each respectively.
Glucose and fructose are found mainly in fruits (especially dried), pastries, candy, jams and honey, whereas galactose is present primarily in the mammary glands of certain animals.

ii) Disaccharide: These carbohydrates contain two 6-carbon sugar molecules like maltose, sucrose and lactose, etc. Maltose is produced during digestion of starches, while lactose is found in milk and it eventually breaks down to galactose and glucose during digestion. Sucrose (or table sugar) is normally found in cane sugar.

iii) Polysaccharide: These carbohydrates contain three or more 6-carbon sugar molecules like starches. Starch is found mainly in potatoes, cereal and bread products such as rice, corn, wheat and barley along with beans and peas. Other polysaccharide products that are ingested to a slight extent in the diet are glycogen, dextrin, pectin and cellulose, all of which are digested in basically the same manner as the starches.

Sources: Carbohydrates are generally obtained from two sources such as:

i. Plant source: glucose- grapes; fructose- honey and all types of sweet fruits; lactose-milk; sucrose- jaggery, cane sugar; starch-potato, wheat, rice; cellulose-vegetables, etc.

ii. Animal source: milk, muscle, liver, etc.

Functions of carbohydrates:

i. Source of energy (calorific value): In the tissue when carbohydrates are oxidized completely then it produce energy. One gram of carbohydrates yields about 4.0 Kcal of energy.

ii. Storage: The chief form of carbohydrate is starch in plant and glycogen in liver and muscles of animal.

iii. Maintenance of blood sugar: Normally 100 ml human blood contains 80 to 120 mg of glucose. This blood glucose level is maintained by the stored glycogen in the liver and muscle.

iv. Synthesis of fats and proteins: By the process of metabolism, carbohydrates are converted into fats, proteins, etc. in the liver.

v. Evacuation of feces: Cellulose is not digested by man but it adds bulk to the stomach and intestinal contents, stimulate peristaltic movement of the intestine and thus helps in evacuation of feces. Thus it acts as roughage.

Daily requirement: In healthy adult human the daily requirement of carbohydrates is about 400 to 500 gm (i.e. 65 to 70% of total food).

Deficiency symptoms: Decrease of sugar in blood causes hypoglycemia and ketosis.

b) Proteins: Proteins, unlike carbohydrates and fats, contain nitrogen in addition to carbon, hydrogen and oxygen. Some protein may contain sulfur and phosphorus.

Definition: Proteins are polymers formed by monomeric units called amino acids. Every cell in the body needs protein. In fact, proteins are found throughout the body with muscle tissue being their major location. Proteins are not used to any great extent for energy, but instead, they are considered as “building blocks” of tissue.

Proteins are made up of long chain like nitrogenous compounds called amino acids. There are over twenty different known amino acids in protein, eight of these cannot be manufactured within the body, and therefore, must be obtained directly from the diet. These are referred to as essential amino acids. The remaining ones, called non-essential, can be synthesized within the body from the nutrients and in from the diet. The eight essential amino acids are: Isoleucine, Leucine, Lysine, Methionine, Phenyl alanine, Threonine, Tryptophan and Valine.

It is interesting to note that the animal protein sources contains all of the essential amino acids and therefore, are generally considered to be “high quality” proteins. On the other hand, protein from vegetables and other sources tend to be “low quality” protein because these foods are missing one or more of these essential amino acids.

While there is some disagreement as to how much protein is needed daily, most nutritionist do agree that no more than one gram of protein per kilogram of body weight is needed daily, In fact the United States Food and Nutrition Board has recommended a daily protein allowance of 0.9 grams per kilogram of body weight for adolescent and adult men and women. This means that a person who weighs 165 pounds (75 kilograms) would require, on a daily basis, approximately 75 grams of protein intake in order to meet the needs for tissue growth and maintenance. People who are heavier would naturally require a greater
amount. It has also been suggested that women who are pregnant should raise their daily protein allowances by as much as 10 grams, whereas young mothers who are nursing young babies should increase their daily intake to 20 grams. Sports person also need more protein as per their event/game.

**Classification of proteins:** Proteins may be classified in many ways. Generally it is classified as follows:

a. Simple Protein: Simple proteins are defined as those proteins, which are made up of only amino acids. Examples: albumin, globulin, histone, etc.

b. Conjugated Protein: The proteins, in which simple proteins remain combined with some non-protein substances, are known as conjugated proteins. Examples: nucleoprotein, phosphoprotein, lipoprotein, glycoprotein, etc.

c. Derived Protein: Those proteins obtained as intermediate products during hydrolysis (digestion) of simple or conjugated proteins are known as derived proteins. Examples: proteins, metaproteins, peptones, polypeptides, etc.

**Sources:** Proteins are obtained from both plant and animal sources such as-

i. Plant Source: wheat, rice, soyabean, pulses, different seeds etc.

ii. Animal source: milk, egg, fish, meat etc.

**Functions of protein:**

i. Calorific value: one gram of protein when completely oxidized yields about 4.1 Kcal of heat energy.

ii. Proteins help in body-structure, growth, repair of damaged tissues due to wear and tear, storage, etc.

iii. Synthetic functions: It helps in the synthesis of plasma proteins, hemoglobin, enzymes, hormones, milk proteins, etc.

iv. Synthesis of cell: Protein is the chief constituent of all parts of the living cell. Hence proteins are necessary to build them up.

**Daily requirement:** In healthy adult human the daily protein requirement is about 1 gm per kg body weight, in infant 3 to 4 gm per kg and in growing children 2 to 3 gm per kg body weight. Sports person required 1.5 to 2.0 gm protein per kilogram of body weight or even more for power events.

**Deficiency symptoms:**

i. Kwashiorkor: It is a severe protein deficiency disease generally observed in children less than one year. It is characterized by discolored hair and skin, bloated belly due to fluid imbalance and thin legs.

ii. Marasmus: It is a disease caused by deficiency of both protein and calories in infants. It is due to inadequate supply of food or poor absorption of digested food from the intestine. It is characterized by gradual wasting of tissues, wrinkled skin and sunken eyes.

c) Fats: Like carbohydrate and protein, fat is also an organic compound made up of carbon, hydrogen and oxygen in different amounts. While fats contain less oxygen than carbohydrates, they also have more carbon and hydrogen. This obviously allows them to be greater fuel providers, but at the same time, a greater cost in terms of oxidation.

**Definition:** Fats are ester of fatty acids with glycerol. The salts of alcohols (glycerol) with organic acids (fatty acid) are known as esters. Fatty acids are insoluble in water but soluble in fat solvents like chloroform, ether, alcohol, etc.

**Classification of fats:** Chemically, a fat molecule is made up of two different groups of atoms; namely, fatty acids and glycerol.

i) Fatty acids: These are higher aliphatic acids which are insoluble in water but soluble in fat solvent like chloroform, ether, benzene, etc. the commonest type of fatty acids occurring in the natural fats generally contain even number of carbon atoms and are straight chain derivatives. According to absence of or presence of double bonds in the chains, the fatty acids are saturated fatty acids and unsaturated fatty acids respectively.

a. Saturated fatty acids: Fats are considered to be saturated fatty acids if the carbon atom chain contains as many hydrogen atoms as it will hold. In other words, the single bond link between the carbon atoms is completely saturated with hydrogen atoms and will not hold any more hydrogen atom.

Examples: palmitic acid, butyric acid, stearic acid, etc.
b. Unsaturated fatty acids: Fats are classified as unsaturated fatty acids if two or more hydrogen atoms, because of the presence of a double bond link between the carbon atoms, are missing from the carbon atom chain. The carbon chain of unsaturated fats contains two less hydrogen atoms for each double bond. The fat molecule is referred to as mono-unsaturated if the carbon chain contains one double bond and polyunsaturated if two or more double bonds are present. Examples: linoleic acid, linolenic acid and arachidonic acid, etc.

ii) Glycerol: It is trihydric alcohol. It is clear, colorless, syrupy liquid of sweet taste. It is miscible with alcohol and water. It is obtained by the hydrolysis of fats.

Fats are stored in the body in the form of triglyceride (three fatty acids joined with glycerol). Triglycerides, also known as neutral fats, are the most common fats of the diet. Other fats found in the body are phospholipids and cholesterol, both of which play important roles in maintaining the membrane structure of all cells. Phospholipids also play an important role in blood clotting while cholesterol is needed for the production of both male and female hormones androgen, estrogen and progesterone.

While fat plays an essential part in our diet, it is generally agreed that most people of developed country (for example: Americans) consume far more fat than they really need. Today, fat makes up about 42 percent of the total average American diet, while most experts in the area of nutrition agree that approximately 20 to 25 percent is adequate.

Although this is a highly controversial subject, there is suggestive evidence that a diet high in saturated fats may be directly or indirectly related to cardiovascular disease. It has been suggested that in order to perhaps avoid this potential risk, a portion of saturated fats should be replaced with unsaturated fats. This has been especially advisable for people who are overweight, those with a history of heart disease (especially middle age people), those people who have sedentary type jobs, or people who hold highly stressful type jobs. Obviously, more research is needed in this area before any final recommendation can be made.

Sources: Fats are obtained from both plant and animal sources such as-

i. Plant Source: mustard oil, coconut oil, groundnut oil, etc. The vegetable oils such as- cottonseed oil, corn oil, peanut oil and soybean oil are good source of unsaturated fats.

ii. Animal source: milk, egg, fish, meat, etc. In addition cream, whole milk, ice cream, butter, margarine, egg-yolks, cheese, lobster and crabs are rich source of saturated fats.

Functions of fats:

i. Calorific value: Fat gives maximum amount of heat. One gram fat yields 9.0 Kcal of heat energy.

ii. Storage: Fats are easily stored for future use.

iii. Protection: The storage (depot) fat protects the vital organs and also acts as cushion and packing tissues.

iv. It supplies essential fatty acids and fat-soluble vitamins like A, D, E, K.

v. Regulation of body temperature: Fat acts as poor heat conductor. Thus subcutaneous fat helps to regulate body temperature.

Daily requirement: In adult the daily fat requirement is about 80 to 100 gm. (i.e. 20% of total food).

Classification and Functions of Vitamins and Minerals

Secondary or protective food: In addition to the organic substances (food) like carbohydrate, protein and fat, human being needs few other things for its growth and maintenance of its functional activities. Those substances are vitamins, inorganic salts or minerals and water, which are known as accessory or protective food.

a) Vitamins:

Definition: Vitamins are organic compounds present in variable minute quantity in natural food stuffs, which are required for the normal growth as well as maintenance of health and life.
Although vitamins are needed in only small amounts, they must be provided in either the diet or by way of supplements since the living cell cannot manufacture them.

**Classification of Vitamins:** Vitamins are generally classified as either water-soluble or fat-soluble.

i) **Water-soluble Vitamins:** The Vitamins, which are soluble in water, are called water-soluble vitamins. Such as-

a. *Vitamin B-complex:* The B-complex group includes thiamine (Vit-B₁), riboflavin (Vit-B₂), pantothenic acid (Vit-B₃), niacin (Vit-B₅), pyridoxine (Vit-B₆), biotin (Vit-B₇), folic acid (Vit-B₉), cyanocobalamine (Vit-B₁₂), choline, nicotinic acid, inositol, etc.

b. *Vitamin C* (ascorbic acid)

ii) **Fat-soluble Vitamins:** The vitamins, which are soluble in fat solvents like chloroform, benzene, etc. are called, fat soluble vitamins. Such as- Vitamin A (Retinol), D (Calciferol), E (Tocopherol) and K (Phylloquinone).

**Provitamins:** The Provitamins are the organic compounds from which the vitamins are synthesized in the animal body. Examples: Carotene—it is present in green and yellow vegetables and fruits. It produces vitamin A.

**Antivitamins:** The substances, which have got structural and chemical group similar to vitamins but without any physiological action or which destroy the vitamins or make the vitamins ineffective are called antivitamins. Examples: Phyrithiamine—Destroy the action of thiamine (Vit-B₁).

Table 8.1: Summary of vitamins sources, functions, deficiency symptoms, diseases and excess intake.

<table>
<thead>
<tr>
<th>Vitamins</th>
<th>Dietary Sources</th>
<th>Major bodily functions</th>
<th>Deficiency symptoms</th>
<th>Diseases</th>
<th>Excess</th>
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<tbody>
<tr>
<td><strong>Water-Soluble</strong></td>
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<tr>
<td>Thiamine (B₁)</td>
<td>Pork, organ meats, whole grains, nuts, legumes, milk, fruits and vegetables</td>
<td>Coenzyme (thiamin phosphate) in reactions involving the removal of carbon dioxide.</td>
<td>(i) Degeneration of peripheral nerves resulting in paralysis, edema and heart failure</td>
<td>Beriberi</td>
<td>None reported</td>
</tr>
<tr>
<td>Riboflavin (B₂)</td>
<td>Widely distributed in foods: meats, eggs, milk products, whole grain and enriched cereal products, wheat germ, green leafy vegetables</td>
<td>Constituent of two flavin nucleotide coenzymes involved in energy metabolism (FAD and FMN).</td>
<td>(i) Fissures at the angles of the mouth. (ii) Reddened lips (iii) Cracks at mouth corner (iv) Dry chaffed skin (v) Eye lesions</td>
<td>(i) Cheilosis (ii) Dermatitis</td>
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<tr>
<td>Pantothenic Acid</td>
<td>Widely distributed in foods: meat, fish, poultry, milk products, legumes, and whole grains</td>
<td>Constituent of coenzyme A, which plays a central role in energy metabolism.</td>
<td>(i) Roughness of skin (ii) Fatigue (iii) Sleep disturbances (iv) Impaired coordination (v) Nausea</td>
<td>Dermatitis</td>
<td>None reported</td>
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<thead>
<tr>
<th>Vitamins</th>
<th>Dietary Sources</th>
<th>Major bodily functions</th>
<th>Deficiency symptoms</th>
<th>Diseases</th>
<th>Excess</th>
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<tbody>
<tr>
<td>Niacin (B₃)</td>
<td>Liver, lean meats, poultry, grains, legumes, peanuts, etc</td>
<td>Constituent of two coenzymes in oxidation-reduction reactions (NAD and NADP)</td>
<td>(i) Skin lesion</td>
<td>Pellagra</td>
<td>Flushing, burning and tingling around neck, face and hands</td>
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<td>(ii) Gastrointestinal lesion.</td>
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<td></td>
<td>(iii) Nervous mental disorders.</td>
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<tr>
<td>Pyridoxine (B₆)</td>
<td>Meats, fish, poultry, vegetables, whole grains, cereals, seeds, etc</td>
<td>Coenzyme (pyridoxal phosphate) involved in amino acid and glycogen metabolism.</td>
<td>(i) Roughness of skin.</td>
<td>(i) Dermatitis</td>
<td>None reported</td>
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<td></td>
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<td>(ii) Irritability</td>
<td>(ii) Acrodyonia</td>
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<td></td>
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<td>(iii) Convulsions</td>
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<td>(iv) Muscular twitching</td>
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<td>(v) Kidney stones</td>
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<td>(i) Production of abnormal, large red blood cells</td>
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<td></td>
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<td>(ii) Gastrointestinal disturbances</td>
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<td>(iii) Diarrhea</td>
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<td>(iv) Red tongue</td>
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<tr>
<td>Folic acid (B₉)</td>
<td>Legumes, green vegetables, whole-wheat products, meats, eggs, milk products, liver</td>
<td>Coenzyme (reduced form) involved in transfer of single-carbon units in nucleic acid and amino acid metabolism.</td>
<td>(i) Decrease of red blood cell</td>
<td>Macrocyclic anemia</td>
<td>None reported</td>
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<td></td>
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<td>(ii) Neurologic disorders</td>
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<td>(iii) Convulsions</td>
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<td></td>
<td></td>
<td></td>
<td>(iv) Convulsions</td>
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<tr>
<td>Cyanocobalamine (B₁₂)</td>
<td>Muscle meats, fish, eggs, dairy products.</td>
<td>Coenzyme involved in transfer of single-carbon units in nucleic acid and amino acid metabolism.</td>
<td>(i) Decrease of red blood cell</td>
<td>Pernicious anemia</td>
<td>None reported</td>
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<tr>
<td></td>
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<td></td>
<td>(ii) Neurologic disorders</td>
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<td></td>
<td></td>
<td></td>
<td>(iii) Convulsions</td>
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<tr>
<td>Biotin</td>
<td>Legumes, vegetables, meats, liver, egg yolk, nuts, etc</td>
<td>Coenzyme required for fat synthesis, amino acid metabolism, and glycogen (animal starch) formation.</td>
<td>(i) Fatigue</td>
<td>Dermatitis</td>
<td>None reported</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>(ii) Depression</td>
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<td>(iii) Nausea</td>
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<td>(iv) Muscular pain</td>
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<td></td>
<td>(v) Roughness of skin</td>
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<tr>
<td>Vitamin C</td>
<td>Citrus fruits, tomatoes, green peppers, salad greens.</td>
<td>(i) Maintains intercellular matrix of cartilage, bone and dentine</td>
<td>(i) Spongy and bleeding gum</td>
<td>Scurvy</td>
<td>Relatively nontoxic and possibility of kidney stones</td>
</tr>
<tr>
<td>(Ascorbic acid)</td>
<td></td>
<td>(ii) Important in collagen synthesis</td>
<td>(ii) Hemorrhage under skin and mucous membrane</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(iii) Degeneration of skin, teeth and blood vessels</td>
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</tr>
<tr>
<td>Fat-Soluble</td>
<td>Provitamin A (β-carotene) widely distributed in green vegetables Retinol present in milk, butter, cheese, fortified margarine</td>
<td>(i) Constituent of rhodopsin (visual pigment). (ii) Maintenance of epithelial tissue (iii) Role in mucopolysaccharide synthesis</td>
<td>(i) Dry scaly eye. (ii) Inability to see in dim light (iii) Keratinization of ocular tissue</td>
<td>(i) Xerophthalmia</td>
<td>Headache, vomiting, peeling of skin, anorexia, swelling of long bones</td>
</tr>
<tr>
<td>Vitamin A</td>
<td></td>
<td></td>
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<td>(ii) Nightblindness</td>
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<tr>
<td>(Retinol)</td>
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<td>(iii) Permanent blindness</td>
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<tr>
<th>Vitamin</th>
<th>Dietary Sources</th>
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<th>Diseases</th>
<th>Excess</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin D</td>
<td>Cod-liver oil, eggs, dairy products, fortified milk, margarine, UV-ray of sun light</td>
<td>(i) Promotes growth and mineralization of bones (ii) Increase absorption of calcium</td>
<td>(i) Softening and bending of leg bones (ii) Decay of bone and teeth (iii) Bone deformities</td>
<td>(i) Rickets (in children) (ii) Osteomalacia (in adult)</td>
<td>Vomiting, diarrhea, loss of weight, kidney damage</td>
</tr>
<tr>
<td>Vitamin E</td>
<td>Seeds, green leafy vegetables, margarines, seeds oil</td>
<td>Function as an antioxidant to prevent cell damage</td>
<td>(i) Weakness (ii) Nausea</td>
<td>Possible anemia</td>
<td>Relatively nontoxic</td>
</tr>
<tr>
<td>Vitamin K</td>
<td>Green leafy vegetables, small amounts in cereals, fruits and meats</td>
<td>Important in blood clotting (involved in formation of prothrombin)</td>
<td>(i) Severe bleeding (ii) Internal hemorrhage</td>
<td>Defective coagulation of blood.</td>
<td>Relatively nontoxic. Synthetic forms at high doses may cause jaundice</td>
</tr>
</tbody>
</table>

**Hyper-vitaminosis:** A condition due to the administration of excess amount of a vitamin is termed as hyper-vitaminosis. Excess amount of water-soluble vitamins when administered in the body are excreted through urine. Thus they cause no toxic effects. But fat-soluble vitamins are more prone to produce toxic symptoms, as their concentration level rises steadily in the body.

Daily ingestion of a moderate to large amount of vitamin A and D eventually can have serious toxic effects. In young children, excessive vitamin A intake causes irritability, swelling of the bones, weight loss and dry, itchy skin. In adults, symptoms can include nausea, headache, drowsiness, loss of hair, diarrhea and loss of calcium from bones, causing brittleness. Discontinuing high intakes of Vitamin-A reverses these symptoms. Kidney damage can result from a regular excess in take of vitamin D.

**Antioxidant Role of Specific Vitamins**

Most of the oxygen consumed during energy metabolism in the mitochondria combines with hydrogen to produce water. Normally, however, approximately 2 to 5 percent of this oxygen will form oxygen containing free radicals such as super oxide (\(O_2^-\)), hydrogen peroxide (\(H_2O_2\)) and hydroxyl (\(OH^-\)) radicals due to electron “leakage” at various steps in the electron transports chain.

When super oxide forms, for example, it dismutates to hydrogen peroxide, normally, super oxide is rapidly converted to \(O_2\) and \(H_2O\) by the enzyme super oxide dismutase. An accumulation of free radicals increases the potential for cellular damage, or oxidative stress, to many biologically important substances. In fact the major effect of oxygen radicals is their affinity to the poly-unsaturated fatty acids that make-up the lipid by layer of the cell membrane. During unchecked oxidative stress, there is a deterioration of the fatty acids in the plasma membrane, which becomes damaged through a chain reaction series of events termed lipid peroxidation. Free radicals also can facilitate the oxidation of low density lipoprotein (LDL) cholesterol, which accelerate the process of atherosclerosis. Oxidative stress ultimately increases the likely hood of cellular deterioration associated with advanced aging as well as cancer, diabetes and coronary artery disease.

Although there is no way to stop oxygen reduction and the production of free radicals, an elaborate natural defense against their damaging...
effects exists within the cytosol and mitochondria of the cell and its surrounding extra cellular space. This defense includes the scavenger enzymes such as catalase, glutathione peroxidase, superoxide dismutase and metal binding proteins. In addition, nutritive-reducing agents such as vitamins A, C, E and the vitamin A-precursor, β-carotene, serve important protective functions. Maintaining a diet that provides appropriate levels of the antioxidant vitamins, especially vitamin C and β-carotene, is linked to a reduced risk of several types of cancers, whereas normal to above normal intake of vitamin E and β-carotene may decrease the heart attack risk.

Exercise and Antioxidant

Although the beneficial effects of physical activity are well known, the potential for possible negative effects is currently being reviewed in the literature. This potential is based on the reasoning that the elevated aerobic metabolism in exercise increases the production of free radicals. Free radical can be produced during exercise in at least two ways. The first is via an electron leak in the mitochondria and the second is during alterations in blood flow and oxygen supply—under perfusion often occurs during intense exercise and is then followed by reperfusion in the recovery period. With exercise, the risk seems to depend on intensity and the individual’s state of training, because exhaustive exercise by untrained individuals is more likely to produce oxidative damage in the active muscle. Now the question is whether physically active individuals are more prone to free-radical damage or whether protective agents with antioxidant properties are required in increased quantities in physically active individuals.

It has been suggested that for well nourished humans, the natural defenses of the body are adequate for its protection. Although a single bout of submaximal exercise increased oxidant production, the natural antioxidant defenses coped effectively. Even when repeated bouts of exercise were performed on consecutive days, various indices of oxidative stress indicated that the body’s antioxidant system was not depleted. However, vitamin E and vitamin C are the strong antioxidant and supplementation of these vitamins, the free radical production is reduced dramatically as reported by many researchers.

Supplementation of Vitamins

Vitamin supplements can reverse the symptoms of vitamin deficiency, once a deficiency is cured, supplements do not further improve a normal status. However, researches have not supported the use of vitamin supplements to improve exercise performance or training performance in nutritionally adequate healthy people. When vitamin intake is at recommended levels, supplements neither improve exercise performance nor necessarily increase the blood levels of these nutrients.

VITAMINS AND EXERCISE PERFORMANCE

Many vitamins serve as coenzyme components or precursors of coenzymes that regulate energy metabolism. Many coaches, athletes, fitness enthusiasts to advocate the use of vitamin supplements. But this approach is simply not supported by research findings. However, B-vitamins play key roles as coenzymes in important energy yielding reactions during carbohydrate, lipid and protein catabolism.

Supplementing the diet with vitamin B₆ an essential co-factor in glycogen and amino acid metabolism, is of no benefit to the mixture metabolized by men/women during intense aerobic exercise. No exercise benefit has been reported for vitamins other than the B-complex group such as vitamin C and E.

It has never been firmly established that a deficiency state for vitamin E exists for normal individuals or that vitamin E supplements above the RDA level are beneficial to stamina, circulatory function, or energy metabolism. Chronic high potency multivitamin-mineral supplementation for well nourished healthy individuals was of no benefit on measures of aerobic fitness, muscular strength and athletic performance.
Mega Vitamins

Although physically active individuals who eat a well balanced diet do not need to take additional vitamins, most scientists believed that there is little harm in taking a multivitamin capsule containing the recommended allowance of each vitamin. It is of great concern, however, that some athletes resort to taking megavitamins, or doses at least ten fold and up to 1000 times the RDA, in hopes that “supercharging” with vitamins will improve exercise performance. Such a practice can be harmful, except in case of specific serious medical illness.

A mega dose of water soluble vitamin C, for example, can raise serum uric acid levels and precipitate gout in people predisposed to this disease. Also some American blacks, Asian and Sephardic Jews have a genetic metabolic deficiency in which vitamin C can lead to hemolytic anemia. In individuals who are iron-deficient, mega doses of vitamin C may destroy significant amount of vitamin B₁₂. In healthy individuals, vitamin C supplements frequently irritate the bowel and cause diarrhea.

Excess vitamin B₆ may produce liver disease and nerve damage. Excess vitamin B₂ can impair vision, whereas mega dose of nicotinic acid inhibits the uptake of fatty acids by cardiac muscle during exercise. Possible side effects of vitamin E mega dose include headache, fatigue, blurred vision, gastrointestinal disturbances, muscular weakness, and low blood sugar. The toxicity to the nervous system of mega doses of vitamin A and the damaging effects to the kidneys of excess vitamin D have been well demonstrated.

Minerals

Minerals are defined as those elements which remain largely as ash when plant or animal tissues are burned. The mineral salts are very much

Fig. 8.3: Role of water soluble vitamins in the metabolism of carbohydrates lipids, and proteins.
(Adapted from Katch, Katch and Mcardle)
essential for our life although they give no energy. The body contains more than 19 minerals all of which must be derived from foods. About 4% of the body weight is made up of minerals.

Classification of minerals: Minerals fall broadly under two groups such as-

a. Macro minerals: Macro minerals are those elements needed in relatively large amounts (around 100 mg per day). Examples: sodium, potassium, calcium, magnesium, phosphorus, chloride, sulfur.

b. Micro minerals: Micro minerals are those elements, which are required in much smaller amounts (less than 2–5 mg per day). Examples: iodine, iron, copper, fluorine, zinc, etc.

Role of Minerals in the Body

Various minerals that help in catabolic and anabolic cellular process. They are important in activating the numerous reactions that release energy during the breakdown of fat, protein and carbohydrate.

### Table 8.2: Summary of minerals sources, functions and deficiency symptoms and excess intake.

<table>
<thead>
<tr>
<th>Minerals</th>
<th>Dietary Sources</th>
<th>Major bodily functions</th>
<th>Deficiency symptoms</th>
<th>Excess intake</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calcium</td>
<td>Milk, cheese, dark green vegetables, dried legumes, etc.</td>
<td>(i) Bone and teeth formation (ii) Nerve transmission (iii) Blood clotting</td>
<td>(i) Muscular cramps and convulsions (ii) Delays blood coagulation (iii) Stunted growth (iv) Osteoporosis (v) May cause rickets</td>
<td>Not reported in humans</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>Milk, cheese, yogurt, meat, poultry, grains, fish, etc.</td>
<td>(i) Bone and teeth formation (ii) Essential part of cell membrane (iii) Loss of calcium</td>
<td>(i) Weakness (ii) Demineralization (iii) Ostomalacia</td>
<td>Erosion of jaw (phossy jaw)</td>
</tr>
<tr>
<td>Potassium</td>
<td>Leafy vegetables, cantelope, lima beans, potatoes, bananas, milk, meats, coffee, tea</td>
<td>(i) Fluid balance (ii) Nerve transmission (iii) Acid-base balance</td>
<td>(i) Muscle cramps, (ii) Irregular cardiac rhythm (iii) Mental confusion (iv) Loss of appetite (v) Can be life threatening</td>
<td>None if kidneys function normally, But poor kidney function causes potassium buildup and cardiac arrhythmias</td>
</tr>
<tr>
<td>Sulfur</td>
<td>Meat, eggs, dairy products, nuts, legumes</td>
<td>(i) Acid-base balance (ii) Liver function</td>
<td>Unlikely to occur if dietary intake is adequate</td>
<td>Unknown</td>
</tr>
<tr>
<td>Sodium</td>
<td>Common table salt, dairy products, meat, eggs, vegetables</td>
<td>(i) Acid-base balance (ii) Body water balance (iii) Nerve function</td>
<td>(i) Muscle cramps, (ii) Mental apathy (iii) Reduced appetite</td>
<td>High blood pressure</td>
</tr>
<tr>
<td>Chlorine</td>
<td>Chlorides are part of salt containing food, table salt (NaCl). Some vegetables and fruits</td>
<td>Important part of extra cellular fluids</td>
<td>Unlikely to occur if dietary intake is adequate</td>
<td>Along with sodium contributes to high blood pressure</td>
</tr>
<tr>
<td>Magnesium</td>
<td>Whole grains, green leafy vegetables</td>
<td>Activates enzymes involved in protein synthesis</td>
<td>(i) Growth failure (ii) Behavioral disturbances</td>
<td>Diarrhea</td>
</tr>
</tbody>
</table>

Contd...
**Minerals and Exercise Performance**

There is no evidence that supplementation benefit of exercise performance for normal individuals receiving the RDA of minerals. An important consequence of prolonged exercise, especially in hot weather, is the loss of water and minerals salts, primarily sodium and some potassium chloride in sweating. Excessive water and electrolyte losses impair heat tolerance and exercise performance and can lead to severe dysfunction in the form of heat cramps, heat exhaustion, or heat stroke.

Vigorous exercise triggers a rapid and co-ordinated release of the hormones vasopressin, rennin, and aldosterone, which reduce sodium and water loss through kidneys. Sodium conservation by the kidneys is increased, even under extreme conditions such as marathon running in warm weather, during which sweat output may be as much as 2 L per hour. Any electrolytes that are lost usually can be replenished by adding a slight amount of salt to the fluid ingested or to the normal daily food intake.

Research findings indicated that ingested so called ‘Athletic Drinks’ is of no special benefit in replacing the minerals lost through sweating.

<table>
<thead>
<tr>
<th>Minerals</th>
<th>Dietary Sources</th>
<th>Major bodily functions</th>
<th>Deficiency symptoms</th>
<th>Excess</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iron (Fe) Eggs, lean meats, legumes, whole grains, green leafy vegetables, fruits</td>
<td>(i) Constituent of hemoglobin (ii) Enzymes involved in energy metabolism.</td>
<td>(i) Weakness (ii) Reduced resistance to infection</td>
<td>(i) Siderosis (ii) Cirrhosis of liver</td>
<td></td>
</tr>
<tr>
<td>Fluorine (F) Drinking water, tea and seafood</td>
<td>May be important in maintenance of bone structure</td>
<td>Higher frequency of tooth decay.</td>
<td>(i) Mottling of teeth (ii) Increased bone density</td>
<td></td>
</tr>
<tr>
<td>Zinc (Zn) Widely distributed in foods: liver, fish, shellfish and many other foods</td>
<td>Constituent of enzymes involved in digestion</td>
<td>(i) Growth failure (ii) Small sex glands</td>
<td>(i) Fever (ii) Nausea (iii) Vomiting (iv) Diarrhea</td>
<td></td>
</tr>
<tr>
<td>Copper (Cu) Meats, drinking water, etc.</td>
<td>Constituent of enzymes associated with iron metabolism.</td>
<td>(i) Anemia (ii) Bone change (rare in humans)</td>
<td>Rare metabolic condition (Wilson’s disease)</td>
<td></td>
</tr>
<tr>
<td>Selenium (Se) Seafood, meats, grains</td>
<td>Functions in close association with vitamin E</td>
<td>Anemia (rare)</td>
<td>(i) Gastrointestinal disorders (ii) Lung irritations</td>
<td></td>
</tr>
<tr>
<td>Iodine (I) Marine fish and shellfish, dairy products, vegetables, iodized salt</td>
<td>Constituent of thyroid hormones</td>
<td>Goiter (enlarged thyroid)</td>
<td>Very high intakes depress thyroid activity</td>
<td></td>
</tr>
<tr>
<td>Chromium (Cr) Legumes, cereals, organ meats</td>
<td>Constituent of some enzymes</td>
<td>Not reported in humans</td>
<td>Inhibition of enzymes</td>
<td></td>
</tr>
</tbody>
</table>

Minerals also form important constituents of hormones. Along with these minerals serve three broad roles in the body which are as follows:

i. They provide structure in the formation of bones and teeth.

ii. In terms of function, they are intimately involved in maintaining normal heart rhythm, muscular contractility, neural conductivity and the acid-base balance of the body.

iii. They play crucial roles in the regulation of cellular metabolism by serving as important parts of enzymes and hormones that modulate cellular activity.
compared with ingesting a well balance diet. For fluid losses in excess of 4 or 5 kg and for prolonged activity in the heat, salt supplements may be necessary and can be achieved with a 0.1 to 0.2 percent salt solution by adding approximately 0.3 tsp of table salt per liter of water.

Strenuous exercise may place a drain on the body’s content of the trace elements. While such trace minerals losses do not necessarily mean that athletes should supplement these micronutrients, it is possible that for men and women with marginal micronutrient intakes any further loss with strenuous exercise needs to be replaced to prevent an over deficiency. Because iron, zinc and copper are highly interactive with each other and compete for the same carrier during intestinal absorption, an excessive intake of one mineral may cause a deficiency in the other.

FOOD REQUIREMENTS

The amount of food necessary each day depends upon a person’s energy needs. These energy needs are directly related to:

i. Periods of rapid growth

ii. Age

iii. Physical activity

During the rapid growing years (12–22 years for boys and 12–18 years for girls), there is a gradual increase in the minimal daily food requirements.

The percent contributes toward the total caloric intake for each of the three foodstuffs are:

| Protein | 14–15 % |
| Fat     | 29–30%  |
| Carbohydrate | 55–56% |

For example, athletes requiring 5000 kcal per day could have their diet divided as follows:

| Protein | 700–750 kcal |
| Fat     | 1450–1500 kcal |
| Carbohydrate | 2750–2800 kcal |

As we become older, our daily energy needs decreases. The following diagram (Fig. 8.4) represent the relationship of age and energy requirements.

BALANCED DIETS

A diet is considered “balanced” when all food groups (six) are represented in appropriate proportions or percentages. This means following the recommended daily allowances for micronutrients and for macronutrients—fats, proteins and carbohydrates. The six groups of foods are fats, proteins, carbohydrates, vitamins, minerals and water.

Nutrition and balanced diets are often difficult to understand, let alone practice. Yet, they are so important to the success. For example: a sedentary young male needs 1gm protein per kilogram of the body weight, 20–25 percent of fat of total calorie and approximately 65 percent of carbohydrate along with other three, i.e. vitamins, minerals and water. If he takes the above proportion are in his daily diet then only to be considered the diet is “balanced”.

Table 8.3: Recommended daily dietary allowances established by the national academy of sciences

<table>
<thead>
<tr>
<th>Gender</th>
<th>Age (years)</th>
<th>Weight (lb)</th>
<th>Weight (kg)</th>
<th>Height (inches)</th>
<th>Height (cm)</th>
<th>Kilo-Calories</th>
<th>Kcal/lb</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td>10–12</td>
<td>77</td>
<td>35.0</td>
<td>55</td>
<td>139.7</td>
<td>2,500</td>
<td>33</td>
</tr>
<tr>
<td></td>
<td>12–14</td>
<td>95</td>
<td>43.1</td>
<td>59</td>
<td>149.9</td>
<td>2,700</td>
<td>28</td>
</tr>
<tr>
<td></td>
<td>14–18</td>
<td>30</td>
<td>59.0</td>
<td>67</td>
<td>170.2</td>
<td>3,000</td>
<td>23</td>
</tr>
<tr>
<td></td>
<td>18–22</td>
<td>147</td>
<td>66.7</td>
<td>69</td>
<td>175.3</td>
<td>2,800</td>
<td>19</td>
</tr>
<tr>
<td>Females</td>
<td>10–12</td>
<td>77</td>
<td>35.0</td>
<td>56</td>
<td>142.2</td>
<td>2,250</td>
<td>29</td>
</tr>
<tr>
<td></td>
<td>12–14</td>
<td>97</td>
<td>44.0</td>
<td>61</td>
<td>154.9</td>
<td>2,300</td>
<td>24</td>
</tr>
<tr>
<td></td>
<td>14–16</td>
<td>114</td>
<td>51.8</td>
<td>62</td>
<td>157.5</td>
<td>2,400</td>
<td>21</td>
</tr>
<tr>
<td></td>
<td>16–18</td>
<td>119</td>
<td>54.0</td>
<td>63</td>
<td>160.0</td>
<td>2,300</td>
<td>19</td>
</tr>
<tr>
<td></td>
<td>18–22</td>
<td>128</td>
<td>58.1</td>
<td>64</td>
<td>162.6</td>
<td>2,000</td>
<td>16</td>
</tr>
</tbody>
</table>

Unfortunately, it’s very hard to find dieting programs that center on balanced diets. That’s because most programs aim to lose weight fast. As consumers of weight loss products, we all want fast results. But fast results come at the expense of nutrition and balanced diets.

**COMPUTATION OF CALORIE AND NUTRIENT REQUIREMENTS**

Before computation of calorie and nutrient requirements, the daily activity to be recorded for the individual. The daily activity and calorie requirement per kilogram of body weight per hour given/classified are as follows:

i. Very light activity (quite sitting, reading, watching TV etc.) — 1.5–2.0 Kcal/kg/hr.

ii. Light activity (Eating, bathing, Dressing, playing Music, etc.) — 2.0–3.0 Kcal/kg/hr.

iii. Moderate activity (Walking, gardening, jogging etc.) — 3.0–3.5 Kcal/kg/hr.

iv. Heavy activity (Swimming, Porters, running, etc.) — 3.5–4.5 Kcal/kg/hr.

v. Exhaustive activity (Weight lifting, Long distance running etc.) — 4.5 and more Kcal/kg/hr.

For example:

Subject: Adult male
Age: 25 years
Weight: 65 kg
B.M.R: 60 Cal/hr

I. **Calorie requirement**

   Activities:
   
   i. Sleep for 8 hours @ 90% of BMR = 60 × 0.9 × 8 = 432 Cal
   
   ii. Non occupational activities:

   a. Eating, shaving, bathing and dressing @ 2 Kcal/kg/hr for 1.5 hrs = 2 × 65 × 1.5 = 195 Cal

   b. Quiet sitting or standing @ 1.5 Cal/kg/hr for 1 hr = 1.5 × 65 × 1 = 98 Cal

   c. Writing, card-playing, gossiping and playing musical instruments @ 1.5 Cal/kg/hr for 5.5 hrs = 1.5 × 65 × 5.5 = 537 Cal

   d. Walking @ 3 Cal/kg/hr for 1 hr = 3 × 65 × 1 = 195 Cal

   iii. Occupational work @ 3.5 Cal/kg/hr for 7 hr = 3.5 × 65 × 7.0 = 1593 Cal

   Total amount of calorie = 3050 Cal

   Ten percent allowance for SDA = 305 Cal

   Total daily calorie requirement = 3355 Cal

II. **Nutrient Requirement**

<table>
<thead>
<tr>
<th>Nutrients</th>
<th>Amounts</th>
<th>Cal</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Proteins @1g/kg</td>
<td>65 × 1 = 65 gm</td>
<td>267</td>
</tr>
<tr>
<td>(1g protein ≡ 4.1 Cal)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Fats @20% of total Cal</td>
<td>(3355 × 0.20)/9</td>
<td>671</td>
</tr>
<tr>
<td>(1g fat ≡ 9 Cal)</td>
<td>= 75 gm</td>
<td></td>
</tr>
<tr>
<td>3. Carbohydrate for the balance of Calorie</td>
<td>[3355 – (267 + 671)]/4 = 604 gm</td>
<td>2417</td>
</tr>
<tr>
<td>(1g carbohydrate ≡ 4 Cal)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**BODY WATER AND WATER BALANCE**

**Water**

Water is one of the most important food for living organisms. The human body contains water (60%), proteins (20%), lipids (10%), minerals (7%) and carbohydrates (3%). The total water is distributed throughout the body as in two main compartments, intracellular (55%), i.e. the fluid within the cells and extra cellular (45%), i.e. the fluid outside the cells. The extra cellular water includes 3 liters plasma and about 11 liters interstitial fluid and lymph. Although water makes up 40 to 60 percent of the total body weight, which is like vitamins and minerals, which is not classified as an energy nutrient.
Sources:

i. Exogenous water: From food and drink.
ii. Endogenous water: From the end product of metabolism.

Functions: Water plays several important roles which are given below:

i. It is an essential constituent of living cell.
ii. It acts as an important medium for osmosis, diffusion, filtration, etc. of the various physical processes.
iii. Body temperature is regulated by water.
iv. Water acts as lubricating agent by which it prevents friction and drying.
v. It helps in the process of absorption from intestine, reabsorption from kidney tubules, synthesis of digestive juices, transport of hormones, etc.
vi. Water helps in making the food bolus.
vii. It helps in dilution of food for activity of enzyme.

Water Intake: A fairly sedentary adult in a normal environment requires about 2.5 L of water each day. For an active person in a warm environment the water requirement often increases to between 5–10 L daily. Three sources provide this water: (1) liquids, (2) foods, and (3) metabolic processes.

i. Water from liquids: The average individual normally consumes 1200 ml or 41 oz of water each day. Of course, during exercise and thermal stress, fluid intake can increase five or six times above normal. However, with proper fluid ingestion, including salt supplements, the actual body weight loss amounted to only 1.4 kg. Fluid loss and replenishment represented between 3.5 and 4 gallons of liquid.

ii. Water in foods: Most foods, particularly fruits and vegetables, contain considerable amount of water (e.g. lettuce, watermelon, cucumber and cantaloupe, pickles, green beans and broccoli, etc.); in contrast, butter, oils, dried meats and chocolate, cookies and cakes etc. have a relatively low water content.

iii. Metabolic water: Water and carbon dioxide form when food molecules become catabolized for energy. This water, termed metabolic water, provides about 25 percent of the daily water requirement of a sedentary
person. The complete breakdown of 100 g of carbohydrate, protein and fat produces 55g, 100g and 107g of metabolic water, respectively. Additionally, each gram of glycogen joins with 2.7g of water as its glucose unit’s link together; subsequently, glycogen liberates this water during its catabolism for energy.

**Water Output:** Water loss from the body occurs in urine, through the skin, as water vapor in expired air, and in feces.

i. **Water loss in urine:** Under normal conditions the kidneys reabsorb about 99 percent of the 140 to 160 L of filtrate formed each day; consequently, the volume of urine excreted daily by the kidneys ranges from 1000 to 1500 ml or about 1.5 quarts.

   Elimination of 1g of solute by the kidneys requires about 15 ml of water. Thus, a person need of water in urine becomes “obligated” to rid the body of metabolic byproducts like urea, an end product of protein breakdown. Using large quantities of protein for energy (as occurs with a high protein diet) actually accelerates the body’s dehydration during exercise.

ii. **Water loss through the skin:** A small quantity of water (perhaps 350 ml), termed *insensible perspiration*, continually seeps from the deeper tissues through the skin to the body’s surface.

   Water loss through the skin also occurs as sweat produced by specialized sweat glands beneath the skin. Evaporation of sweat’s water component provides the refrigerator mechanism to cool the body. Daily sweat rate under normal conditions amounts to between 500 and 700 ml. This by no means reflects sweating capacity; a well-acclimatized person can produce up to 12 L of sweat (equivalent of 10 to 12 kg) at a rate of 1 L per hour during prolonged exercise in a hot environment.

iii. **Water loss in feces:** Intestinal elimination produces between 100 and 200 ml of water loss because water constitutes approximately 70 percent of fecal matter. The remainder comprises nondigestible material including bacteria from the digestive process, and the residues of digestive juices from the intestine, stomach and pancreas. With diarrhea or vomiting, water loss increases in between 1500 and 5000 ml.
Physical activity and environmental factors play an important role: The loss of body water represents the most serious consequence of profuse sweating. The severity of physical activity, environmental temperature and humidity determine the amount of water lost through sweating. Relative humidity (water content of the ambient air) affects the efficiency of the sweating mechanism in temperature regulation. The air becomes completely saturated with water vapor at 100 percent relative humidity. This blocks any evaporation of fluid from the skin surface to the air, thus minimizing this important avenue for body cooling. On a dry day, however, the air can hold considerable moisture, and fluid rapidly evaporates from the skin. Thus, the sweat mechanism functions at optimal efficiency and body temperature remains regulated. Interestingly, a decrease in plasma volume occurs when sweating causes a fluid loss equal to 2 or 3 percent of body mass. Fluid loss places a significant strain on circulatory function, which ultimately impairs exercise capacity and thermoregulation. Monitoring changes in body weight provides a convenient method for assessing fluid loss during exercise and or heat stress. Each 0.45 kg (1 lb) of body weight loss corresponds to 450 ml (15 oz) of dehydration.

**Water Requirement in Exercise**

The most serious consequence of profuse sweating is the loss of body water. The amount of water loss through sweating depends on the severity of the physical activity and environmental temperature. The relative humidity is also an important factor affecting the efficiency of the sweating mechanism in temperature regulation. Staying hydrated is particularly important during exercise. Adequate fluid intake is essential to comfort, performance and safety. The longer and more intense exercise, the more important it is to drink the right kind of fluids. Studies have found that athletes who lose as little as two percent of their body weight through blood volume may also lead to muscle cramps, dizziness, fatigue and heat illness.

There is wide variability in sweat rates, losses and hydration levels of individuals, it is nearly impossible to provide specific recommendation or guidelines about the type or amount of fluids athletes should consume. Finding the right amount of fluid to drink depends upon a variety of individual factors including the length and intensity of exercise and other individual differences.

**Recommended Oral Rehydration Solution**

The ideal oral rehydration solution contains a carbohydrate concentration of between 5 and 8 percent. Oral rehydration solutions within this range generally permit carbohydrate replenishment with out hindering water uptake, fluid homeostasis, and temperature regulation compare to ingesting plain water during prolonged exercise in the heat. If the duration of the intense aerobic effort is relatively short (less than one hour) and the thermal stress is high, fluid replenishment is of utmost importance to health and safety, and it is advisable to consume a diluted carbohydrate-electrolyte solution (less than 5% carbohydrate). There is a little difference between liquid glucose, sucrose, or starch as the ingested carbohydrate fuel source during exercise. Fructose is not desirable because its absorption by the gut does not involve by the active co-transport process required for glucose-sodium. Therefore, fructose absorption is relatively slow and promotes less fluid uptake than an equivalent amount of glucose. The optimal carbohydrate replacement rate is between 30 and 60 gm per hour ingested at least 30 minutes before the time when fatigue would normally occur with out a carbohydrate supplement.

**Body Weight Control and Exercise**

There are two components to the weight control equation; namely, the number of calories that you consume and the number of calories that you burn. Under normal conditions, the objective in weight control is to achieve a balance between the two. If an imbalance does occur it will generally result in either a loss or possibly a gain in weight. For
example, if more calories than needed for energy expenditure are taken in, a weight gain usually results. The reverse is also true.

An individual can lose or gain weight in one of three ways:
  i. By manipulating the number of calories taken in.
  ii. By manipulating the number of calories burned up.
  iii. By manipulating both the intake and expenditure.

Most people, when attempting to lose a few kilograms of body weight, generally think of cutting down on their caloric intake (by dieting) while giving very little attention to the number of calories that they burn. Although the result is usually some weight loss and a reduction in lean body weight, its generally just for a short period of time and is usually not retained. Under this system where weight loss is accomplished only by dieting, a feeling of deprivation and resentment is usually developed over a period of time. What normally happens is that people often revert back to their old previous eating habits and gain weight in terms of body fat. Most nutritionists and exercise physiologists agree that a combination of reduced intake and increased expenditure is generally considered the best. In fact, the majority of research in recent years has concluded that the best method of losing weight is through a combined exercise (progressive cardio respiratory-endurance type) and diet program over a relatively long period of time. This type of weight-reduction program is designed to allow for a slow and gradual weight loss of between 0.5 and 1.0 kg per week without a large reduction in the daily food intake. However, it has been suggested that individuals should not lose more than 1.0 kg per week. A weight loss of 1.0 kg per week is a considerable amount when one considers that this would total around 47 kg if carried on for 12 months or one year. It should also be mentioned that this combined and diet long-range program is also designed to not only reduce body weight to optimum levels without necessarily lowering lean body weight, but also to perhaps create new and long lasting eating and exercising habits.

At this point, it should be mentioned that in some circles a general misconception concerning exercise and weight control is that exercise is not really effective in weight reduction because appetite is automatically increased in direct proportion to the increased exercise. There is no doubt that laborers such as lumberjacks and farmers who perform daily 8 hours of hard physical work consume twice as many daily calories as sedentary people. Also, athletes such as marathon runners and cross-country skiers who devote a considerable amount of time (in some cases, 8 hours or more) to strenuous training consume a large amount of calories per day (around 6,000 kcal) as compared to sedentary (between 2,000 and 3,000 kcal) people. Apparently, this high caloric intake for these athletes, however, is needed to meet their energy demands for training since some of the athletes have very low percent of body fats.

On the other hand, there is a considerable amount of evidence available to indicate that if exercise is performed for short to moderate periods of time (such as one hour per day) daily, appetite and food intake is not increased regardless of whether the work is mild or vigorous. But instead, most people’s appetite will generally decrease (especially if they are used to sedentary style of living).

Another misconception concerning exercise and weight control is the amount of time required by exercising to reduce body weight, in other words, some people claim that a person must spend an unbelievable amount of time and effort to lose just half kg of fat. For example, on a short-time weight-reduction exercise program, this is probably true since in order to lose a pound of stored fat, a deficit of 3,500 kcal is equal to 3,087 ft-lb of work. What this actually means in terms of physical activity is playing golf (walking and not riding in a cart) for something like 20 hours, leisure walking for something like 20 to 25 hours, or performing some other ridiculous feat of work. A person who is overweight and wants to lose 5 to 10 kg would become very discouraged and upset if he thought that he had to spend that much time in one or two days or even in a week on order to lose one kg. In fact, he probably would choose dieting over existing if he felt that it was going to require that much time. On the other hand, what most people tend to forget is that the number of calories expended during physical activity is cumulative and may take place over a prolonged period of time.
other words, a caloric deficit of 3,500 kcal is equal to half kg of weight loss no matter if it occurs in one day, over a one-month period, or in one year.

It is important to remember that the speed by which an effective weight loss can be brought about will naturally depend upon the individual’s personal goals, their motivation, how much they reduce the intake of food, and how much exercise they actually get involved in.

Some typical caloric values of several physical activities are only average energy values for normal people and should not be interpreted as absolute values since the number of kilocalories expended for any physical activity will fluctuate from person to person. These values can be used as a helpful guide in calculating how much extra daily activity one needs in a weight-reducing program.

**TIMING OF NUTRITIONAL INTAKE**

Athletes are concerned that they are properly fueled prior to exercise or competition. Although most realize the importance of eating, many do not understand the timing of the pre-exercise or pre-event meal and what this meal should consist of. However, over the last few years a greater understanding has been attained due to studies examining the effects of food and fluid consumption prior to and during exercise and completion.

**The Pre-Competition Meal**

It is generally accepted that athletes benefit greatly from having meal before practice or competition as opposed to performing in a fasted state (ACSM Joint Position Statement, 2000). The meal before game contributes very little to the glycogen content of the muscle. However, it helps to ensure blood glucose levels and prevent feelings of hunger.

These guidelines are recommended for the pre-competition meal:

a. Avoid foods that are even mildly distasteful to an individual athlete—no matter how well they may serve nutritional objectives. An athlete may get sick even though the food is excellent.

b. Avoid irritating foods, such as highly spiced foods and roughage.

c. Avoid gas-forming foods: onions, cabbage, apples and baked beans, etc.

d. Low in fat and fiber to facilitate gastric emptying and gastrointestinal distress.

e. Hold moderate intake of protein foods because their metabolism results in fixed acids. In large quantities, this could result in an undesirable acidosis.

f. Fluid can best be supplied by bouillon (which supplies sodium, which is excreted in perspiration during an event). Many athletes will prefer milk or juices, and if experiences show no ill effects it is probably wise to accede to this preference. Sufficient in fluid to maintain hydration is suggested.

g. High in carbohydrate to maintain blood glucose and maximize filling of glycogen stores in the body.

h. Made up of foods familiar to the athlete should be preferred.

i. Ideally, the meal should be eaten approximately 3 to 4 hours before the event and comprise 200 to 300 g of carbohydrate.

**Food Supplements during Competition**

The importance of carbohydrate supplementation during competition has begun to attain full acceptance and its efficacy has achieved scientific merit. These guidelines are recommended for the during-competition meal:

i. Carbohydrates during exercise maintain blood glucose levels and improve exercise performance. In addition, the carbohydrate supplement did not cause any elevation in plasma insulin concentrations.

ii. Continually providing carbohydrate at 15 to 20 min intervals during the first 2-hour activity may be more beneficial.

iii. The composition of the carbohydrate should be primarily glucose but a combination of glucose and fructose may be used without gastric problems.

iv. The carbohydrate consumed can be in liquid, solid, or gel form as long as the athlete drinks adequate fluids.

v. If the athletes enter the competition with an inadequate glycogen supply, they would benefit from carbohydrate supplementation during the event.
The Post-Competition Meal

The timing of the post exercise or post-competition meal is important. It is generally recommended that the post exercise meal be eaten within 2 to 4 hours. However, the closer the meal is to the conclusion of the exercise or competition, the greater the opportunity to maximize glycogen loading. The following guidelines are recommended for the post-competition meal:

i. Carbohydrates with a high glycemic index are essential for the meal due to higher muscle glycogen content.

ii. Foods with a high glycemic index are digested quickly and raise blood glucose levels fairly rapidly.

iii. Protein consumption is necessary for muscle repair and other anabolic processes within the muscle.

iv. A combined protein and carbohydrate supplement provided immediately post exercise might enhance the anabolic processes after resistance exercise.

Vegetarian Diet

To eat a healthy diet and to increase carbohydrate intake, many athletes have adopted vegetarianism. However, athletes can survive on vegetarian diet. But the athletes who are strict vegetarians (not lacto-vegetarian) must be very careful in the selection of the plant foods they eat to provide a good balance of the essential amino acids and adequate sources of vitamin A, riboflavin, vitamin B₁₂, vitamin D, calcium, iron and sufficient calories.

Diets Affect Performance

Carbohydrate is the prime source of energy during exhaustive work; both fat and carbohydrates are the sources of energy during steady state activities. The carbohydrate molecule contains more oxygen than does fat. Add to this the fact that carbohydrate is a more efficient precursor of energy than fat (requires less oxygen to produce the same amount of energy); this makes it the preferable energy-producing food. Studies conclusively demonstrate that diets lacking in carbohydrates have deleterious effects on work performance.

Muscle Glycogen Loading or Supercompensation

The amount of glycogen resynthesized in skeletal muscle can be increased to values much higher than normal by following one or more of the following diet and or exercise procedures.

1. The first of these procedures is the endurance athletes who consume a high carbohydrate diet for 3 to 4 days after several days on a normal

Fig. 8.7: The relationship between muscle glycogen content and work time.
mixed diet may increase their glycogen stores from the normal 15 grams to around 25 grams per kilogram of muscle. During the period of the high carbohydrate diet, no exhausting exercise should be performed.

2. A second procedure for loading the muscle with glycogen combines exercise and diet. In this procedure the muscles that are to be loaded are first exhausted of their glycogen stores through exercise; the individual then follows a high carbohydrate diet for a few days. This routine has been shown to double the glycogen stores.

3. A third procedure for glycogen loading calls for exercise and two special diets. Exercise is once again used to induce glycogen depletion. The individual then follows a diet very low in carbohydrates but high in fat and protein for 3 days, after which a high carbohydrate diet is followed for an additional 3 days. This procedure has been shown to increase the glycogen stores to levels approaching 50g/kg and could result 700 gm of stored glycogen or 2800 kcal of ready energy.

A degree of caution should be observed whenever glycogen loading is attempted.

i. Exercise-induced depletion of glycogen is followed by a fat and protein diet, causes a feeling of fatigue.

ii. Possibility of a reduced niacin intake during the carbohydrate-rich diet.

iii. Glycogen loading results in an increased muscular storage of water. A feeling of stiffness and heaviness is thus often associated with loading of the muscle.

**SUMMARY**

i. Nutrition is the combination of processes by which the living organism receives and utilizes the materials necessary for the maintenance of its functions and for the growth and the renewal of its components. All nutrients that are collected by the living organisms from their surroundings are not considered as food. Nutrients do not require digestion. The essential substance like minerals, vitamins and water are collectively called nutrients.

ii. By taking those edible substances, due to which growth, nutrition, yield of energy, and

---

**Fig. 8.8:** Muscle glycogen stores in the muscle followed by three different processes
building new tissue of the living body take
place, are considered as food. Food is divided
into two types according to their functions
such as- (i) primary or nutritive food (ii)
secondary or protective food. Fat, protein,
carbohydrates are called nutritive food and
vitamins, minerals and water are known as
protective food. Nutritive foods provide us
energy and protective foods protect our body
against the disease.

iii. The Provitamins are the organic compounds
from which the vitamins are synthesized in
the animal body. The substances, which have
got structural and chemical group similar to
vitamins but without any physiological action
or which destroy the vitamins or make the
vitamins ineffective are called antivitamins.
A condition due to the administration of
excess amount of a vitamin is termed as hyper-
vitaminosis. Excess amount of water-soluble
vitamins when administrated in the body are
excreted through urine cause no toxic effects.
But fat-soluble vitamins are more prone to
produce toxic symptoms, as their
concentration level rises steadily in the body.

iv. Vitamin supplements can reverse the
symptoms of vitamin deficiency, once a
deficiency is cured, supplements do not
further improve a normal status. Many
coaches, athletes, fitness enthusiasts to
advocate the use of vitamin supplements. But
this approach is simply not supported by
research findings.

v. The mineral salts are very much essential for
our life although they give no energy. The
body contains more than 19 minerals all of
which must be derived from foods. Various
minerals that help in catabolic and anabolic
cellular process. They are important in
activating the numerous reactions that release
energy during the breakdown of fat, protein
and carbohydrate. Minerals also form
important constituents of hormones.

vi. There is no evidence that supplementation
benefit of exercise performance for normal
individuals receiving the RDA of minerals.
An important consequence of prolonged

vii. A diet is considered “balanced” when all food
groups (six) are represented in appropriate
proportions or percentages. This means
following the recommended daily allowances
for micronutrients and for macronutrients—
fats, proteins and carbohydrates. The six
groups of foods are fats, proteins,
carbohydrates, vitamins, minerals and water.
Water is one of the most important foods for
living organisms.

viii. The body’s water content remains relatively
stable overtime. Although considerable water
output occurs in physically active individuals,
appropriate fluid intake rapidly restore and
imbalance in the body’s fluid level. A fairly
sedentary adult in a normal environment
requires about 2.5 L of water each day. For
an active person in a warm environment the
water requirement often increases to between
5 to 10 L daily. Three sources provide this
water: (1) liquids, (2) foods, and (3) metabolic
processes.

ix. There are two components to the weight
control equation; namely, the number of
calories that you consume and the number
of calories that you burn. Under normal
conditions, the objective in weight control
is to achieve a balance between the two. If
an imbalance does occur it will generally
result in either a loss or possibly a gain in
weight. For example, if more calories than
needed for energy expenditure are taken in,
a weight gain usually results.

x. Athletes are concerned that they are properly
fueled prior to exercise or competition.
Although most realize the importance of
eating, many do not understand the timing
of the pre-exercise or pre-event meal and
what this meal should consist of. However,
over the last few years a greater understanding
has been attained due to studies examining
the effects of food and fluid consumption prior to and during exercise and completion.

Carbohydrate is the prime source of energy during exhaustive work; both fat and carbohydrates are the sources of energy during steady state activities. The carbohydrate molecule contains more oxygen than does fat. Add to this the fact that carbohydrate is a more efficient precursor of energy than fat (requires less oxygen to produce the same amount of energy); this makes it the preferable energy-producing food.

**Review Questions**

1. Define food. What do you mean by nutritive and protective food? What are the six categories of nutrients? Write the name of all essential acids.

2. What is an appropriate protein allowance for a (i) normally active male and female, (ii) growing children and (iii) Shot-put thrower?

3. What are the essential amino acids?

4. What are the deficiency symptoms of vitamin A and D? State the sources of vitamin A and C.

5. Although fats provide more energy than carbohydrates, yet carbohydrates are more suitable than fats, why?

6. Define nutrients? State the physiological functions of three fat and water soluble vitamins.

7. Name the nutrients the deficiency of which produces the following diseases: (a) Kwashiorkor; (b) Scurvy; (c) Osteomelacia; (d) Xerophthalmia; (e) Marasmus; (f) Pellagra; (g) Goiter; (h) Rickets

8. Mentioned the nutritional role of the following in the human body: (a) Iron; (b) Iodine; (c) Vitamin D; (d) Carbohydrates; (e) Thiamin; (f) Calcium; (g) Vitamin A; (h) Protein.

9. Discuss the value of using protein supplements to enhance performance in strength and endurance event.


11. How does the body regulate electrolyte balance during acute exercise and chronic exercise?

12. Describe the preferred pre-competition and post competition meal. Discuss about the food supplements during competition.

13. What is vegetarian diet? How diet affects sports performance?

14. Describe the proper dietary regimen to glycogen load the muscle prior to an exhaustive event lasting three to four hours.

15. Discuss the value of consuming carbohydrate during and after endurance exercise.
Age, Sex and Physical Performance

GROWTH, DEVELOPMENT AND THE YOUNG ATHLETE

Growth, development and maturation are terms that can be used to describe changes starting at conception and continuing through adulthood that occur in the body. Growth refers to an increase in the size of the body or any of its parts. Development refers to differentiation along specialized lines of function, so it reflects the functional changes that occur with growth. Finally, maturation refers to the process of talking on the adult form and becoming fully functional being considered. For example, skeletal maturity refers to having a fully developed skeletal system in which all bones have completed normal growth and ossification, whereas sexual maturity refers to having a fully functional reproductive system. The state of a child’s or adolescent’s maturity can be defined by:

- a. Chronological age
- b. Skeletal age
- c. Stage of sexual maturation

Chronological Age

Chronological age refers to the period that has elapsed beginning with an individual’s birth and extending to any given point in time. Chronological age is used in research and in test norm development as a measure to group individuals. Developmental research looks for age-related differences or behavior changes as a function of age.

Using chronological age provides a means to roughly assure the equivalence of such factors as physical experience, social interaction, learning, and
acculturation among others. Chronological age is not necessarily a predictor of an individual’s stages of development, as the rate at which individual’s progress through stages may not be identical. Problems in using chronological age include such issues as school readiness and the evaluation of premature infants. As medical technology has advanced in the treatment of premature infants, chronological age has been challenged as an appropriate measure for this group with gestational age or durational pregnancy being proposed as a means of adjusting chronological age.

Developmental Norms

Developmental norms are defined as standards by which the progress of a child’s development can be measured. For example, the average age at which a child walks, learns to talk, or reaches puberty would be such a standard and would be used to judge whether the child is progressing normally. Norms have also been used as a basis for the “ages and stages” approach to understanding child development, made famous most notably by Yale University pediatrician and educator Arnold Gesell and University of Chicago educator Robert Havighurst. In using the idea of norms, Havighurst presented a set of developmental tasks tied closely to what behavior one might observe at what age. These sets of developmental tasks became a tool for teachers to use to help judge the appropriateness of certain types of curriculum for children of certain ages or developmental levels. While norms are usually thought of as being age-related, norms can also be tied to other developmental variables such as race, ethnicity and sex.

There are early and late maturers, such as (Fig. 9.1) child A and child B whose individual differences in variables such as strength and speed at age 8 are not predictive of values at age 13.

Superimposed on these variations in rate of maturation are inherent (geneticall-based) interindividual differences, which cause one child to be more fit than another, even when body size and level of biologic development are equivalent. Child C in the figure is fortunate and will always be able to lift more weight for a longer time than child A.

INFLUENCE OF GROWTH FACTORS ON PHYSICAL FITNESS

Physical growth is the most important factor in the development of physical responses to exercise during the childhood years. Moreover, differences in the rate of growth are largely responsible for individual differences in physical performances in the pediatric age group. A 16 year old boy has three time greater maximal oxygen uptake than he had back at age 5. Grip strength in girls increases threefold over the same age span. A teenager runs more economically (with a lower VO₂ per kg.) at a given speed than when he was a small child.

These are all expression principally of increases in body size. Between the ages of 6 and 16 years, the lungs in males grow approximately from a total capacity of 1937 ml to 5685 ml and the weight of the heart grows from 95 gm to 258 gm. These increases are manifest in the development in maximum minute ventilation and stroke volume as children grow. Muscle strength improves as a consequence of increased volume of muscle tissue. Estimated total body muscle mass in girls grows from 7 kg at age six years to 23 kg by adolescence. As leg length increase with age, stride frequency at a given speed declines, resulting in a lower overall oxygen requirement for running.

Fig. 9.1: Effects of timing of biologic maturation and inherent physical fitness on physiologic performance development. Child A, early maturer; child B, late maturer and child C, inherently more highly fit
Biologic Age

It has been observed that a program of hormonal activity that stimulates somatic growth proceeds during the childhood and adolescent years. This evolution of body size increase is one example of biologic maturation, a series of developmental changes that culminates in the adult stage or complete biologic maturation.

The extent of biologic maturation can be estimated by percentage of estimated adult height and weight, wrist bone age as measured by radiography and after the onset of puberty by the progression of appearance of secondary sexual characteristics. These are all indicators of different aspects of biologic growth, but since all are affected by similar anabolic hormonal influences, they typically cluster and track together as children grow.

Table 9.1: Average height and weight of boys at different ages (Indian)

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
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<tbody>
<tr>
<td>Birth</td>
<td>50.5</td>
<td>3.3</td>
</tr>
<tr>
<td>3 months</td>
<td>61.1</td>
<td>6.0</td>
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<tr>
<td>6 months</td>
<td>67.8</td>
<td>7.8</td>
</tr>
<tr>
<td>9 months</td>
<td>72.3</td>
<td>9.2</td>
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<td>14.6</td>
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<td>4 years</td>
<td>102.9</td>
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<tr>
<td>5 years</td>
<td>109.9</td>
<td>18.7</td>
</tr>
<tr>
<td>6 years</td>
<td>116.1</td>
<td>20.7</td>
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<tr>
<td>7 years</td>
<td>121.7</td>
<td>22.9</td>
</tr>
<tr>
<td>8 years</td>
<td>127.0</td>
<td>25.3</td>
</tr>
<tr>
<td>9 years</td>
<td>132.2</td>
<td>28.1</td>
</tr>
<tr>
<td>10 years</td>
<td>137.5</td>
<td>31.4</td>
</tr>
<tr>
<td>11 years</td>
<td>140.0</td>
<td>32.2</td>
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<td>147.0</td>
<td>37.0</td>
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<tr>
<td>13 years</td>
<td>153.0</td>
<td>40.9</td>
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<td>14 years</td>
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<td>47.0</td>
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<td>15 years</td>
<td>166.0</td>
<td>52.6</td>
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<tr>
<td>16 years</td>
<td>171.0</td>
<td>58.0</td>
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<tr>
<td>17 years</td>
<td>175.0</td>
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<tr>
<td>18 years</td>
<td>177.0</td>
<td>65.0</td>
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</table>

Source: Nutrient requirements and recommended dietary allowances for Indians, ICMR, 1990

Table 9.2: Average height and weight of girls at different ages (Indian)

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth</td>
<td>49.9</td>
<td>3.2</td>
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<td>3 months</td>
<td>60.2</td>
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<tr>
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<td>126.4</td>
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<tr>
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</tr>
<tr>
<td>18 years</td>
<td>164.0</td>
<td>54.4</td>
</tr>
</tbody>
</table>

Source: Nutrient requirements and recommended dietary allowances for Indians, ICMR, 1990

Growth and Development of the Tissues

To understand the physical capabilities of children and the potential impact that sport activity can have on young athletes. We should consider the physical state of their body. Growth and development of some tissues are as follows:

a. Height and weight
b. Bone
c. Muscle
d. Body Fat
e. The nervous system

Height and Weight

These two variables are most useful when we examine their rates of change with growth. Change in height is assessed in terms of centimeters per year and change in weight in terms of kilograms.
per year. Figure 9.2 shows that height increases rapidly during the first two years of life. In fact the child reaches about 50 percent of adult height by age 2. After this, height increases at a progressively slower rate throughout childhood; thus there is a decline in the rate of its change. Just prior to puberty, the rate of change in height increases markedly, followed by an exponential decrease in the height growth rate until full height is attained at a mean age of about 16.5 years in girls and 18.5 years in boys. The peak rate of growth in height occurs at approximately 11–12 years in girls and 13–14 years in boys. The same overall trend for the rate of change in weight is revealed from the Figure 9.2.

**Fig. 9.2:** Change of height and weight with age of boys and girls (up to maturity)
As with height, the peak rate of growth in body weight occurs at approximately 12.0 years in girls and 14.5 years in boys, which is slightly later than height.

**Bone**

Bones are formed through ossification, which spreads from primary (diaphysis) and secondary (epiphysis) ossification centers. Injury in the epiphysis would cause early termination of growth. Exercise, along with an adequate diet, is essential for proper bone growth. Exercise affects primarily bone width, density, and strength but has little or no affect on length.

Competitive baseball, specially pitching motion, carries the highest risks of epiphyseal injury. Sometimes Tennis and Swimming also carry higher risks for young athletes.

**Muscle**

Muscle mass increases steadily along with weight gain from birth through adolescence. In males, the rate of muscle mass increase peaks at puberty, when testosterone production increases dramatically. On the other hand girls do not experience this sharp increase in muscle mass. Muscle mass increases in boys and girls result primarily from fiber hypertrophy with little or no hyperplasia. The increase in muscle mass with growth and development is accomplished primarily by hypertrophy of individual muscle fibers through increases in their myofilaments and myofibrils. Muscle length increases through the addition of sarcomeres and by increases in the length of existing sarcomeres.

Muscle mass peaks in girls between ages 16 and 20 years and in boys between 18 and 25 years, though it can be increased more through diet and exercise.

**Body Fat**

Fat cells can increase in size and numbers throughout the life. Fat storage occurs through increase in the size of existing fat cells and by increasing the number of fat cells. It appears that existing fat cells, as they become full, signal the need for the development of the new fat cells.

The amount of fat that accumulates with grows and aging depends on diet, exercise habits and heredity. Heredity is unchangeable, but both diet and exercise can be altered to either increase or decrease the body fat storage.

At physical maturity, the body’s fat content averages 15 percent in males and 25 percent in females. The differences are caused primarily by higher testosterone level in males and higher estrogen levels in females.

**Nervous System**

As children grow, they develop better balance, agility and coordination as their nervous systems develop. Myelination of the nerve fibers must be completed before fast reactions and skilled movement can occur, because conduction of an impulse along a nerve fiber is considerably slower if myelination is absent or incomplete. Myelination of the cerebral cortex occurs most rapidly during childhood, but continues well beyond puberty. Although practicing an activity or skill can improve performance to a certain extent, the full development of that activity or skill is dependent on full maturation of the nervous system.

**Effect of Exercise on Growth**

It is one of the very important aspect that how physical activity during childhood and adolescence, particularly athletic participation, affect physical...
growth. This is a major concern of parents, coaches, and physical educators. Can this stress of intense physical activity in the growing years impair linear and visceral growth?

From an early report indicated that delayed statural maturation in children undertaking hard labor in poor socioeconomic conditions. However, exercise along with an adequate diet, is essential for proper bone growth. Exercise affects primarily bone width density and strength but has little or no effects on length.

**PHYSICAL PERFORMANCE IN YOUNG ATHLETES**

The function of almost all physiological systems improves until full maturity is reached or shortly before. After that, function plateaus for a period

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**Figs 9.4(A and B):** Change of (A) – fat and fat free mass and (B) skin fold thickness (four sites) of boys and girls with increase in age (adopted from physiology of sports and exercise, Wilmore and Costill)
Fig. 9.5: Changes in motor ability from the ages of 6 years to 17 years boy and girls (adopted from physiology of sports and exercise, Wilmore and Costill)

of time before starting to decline with advancing age. The following changes in the young athlete that accompanies the growth and development.

- Motor ability
- Strength
- Pulmonary function
- Cardiovascular function
- Aerobic capacity
- Running economy
- Anaerobic capacity, and
- Thermal stress
Motor Ability

The motor ability of boys and girls generally increases with age for the first 18 years although girls tend to plateau at the age of puberty. This improvements result primarily for the development of neuromuscular and endocrine system and secondarily from the children’s increased activity.

The plateau observed in the girls at puberty is likely explained by two factors. The increase in estrogen level at puberty leads to increase fat deposition. Performance stands to decrease as fat increases. Probably of greater importance, though, is that around puberty many girls assume a much more sedentary lifestyle than boys. This is largely a matter of social conditioning, as boys are encouraged to be more active and athletic than girls. As these girls become less active, their motor abilities tend to plateau.

Strength

Strength improves as muscle mass increases with age. Gains in strength also depend on neural maturation, because neuromuscular control is limited until myelination has been completed usually around sexual maturity. Girls are experiences a more gradually increase in strength and do not exhibit a marked change in their rate of change gain with puberty.

It is well established that testosterone increases skeletal muscle bulk and strength. Changes in muscle strength at puberty are closely associated with a rise in testosterone levels. Men who have depressed gonadal function characteristically also demonstrate low fat free mass. The decline in testosterone as men age parallels a fall in muscle strength. Testosterone also increases strength by some means other than increasing muscle mass. Neuromuscular transmission may be facilitated by testosterone, and the cognitive effects of androgens might alter motivation to train at higher intensity.

Pulmonary Function

Lung function changes markedly with age. All lung volumes increase until growth is completed. Peak flow rates follow the same pattern. Until physical maturity, maximal ventilatory capacity and maximal expiratory ventilation increase in direct proportion to the increasing body size during exhaustive exercise.

A cross sectional study showed that maximal expiratory ventilation averages about 40 lit/min for 4 to 6 years old boys and increases to 110 to 140 lit/min at full maturity. Girls also follow the same patterned, but their absolute values remained considerably lower, primarily due to their smaller size of the body. These changes are associated with the growth of the pulmonary system, which parallels the general growth patterns of children.

Developmental Change in Ventilatory Components

The changes that occur in the components of ventilation as children grow, both at rest and during exercise, have been well described. Much of this development parallels increases in lung size. Between the age 5 and 14 years, the total capacity of the lung increases from approximately 1,400 to 4,500 cm³ It has been reported that lung volume correlated closely with both height and height cubed, irrespective of sex.

Ventilatory Equivalent for Oxygen

Children also hyperventilate in relation to the aerobic metabolic requirements of physical work. This manifested by their greater ventilatory equivalent for oxygen (VE/VO₂) at all levels of exercise. A gradual decline in VE/VO₂ occurs continuously through the childhood years. It has been reported by many scientists that a gradual decline in VE/VO₂ at maximal exercise between the ages of 8 and 16 years, with values falling from about 34 to 24. No differences were seen in these changes between boys and girls.

Cardiovascular Function

Heart size is directly related to body size, so children have smaller hearts than the adults. As a result of this and a smaller blood volume the child has a smaller stroke volume capacity. The higher maximum heart rate in the children can only partially compensate for this lower stroke volume capacity and thus maximal cardiac output is lower than that of an equal trained adult. Blood pressure is directly related to body size- it is lower in children
than adults but increases to adult levels in the late teen years.

Even with increase heart rate, a child’s cardiac output remains less than an adult. In submaximal exercise, an increase in the arteriovenous oxygen difference ensures adequate oxygen delivery to the active muscles. But at maximal work rates oxygen delivery limits performance in activities other than those in which the child merely needs to move his or her body mass.

**Heart Rate**

Resting heart rate falls progressively during childhood. When measured in the basal state, the average child experiences a decrease from 80 bpm at age 5 to 62 bpm at age 15 years. Before age 10 years there are little gender differences, but above this age the basal heart rate is about 3 to 5 bpm greater in girls. Seated resting values are typically 15 bpm higher than those obtained in basal conditions (postabsorptive, lying quietly for at least 30 minutes).

The decline in resting heart rate with age parallels that of weight-relative basal metabolic rate (BMR), and it is tempting to conclude that this association is causal, i.e. that the fall is BMR as body size increases is the result of a decline in resting heart rate, while other factors responsible for metabolic rate (cardiac stroke volume, peripheral arteriovenous oxygen uptake) grow in relation to body size. The fall in resting heart rate with age during childhood is not caused by changes in autonomic influence, since a similar decline is observed after sympathetic and parasympathetic blockade.

Both longitudinal and cross-sectional studies in children have indicated that maximal heart rate does not change during childhood. It is important to recognize, then, that formulae used for estimating maximal heart rate for age (such as 220 minus age) are inappropriate for children. Maximal heart rates in studies of children typically range from 195 to 205 bpm and are influenced by testing modality. Peak heart rates obtained during cycle testing are typically about 5 bpm less than those obtained during treadmill exercise and values during treadmill running are usually higher than with walking protocols. These findings mimic those observed in adults. Most studies have shown no difference in maximal heart rates during exercise in boys and girls. Although maximal heart rate is stable during childhood, the inter-individual variability in considerable. Typical standard deviations of 5 to 9 bpm has been reported in most studies.

Since resting heart rate falls with age in children while maximal values are stable, the difference (heart rate reserve) must increase. Between the ages of 6 and 12 years, for instance, this reserve increases from 120 to 133 bpm in the average boy. Such changes may contribute to improved aerobic capacity during the childhood.

Like those at rest, heart rate values at a given submaximal work level progressively decline as the child ages. This makes sense, since absolute oxygen uptake and cardiac output do not change with age at a specific work load, while stroke volume increases with body size. As a result, heart rate must fall. For instance, submaximal heart rate declines from about 140 to 100 bpm in males between ages 8 and 18 years while pedaling at a work load of 30 watts.

Heart rate relates linearly to work intensity at moderate levels of exercise, but at high loads the heart rate tapers as work rate increases. This phenomenon, not well explained, appears to be qualitatively and quantitatively similar in children and adults. The point at which the heart rate begins to decelerate as work increases (the “heart rate deflection”) has been reported to relate to ventilatory markers of the anaerobic threshold in both children and adults.

The rate of decline in heart rate after exhaustive exercise decreases as the child ages. Washington et al, reported one-minute recovery heart rates of 133, 138 and 148 bpm in boys grouped by surface area as <1.0 m², 1.0 to 1.19 m² and >1.2 m², respectively. Factors responsible for the more rapid recovery of heart rate after peak exercise in the smaller child are uncertain.

**Stroke Volume**

Since maximal heart rate during childhood is stable, it follows that (1) the rise in maximal cardiac output in children as they grow must be entirely due to an increase in stroke volume, and (2) during the same time period heart rate does not contribute to any increases in absolute maximal oxygen uptake.
Similarly, it can be summarised that maximal stroke volume increases in proportion to body dimension in a manner similar to that of oxygen uptake.

Echocardiographic and cardiac catheterization studies of children indicate that resting stroke volume relates closely to both body weight and surface area. Typical values are 42 ml/m² in the supine position and 35 ml/m² in upright position. Absolute values have been reported as 5 ml at birth, 25 ml at age 5 years and 85 ml at age 15 years.

Values for stroke volume at maximal exercise demonstrate a similar close relationship to body size/weight as they do at rest. Scientists reported that stroke volumes from cardiac output estimates based on the carbon dioxide re-breathing technique within 15 seconds after exhaustive cycle exercise testing. Values for stroke volume related to body surface area remained stable across the age range of 9 to 20 years, averaging 65 ml/m² in males and 56 ml/m² in females.

**Cardiac Output**

Stroke volume increases with age in children in close relationship with body dimensions while resting heart rate falls, it would be expected that resting cardiac output relative to body weight or surface area would progressively decline during the childhood. Limited experimental data support this conclusion. However, some study of healthy subjects demonstrated a decline in resting cardiac output of 6 l/min/m² at age 4 to 4 l/min/m² during adolescence.

Likewise, if stroke volume-body size relationships remain constant at maximal exercise during childhood while peak heart rate is unchanged, maximal cardiac output should parallel to body dimensions or weight. This is also borne out by the experimental data. A cross-sectional study indicated that absolute maximal cardiac output increased from 12.5 to 21.1 l/min/m² in males between 10 and 20 years of age and from 10.5 to 15.5 l/min in girls of the same ages. Maximal

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Figs 9.6(A to D): Submaximal a) heart rate, b) stroke volume, c) cardiac output and d) arterio-venus O₂ difference in an 8 year boy and fully mature man at fixed rate of O₂ uptake (adopted from physiology of sports and exercise, Wilmore and Costill)
cardiac output relative to body surface area remained essentially unchanged during this age span, with a average of 12.2 and 10.5 l/min/m² for the boys and girls, respectively.

**Blood Pressure**

During exercise (rhythmic) the rise in systolic blood pressure is proportional to exercise intensity and to the overall metabolic level. Such pattern is operative in all healthy individuals, irrespective of age. Diastolic blood pressure, which depends primarily on the peripheral vascular resistance, changes little (or even decrease slightly) with exercise. However, for a given level of exercise, a small child responds with a lower increase in systolic and diastolic pressures than does an adolescent. With increase in mean blood pressure, total peripheral resistance decreases progressively. In spite of the lower arterial pressure in children, their peripheral resistance is higher than in adults. There seem to be no gender differences in total peripheral resistance.

A lower exercise blood pressure in the young child is in line with the lower cardiac output and stroke volume. There is no reason to assume that such an age related pressure difference is either materially beneficial or detrimental to the working capacity of the young child.

**Aerobic Capacity**

As pulmonary and cardiovascular function improves with continued development, so does aerobic capacity. VO₂ max express in lit/min, peaks between ages 17 and 21 years in males and between 12 and 15 years in females, after which it steadily decreases. When VO₂ max is expressed relative to body weight, it plateaus in males from age 16 to 25 years, but begins its decline at about age 15 years in girls. However, expressing VO₂ max relating to body weight might not provide an accurate estimate at aerobic capacity. Such VO₂ max values do not have reflect the significant gains in endurance performance capacity that are noted with both maturation and training.

The child’s lower VO₂ max value (lit/min) limits endurance performance unless body weight is the major resistance to movement, such as in distance running. When expressed relative to body weight a child’s VO₂ max is similar to adults yet in activities such as distance running. A child’s performance is far inferior to adult performance because of differences of economy of effort.

**Running Economy**

Children cannot maintain as first a running pace as the adult because of basic differences in economy of effort. Running economy increases and this improve their distance-running pace even if the children are not training and if their VO₂ max values do not increase.

Some scientist have hypothesized that the following factors that can change growth and development explain at least in part the lower running economy in children and its improvement its maturation:

- Stride frequency
- Gait mechanics

![Figs 9.7(A and B): Changes in maximal oxygen uptake with age (adopted from physiology of sports and exercise, Wilmore and Costill)]
Musculotendinous elastic energy storage
Surface area to body mass ratio
Changes in body composition
Thermal response to exercise
Substrate utilization
Anaerobic capacity
Ventilatory efficiency

Of these factors, thus far only stride frequency has been proven to be important.

**Anaerobic Capacity**

The child’s ability to perform anaerobic activities is limited. A child has a lower glycolytic capacity, possibly because of a limited amount of Phosphofructokinase enzyme.

Children cannot attain high respiratory exchange ratio during maximal and exhaustive exercise, suggesting less lactate production. Mean anaerobic capacity and peak power outputs are lower in children than in adults.

**Thermal Stress**

Thermal stress including heat and cold induced illness or injury is more susceptible in children than adults. Children are capable of less evaporative heat loss than adults because children sweat less (less sweat is produced by each active sweat gland). Young boys acclimatized to heat more slowly than adults do. Children appear to have greater conductive heat loss than adults, which should replace children at greater risk for hypothermia in cold environments. Until more is known about children’s susceptibility to thermal stress, a conservative approach should be used for children who exercise in temperature extremes.

**TRAINING THE YOUNG ATHLETE**

The young athlete is physiologically unique from the adult and must be considered differently. Training can improve the strength, aerobic capacity and anaerobic capacity of the young athlete. Generally, the youngster will adapt well to the same type of training routine used by the mature athlete. But training programs for children and adolescents should be designed specifically for each age group, keeping in mind the development factors associated with that age. In this section, it has been discussed the issue of most concern for young athletes who are involved in:

i. Resistance (strength) training
ii. Aerobic training
iii. Anaerobic training

**Resistance (Strength) Training**

For many years, the use of resistance training to increase muscular strength and endurance in prepubescent and adolescent boys and girls was highly controversial. Boys and girls were discouraged from using free weights for fear that they might injure themselves and prematurely stop the growth process. Furthermore, many scientists speculated that resistance training would have little or no effect on the muscles of prepubescent boys because their levels of circulating androgens were still low.

Studies on animals suggest that heavy-resistance exercise can lead to stronger, broader and more compact bones. Several studies have been conducted in which both prepubescent and adolescent children have participated in resistance training. From these studies it has been concluded that the risk of injury is very low. In fact, resistance training might offer some protection against injury for example, by strengthening the muscles that cross a joint.
Several studies conducted in the mid-1980s demonstrated that prepubescent boys and girls can participate safely in resistance training and they can gain substantial strength. In one study, prepubescent boys and girls took part in a 9-week progressive resistance-training program. They exercised 25 to 30 minutes per day, 3 days each week. Their mean strength increase was 42.9 percent, compared to a 9.5 percent increase in nontraining control group. In a second study, 16 prepubescent males between ages 6 and 11 participated in a 14-week strength-training program using isokinetic techniques with hydrolic resistance, while another 10 boys served as nontraining controls. Isokinetic strength increased between 18 percent and 37 percent in the training group. None of the subjects demonstrated any damage to the epiphysis, bones or muscles as a result of strength training.

In a final study, 33 prepubescent, pubescent and postpubescent males underwent a 9-week resistance-training program. All three groups had significant strength gains. Researchers hypothesized that the pubescent group would experience the greatest strength gains because testosterone levels increase dramatically during this period. In fact, the prepubescent group made greater gains than the pubescent group in several of the strength tests.

The mechanisms allowing strength changes in children are similar to those for adults, with one minor exception: prepubescent strength gains are accomplished largely without any change in muscle size. The mechanisms responsible for strength increase in prepubescent boys are:

i. Improved motor skill coordination
ii. Increased motor unit activation
iii. Other underdetermined neurological adaptation

Strength gains in the adolescent result primarily from neural adaptations and increase in both muscle size and specific tension. Strength is influenced by the amount of fat-free mass, testosterone concentrations, the extent of nervous system development, and the differentiation of fast twitch and slow twitch muscle fibers. The early gains in strength up through puberty are largely the result of changes in neuromuscular patterns.

For actual training programs, resistance training for children should be prescribed in much the same way as is done in adults. Specific guidelines were established by a group representing eight different professional organizations. However, competent instructors who have been trained specifically to work with children must carefully supervise any youth resistance-training program. Furthermore, resistance training should be only one part of more comprehensive fitness program for this age group.

**Strength Training Recommendations for Prepubescent Children**

**Equipment:** The following equipments are recommended for introducing strength training of children.

i. Strength training equipment should be of appropriate design to accommodate their size and degree of maturity of the prepubescent.
ii. It should be cost effective.
iii. It should be safe, free of defects and inspected frequently.
iv. It should be located in an uncrowned area free of obstructions with adequate lighting and ventilation.

**Program Consideration**

i. A preparticipation physical examination is mandatory.
ii. The child must have the emotional maturity to accept coaching and instruction.
iii. There must be adequate supervision by coaches who are knowledgeable about strength training and the special problems of prepubescent.
iv. Strength training should be a part of an overall comprehensive program designed to increase motor skills and level of fitness.
v. Strength training should be preceded by a warm-up period and followed by a cool down.
vi. Emphasis should be on dynamic concentric contractions of muscle while giving the training program.
vii. All exercises should be carried through a full range of motion.
viii. Competition is prohibited at that period.
ix. No maximum lift should ever be attempted.
Prescribed Program

i. Training is recommended two or three times a week for 20 to 30 min periods.

ii. No resistance should be applied until proper form is demonstrated. Six to fifteen repetitions equal one set; one to three set per exercise should be done.

iii. Weight or resistance is increased in 1 to 3-lb increments after the prepubescent does 15 repetitions in good form.

Table 9.3: Basic guidelines for resistance exercise progression in children

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>7 or younger</td>
<td>Introduce child to basic exercises with little or no weight; develop the concept of a training session; teach exercise techniques; progress from body weight calisthenics, partner exercises, and lightly resisted exercises; keep volume low.</td>
</tr>
<tr>
<td>8–10</td>
<td>Gradually increase the number of exercises; practice exercise technique in all lifts; start gradual progressive loading of exercises; keep exercises simple; gradually increase training volume; carefully monitor toleration to the exercises stress.</td>
</tr>
<tr>
<td>11–13</td>
<td>Teach all basic exercise techniques; continue progressive loading of each exercise; emphasize exercise techniques; introduce more advanced exercises with little or no resistance.</td>
</tr>
<tr>
<td>14–15</td>
<td>Progress to more advanced youth programs in resistance exercise; add sport-specific components; emphasize exercise techniques; increase volume.</td>
</tr>
<tr>
<td>16 or older</td>
<td>Move child to entry-level adult programs after all background knowledge has been mastered and a basic level of training experiences has been gained.</td>
</tr>
</tbody>
</table>

Aerobic and Anaerobic Training

It has been well establish that training prepubescent children did not change their VO₂ max values. Even, without significant increases in VO₂ max, the running performance of the children studied did improve substantially. They could run a fixed distance faster following the training program. More recent studies have found small increase in aerobic capacity with training in prepubescent children, but these increases are less than would be expected for adolescents or adults. More substantial changes in VO₂ max appear to occur once children have reached at puberty. The reasons for these findings are not well defined. Because stroke volume appears to be the major limitation to aerobic performance in this age group, it is quite possible that further increases in aerobic capacity depend on heart growth.

Anaerobic training appears to improve children’s anaerobic capacity. Following training children have-

i. Increased resting levels of phosphocreatine, ATP and glycogen

ii. Increased phosphofructokinase activity

iii. Increased maximal blood lactate levels

Ventilatory threshold, a noninvasive marker of lactate threshold, has also been reported to increase with endurance training in 10 to 14 year old boys.

GROWTH AND MATURATION

Many people have wondered what effect physical training might have on growth and maturation. Regular training has no apparent effect on growth in height. It does however, affect weight and body composition. Generally, regular training results in:

i. Decreased total body fat

ii. Increased fat-free mass

iii. Increased total body mass

However, the gains in fat-free mass are generally limited to boys. As for maturation, the age at which peak height velocity occurs is generally not affected by regular training, neither is the rate of skeletal maturation. But the data concerning the influence of regular training on indices of sexual maturation are not at all clear. Although some data suggest that menarche (the initial onset of menstruation) is delayed in highly trained girls, these data are
confounded by a number of factors that were not controlled for in the analysis. Malina concludes his review with the following statement: “Responses of the developing individual to the physical activity of regular training are probably not sufficient to alter geotropically programmed growth and maturation processes. Thus, training has no apparent effect on stature and on maturation as ordinarily assessed in growth studies.”

AGING AND EXERCISE

The area of exercise physiology relates to the various physiological effects, training adaptation and basic principles and guidelines for constructing an individualized cardiorespiratory endurance exercise program for the aged that accompany the aging process.

The level of physical activity begins to decline soon after people reach adult maturity. Considering the importance of exercise for maintaining muscle and cardio-respiratory health, it is not surprising that adult inactivity can lead to deterioration of one’s capacity and tolerance for strenuous effort. The changes in physiological function and physical performance which are discuss in details below.

Physiological Changes Accompanying the Aging Process

At the outset, there does not appear to be any specific threshold age for which performance deteriorates. Most of the physiological functions apparently have their own individual peaks and declines with age. Research shows that most systems and functions, after reaching their peak, will level off for a period of time before gradually decreasing with age. Recent evidence indicates that physical activity may retard or slows down the rate of decline that is associated with aging. Some of the physiological changes accompanying the aging process are shown in the following Table 9.4.

### Table 9.4: Physiological changes accompanying the aging process

<table>
<thead>
<tr>
<th>No.</th>
<th>Physiological Functions</th>
<th>Ageing</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Muscle size</td>
<td>Decreased</td>
</tr>
<tr>
<td>2</td>
<td>Muscle strength</td>
<td>Decreased</td>
</tr>
<tr>
<td>3</td>
<td>Lean body weight</td>
<td>Decreased</td>
</tr>
<tr>
<td>4</td>
<td>Percent body weight</td>
<td>Increased</td>
</tr>
<tr>
<td>5</td>
<td>Basal metabolic rate</td>
<td>Decreased</td>
</tr>
<tr>
<td>6</td>
<td>Maximal heart rate</td>
<td>Decreased</td>
</tr>
<tr>
<td>7</td>
<td>Overall heart size (specially the left ventricular cavity)</td>
<td>Decreased</td>
</tr>
<tr>
<td>8</td>
<td>Cardiac muscle strength</td>
<td>Decreased</td>
</tr>
<tr>
<td>9</td>
<td>Maximal stroke volume</td>
<td>Decreased</td>
</tr>
<tr>
<td>10</td>
<td>Maximal cardiac output</td>
<td>Decreased</td>
</tr>
<tr>
<td>12</td>
<td>Maximal blood flow</td>
<td>Decreased</td>
</tr>
<tr>
<td>13</td>
<td>Elasticity of blood vessels</td>
<td>Decreased</td>
</tr>
<tr>
<td>14</td>
<td>Blood pressure</td>
<td>Increased</td>
</tr>
<tr>
<td>15</td>
<td>Capillary density of muscle</td>
<td>Unchanged</td>
</tr>
<tr>
<td>16</td>
<td>Maximal oxygen uptake (VO₂ max)</td>
<td>Decreased</td>
</tr>
<tr>
<td>17</td>
<td>Vital capacity</td>
<td>Decreased</td>
</tr>
<tr>
<td>18</td>
<td>Maximal expiratory ventilation (Vₑ max)</td>
<td>Decreased</td>
</tr>
<tr>
<td>19</td>
<td>Pulmonary diffusion capacity</td>
<td>Decreased</td>
</tr>
<tr>
<td>20</td>
<td>Ratio of residual volume to the total lung capacity (RV/TLC)</td>
<td>Increased</td>
</tr>
<tr>
<td>21</td>
<td>Reaction time</td>
<td>Decreased</td>
</tr>
<tr>
<td>22</td>
<td>Movement time</td>
<td>Decreased</td>
</tr>
<tr>
<td>23</td>
<td>Bone density</td>
<td>Decreased</td>
</tr>
<tr>
<td>24</td>
<td>Flexibility</td>
<td>Decreased</td>
</tr>
<tr>
<td>25</td>
<td>Elasticity of lung tissue and chest wall</td>
<td>Decreased</td>
</tr>
</tbody>
</table>
**Muscle size and Strength**

As an individual gets older, there is a decline in muscle size. It is believed that this decline is due, in part, to a reduced amount of protein as well as a decline in the number and size of muscle fibers. While it is not completely clear, it has been suggested that the decline in the number of muscle fibers may be due to degenerative diseases affecting the nerve fibers. Increases in strength are highly related to muscle fiber hypertrophy.

On other words, strength increases parallel to increases in muscle size. As people get old, there is also a parallel decrease in muscular strength, which probably results from the decline in muscle size. The decline in strength is a gradual one following the age of about 35 to 45 years. However, even at the age of 60, the decline in strength does not appear to exceed 20 percent of an individual’s maximum strength.

**Body Fat**

With advancing age, there is a general trend to accumulate increase in body fat. There are several reasons normally given for this increase in body fat with advancing age. Such as:

i. Decrease one’s ability to release or mobilize stored fatty acids from adipose tissue for energy fuel, results in less fatty acids being burned up.

ii. Increase of the food intake but become less active.

iii. Take more calories than burned up.

iv. Besides heredity the amount of fat gained depends on eating and exercise habits.

Normally active and sedentary men and women gradually gain body weight from age 20 to 60, despite a gradual reduction in fat free body tissue, muscle and bones. But this age related tendency for greater fatness and less fat free body mass is not constant throughout the life.

**Lean Body Weight**

As one gets older, their lean body weight decreases. The lean body weight with age parallel with the increases and decreases in muscle mass. However, the decrease in lean body weight is due to the decrease in muscle size along with the decline in calcium and phosphorus content of the bones. The amount of decline in lean body weight also controlled by the eating and exercise habits.

**Basal Metabolic Rate**

Basal metabolic rate (BMR) decreases gradually with increasing age. The rate of decline from the age of 3 through 80 years is around 3 percent per decade. Between the ages of 20 and 30, this decline apparently indicates an improved metabolic efficiency. A decline for older people past 30 may be due to the decrease in lean body weight that accompanies age.

**Respiratory Changes**

Both vital capacity and forced expiratory volume decrease linearly with age. Residual volume increases and total lung capacity remain unchanged. This increases RV: TLC ratio, meaning that less air can be exchanged with each breath. Maximum expiratory ventilation also increases with age.

Pulmonary changes that accompany age are primarily caused by a loss of elasticity in the lung tissue and the chest wall. With a reduced elasticity, it naturally decreases their mobility and as a result, increases the effort of breathing. However, older athletes have only slightly decreased pulmonary ventilation capacity. For them, the primary limitation of VO₂ max appears to be decreased oxygen transport to the muscle. Furthermore, arteriovenous oxygen difference is decreased, indicating that less oxygen is extracted by their muscles.

**Cardiovascular Changes**

Cardiovascular functions are also change with increase in age. These are maximum heart rate, stroke volume, cardiac output, etc.

Maximum heart rate (HR max) decreases slightly less than one beat/min per year with increasing age. The average maximum heart rate for a certain age can be estimated/predicted by the following equation:

\[ \text{HR max} = 220 - \text{age} \]

However, this only estimates the average value for a given age. Individual values can deviate by ±20 beats/min or more from the predicted value. For example, the equation predicts that a sixty-year-old would have a HR max of 160 beats/min, but actual HR max might be as low as 140 beats/min or high as 180 beats/min.
Maximal stroke volume (SV max) and cardiac output also appear to decrease with age. Stroke volume can well be maintained by older athletes who have continued to train, but it will still be less than in younger athletes. Peripheral blood flow also decreases with age; however, in trained older athletes this is offset by an increased submaximal arteriovenous oxygen difference.

The decrease in VO2 max with aging and inactivity is largely explain by a decrease in HR max, SV max and arteriovenous differences. The decrease in HR max is due largely to decrease in sympathetic nervous system activity and alteration in the cardiac conduction system. The decrease in SV max is due primarily to increase total peripheral resistance due to a reduced compliance in the arteries with aging and to possible reductions in left ventricular contractility. The decrease in arteriovenous difference is related to the reduction in blood flow to the active muscles, which is possible due to the reduced cardiac output.

It is unclear how much of the decrease in cardiovascular function with aging is due to physical aging along and how much is due to deconditioning because of decreased activity. However, many studies indicate that these changes are minimized in older athletes to continue to train, which seems to indicate that inactivity might play a larger role than physical aging.

**Nervous System**

The reaction time and movement time slow down with increasing age. A slower conduction time along both the afferent and efferent nerve fibers (in peripheral nervous system) may account for some of the decrement in reaction time and movement time. The major cause is more directly related to the degeneration of the central nervous system. The excessive activity of nerve cells may have beneficial effects in preventing cognitive (reaction time) decrements in performance with increasing age.

**Bone Density**

Bone density decreases with increasing age, which means that elderly people are much more prone for bone injuries than young people whose bones have obtained full growth and maturity. This is due to a decrease in minerals (calcium and phosphorus) found in the bone, which makes the bones less dense, more porous and harder to heal from an injury. The decline in calcium and phosphorus generally starts in the early forties. The extra intake of these minerals may actually slow down or reduce their loss. The physical activity (endurance type) apparently has a beneficial effect on curbing mineral losses.

**Training Adaptation in the Aged**

The older person can adapt with various physiological training and can improve the different physiological components which are deteriorating due to ageing. Those parameters are summarized in the following Table 9.5.

**Basic Principles and Guidelines for Constructing Individualized Cardiorespiratory Endurance Exercise Program for the Aged**

(a) **Exercise Prescription**: In prescription a cardiorespiratory endurance exercise program, four basic factors are involved. They are:

(i) **Intensity**: This factor is the most critical of all in developing cardiorespiratory endurance fitness. It depends upon an individual’s present level of
fitness, their present health condition and the length of duration of the training. The intensity of work can be expressed in several ways including a percentage of maximum heart rate, a percentage of maximal oxygen consumption, number of calories consume or in METS. During submaximal and aerobic work, it has been well established that heart rate increases linearly with energy cost of the work. Because of these and for practical reasons, exercise heart rate has been used by many research workers for determining not only the physiological stress of the work, but also developing various training programs.

Evidences now indicates that a sufficient amount of cardiorespiratory endurance can be accomplished by training at somewhere between 60 and 90 percent of maximum heart rate. This represents a maximum oxygen uptake level of 50 to 80 percent. The lower heart rate figure of 60 percent of maximum represents a minimum threshold level for which it must reach in order for improvements to take place. Training at levels below this apparently results in little or no cardiorespiratory improvements.

The concept of training at individual heart rate threshold level automatically accounts for any improvement that might take place during the training program. For example, as a person’s fitness level improves and exercise heart rate for a standard workload decreases, then they automatically must work more in order to reach their minimum threshold level.

(ii) Duration: In training for cardiorespiratory endurance, it should be kept in mind that the duration and intensity of the work are interrelated. For example, research shows that improvements in cardiorespiratory endurance (about 15–20%) can be noticed with high-intensity (heart rates around 85–90% of maximum) work lasting for only 5 to 10 minutes per day. However, low intensity (heart rates around 65–75% of maximum) work shows little (about 5%) or no improvement for this period of time. The continuous training at a low intensity level for duration in between 30 to 60 minutes per day will result in significantly greater improvements than training at low intensity for short periods of time.

In general the adult (middle age and older people) do not really enjoy or tolerate exercise at a high intensity level and because running requires more energy than leisurely walking. To work-up for the lower calorie expenditure at the low intensity training level, an individual can merely walk longer and more often and achieve basically the same results as a person working to near exhaustion for a relatively short period of time.

<table>
<thead>
<tr>
<th>No.</th>
<th>Variables</th>
<th>Training effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Heart rate at standard submaximal work loads</td>
<td>Decreased</td>
</tr>
<tr>
<td>2</td>
<td>Stroke volume at standard submaximal work loads</td>
<td>Increased</td>
</tr>
<tr>
<td>3</td>
<td>Blood volume</td>
<td>Increased</td>
</tr>
<tr>
<td>4</td>
<td>Total hemoglobin</td>
<td>Increased</td>
</tr>
<tr>
<td>5</td>
<td>Oxygen pulse</td>
<td>Increased</td>
</tr>
<tr>
<td>6</td>
<td>Blood pressure (resting)</td>
<td>Decreased</td>
</tr>
<tr>
<td>7</td>
<td>Vital capacity</td>
<td>Increased</td>
</tr>
<tr>
<td>8</td>
<td>Maximal expiratory ventilation ($V_e$ max)</td>
<td>Increased</td>
</tr>
<tr>
<td>9</td>
<td>Maximal oxygen uptake ($VO_2$ max)</td>
<td>Increased</td>
</tr>
<tr>
<td>10</td>
<td>Physical work capacity</td>
<td>Increased</td>
</tr>
<tr>
<td>11</td>
<td>ECG abnormalities</td>
<td>Decreased</td>
</tr>
<tr>
<td>12</td>
<td>Muscle strength</td>
<td>Increased</td>
</tr>
<tr>
<td>13</td>
<td>Lean body weight</td>
<td>Increased</td>
</tr>
<tr>
<td>14</td>
<td>Percent body fat</td>
<td>Decreased</td>
</tr>
<tr>
<td>15</td>
<td>Serum cholesterol and triglyceride levels</td>
<td>Decreased</td>
</tr>
<tr>
<td>16</td>
<td>Flexibility</td>
<td>Increased</td>
</tr>
</tbody>
</table>
On the other hand, the training duration time refers to the length of time that the subject’s heart rate is kept within the prescribed training threshold level.

(iii) **Frequency:** In order to develop one’s cardiorespiratory responses capacity, daily exercise and training is not necessary. In fact, 3 to 5 days per week is an optimal number of workouts for developing cardiorespiratory fitness. Once a regular exercise routine has been established and the workouts have become enjoyable than the frequency of workouts may be extended to more than 3 to 5 days per week. It is important however, not to initially start out training everyday of the week since chances are good that the individual, after a couple of weeks will become completely exhausted and will more than likely quit the program.

(iv) **Mode of activity:** In general, it is agreed that activities involving the entire body such as walking, jogging, running, swimming, hiking, bicycling, canoeing, game like activities such as basketball, soccer and aerobic dancing produce the best improvements of the cardiorespiratory fitness. On the other hand, activities that are somewhat low in energy cost such as golf, bowling, softball and most calisthenics do little in way in developing physical fitness. In addition, short, anaerobic type activities that call for explosive power and speed do little in developing the aerobic fitness.

While weight-training programs may improve muscular strength and muscular endurance, they have little or no significant effects on developing one’s cardiorespiratory fitness. Because isometric type weight training exercises results in uncommonly high blood pressures, they are absolutely not recommended for the elderly and especially people with cardiovascular disease.

An outline program to develop physical fitness (safe and effective) for the old man and woman involves (a) calisthenics (for warm-up) (b) a run-walk program for developing the cardiorespiratory fitness and (c) static stretching for improving the joint mobility and preventing soreness. The subjects, in the run-walk phase, work at their own cadence and stride length that is normal and comfortable without any consideration for regulating time.

Phase I: Calisthenics for 15 to 20 minutes

Phase II: Run-walk program for 15 to 20 minutes

Phase III: Static stretching to prevent soreness and to improve joint mobility for 15 to 20 minutes.

### Table 9.6: Age–predicted maximum heart rate and cardiorespiratory training heart rates that represent the minimum and upper threshold levels

<table>
<thead>
<tr>
<th>Age(yrs)</th>
<th>Age-predicted (220-age) Maximal heart rate (beats/min)</th>
<th>Training heart rates</th>
<th>Minimum Threshold Level 60% Heart rate</th>
<th>Upper Threshold level 90% Heart rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>20</td>
<td>200</td>
<td>120</td>
<td>180</td>
<td></td>
</tr>
<tr>
<td>25</td>
<td>195</td>
<td>117</td>
<td>176</td>
<td></td>
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<tr>
<td>30</td>
<td>190</td>
<td>114</td>
<td>171</td>
<td></td>
</tr>
<tr>
<td>35</td>
<td>185</td>
<td>111</td>
<td>167</td>
<td></td>
</tr>
<tr>
<td>40</td>
<td>180</td>
<td>108</td>
<td>162</td>
<td></td>
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<tr>
<td>45</td>
<td>175</td>
<td>105</td>
<td>158</td>
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<td>50</td>
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<td>153</td>
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<td>60</td>
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<td>65</td>
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<td>70</td>
<td>150</td>
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<td>135</td>
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<td>75</td>
<td>145</td>
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<td>85</td>
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<td>122</td>
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</tr>
<tr>
<td>90</td>
<td>130</td>
<td>78</td>
<td>117</td>
<td></td>
</tr>
</tbody>
</table>
SPORT PERFORMANCE

Records in running, swimming, cycling and weight lifting suggest that human beings are in their physical prime during the late 20s or early 30s. Using a cross-sectional approach, comparing these records with national and world records for masters athletes in these events allows to examine the effects aging has on the best performers. Unfortunately, there are little longitudinal information about the effects of aging on performance, because few studies have enabled to follow physical performance in selected individuals over the span of their athletic careers.

Running Performance

Running performance decreases with age and the rate of this decline appears to be independent of distance. Performance records for both 100-m and 10-km runs decrease by about 1 percent per year from age 25 to 60. Beyond age 60, the records for men slow by nearly 2 percent per year. A sprint running test of 560 women between ages 30 and 70 revealed a steady decrease in maximal running velocity of 8.5 percent per decade. The patterns of change are about the same in both sprint and endurance running performances.

Swimming Performance

Swimming performance is affected by the aging process as much the same manner as running. The average velocities for record performances in the 100-m front crawl decrease by about 1 percent more than the running performance.

CHANGES IN MUSCULAR STRENGTH WITH AGING

It is well documented that a person’s maximal strength decreases with increasing age. Is this case due to an unavoidable effect of aging or the typical decrease in physical activity that often accompanies getting older? The answer appears to be both.

From the Figure. 9.11 below, it is apparent that strength training remains highly effective in maintaining muscular strength throughout life. However, after about age 60, strength levels fall more rapidly, independent of training. This is probably influenced by marked changes in the hormonal milieu. Both testosterone and growth

![Figure 9.10: Age-predicted maximum heart rate and cardiorespiratory training heart rates that represent the minimum and upper threshold levels.](image-url)
hormone appear to decline more dramatically after about the age of 60. Reduction in the circulating concentration of these hormones will result in a shift in the balance between muscle protein synthesis (anabolism) and protein breakdown (catabolism). The decreased strength is due to atrophy of muscle fibers. It is important to notice that with strength training, the maximal strength of a 60-year-old can exceed that of his untrained sons! And, several studies have demonstrated that strength gains are possible even at 90-year-old. So it is never too late to begin a strength training program.

**MUSCLE FIBER TYPE AND AGING**

There have been conflicting reports and myths developed regarding fiber type changes with aging. Cross-sectional studies of postmortem bodies between age 15 and 83 have suggested that fiber type composition is unchanged throughout life. This is also supported by comparing muscle biopsy results of younger and older endurance athletes. In contrast, one longitudinal study of a group of runners examined in 1974 and again 1992, suggested that training could play a role in fiber distribution. Those athletes who continued training showed unchanged fiber composition. Those who stopped training appeared to have greater slow-twitch fiber percentage. This was primarily due to selective atrophy of the fast fibers. This is not difficult to explain since they are seldom recruited. There is also some evidence that the actual number of fast motor units decreases slightly with aging after age 50, about 10 percent per decade. The reasons or mechanisms for such a change are unclear. So, the net effect of aging for the endurance athlete is unchanged fiber composition or a slight relative increase in slow fiber type due to selective fast fiber loss. The Fast motor units do not become slow motor units.

**MUSCLE ENDURANCE CAPACITY AND AGING**

The good news for the endurance athlete is that there appears to be little change in skeletal muscle oxidative capacity with age, as long as training is maintained. The number of capillaries per unit area of muscle is the same in young and old endurance athletes. Oxidative enzyme levels are similar or slightly lower in older athletes. This small decrease is probably attributable to decreased training volume in the older athletes. Furthermore, it appears that the older individual who starts endurance training retains the potential to improve muscle endurance capacity.

**SUMMARY**

1. Growth refers to an increase in the size of the body or any of its parts. Development refers to differentiation along specialized lines of function, so it reflects the functional changes that occur with growth. Finally, maturation refers to the process of talking on the adult.

![Age related change of muscular strength in trained and untrained men.](image-url)
form and becoming fully functional being considered. Chronological age refers to the period that has elapsed beginning with an individual's birth and extending to any given point in time. It has been observed that a program of hormonal activity that stimulates somatic growth proceeds during the childhood and adolescent years. This evolution of body size increase is one example of biologic maturation, a series of developmental changes that culminates in the adult stage or complete biologic maturation.

2. The extent of biologic maturation can be estimated by percentage of estimated adult height and weight, wrist bone age as measured by radiography and after the onset of puberty by the progression of appearance of secondary sexual characteristics. Change in height is assessed in terms of centimeters per year and change in weight in terms of kilograms per year. The peak rate of growth in height occurs at approximately 12.0 years in girls and 14.0 years in boys. The same overall trend for the rate of change in weight is revealed from the Figure in the text. As with height, the peak rate of growth in body weight occurs at approximately 12.0 years in girls and 14.5 years in boys, which is slightly later than height.

3. The function of almost all physiological systems improves until full maturity is reached or shortly before. After that, functional plateaus for a period of time before starting to decline with advancing age. The motor ability of boys and girls generally increases with age for the first 18 years although girls tend to plateau at the age of puberty. Strength improves as muscle mass increases with age. Gains in strength also depend on neural maturation, because neuromuscular control is limited until myelination has been completed usually around sexual maturity. Girls experience a more gradually increase in strength and do not exhibit a marked change in their rate of change gain with puberty.

4. Lung function changes markedly with age. All lung volumes increase until growth is completed. Peak flow rates follow the same pattern. Heart size is directly related to body size, so children have smaller hearts than the adults. As a result of this and a smaller blood volume the child has a smaller stroke volume capacity.

5. Resting heart rate falls progressively during childhood. The decline in resting heart rate with age parallels that of weight-relative basal metabolic rate (BMR) and it is tempting to conclude that this association is causal i.e. the fall is BMR as body size increases is the result of a decline in resting heart rate, while other factors responsible for metabolic rate (cardiac stroke volume, peripheral arteriovenous oxygen uptake) grow in relation to body size. Both longitudinal and cross-sectional studies in children have indicated that maximal heart rate does not change during childhood. Since resting heart rate falls with age in children while maximal values are stable.

6. Since maximal heart rate during childhood is stable, it follows that (1) the rise in maximal cardiac output in children as they grow must be entirely due to an increase in stroke volume and (2) during the same time period heart rate does not contribute to any increases in absolute maximal oxygen uptake. Similarly, it can be summarised that maximal stroke volume increases in proportion to body dimension in a manner similar to that of oxygen uptake. Stroke volume increases with age in children in close relationship with body dimensions while resting heart rate falls, it would be expected that resting cardiac output relative to body weight or surface area would progressively decline during the childhood.

7. During exercise (rhythmic) the rise in systolic blood pressure is proportional to exercise intensity and to the overall metabolic level. Such pattern is operative in all healthy individuals, irrespective of age. A lower exercise blood pressure in the young child is in line with the lower cardiac output and stroke volume.

8. As pulmonary and cardiovascular function improves with continued development, so does aerobic capacity. VO₂ max express in lit/min, peaks between ages 17 and 21 years in
males and between 12 and 15 years in females, after which it steadily decreases. Children can not maintain as a first a running pace as the adult because of basic differences in economy of effort. Running economy increases and this improve their distance-running pace even if the children are not training and if their VO₂ max values do not increase. The child’s ability to perform anaerobic activities is limited.

9. Training can improve the strength, aerobic capacity and anaerobic capacity of the young athlete. Generally, the youngster will adapt well to the same type of training routine used by the mature athlete. But training programs for children and adolescents should be designed specifically for each age group, keeping in mind the development factors associated with that age. Boys and girls were discouraged from using free weights for fear that they might injure themselves and prematurely stop the growth process. Furthermore, many scientists speculated that resistance training would have little or no effect on the muscles of prepubescent boys because their levels of circulating androgens were still low. However, it has been established that strength training on children with proper guidance is beneficial.

10. The area of exercise physiology relates to the various physiological effects, training adaptation and basic principles and guidelines for constructing an individualized cardiorespiratory endurance exercise program for the aged that accompany the aging process. The level of physical activity begins to decline soon after people reach adult maturity. As an individual gets older, there is a decline in muscle size. It is believed that this decline is due, in part, to a reduced amount of protein as well as a decline in the number and size of muscle fibers. With advancing age, there is a general trend to accumulate in increase in body fat. As one gets older, their lean body weight decreases.

11. Cardiovascular functions are also change with increase in age. These are maximum heart rate, stroke volume, cardiac output, etc. The reaction time and movement time slow down with increasing age. Bone density decreases with increasing age, which means that elderly people are much more prone for bone injuries than young people whose bones have obtained full growth and maturity.

12. Running and swimming performance decreases with age and the rate of this decline appears to be independent of distance. It is well documented that a person’s maximal strength decreases with increasing age. Is this due to an unavoidable effect of aging or the typical decrease in physical activity that often accompanies getting older? The answer appears to be both.

**Review Questions**

1. Define growth and development? What is maturity status? Differentiate growth, development and maturation.
2. What do you mean by growth spurt? Discuss with the growth curve.
3. What typical change takes place in various physical and physiological variables during growth and development period?
4. What are the effect of exercises on growth? What changes occur in stroke volume for a fixed rate of work as the child grows?
5. What changes occur in sub-maximal and maximal heart rate as the child grows?
6. How dangerous is resistance training in children? What advised would you give to these youngsters if they wanted to improve their strength? Can they improve strength, and if so, how does this occur?
7. How does physical activity and regular training affect the growth and maturation process?
8. What cardiovascular changes occur during ageing? How do these changes affect maximal oxygen uptake capacity?
9. Describe the changes in VO₂ max with age. How do trained individuals differ from untrained subjects?
10. Discuss the changes in maximum heart rate with age. How does training alter this relationship?
Physical and physiological differences between men and women

Gender differences in endurance performance and training
- The maximal oxygen consumption
- The lactate threshold
- Efficiency
- Fat metabolism
- Muscle strength and power
- Training volume

Physiological adaptations to exercise training
- Athletic ability
- Menstruation and athletic performance
- Menopause
- Menstrual dysfunction
- Pregnancy
- Eating disorders
- Amenorrhea
- Female athlete triad

Chapter 10

Women Athletes

Physical and physiological differences between men and women

The body of the men and women reacts differently to varying degrees of physical stress, and never two bodies react exactly the same way to the same physical stress. For everyone to get the maximum benefit from training, and trainer must be aware of these differences and plan the training schedule to provide maximum benefit for everyone. They must also be aware of the physiological differences between men and women. While they require equal efforts of men and women during the training period, they must also realize that women have physiological limitations which generally preclude equal performance. The following paragraphs describe the most important physical and physiological differences between men and women.

Body size

The average 18-year-old man is 68.0 to 70.2 inches tall and weighs 125.0 to 144.8 pounds, whereas the average woman of the same age is 60.0 to 64.4 inches tall and weighs 110.0 to 126.6 pounds. This difference is size affects the absolute amount of physical work that can be performed by men and women. Accordingly, the body surface area is also varying between men and women of same ages.

Until puberty, females and males do not differ significantly in most measurements of body size and composition. But at puberty, due to influences of estrogen and testosterone hormones, body composition begins to change markedly. Estrogen causes increased fat deposition in females, particularly in the hips and thighs and an increased rate of bone growth, such that bones of females reach their final length earlier than in male.

Muscles

Men have 50 percent greater total muscle mass, based on weight, than do women. A woman who is the same size as her male counterpart is generally only 80 percent as strong. Therefore, men usually have an advantage in strength, speed and power over women.
In males testosterone causes increased bone formation, which leads to larger bones, as well as increased protein synthesis, which leads to increased muscle mass. As a result, adolescent males are larger and more muscular than females, and these characteristics continue into adulthood.

**Body Fat**

Women carry about 10 percentage points more body fat than do men of the same age. Men accumulate fat primarily in the back, chest and abdomen; women gain fat in the buttocks, arms and thighs. Also, because the center of gravity is lower in women than in men, women must overcome more resistance in activities that require movement of the lower body. So, women are carrying extra body weight as subcutaneous adipose tissue as compare to their men counterparts which hinder the speed ability. The total body fat percent of young sedentary women is about 25 percent whereas a sedentary men of same age is 5 to 7 percent less as female.

In female estrogen hormone influence fat deposition particularly in the thighs and hips and this increase in fat deposition is the result of lipoprotein lipase activity in these areas. This enzyme is mainly responsible for storing fat in adipose tissue. The decreased lipolytic activity makes it difficult for women to lose fat from these areas.
Table 10.1: Relative body fat% for men and women of various ages

<table>
<thead>
<tr>
<th>Age Groups (Years)</th>
<th>Relative body fat %</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Men</td>
</tr>
<tr>
<td>15–19</td>
<td>13–16</td>
</tr>
<tr>
<td>20–29</td>
<td>15–20</td>
</tr>
<tr>
<td>30–39</td>
<td>18–26</td>
</tr>
<tr>
<td>40–49</td>
<td>23–29</td>
</tr>
<tr>
<td>50–59</td>
<td>26–33</td>
</tr>
<tr>
<td>60–69</td>
<td>29–33</td>
</tr>
</tbody>
</table>

Bones

Women have less bone mass than men, but their pelvic structure is wider. This difference gives men an advantage in running efficiency. Estrogen increases the growth rate of bone allowing the final bone length to be reached within the 2 to 4 years following the onset of puberty. As a result female grow very rapidly for the first few years following puberty, then cease to grow. Males have a much longer growth phase, allowing them to attain a greater height. Because of these differences, compare to fully matures males, fully matures females are on average nearly:

i. 13 cm shorter
ii. 14 to 18 kg lighter to total weight
iii. 18 to 22 kg lighter in fat free mass
iv. 3 to 6 kg heavier in fat mass
v. 6 percent to 10 percent higher in relative body fat

Anthropometric Measurements

Anthropometric measurements at maturity differ substantially between the sexes. Women have narrower shoulders, broader hips, and smaller chest diameters and tend to have more fat in the hips and lower body, whereas men carry more fat in the abdomen and upper body. The below represents the skinfold thickness (four sites), circumference of chest, abdomen, hips and thigh and four diameters of both young and middle aged men and women.

Heart Size and Heart Rate

The average woman’s heart is 25 percent smaller than the average man’s. Thus, the man’s heart can pump more blood with each beat (i.e. stroke volume). The larger heart size contributes to the

Table 10.2: Anthropometric measurements for young and middle aged men and women

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Young*</th>
<th>Middle-aged**</th>
<th>Women</th>
<th>Middle-aged**</th>
</tr>
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<tbody>
<tr>
<td>Skinfolds</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>i. Triceps</td>
<td>7.9</td>
<td>18.5</td>
<td>12.8</td>
<td>22.2</td>
</tr>
<tr>
<td>ii. Suprailliac</td>
<td>19.3</td>
<td>22.0</td>
<td>17.2</td>
<td>17.3</td>
</tr>
<tr>
<td>iii. Abdomen</td>
<td>16.0</td>
<td>30.0</td>
<td>15.1</td>
<td>29.6</td>
</tr>
<tr>
<td>iv. Thigh</td>
<td>14.9</td>
<td>22.2</td>
<td>31.8</td>
<td>33.1</td>
</tr>
<tr>
<td>Circumference</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>i. Chest</td>
<td>97.4</td>
<td>96.3</td>
<td>85.2</td>
<td>87.1</td>
</tr>
<tr>
<td>ii. Abdomen</td>
<td>84.0</td>
<td>91.1</td>
<td>75.3</td>
<td>82.7</td>
</tr>
<tr>
<td>iii. Hips</td>
<td>96.9</td>
<td>98.4</td>
<td>95.9</td>
<td>97.5</td>
</tr>
<tr>
<td>iv. Thigh</td>
<td>58.0</td>
<td>59.0</td>
<td>57.0</td>
<td>57.6</td>
</tr>
<tr>
<td>Diameters</td>
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</tr>
<tr>
<td>i. Biacromial</td>
<td>40.4</td>
<td>41.5</td>
<td>36.5</td>
<td>36.7</td>
</tr>
<tr>
<td>ii. Bi-iliac</td>
<td>28.4</td>
<td>31.4</td>
<td>28.4</td>
<td>31.2</td>
</tr>
<tr>
<td>iii. Knee</td>
<td>9.5</td>
<td>10.1</td>
<td>8.9</td>
<td>9.6</td>
</tr>
<tr>
<td>iv. Elbow</td>
<td>6.0</td>
<td>—</td>
<td>7.0</td>
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</tbody>
</table>

*: Data from Wilmore Behnke (1969 and 1970)

**: Data from Pollock et al. (1975 and 1976)
slower resting heart rate (five to eight beats a minute slower) in males. The normal resting heart rate of young adult males is 72 beats/min whereas the normal heart rate of women of similar age is 76 beats/min. This lower heart rate is evident both at rest and at any given level of submaximal exercise. Thus, for any given work rate, the faster heart rate means that most women will become fatigued sooner than men and hence working efficiency is also less in case of women.

For the same rate of work, trained women generally have cardiac outputs similar to those of comparably trained men, but this is achieved by higher heart rates and lower stroke volumes. The lower stroke volume in women is due to their smaller left ventricle and lower blood volume, both the result of women’s smaller body size.

Flexibility

Flexibility, which may be define as the athlete’s ability to move a joint through a normal range of motion without undue musculotendinous stress. It has been advocated for enhanced performance as well as reduced injury rates.

Women generally are more flexible than their men counterpart which may be due to the hip flexion is greater in women than men. Also some scientists studied number of male and female athletes of different sports and found that the women were more flexible as compare to men athletes.

Lungs and Respiratory Response

The lung capacity of men is 25 to 30 percent greater than that of women. This gives men still another advantage in the processing of oxygen and in doing aerobic work such as running, swimming, cycling, etc. The bigger size in lung in male than female is due to their bigger trunk size. Again the bigger trunk size is due to they are taller in height as compare to women.

The differences between men’s and women’s respiratory responses to exercise are also due largely to body size differences. Breathing frequency when working at the same relative power output differs little. However, if consider the same absolute power output, women tend to breathe more rapidly than men, probably because when both subjects are at the same absolute power output the women is working at a higher percentage of her VO2-max.

Tidal volume and ventilatory volume are generally smaller in women at the same relative and absolute power outputs, up to and including maximal levels. Most highly trained female athletes have maximal ventilatory volumes below 125 L/min., but highly trained men have maximal values of 150 L/min. and higher, some exceeding even 250 L/min. Again these differences are also closely associated with body size.

Response to Heat

A woman’s response to heat stress differs somewhat from a man’s. Women sweat less, lose less heat through evaporation and reach higher body temperatures before sweating starts. Nevertheless, women can adapt to heat stress as well as men. Regardless of gender, players with a higher level of physical fitness generally better tolerance and adapt more readily to, heat stress than do less fit sports person.

Other Factors

Knowing the physiological differences between men and women is just the first step in planning physical training for a group. Trainers/Coaches need to understand other factors too. Women can exercise during menstruation; it is, in fact, encouraged. However, any unusual discomfort, cramps, or pains while menstruating should be medically evaluated. Pregnant women cannot be required to exercise without a doctor’s approval. Generally, pregnant women may exercise until they are close to childbirth if they follow their doctors’ instructions. The safety and health of the mother and fetus are primary concerns when dealing with exercise programs. Vigorous activity does not harm women’s reproductive organs or cause menstrual problems. Also, physical fitness training need not damage the breasts. Properly fitted and adjusted bras, however, should be worn to avoid potential injury to unsupported breast tissue that may result from prolonged jarring during exercise. Although female players must sometimes be treated differently from males, women can reach high levels of physical performance. Trainers must use common sense to help both male and female achieve acceptable levels of fitness.
GENDER DIFFERENCES IN ENDURANCE PERFORMANCE AND TRAINING

Historically, there is no doubt that sport has been a center of faulty assumptions and sexism where female athletes are concerned. Social issues, and misunderstanding about female physical and medical limitations (or the presumption of limitations) conspired to slow the development of female performance for many years (the marathon for women was only added to the Olympic schedule in 1984), but this is not true at present, at least among young athletes. Among master’s athletes, we still see greatly reduced participation by the older female age groups. This participation difference will no doubt diminish over the next couple of decades. As a result, performances by the oldest females will probably improve more rapidly than those of the oldest males, as this new generation of well trained young female athletes’ moves into age-group competition, and are joined by more and more talented “late bloomers.”

“Old” Social norms and habits are still having negative consequences on participation and performance by older (50 + years) females. Modern female athletes have repeatedly demonstrated these norms (“women are not built to run long distances”) are not true. Currently, teenage girls are encouraging their formally sedentary mothers and even grandmothers to take up exercise. This transfer of knowledge and norms Upstream is the reverse of what we traditionally seen in males (Father teaching his boy all he knows). However, this is a transitional period for women in sport, so the knowledge transfer across generations is helping to speed the development of women’s masters sport.

Having said all that, there are some physiological differences between the sexes that impact performance in females independent of age. Some years ago, when the marathon was first becoming a competitive event for women, the rapid improvement in female times led some to predict that female performances would soon equal those of men in the marathon. This has not happened, and it will not. The current world record for women is 2:21, compared to 2:06:50 for the men, a difference in speed of about 10 percent. This same 10 percent gap is present across the distance running performance spectrum. The reason for the performance gap is not that women don’t train as hard as men. There are some important physiological differences between the sexes that cannot be overlooked or overcome.

The Maximal Oxygen Consumption

The “typical” young untrained male will have an absolute VO₂ max of 3.5 liters/min, while the typical same-age female will be about 2 liters/min. This is a 43 percent difference! Where does it come from? First, much of the difference is due to the fact that males are bigger in size, on average, than females. The humans are all (sort of) geometrically similar, so heart size scales in proportion to lean body size. If we divide VO₂ by bodyweight, the difference is diminished (45 ml/min/kg vs 38 ml/min/kg) to 15 to 20 percent, but not eliminated. What is the source of this remaining difference?

If we compare average body fat in males and females, we find part of the answer. Young untrained women average about 25 percent body fat compared to 15 percent in young men. So, if we factor out body composition differences by dividing VO₂ by lean body mass (Bodyweight minus estimated fat weight)) the difference in maximal O₂ consumption decreases to perhaps 7 to 10 percent. Keep in mind though that this is only a meaningful exercise on paper. A female athlete cannot expect to improve her performance by reducing her body fat down to the sub 7 percent levels that are often observed in elite males. The health consequences for the female are too severe!

To find an explanation for the remaining 10 percent difference we must go back to the key limitation on VO₂ max, oxygen delivery. On average females have lower blood hemoglobin (13.5 gm percent) content than males, up to 10 percent lower. Finally, there is some evidence, that the female heart is slightly smaller relative to body size than the male heart. Several ECG and echocardiographic studies also suggest that the young female heart exhibits less enlargement in response to either endurance or resistance training than the male heart. This may be due to differences in androgen receptor density in the female heart. A smaller heart would be expected to be a less effective pump as well.
Slightly lower oxygen carrying capacity of the blood (lower hemoglobin levels) plus a somewhat smaller or less adaptive heart are sufficient to account for the gender differences in maximal oxygen consumption that are independent of body size and fat percentage.

It is worth noting here the results of a 1993 study by Spina et al. Their data suggested that in previously sedentary older men and women (60 to 65 years old) who trained for 9 months to a year, both men and women increased their VO2 max by the same amount (an average of 20 percent). However, the

![Fig. 10.4: Changes in maximum oxygen uptake capacity in male and female with increase in age](image)

**Table 10.3: Maximal oxygen uptake (ml/kg/min) in various population groups of both sexes**

<table>
<thead>
<tr>
<th>Non Athletes</th>
<th>Age</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>10–19</td>
<td>47–56</td>
<td>38–46</td>
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<td></td>
<td>20–29</td>
<td>43–52</td>
<td>33–42</td>
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<td>40–49</td>
<td>36–44</td>
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<td>50–59</td>
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<td>60–69</td>
<td>31–38</td>
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<tr>
<td></td>
<td>70–79</td>
<td>28–35</td>
<td>20–27</td>
</tr>
<tr>
<td>Athletes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseball/soft ball</td>
<td>18–32</td>
<td>48–56</td>
<td>52–57</td>
</tr>
<tr>
<td>Basketball</td>
<td>18–30</td>
<td>40–60</td>
<td>43–60</td>
</tr>
<tr>
<td>Bicycling</td>
<td>18–26</td>
<td>62–74</td>
<td>47–57</td>
</tr>
<tr>
<td>Canoeing</td>
<td>22–28</td>
<td>55–67</td>
<td>48–52</td>
</tr>
<tr>
<td>Football</td>
<td>20–36</td>
<td>42–60</td>
<td>—</td>
</tr>
<tr>
<td>Gymnastics</td>
<td>18–22</td>
<td>52–58</td>
<td>36–50</td>
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<tr>
<td>Ice Hockey</td>
<td>10–30</td>
<td>50–63</td>
<td>—</td>
</tr>
<tr>
<td>Jockey</td>
<td>20–40</td>
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<td>—</td>
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<tr>
<td>Orienteering</td>
<td>20–60</td>
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<td>Racquetball</td>
<td>20–35</td>
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<td>Rowing</td>
<td>20–35</td>
<td>60–72</td>
<td>58–65</td>
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<tr>
<td>Skiing, alpine</td>
<td>18–30</td>
<td>57–68</td>
<td>50–55</td>
</tr>
<tr>
<td>Skiing, nordic</td>
<td>20–28</td>
<td>65–94</td>
<td>60–75</td>
</tr>
<tr>
<td>Ski jumping</td>
<td>18–24</td>
<td>58–63</td>
<td>—</td>
</tr>
<tr>
<td>Soccer</td>
<td>22–28</td>
<td>54–64</td>
<td>50–60</td>
</tr>
<tr>
<td>Speed skating</td>
<td>18–24</td>
<td>56–73</td>
<td>44–55</td>
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<tr>
<td>Swimming</td>
<td>10–25</td>
<td>50–70</td>
<td>40–60</td>
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<tr>
<td>Track &amp; field, discus</td>
<td>22–30</td>
<td>42–55</td>
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</tr>
<tr>
<td>Track &amp; field, running</td>
<td>18–39</td>
<td>60–85</td>
<td>50–75</td>
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<tr>
<td></td>
<td>40–75</td>
<td>40–60</td>
<td>35–60</td>
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<tr>
<td>Track &amp; field, shot put</td>
<td>22–30</td>
<td>40–46</td>
<td>—</td>
</tr>
<tr>
<td>Volleyball</td>
<td>18–22</td>
<td>—</td>
<td>40–56</td>
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<tr>
<td>Weightlifting</td>
<td>20–30</td>
<td>38–52</td>
<td>—</td>
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<tr>
<td>Wrestling</td>
<td>20–30</td>
<td>52–65</td>
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</tbody>
</table>

Adopted from Wilmore and Costill (2005)(3)
mechanism of improvement was different. The men improved primarily by increasing maximal cardiac output due to higher stroke volume. However, the older women did not demonstrate any increase in cardiac performance, but rather increased oxygen consumption by improving oxygen extraction by the working muscles, due to greater capillarization and more number of mitochondria. This data supports previous studies in 60+ year old women that show no cardiac hypertrophy in response to endurance training.

It has been reported that females demonstrate a different pattern of cardiac adaptation to exercise, which may become more dissimilar with age. They also generally have a lower hemoglobin level by several percent. The net effect is a small but significant difference in maximal oxygen consumption, even among similarly trained males and females, and after scaling for differences in size and body composition.

It is important to make note of the fact that these differences are “on average”. In reality, there are many women with significantly higher VO2 max values than average men. However, if we look at the “best of the best”, the differences persist. Using Cross Country skiing as an example (Norway), the highest reliable values for VO2 max recorded in national team Cross Country skiers are about 90 ml/min/kg. The very best Norwegian woman has been measured at 77 ml/min/kg, a 17 percent difference was noted. So, while this woman will outperform 99.9 percent of all men, she will not outperform the national team level of males.

The question here is, do women demonstrate a different pattern or capacity for peripheral adaptations then men? As best as it was known, the answer is NO.

First, Female skeletal muscle is not distinguishable from male skeletal muscle. Second, within some margin of error, the fiber type distribution (percentage of slow versus fast fibers) is not different in the male and female population. Third, male and female skeletal muscle responds similarly to endurance exercise. Finally, elite female endurance athletes have similar lactate threshold values compared to men when expressed as a percentage of their VO2 max. Elite women perform at the same high percentage of their maximal oxygen consumption as their male counterparts.

Earlier it was proposed that women would actually perform better at ultra-endurance type activities. This theory has been disproved both in the laboratory and in practice as a performance difference persists in the ultra marathon events. It has been suggested that women had an edge in the really long events. They discussed a study in which a group of male and female runners who were matched for marathon time were raced head to head in the Comrades marathon, a 90k race. The women won by 54 minutes, suggesting a female edge in longer events. The problem with this study is that when you match men and women for performance, the women are relatively better runners and probably have a higher slow twitch fiber percentage. This advantage becomes bigger in an ultra distance event.

The fact remains that the performance gap between male and female record holders in the really long running races 50k to 6 days is actually more on the order of 15 to 20 percent, instead of the 10 percent difference for the standard distances. Part of this larger gap may be to lower participation, and the fact that the most talented females have not yet tested themselves over the ultra distances. But at elite level the gap may not disappear.

Efficiency

The next component of endurance performance is efficiency which of course has different constraints, depending on the sport. The research information comparing the efficiency of female and male
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athletes is both sparse and inconclusive. In running, for example females have been found to be more, less and equally efficient compared to males depending on the specific study. Some of this confusion comes down to how the differences in bodyweight and body fat were accounted for.

After looking over some of the research comparing running economy between genders, it may be argued that any inherent economy differences in male and female runners are smaller than the individual variation in running economy that is observed among runners, independent of gender. It may be supported that argument by suggesting the differences in VO2 max observed between elite males and females are sufficient to explain the “10 percent gap” without other factors being involved.

Now, if we look at efficiency/economy differences in other sports, things mostly boil down to body shape/anthropometric differences. In situations like running or cycling, these may actually favor females in general, due to narrower upper bodies for a given total body mass, and potentially less wind or water drag. It has been seen the differences in VO2 max alone are sufficient to explain the gender performance gap in rowing.

**Fat Metabolism**

In 70s, a theory got started that said “Since women have more fat stores, they will be better at utilizing fat during endurance performance when glycogen stores are depleted.” One of the supporting pillars of the theory was that it had been noticed by one female runner/author how “fresh” many female runners looked as they crossed the finish line! This shaky theory was crushed under the harsh light of science. In 1979, Costill and colleagues compared males and females who were equally trained during a 60 minute treadmill run. There were no differences in any measures of fat metabolism was observed. They even took some muscle out of the runners’ legs and tested it in a test tube. Still no difference was noticed. This is an often repeated finding among similarly trained males and females. There is no gender difference in the ability of men and women to burn fat!

Of the three critical components of endurance performance, the only one that is clearly and consistently depressed in females is the maximal oxygen consumption. Even after accounting for differences in bodyweight and body fat percentage, a gap of roughly 10 to 15 percent remains.

**Muscle Strength and Power**

Although maximal muscular strength and anaerobic power has little to do with endurance performance, there are many events which can be classified as “power-endurance” events. These events ranging from 2 to about 8 minutes require some combination of aerobic and anaerobic capacity. For this reason, it is important to also consider this “anaerobic” component of the performance. When it is discussed about anaerobic capacity, the critical determinant is muscle mass. Females, on average, have less total muscle mass than males. As a result, maximal strength measures as well as maximal power measures (power = force/time) are reduced. Gross measures of upper body strength suggest an average 40 to 50 percent difference between the sexes, compared to a 30 percent difference in lower body strength. About power, Maud and Schultz compared 52 men and 50 women, all about 21 years old using a maximal power test on a bicycle ergometer. Peak power was about 60 percent lower for the females when comparing absolute values. But, the men were heavier. Peak power per kg bodyweight was more similar, 9.3 watts/kg vs 7.9 watts/kg for the women, an 18 percent difference. Finally, when power outputs were adjusted for fat-free mass, the values were 10.4 watts/kg and 9.9 respectively. This 5 percent difference was not statistically significant. Numerous other studies using different techniques have also demonstrated that when it has been look at muscle quality, male and female muscle is not different. Within the accuracy of current comparative techniques, it appears that the strength and power differences between the sexes are a function of muscle quantity only. Biomechanical differences probably play a role in some situations, but this will be very sport specific.

**Training Volume**

There are small but important differences in the recovery capacity of male and females, at least when pushed to the extremes of elite level training. Again for example of world class Cross Country skiers, it appears that the best women perform optimally at
a training volume that is perhaps 10 to 15 percent lower than that observed in the best men. Increasing the volume in the women does not improve results, and often leads to overtraining. The general consensus is that the difference lies in the higher average testosterone levels of males. Remember, testosterone is an anabolic hormone. This means it is critical for tissue growth and repair. It has been reported that only one of the Norwegian female national team skiers has been able to maintain the average yearly training volume (measured in hours) that is maintained by the entire Russian female team. At any rate, we should be aware that there is probably a small gender difference in recovery capacity from hard or high volume training, in addition to the individual variation that is observed.

Feminity

The inherent hormonal and morphological factors are responsible for femininity and masculinity and not strenuous exercise as some people would like to believe. There is no evidence to indicate that participation in vigorous exercise and sports will masculinize women. On the contrary it is a generally accepted fact that such activity tends to make a women more graceful and feminine because it brings about better muscle tones, replaces fatty tissue with from musculature, and improves overall fitness, just to mention a few. It has been agreed that femininity is the general rule among today and female athlete rather than the exception.

Physiological Adaptations to Exercise Training

Basic physiological function both at rest and during exercise changes substantially with physical training. The emphasizing areas in which how women adapt to chronic exercise as compare to men.

(i) Body composition: With either cardiorespiratory endurance training or strength training, both women and men experience-
- Losses in total body mass
- Losses of fat mass
- Losses of relative fat and
- Gains in fat free mass

Women generally gained much less in fat-free mass (FFM) than men do. With the exception of FFM, the magnitude of the change of body composition appears to be related more to the total energy expenditure associated with the training activities than to the participant’s sex. As for FMM, significantly more is gained in response to strength training than with endurance training, and the magnitude of these responses is much less in women, due to their hormonal difference.

(ii) Neuromuscular adaptation: To prescribing strength training programs for girls and women was not believed capable of gaining strength due to their extremely low levels of the male anabolic hormones until 1970s. Paradoxically, many people also generally feared that strength training would masculinize women. During the 1960s and 1970s, however, it became evident that many of the female athletes of United States were not doing better in international competition mainly because they were weaker than their competitors. Gradually research demonstrated that women can gain strength considerably from strength training programs and that strength gain programs usually not accompanied by large increases in muscle bulk. It has been reported that women can experience that major increases in strength (20–40 %) as a result of resistance training and the magnitude of these changes is similar to that in men. These gains are likely due more to neural factors, because the increase in muscle mass is generally small.

(iii) Cardio-respiratory adaptation: Cardiovascular and respiratory changes that accompany cardiorespiratory endurance training do not appear to be sex specific. Women experience the same relative increase in VO₂ max that men experience with cardiorespiratory endurance training. Women can experience major increases in endurance capacity of 10 to 40 percent with aerobic training. So, women respond to physical training in the same manner as men do.

Athletic Ability

Women are outperformed by men in almost all sports, events or activities. This is quite obvious in activities such as the shot put in track and field,
where high levels of upper body strength are crucial to successful performance. In 400 m freestyle swimming, however, the swimming time for women in the 1924 Olympic Games was 16 percent slower than that of men, but this difference decreased to 11.6 percent in the 1946 Olympics, and to only 6.9 percent in the 1984 Olympics. The fastest women’s 800 m freestyle swimmer in 1979 swam faster than the world record holding man for the same distance in 1972. Therefore, in this particular event the gap between the sexes is narrowing, and this is also true for other events and for other sports. Unfortunately, making valid comparisons through the years has been difficult because the degree to which an activity has been emphasized, or its popularity is not constant and other factors, such as opportunities to participate, coaching, facilities, and training techniques, have differed considerably between the sexes over the years.

**Menstruation and Athletic Performance**

Alterations in athletic performance experienced during different phase of the menstrual cycle are subject to considerable individual variability. Some woman have absolutely no noticeable change in their performance ability at anytime during their menstrual cycle, yet other have considerable difficulty in either the pre flow or the early flow phase or during both. Several studies have suggested that athletic performance is best during the immediate post flow period up to the 15th day of the cycle, with the first day of the corresponding to the initiation of the flow.

Number of studies has been done that and tried to show some decrease in performance during menstruation. Whenever some decrease in performance was actually found it is difficult to sufficiently isolate the variable such that the decrease can be explicitly linked to menstruation. In other words, there is no conclusive clinical proof that getting period negatively affects the athletic performance. In addition, there is a great deal of individual variation among women. For example, in a study of Finnish female athletes at the Helsinki Olympic Games, five set personal bests while menstruating. In another study of Prague track and field athletes, 29 percent of the competitors attested that they had done the same. Results vary wildly from study to study, which shows that individual physical variation combined with psychological factors around menstruation produces highly diverse outcomes with regard to performance during menstruation. In terms of the effect of exercise on menstruation, no clinical evidence has been found to show that moderate regular exercise affects the cycle negatively. In the next section on amenorrhea, it is pointed out some of the problems associated with heavy training and low body fat levels. The majority of women find that exercise alleviates many of the negative symptoms associated with premenstrual and menstrual discomfort.

However, from currently available information, it can be concluded that performance in some women can be affected by the phase of their menstrual cycle, but that many, if not most, women are not affected. Any women who experiences premenstrual syndrome or dysmenorrhea will likely not perform as well while she is experiencing symptoms. For these women, some degree of control over their menstrual cycle is possible through the use of low dose oral contraceptives.

**Menarche**

The menarche is the first menstrual flow of women. Delayed menarche has been reported in young athletes involved in certain sports and activities, such as gymnastics and ballet. The mean age for menarche is generally 12.0 years. For gymnasts, the mean age appears to be closer to 15 years. Frisch has hypothesized that menarche is delayed 5 months for each year for training prior to menarche, implying that training causes delayed menarche. Malina, however, has postulated that late maturers, such as those with delayed menarche, are more likely to be successful in sports such as gymnastics because of their small, lean bodies. However, there is not strong evidence to supports the contention that the intense training for the sports delayed menarche.
MENSTRUAL DYSFUNCTION

Female athletes can experience disruptions of their normal menstrual cycle. These disruptions are collectively referred to as menstrual dysfunction, of which there are several types.

i. Eumenorrhea: It is the term for normal menstrual function.

ii. Oligomenorrhea: It refers to abnormality infrequent or scant menstruation.

iii. Primary amenorrhea: Primary amenorrhea refers to the absence of menarche in women 18 years of age and older women who never began menstruating.

iv. Secondary amenorrhea: Some athletes with previously normal menstrual function have reported the absence of menstruation for months or even years when they have trained intensely in sports such as figure scatting, ballet, gymnastics, body building, cycling and distance running. This phenomenon is referred to as secondary amenorrhea.

The prevalence of secondary amenorrhea and oligomenorrhea among athletes is not well documented, but is estimated to vary from approximately 5 to 40 percent or higher, depending on the sports or activity and the level of competition. A high percentage of female athletes in endurance and appearance sports performance secondary amenorrhea, where normal menstrual function is lost for months or even years. This appears to be reversible with reductions in the intensity and volume of training and an increase in caloric intake. The cause of these conditions in athletes is unknown, but current evidence implicates inadequate nutrition as a primary cause of secondary amenorrhea. In addition, hormonal changes from exercise and training might disrupt gonadotropic hormone releasing hormone secretion, which is needed to direct the normal cycle.

The high level training leads to menstrual dysfunction, but the true cause might involve one or more factors associated with high level training. Some factors that have been proposed include the following:

a. Previous history of menstrual dysfunction: A previous history of menstrual dysfunction can be one of a number of factors, but is probably not the primary factor, in the development of secondary amenorrhea.

b. Acute effects of stress: The amenorrhic runners associated more stress with their training than did eumenorrhic runners. However, scientific tests faild to detect any differences in the amenorrhic runners’ levels of anxiety, depression and other states that reflects stress.

c. High quantity and intensity of training: It has been established that high quantity of training is associated with secondary amenorrhea. However, the effect of training intensity on menstrual function has not been well documented. Training at a high intensity, because of the high physical stress placed on the body, might be more closely linked with secondary amenorrhea.

d. Low body weight and body fat: Excessive leanness, undernutrition, or both, have long been associated with amenorrhea.

e. Inadequate nutrition and eating disorder.

f. Hormonal alterations.

A Proactive Approach

The American College of Sports Medicine (ACSM) has taken a proactive approach to menstrual dysfunction in athletes, recommending intervention within three months of the onset of amenorrhea. Their guidelines recommend a nonpharmacological behavioral approach, together with diet and training interventions, as follows:

- Reduce training level by 10 to 20 percent;
- Gradually increase total energy intake;
- Increase body weight by 2 to 3 percent;
- Maintain daily calcium intake at 1,500 mg.

Skin blood flow and the sweating response during rest and activity are also influenced by the menstrual cycle. Scientists have found that a significantly higher core temperature is required to initiate sweating during the luteal phase. Although this change in thermoregulatory sensitivity does not affect the ability to exercise, it is worth taking account of menstrual cycle phase when evaluating thermoregulatory dynamics during exercise and thermal stress.
The majority of published studies agree that neither menstrual phase (follicular vs luteal) nor menstrual status (menstruating vs non-menstruating) significantly alters or limits exercise performance.

However, the combination of intensive exercise (particularly during the pre-pubertal years) and under-nutrition can have an adverse impact on reproductive function and sexual maturation, leading to either primary or secondary amenorrhea.

If an athlete does become amenorrheic, medical treatment should be considered in order to maintain long-term health and reduce the risk of fractures of bones.

**Pregnancy**

Historically the question of what women should do during pregnancy was a highly class-based one. Upper- and middle-class women of previous centuries were kept in confinement and forced leisure. One of the important truisms about activity during pregnancy is that pregnancy is not the time to begin a strenuous exercise program. Don’t start German Volume Training or any kind of masochistic weight program. In fact, pregnancy is not the time to make any drastic physical changes. However, women who are already accustomed to regular activity and exercise generally find pregnancy no disruption to their normal routine.

The female body has had plenty of evolutionary time to adapt to the stress of pregnancy and compensates for the extra demand in a variety of ways. For example, oxygen consumption/aerobic capacity can increase up to 30 percent during pregnancy in non-exercisers and even more in women who exercise. In addition, strenuous exercise can be exceedingly uncomfortable in the last part of pregnancy. However, anecdotal and clinical evidence shows that fitter women tend to have easier pregnancies and shorter deliveries with fewer complications. Many women also find that exercise during pregnancy helps alleviate fatigue and keep energy levels up. In addition, exercise has been shown to reduce the gain of subcutaneous (under the skin) fat associated with extra caloric intake during pregnancy. Thus, regular and moderate exercise during pregnancy can have many positive effects.

One of the most typical problems encountered by pregnant women is back pain. Their center of gravity shifts and extra weight is added over nine months. Women tend to slouch the shoulders and arch the lower back to compensate for these changes, which of course leads to discomfort. A strong abdominal column as well as a strong back gained through weight training before pregnancy can alleviate much of this problem.

Pregnant women are especially prone to overheating during exercise, so monitoring temperature, avoiding exercise in hot, humid environments, ensuring that there are mechanisms for body cooling (sweating, loosening of clothing, etc., and drinking lots of water are key factors).

Exercise during pregnancy, four major physiological concerns is associated. These are as follows:

i. The acute risk associated with reduced blood flow to the uterus leading to fetal hypoxia.

ii. Fetal hyperthermia associated with the increase in the mother's internal body temperature during prolonged aerobic type of exercise or exercise under conditions of heat stress.

iii. Reduced carbohydrate availability to the fetus as the mother's body uses more carbohydrate to fuel her exercise.

iv. The possibility of miscarriage and the final outcome of pregnancy.

Although there are several concerns over the health of the fetus during maternal exercise, the risk to the fetus from women performing aerobic exercise during pregnancy appears to be low, particularly if guidelines for exercising during pregnancy are followed.

**Guidelines for the Prescription of Aerobic Exercise During Pregnancy:**

i. Obtained medical clearance prior to exercise.

ii. Swimming, cycling (except weight bearing exercise) is preferable.

iii. Exertion levels should be determined on an individual basis.

iv. Avoid strenuous exertion during the first trimester.

v. Increases in exercise quantity and quality should be very gradual for previously inactive women.
vi. Avoid exercise in warm/humid environments.
vii. Drink liquids before and after exercise to ensure adequate hydration.
ix. Periodic rest intervals may be helpful to minimize hypoxia or thermal stress to the fetus.

**EAT**ING **D**ISORDERS

Eating disorder, such as anorexia nervosa and bulimia nervosa, are more common in women than in men and for athletes, are especially common in appearance sports, endurance sports and weight classification sports. Athletes seem to be at a higher risk for eating disorders than the general population.

**Anorexia Nervosa**

Anorexia nervosa is a disorder characterized by
- Refusal to maintain more than the minimal normal weight based on age and height.
- Distorted body image,
- Intense fear of fatness or gaining weight and
- Amenorrhea

**Bulimia Nervosa**

Bulimia nervosa is characterized by
- Recurrent episodes of binge eating,
- A feeling of lack of control during these binges and
- Purging behavior, which can include self-induced vomiting, laxative use and diuretic use.

**Warning Signs for Anorexia Nervosa and Bulimia Nervosa**

Warning signs for Anorexia nervosa:
- i. Dramatic loss in weight
- ii. A preoccupation with food, calories and weight
- iii. Wearing baggy or layered clothing
- iv. Relentless, excessive exercise
- v. Mood swings
- vi. Avoiding food related social activities

Warning signs for Bulimia nervosa:
- i. A noticeable weight loss or gain
- ii. Excessive concern about weight
- iii. Bathroom visits after meal
- iv. Depressive moods
- v. Strict dieting followed by eating binges
- vi. Increasing criticism of one's body

**AMENORRHEA**

Amenorrhea is the clinical term for cessation of menstrual periods with possible related loss of ovulation. This is generally seen in women who train very heavily and/or have body fat below 10 to 14 percent; thus, athletes, bodybuilders and models may all experience this condition. In essence, menstruation has ceased because the body does not feel that it has sufficient resources to nurture a fetus. Interestingly, there seems also to be a relationship between intense athletic training in young girls and delayed initial onset of menstruation, although the exact variables are hard to determine. It may be that lean girls are more athletically predisposed anyway, and the delay of menarche (onset of first menstruation) is merely an adjunct rather than a result. One of the main concerns around amenorrhea is the concomitant loss of bone density. In other words, skeletal bone mineral loss has been observed to be related to lack of menstruation.

However, research has demonstrated that amenorrhea is not caused by low body fat per se. Rather, it appears to be triggered by a long-term negative energy balance, which can result in a low body fat. Energy balance is the relationship between calories in and calories expended. If a female athlete consistently maintains a negative energy balance for a long-time (in other words, if she does not eat enough to fuel her activity), then this is what stimulates loss of menstruation, not a particular body fat level in and of itself. Indeed, female athletes and bodybuilders who are sensible about their nutrition and dieting practices may find that they do not lose their periods even though they get quite lean.

Although some women who re-start menstruation can make some moderate gains back, it appears that long-term bone mineral loss related to amenorrhea is largely irreversible. With the increasing concern about osteoporosis in our society today, this is a critical concern for female
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data is still sketchy on the bone density concerns of developing girls, although perhaps this problem in general might be somewhat alleviated by weight training, which is known to increase bone density. Along with the problem of bone mineral loss initiate the problem of stress fractures. The possibility of stress fractures raises some important questions. Is, for example, the increased bone density of the marathon runner as compared to her sedentary colleague enough to withstand the actual activity of marathon running? At what point do the load-bearing exercises, which improve bone density, become too much load for the bone to handle?

Obviously there are more variables involved in the problem of amenorrhea and bone density loss. Diet, training and other environmental factors are also significant considerations. However, amenorrhea as a long-term clinical state is highly undesirable in any case.

**Menopause**

Surprisingly, there have been few studies done on menopausal women and exercise. The main concern for menopausal and postmenopausal women is the drop in estrogen levels which is associated with bone density loss. This is especially problematic for those women who may have been amenorrheic in their youths, since there is likely some bone density loss which has already taken place. Weight training, however, has been shown to have very beneficial effects irrespective of age. Obviously a menopausal woman isn't going to respond in the same way as a teenage girl, but the positive consequences of weight training are undeniable. Within every age group, active women fare better than inactive women in just about every test. Some studies showed that exercise can reduce and/or delay much of the symptomology (hot flashes, anxiety, etc.) associated with menopause. If one has been inactive, menopause is a good time to become active. It’s never too late to begin to strength training and engage in regular exercise. Excess body fat is associated with a host of later-life problems such as heart disease and diabetes. As increasing one’s muscle mass contributes dramatically to a decrease in injuries and chronic problems, as well as to a loss of excess body fat. One important point for aging women: in the calculation of body fat percentages, the same actual caliper measurement (say, 15 mm) will mean different body fat percentage readings depending on age. This is due to inter-abdominal fat deposition with age. In other words, the older you get, the more fat you accumulate on your internal organs as opposed to beneath your skin (subcutaneous fat). Thus the “healthy” range of percentages increases with numerical age.

**Female Athlete Triad:** In the early 90s, it became apparent that there is a reasonably strong association between eating disordered, secondary amenorrhea and bone mineral disorder. This has been termed the female athlete triad and it appears that the triad might start with disordered eating. An athlete who has disordered eating start to experience disordered menstrual function, which eventually leads to secondary amenorrhea. Again secondary amenorrhea leads to bone mineral disorders. However, number of research are going on there relationship among the female athletes.

**Summary**

i. The body of the men and women reacts differently to varying degrees of physical stress, and never two bodies react exactly the same way to the same physical stress. For everyone to get the maximum benefit from training, and trainer must be aware of these differences and plan the training schedule to provide maximum benefit for everyone. They must also be aware of the physiological differences between men and women. While they require equal efforts of men and women during the training period, they must also realize that women have physiological limitations which generally preclude equal performance.

ii. This difference in size affects the absolute amount of physical work that can be performed by men and women. Accordingly, the body surface area is also varying between men and women of same ages. Men have 50 percent greater total muscle mass, based on
weight, than do women. A woman who is the same size as her male counterpart is generally only 80 percent as strong. Therefore, men usually have an advantage in strength, speed and power over women.

iii. Women carry about 10 percent more body fat than do men of the same age. Men accumulate fat primarily in the back, chest and abdomen; women gain fat in the buttocks, arms and thighs. Women have less bone mass than men, but their pelvic structure is wider. This difference gives men an advantage in running efficiency.

iv. Anthropometric measurements at maturity differ substantially between the sexes. Women have narrower shoulders, broader hips, and smaller chest diameters and tend to have more fat in the hips and lower body, whereas men carry more fat in the abdomen and upper body. The average woman’s heart is 25 percent smaller than the average man’s. Thus, the man’s heart can pump more blood with each beat (i.e., stroke volume). The larger heart size contributes to the slower resting heart rate (five to eight beats a minute slower) in males. The normal resting heart rate of young adult men is 72 beats/min. whereas the normal heart rate of women of similar age is 76 beats/min. Women generally are more flexible than their men counterpart which may be due to the hip flexion is greater in women than men. The lung capacity of men is 25 to 30 percent greater than that of women. This gives men still another advantage in the processing of oxygen and in doing aerobic work such as running, swimming, cycling, etc.

v. Knowing the physiological differences between men and women is just the first step in planning physical training for a group. Trainers need to understand other factors too. Women can exercise during menstruation; it is, in fact, encouraged. The “typical” young untrained male will have an absolute VO₂ max of 3.5 liters/min, while the typical same-age female will be about 2 liters/min. This is a 43 percent difference and this difference may be due to the fact that males are bigger, on average, than females and also having big heart and lung size with more muscle mass.

vi. Some of the research comparing running economy between genders, it may be argued that any inherent economy differences in male and female runners are smaller than the individual variation in running economy that is observed among runners, independent of gender. It may be supported that argument by suggesting the differences in VO₂ max observed between elite males and females are sufficient to explain the “10 percent gap” without other factors being involved.

vii. Maximal muscular strength and anaerobic power has little to do with pure endurance performance, there are many events which can be classified as “power-endurance” events. These events ranging from 2 to about 8 minutes require some combination of aerobic and anaerobic capacity. For this reason, it is important to also consider this “anaerobic” component of the performance.

viii. Alterations in athletic performance experienced during different phase of the menstrual cycle are subject to considerable individual variability. Some woman have absolutely no noticeable change in their performance ability at anytime during their menstrual cycle, yet other have considerable difficulty in either the pre flow or the early flow phase or during both. Several studies have suggested that athletic performance is best during the immediate post flow period up to the 15th day of the cycle, with the first day of the corresponding to the initiation of the flow.

ix. Delayed menarche has been reported in young athletes involved in certain sports and activities, such as gymnastics and ballet. The mean age for menarche is generally 12.0 years. For gymnasts, the mean age appears to be closure to 15 years. Female athletes can experience disruptions of their normal menstrual cycle. These disruptions are collectively referred to as menstrual dysfunction, of which there are several types: Eumenorrhea, Oligomenorrhea, Primary
**Amenorrhea and Secondary Amenorrhea.** A high percentage of female athletes in endurance and appearance sports performance secondary amenorrhea, where normal menstrual function is lost for months or even years. This appears to be reversible with reductions in the intensity and volume of training and an increase in caloric intake.

The female body has had plenty of evolutionary time to adapt to the stress of pregnancy and compensates for the extra demand in a variety of ways. For example, oxygen consumption/aerobic capacity can increase up to 30 percent during pregnancy in non-exercisers and even more in women who exercise. In addition, strenuous exercise can be exceedingly uncomfortable in the last part of pregnancy, which is perhaps the body’s way of telling mom to lay off for a while till things get back to normal.

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**REVIEW QUESTIONS**

1. How do men and women differ physically and physiologically?
2. How do female compare with males with respect to body composition?
3. How do men and women compare relative to upper and lower body strength? Can women gain strength with resistance training?
4. Discuss the Gender Differences in Endurance Performance and Training.
5. What cardiovascular differences exist between female and males with respect to sub maximal exercise? Maximal exercise?
6. What are the difference between male and female of physiological adaptations to exercise and training?
7. Why are female and male performances in motor skills similar up to the age of puberty, and yet considerably different once puberty has been attained?
8. How does the menstrual cycle influence athletic performance in female athletes? Discuss with suitable example.
9. What do you mean by menstrual dysfunction? What are some of the possible reason that woman athletes in intensive training will, in some cases, stop menstruating for intervals of several months to several years or more?
10. What are the risks associated with training during pregnancy? What are the guidelines of aerobic exercise during pregnancy?
11. What are the two majors’ eating disorders and what is the level of risk of elite female athletes for these eating disorders? Writes notes on the following: (a) Amenorrhea, (b) Menopause, (c) Eating disorders.
12. What are the difference between men and women in their exercise response when exposed to intense heat and humidity?
The animal kingdom can be broadly classified into two groups, depending upon their body temperature. Those who can maintain their body temperature relatively constant in the face of wide variations of environmental temperature are known as warm-blooded animal or homeotherms, whereas those whose body temperature fluctuates with fluctuations of the environmental temperature are termed as cold-blooded animals or poikilotherms. In the course of evolution, from poikilotherms to homeotherms, there exists another group who are known as hybernants, going into hibernation in winter, otherwise behaving like the worm-blooded animals in the remaining period. Hibernating animals do not require an external source of heat to raise their body temperature to normal and can rouse themselves probably by activating their large store of brown fat.

Even in homoiotherms, who are capable of maintaining constant body temperature, it has been found that different parts of the body, even different organs, have different temperatures and this is a normal phenomenon. However, the average temperature as measured has been found to lie within a constant range, hence justifying the statement of having a constant body temperature.

**Regulation of Body Temperature**

Although a large amount of heat is produced and lost from the body constantly, yet the body temperature remains constant within a limited range. This indicates that there is strong machinery, which keeps an exact balance between gain and loss of heat and thereby maintains a constant body temperature. The physiological process of heat
production in the body is known as thermogenesis. Dissipation of bodily heat by means of radiation, evaporation, etc., is called thermolysis. The mechanism by which body temperature is normally adjusted is known as the thermotaxis. The controlling mechanism consists of the following:

**MECHANISM OF HEAT PRODUCTION**

The human body primarily as a result of all the internal metabolic processes gains heat. However, there may be a small gain by radiation and convection from the external environment, such as the sun, a newly resurfaced blacktop road, desert sand and rock, etc. if they are at a higher temperature than the body. A small gain may also be obtained through conduction if the body is exposed to a source having a high temperature such as hot water. The amount taken in with hot food and drink is almost negligible. Heat production from metabolism takes place in the tissues. Although every tissue contributes to this, the skeletal muscles furnish the largest amount. Therefore, increasing heat production is obtained primarily by increasing muscular activity. In strenuous activity, heat production from metabolism has been known to increase by as much as 30 times its normal. In bitter cold weather, the metabolic heat production is achieved by an involuntary response known as shivering, which may in fact, increase the metabolic processes by as much as 400 percent. The human body generally starts to shiver when the skin temperature has dropped to approximately 19°C (66°F).

**MECHANISM OF HEAT LOSS**

There are three ways by which heat is lost from the body: (i) Skin (ii) lungs and (iii) excretion. Skin is the most important organ among the three and approximately 85 percent of heat is lost via this organ, although this depends on external and internal conditions. Thus, the bodily changes that regulate the exchange of heat between the body and the environment are referred to as physical heat regulation.

i) *From the skin (Proportional to the total surface area):* Heat is lost from the skin by the following means:
   a. Conduction
   b. Convection
   c. Radiation
   d. Evaporation

a. **Conduction:** Heat may be lost from the skin by conduction, as in coming in direct contact with an object possessing a colder temperature. Heat is always conducted from the warmer to the colder
object. If the outside object is warmer than the human body, then heat is gain by conduction.

b. Convection: When the body temperature is higher than the surrounding air, heat is lost from the skin by way of convection. Thus, the surrounding air is warm by air currents from the body. The convection currents replace warmed air with cold air and they also replace moist air with relatively dry air. The amount of heat loss from convection depends on the temperature as well as the speed by which the air flows over the surface of the body. We gain heat by convection when the surrounding air is warmer than the body.

c. Radiation: Heat is lost from the skin by radiation when the body temperature is higher than the surrounding object such as walls and furniture. On the other hand, we gain heat by way of radiation when the external objects are warmer than the human body. Radiation is based on the principle that molecules within the body are constantly vibrating, and as a result, heat is being given off in the forms of electromagnetic waves.

d. Evaporation: Heat is lost from the skin by evaporation of sweat from the body surface since the evaporation of any given fluid utilizes and hence removes that from the surrounding objects and air. In order for evaporation to be an effective mechanism for cooling the body, the sweat from our body must be changed into a gaseous water vapor at the body surface. Energy, in the form of heat, is needed for this change and it is absorbed from the body surface. This absorbing of heat energy results in cooling.

During exercise, the major portion of heat loss is through evaporation of sweat from the body surface. In addition, when air temperature is higher than that of the human body, the majority of heat is lost by evaporation. The amount and rate of heat lost by evaporation depends upon the movement of air across the surface of the body as well as the relative humidity (RH) of the air. The lower the humidity, the greater the heat loss via evaporation; whereas the higher the humidity (air highly saturated with water vapor) the smaller the heat loss. During vigorous exercise, especially in a hot and humid environment, perspiration can exceed as much as 4 liters per hour. Although the rate of perspiration changes with the temperature, there is some perspiration even at low temperatures. Perspiration of this nature is referred to as insensible water loss. Insensible water loss leaves the body at all times unless the ambient humidity is 100 percent RH. This moisture or extra cellular fluid diffuses through the skin, through the pores of the sweat glands and from the lungs.

![Fig. 11.2: The complex interaction between the body’s mechanisms for heat balance and environmental conditions](Adopted from the book Physiology of Sports and Exercise: J. Wilmore & D. Costill.)
NERVOUS SYSTEM AND THERMOTAXIS

There are several centers in the spinal cord and the brain, which control such activities as vasoconstriction, vasodilatation, sweating, muscle tone and shivering. In order for body temperature to remain at or near a constant level, the integration of these various activities is essential.

(i) Role of cerebrum: Removal of cerebrum makes very little changes. The regulating capacity only becomes slightly restricted. The human responds normally to external heat or cold but the body temperature falls if kept in the cold room for a long-time.

(ii) Role of hypothalamus: The heat-regulating center lies in the hypothalamus. The role of the thermoregulatory system in regulating body temperature is accompanied by two centers: (a) Anterior hypothalamus center, (b) Posterior hypothalamus center.

a. Anterior hypothalamus center: This center controls heat-dissipating events. For instance, if this center is stimulated, the blood vessels of the skin are dilated, thus resulting in a greater flow of blood to the surface. Also, impulses are transmitted to the sweat glands, increasing perspiration, and to the respiratory center, causing panting. All these physiological responses act to increase the rate of heat loss from the body, and thereby prevent overheating of the body. Destruction of this center causes a person to react normal in a cold environment, but in hot climates the common methods of losing heat (conduction, radiation, convection and evaporation) are inoperative and hence the body temperature rises.

b. Posterior hypothalamus center: The posterior hypothalamus center calls on heat conservation events, and thereby prevents chilling of the body. For example, if the center is stimulated, the blood vessels of the skin are constricted, thus reducing the blood flow to the surface. In addition, it is well known that the surface hairs on the body are also stimulated to become erect, and thus they come into play to reinforce the insulating layer of air surrounding the skin. At the same time, shivering is brought about through the same mechanism to increase heat production. As a result of this centers destruction, exposure of the person to cold climate does not increase metabolism or heart rate; therefore, heat production lags and the body temperature drops.

The hypothalamus responds reflexly to afferent impulses initiated by the thermo receptors in the skin, and to changes in the temperature of the arterial blood that flows through it. The skin receptors react to changes in the environmental temperature, whereas the hypothalamus receptors respond to small temperature changes (as little as 0.2 to 0.5°F) of the arterial blood flowing through them.

(iii) Role of autonomic nervous center: Only the parasympathetic division, e.g. salivary secretion, secretion of glands of the pharynx and respiratory tract, mediates a few thermal responses and local vasodilatation followed by activity. Greater part of the generalized thermal responses in visceral effectors is due to sympathetic control, e.g. constriction of peripheral vessels, erection of hair and feathers, liberation of epinephrine and norepinephrine, sweating and cutaneous vasodilatation. It has been definitely established that adrenal medulla is an integral part of the sympathetic system.

(iv) Role of spinal cord in heat regulation: Spinal cord is the connecting path between the heat-regulating centers in the hypothalamus, peripheral thermo receptors and effector organs (muscles). The cervical segments of the spinal cord transmit greater part of the sympathetic outflow, which regulates peripheral circulation and hence heat regulation. Spinothalamic tracts of the spinal cord carry the efferent impulse for shivering from higher centers. Effect of section through spinal cord on thermoregulation depends upon the level. When the section of the cord is made above or through the level of sympathetic outflow (cervical segments), gross disturbance of temperature regulation occurs. Transection of the spinal cord from the level of upper thoracic segments downwards abolishes sweating and shivering below the level of transection, i.e. in the paralyzed parts.

(v) Role of motor fibers of the cerebrospinal system in heat regulation (Muscle tone): Muscle tone alone (even without locomotion and exercise) is a continuous source of heat production. Central
nervous system maintains the muscle tone (thermal muscle tone) by continuous discharge of impulses to the muscles via motor fibers. Exaggerated “thermal muscle tone” to the extent of tremor is described as shivering. Shivering impulses from the shivering center are not transmitted via the sympathetic system but via the motor fibers of cerebrospinal system.

Role of Endocrine Glands

The effects of several hormones cause the cells to increase their metabolic rates. This affects heat balance because increased metabolism increases heat production. Cooling the body stimulates thyroxine release from the thyroid gland, which can elevate the metabolic rate throughout the body by more than 100 percent. Also, epinephrine and norepinephrine mimic and enhance the activity of the sympathetic nervous system. Thus, they directly affect the metabolic rate of virtually all body cells.

Thyroid

When the thyroid gland is stimulated, large quantities of the hormone called thyroxin is released into the blood. The thyroid hormone continues to be active for as long as 4 to 8 weeks after its release into the blood. Large secretions of the thyroid hormone can cause the basal metabolic rate (BMR) to increase as much as 200 percent of normal. It is generally known to increase the quantities of most of the cellular enzymes. Hence, this may explain its metabolic effects. Cold stimulates and heat reduces thyroid secretion. In cold, excess thyroid-stimulating hormone (TSH) is liberated from the anterior pituitary and thereby excess thyroid hormones are secreted from the thyroid gland in controlling low body temperature. In cretinism and myxoedema body temperature is subnormal. Thyroidectomised animals cannot maintain the normal body temperature.

Anterior Pituitary

Thyrotrophic hormone stimulates secretion of thyroxine and helps in the maintenance of body temperature. Adrenocorticotropic hormone (ACTH) is secreted under increased or decreased body temperature (cold stress or heat stress respectively).

Adrenal Medulla

It helps in both ways. Cold reflexly stimulates adrenaline secretion, which increases heat production by stimulating metabolism. When the adrenal medulla is stimulated, large amounts of epinephrine and norepinephrine hormones are released into the blood. These two hormones have the ability to increase the basal metabolic rate and therefore increase the heat production. The exact means by which this is accomplished is not clear, however, according to some authorities researching in this area, epinephrine and norepinephrine enhances the breakdown of glycogen into glucose as well as increase the rate of some of the enzymatic reactions that promote oxidation of foods. Since these two hormones may cause constriction of the cutaneous blood vessels, they are also of value in conservation of body heat.

Adrenal Cortex

Adrenal corticoid secretion is stimulated by the increase or decrease of environmental temperature. Usually a low body temperature has been noted in Addison’s disease (hypofunction of adrenal cortex). The hypothalamus contains high concentration of 5-hydroxytryptamine (serotonin) and noradrenaline (norepinephrine). The effect of these two amines, so far the temperature regulation is concerned, varies in different animals. It is presumed that these hypothalamic amines play some part in the regulation of body temperature in normal and in pathological states. The effect of these two amines, so far the temperature regulation is concerned, varies in different animals. It is presumed that these hypothalamic animals play some part in the regulation of body temperature in normal and in pathological states.

Role of Sweat Glands

When either the skin or the blood is heated, the hypothalamus sends impulses to the sweat glands, commending them to actively secrete sweat that moistens the skin. The hotter the person is, the more sweat he produces. The evaporation of this moisture removes heat from the skin’s surface.
Role of Muscles

Smooth Muscle Around Arterioles
When the skin and blood are heated, the hypothalamus sends signals to the smooth muscle in the walls of the arterioles that supply the skin, causing them to dilate. This increases blood flow to the skin. The blood carries heat from the deeper parts of the body to the skin, where the heat dissipates to the environment through conduction, convection, radiation or evaporation.

Skeletal Muscle
Skeletal muscle is called into action when one needs to generate more body heat. In a cold environment, the thermo receptors in the skin relay signals to the hypothalamus. Similarly, whenever the blood temperature drops, the change is noted by the central receptors in the hypothalamus. In response to this neural input, the hypothalamus activates the brain centers that control muscle tone. These centers stimulate shivering, which is a rapid, involuntary cycle of contraction and relaxation of skeletal muscles. This increased muscle activity generates heat to either maintain or increase the body temperature.

Physiological Response to Exercise in the Heat
During exercise in the heat, the heat loss mechanism compete with the active muscles for more of the limited blood volume. Thus neither area is adequately supplied under extreme conditions. Though cardiac output may remain reasonably constant, the stroke volume may decline, resulting in a gradual upward drift in heart rate. Oxygen uptake also increases during constant rate exercise in the heat lead weating increase during exercises in the heat and this can quickly had to dehydration and excessive electrolyte loss. To compensate, the release of aldosterone and ADH increase, causing sodium and water retention, which can expand the plasma volume.

Risks During Exercise in the Heat
Air temperature alone is not an accurate index of the total physiological stress imposed on the body in a hot environment. At least four variables must be taken into account:

i. Air temperature
ii. Humidity
iii. Air velocity and
iv. The amount of radiation
All these influence the degree of heat stress experience by the person. The contribution of each of these factors to the total body heat stress are not clear, because the contribution vary with changing environmental conditions.

With an air temperature of 23°C, an individual exercising on a bright, sunny day with no measurable wind experiences considerably more heat stress than someone exercising the same air temperature but under cloud cover and with a slight breeze. At temperatures above 30–32°C, radiation, conduction and convection substantially at to the body’s heat load rather than acting as avenues for heat loss.

Human Limitations in the Heat
There are some problems that can be caused by exercising in the heat with which coaches, trainers and athletes should be familiar. The following problems are common in the athlete which are discussed:

Heat Stress
The heat stress is not accurately reflected by air temperature alone. Humidity, air velocity (or wind) and thermal radiation also contribute to the total heat stress that one experiences when exercising in the heat. Actually the heat stress is the sum of the metabolic and environmental heat loads. The total thermal load is related to the exercise intensity (metabolic load), the environmental temperature, and the evaporative potential of the environment (itself related to the ambient water vapor pressure, or humidity).

Heat Strain
The bodily effect of heat stress, that is, the relative elevation of body core temperature, average skin temperature, and rate over that occurring in a cool environment. It is quiet obvious that exercise in environmental surroundings of high temperature
and high relative humidity can place severe strains upon the cardiovascular system.

**Heat Related Disorders**

Exposure to the combination of external heat stress and the inability to dissipate metabolically generated heat can lead to mainly three heat-related injuries:

- Heat cramps
- Heat exhaustion
- Heat stroke

**Heat cramps**: Heat cramps, the least serious of the three heat disorders, is characterized by severe cramping of the skeletal muscles. It primarily involves the muscles that are most heavily used during exercise. This disorder is probably brought on by the mineral losses and dehydration that accompany high rates of sweating, but a cause and effect relationship has not been fully established. Heat cramps are treated by moving the stricken individual to a cooler location and administration fluids or a saline solution.

**Heat exhaustion**: Heat exhaustion is typically accompanied by such symptoms as extreme fatigue, breathlessness, dizziness, vomiting, fainting, cold and clammy or hot and dry skin, hypotension (low blood pressure) and a weak, rapid pulse. It is caused by the cardiovascular system’s inability to adequately meet the body’s needs. Recall that during exercise in heat, the active muscles and the skin, through which excess heat is lost, compete for their fare share of the total blood volume. Heat exhaustion results when these simultaneous demands are not met. The disorder typically occurs when the blood volume is reduced, either by excessive fluid loss or by mineral loss from sweating. With heat exhaustion, the thermoregulatory mechanisms are functioning but cannot dissipate heat quickly enough because there is insufficient blood volume to allow adequate distribution to the skin. Although the condition often occurs during mild to moderate exercise in the heat, it is not generally accompanied by a high rectal temperature. Some people who collapse from heat stress exhibit symptoms of heat exhaustion, but have internal temperatures below 39°C (102.2°F). People who are poorly conditioned or unacclimatized to the heat are more susceptible to heat exhaustion.

Treatment for victims of heat exhaustion involves rest in a cooler environment with their feet elevated to avoid shock. If the person is conscious, administration of salt water is usually recommended. If the person is unconscious, medically supervised intravenous administration of saline solution is recommended. If allowed to progress, heat exhaustion can deteriorate to heat stroke.

**Heat syncope**: Heat syncope (fainting) is rare in a conditioned athlete. In most instances, it occurs when individuals stand for a prolonged period of time in the heat or exercise for a prolonged period in an upright position. Heat syncope is caused by a pooling of blood in the vasculature of the limbs and skin because of excessive ambient temperatures. In response to the hot environment, the cutaneous vesicles of the skin dilate to allow for greater cooling. The increase in vasodilatation reduces the volume of blood that is returned to the heart, which decreases cardiac output and lowers blood pressure. Blood flow to the brain is therefore reduced, resulting in a syncopic episode.

The medical diagnosis of heat syncope is based on a fainting spell with the absence of an elevated rectal temperature. Before the syncopic episode occur the patient may experience nausea, weakness, tunnel vision or vertigo. Treatment for heat syncope is to replace any fluid and electrolyte deficits and have the patient lie in a horizontal position with the feet elevated. The horizontal position allows for a greater venous return to the heart. Subsequently, cardiac output and blood pressure increase, resulting in a return of normal blood volume to the brain.

**Heat stroke**: Heat stroke is a life-threatening heat disorder that requires immediate medical attention. It is characterized by:

i. A rise in internal body temperature to values exceeding 40°C (104°F)
ii. Cessation of sweating
iii. Hot and dry skin
iv. Rapid pulse and respiration
v. Usually hypertension (high blood pressure)
vi. Confusion
vii. Unconsciousness

If left untreated, heat stroke progresses to coma and death quickly follows. Treatment involves rapidly cooling the person’s body in a bath.
of cold water or ice or wrapping the body in wet sheets and fanning the victim.

This disorder is caused by failure of the body’s thermoregulatory mechanisms. Body heat production during exercise is dependent on exercise intensity and body weight, so heavier athletes run a higher risk of overheating than lighter athletes when exercising at the same rate, assuming both have about equal heat acclimatization.

In case of athlete, heat stroke is not a problem associated only with extreme conditions. Studies have reported rectal temperatures above 40.5°C (105ºF) in marathon runners who successfully completed races conducted under relatively moderate thermal conditions (for example, 70°F and 30 percent relative humidity). Even in shorter events, the body’s core temperature can reach life-threatening levels. As early as 1937, Robinson observed rectal temperatures of 41°C (105.8ºF) in runners competing in events lasting only about 14 minutes, such as the 5,000-m race. Following a 10,000-m race conducted with an air temperature of 29.5°C (85°F), 80 percent relative humidity and bright sun, one runner who collapsed had a rectal temperature of 43°C (109.4ºF). Without proper medical attention, such fevers can result in permanent central nervous system damage or death. Fortunately, this runner was rapidly cooled with ice and recovered without complications.

Prevention of Hyperthermia

We can do little about environmental conditions. Thus, in threatening conditions, athletes must decrease their effort in order to reduce their heat production and their risk of developing Hyperthermia (high body temperature). All athletes, coaches and sports organizers should be able to recognize the symptoms of Hyperthermia. Fortunately, all the subjective sensations are well correlated with the body temperature. Although there is generally little concern when rectal temperature remains below 40°C (104ºF) during prolonged exercise, athletes who experience throbbing pressure in their head and chills should realize that they are rapidly approaching a dangerous situation that could prove fatal if they continue to exercise.

Prevention of Heat Illness

i. Encourage a pre-season training program. In other words, a period of heat acclimatization should be carried out before working in the heat.

ii. Practice in lightweight uniforms. Long-sleeved jerseys and stockings (such as in football) should not be worn until the weather turns cool.

iii. Players should be weighed before and after workouts. An individual who was lost over 5 lb (2.3 kg) should be observed very carefully. Individuals who lose over 10 lbs (4.5 kg) in a practice should be considered in the danger zone.

iv. Provide for adequate salt and water replacement. Water, in some form, should be allowed during all practices and games.

v. Allow for sufficient rest periods during practice.

vi. Workouts and distance runs should be conducted during the cool-part of the day (generally early mornings and late evenings) on hot days.

vii. Conduct daily dry and wet bulb readings. The relative humidity should also be recorded daily. When the wet-bulb temperature is over 50°F, all members of the team should be alerted (especially those teams that have to have their bodies covered with uniforms such as football players). Many water and rest breaks should be allowed. When wet-bulb temperatures are above 75°F, practices should be conducted in shorts or they should be cancelled. Note that the American college of Sports Medicine has issued a position statement concerning distance running in heat and their position is that distance races (16 kilometers or 10 miles) should not be conducted when the wet-bulb temperature exceeds 82.4°F or 28.0ºC. It is also important to remember that whenever relative humidity is around 95 percent or higher, precautions should be taken at all temperature levels.

Practical Recommendations for Fluid Replacement

The primary aim of fluid replacement is to maintain plasma volume so that circulation and sweating
progress at optimal levels. Ingesting “extra” water before exercising in the heat provides some thermoregulatory protection. It delays the development of dehydration, increases sweating during exercise and brings about a smaller rise in body temperature compared to exercising without prior fluids. In this regard, it is wise to consume 400 to 600 ml of cold water 10 to 20 minutes before exercising. Doing this, however, does not eliminate the need for continual fluid replacement during exercise.

**BODY FLUID BALANCE: SWEATING**

Under some conditions, the temperature of the environment approaches and can exceed both the skin and deep body temperatures. This makes evaporation far more important for heat loss because radiation, convection and conduction are less effective as environmental temperature rises. In fact, these mechanisms can lead to heat gain in extreme environmental conditions. Increased dependence on evaporation means an increased demand for sweating.

The sweat glands are controlled by stimulation of the hypothalamus. Elevated blood temperature causes the hypothalamus to transmit impulses through the sympathetic nerve fibers to the millions of sweat glands distributed over the body’s surface. The sweat glands are tubular structures extending through the dermis and epidermis, opening onto the skin.

Sweat is formed by the filtration of plasma. As the filtrate passes through the duct of the gland, sodium and chloride are gradually reabsorbed back into the surrounding tissues and then into the blood. During light sweating, the filtrate sweat travels slowly through the tubules, allowing time for almost complete reabsorption of sodium and chloride. Thus, the sweat that forms during light sweating contains very little of these minerals by the time it reaches the skin. However, when the sweating rate increases during exercise, the filtrate moves more quickly through the tubules, allowing less time for reabsorption. As a result, the sodium and chloride content of the sweat can be considerably higher.

The mineral content of each subject’s sweat is significantly different in trained and untrained subjects. With training and repeated heat exposure, aldosterone can strongly stimulate the sweat glands, causing them to reabsorb more sodium and chloride. Unfortunately, the sweat glands apparently do not have a similar mechanism for conserving other electrolytes. Potassium, calcium and magnesium for example, are normally found in the same concentrations in both sweat and plasma.

While performing heavy exercise in hot conditions, the body can lose more than 1L of sweat per hour per square meter of body surface. This means that during intense effort on a hot and humid day (high level of heat stress), an average-sized individual (50 to 75 kg) might lose 1.5 to 2.5 L of sweat, or about 2 to 4 percent of body weight each hour. A person can lose a critical amount of body water in only a few hours of exercise in these conditions.

A high rate of sweating reduces blood volume. This limits the volume of blood available to supply the needs of the muscles and to prevent heat build up, which in turn, reduces performance potential particularly for endurance activity. In long distance runners sweat losses can approach 6 to 10 percent of body weight. Such severe dehydration can limit subsequent sweating and make the individual susceptible to heat related illness.

Loss of both mineral and water by sweating triggers the release of aldosterone and antidiuretic hormone (ADH). The aldosterone is responsible for maintaining appropriate sodium levels and ADH maintains the fluid balance. During acute exercise the heat and during repeated days of exercise in the heat, this hormone limits sodium excretion from the kidneys. More sodium is retained by the body, which in turn more water retention. Because of this, plasma and interstitial fluid volumes can increase 10 to 20 percent. This allows the body to retain water and sodium in preparation for additional exposures to the heat and subsequent sweat losses.

Similarly, exercise and body water loss stimulate the posterior pituitary gland to release ADH. This hormone stimulates water reabsorption from the
kidneys, which further promoted fluid retention in the body. Thus, the body attends to compensate for mineral and water loss during periods of heat stress and heavy sweating by reducing their losses in urine.

**Exercise in the Heat**

Exercise in hot climates is a more serious problem than exercise in the cold. In a cold climate the increased metabolic heat production combats the increased heat loss to the environment, but in a hot climate metabolism and environment combine to increase heat gain in body tissues. The problem is further complicated by the fact that when environmental temperature approaches skin temperature (approximately 92ºF), heat loss through convection and radiation gradually comes to an end, so that at temperatures above skin temperature the only means for heat loss is evaporation of sweat. Radiation and convection reverse their direction and add heat to the body.

Sweating then, is the only avenue for heat loss at temperatures above skin temperature, and it is the most important avenue at temperatures that approach skin temperature. At this point it is most important to understand that the mere process of sweating is not in itself effective in dissipating heat; liquid sweat must be converted to a gas by evaporation before any heat loss occurs. Sweat that merely rolls off is virtually ineffective, but large heat losses can result when the weather is so dry that the liquid evaporates from the skin rapidly. Under such conditions sweating is imperceptible. For these reasons, exercise in the heat will be discussed as two separate and distinct environmental problems: (a) Hot and dry environment, (b) Hot and humid environment.

**Hot and Dry Environment**

When a person works or plays in hot and dry environment, cooling of the skin is brought about by evaporation of sweat. There is no problem because dry air can absorb considerable moisture before becoming saturated. Cooling the skin is not the desired end result, however, it is the internal environment that must be cooled at all costs. To retain a normal core temperature, heat must be transported from the core to the skin, and this requires adjustments from the normal, resting circulatory state. The arteriovenous anastomoses of the microcirculation open up, along with precapillary sphincters, to increase flow through the skin and subcutaneous tissues. This results in greater volumes of slow-moving blood in and close to the skin for better transfer of heat to the evaporative surfaces and thus in better cooling.

Along with the improved cooling, however the volume of the circulatory system has increased by a considerable amount. Under these conditions, venous return to the heart is somewhat impaired and this results in a decreased stroke volume (in accord with Starling’s law). To maintain a constant cardiac output for the demands of both exercising muscles and skin circulation, the heart rate must increases. Because increases in rate depress cardiac efficiency, exercise at temperatures close to or above skin temperature can impose very severe loads on the cardiovascular system, even when the air is relatively dry.

Since the entire process of heat dissipation now depends on elimination of water in perspiration, it is obvious that dehydration is a distinct possibility. A man walking in the desert (temperature 100ºF) will lose approximately one quart of water per hour. Furthermore, their extensive desert experimentation indicates that voluntary thirst results in adequate water replacement during rest but not during work or exercise.

**Hot and Humid Environment**

When the air surrounding an individual is not only hot but is also loaded with moisture, evaporative cooling is impaired because evaporation cannot take place unless volumes of air are available to take-up the water vapor given off. To illustrate this, let us take the extreme example where the air is completely saturated (100 percent relative humidity) and the air temperature is higher than the skin temperature. Under these conditions no heat dissipation can occur. Consequently, the metabolic heat accumulates and raises body temperature, until death ensues (108ºF to 110ºF). One may therefore conclude that the problems in a hot, dry atmosphere are related to increased cardiovascular loads and dehydration if water intake...
is insufficient. In a hot, humid climate the same problems exist and are aggravated by a lesser ability to unload water vapor into an already loaded ambient atmosphere. It is clear that although the temperature is lower in the hot, wet situation, it is considerably more stressful in terms of heart rate response than the hot, dry climate.

**Acclimatization to Exercise in the Heat**

Repeated prolonged exercise bouts in the heat cause gradual improvement in the ability to eliminate excess body heat, which reduces the risk of heat exhaustion and heat stroke. This process, termed heat acclimatization, results in many adjustments in sweating and blood flow. Though the total amount of sweat produced during exercise in the heat might not change with heat acclimatization, the amount of sweat produced often increases in the most exposed body areas and in the areas that are most effective at dissipating body heat. At the beginning of exercise, sweating starts earlier in an acclimatized person, which improves heat tolerance. As a result, skin temperatures are lower. This increases the temperature gradient from deep in the body to the skin and the environment. Because heat loss is facilitated, less blood must flow to the skin for body heat transfer, so more blood is available for the active muscles. Also, blood pressure following heat acclimatization appears to be more stable and adequately regulated during exercise. The reduction in the salt content of the sweat is believed to be due to an increased amount of aldosterone hormones released into the blood stream during heat exposure. Without this decline in salt concentration, the human body (in most cases) would suffer a salt deficit.

In addition, the sweat produced is more dilute following training in the heat, so the body’s mineral stores are conserved more efficiently. Because training enhances the body’s heat loss capacity for a specified level of work, body temperatures are lower following training in the heat than they are before training. Also, after training heart rate increases less in response to standardized submaximal exercise. This adaptation results from an increased blood volume, reduced blood flow to the skin or both. Either of these changes increases the stroke volume. Although some investigators have found that an increase in blood volume accompanies heat acclimatization, this change is temporary and probably relates to the body’s efforts to retain sodium, thereby expanding the plasma volume.

In addition, following heat acclimatization, more work can be done before the onset of fatigue or exhaustion. Recall that exercise at a given intensity in the heat requires the use of more muscle glycogen than the same effort done in cooler air. As a result, repeated days of training in the heat can rapidly deplete muscle glycogen and cause chronic fatigue in unacclimatized people. Heat acclimatization reduces the rate of muscle glycogen use by as much as 50 to 60 percent, reducing this risk.

**Achieving Heat Acclimatization**

Heat acclimatization requires more than mere exposure to a hot environment. It is dependent on

i. The environmental conditions during each exercise session
ii. The duration of heat exposure
iii. The rate of internal heat production (exercise intensity).

An athlete must exercise in a hot environment to attain acclimatization that carries over to exercise in a heat. Simply sitting in a hot environment, such as a sauna, for long periods each day will not prepare the individual for physical exertion in the heat.

Although most individuals must be exposed to the heat to gain full adjustment, they can gain partial heat tolerance simply by training, even if it is done in a cooler environment. Interestingly, when athletes become acclimatized to a given level of heat stress, they can also perform better in cooler environments. But to gain maximum benefits, athletes who train in environments cooler than those in which they will be competing should achieve heat acclimatization prior to the contest or event. This will improve their performances and reduce the associated physiological stress and risk of heat injury.
If athletes must compete in hot weather, at least part of their training should be conducted in the warmest part of the day. Early morning and evening training will not fully prepare an athlete to tolerate the midday heat. Normal workouts in the heat for 5 to 10 days should provide nearly total heat acclimatization. Workout intensity should be reduced to 60 to 70 percent during the first few days to prevent excessive heat stress. Of course, care must be taken to guard against heat injuries such as heat stroke and heat exhaustion. Those in training should be alert to any symptoms and should consume as much fluid as possible.

EXERCISE IN THE COLD

Increasing year-round participation in such sporting activities as the triathlon, scuba diving, running, cycling, and long-distance swimming has sparked new interest in, and concerns about, exercise in the cold. In addition, some occupations require employees to work in cold conditions that can limit their performance. For these reasons, understanding the physiological responses and health risks associated with cold stress are important issues in exercise science. The cold stress here as any environmental condition that causes a loss of body heat that threatens homeostasis. In the following discussion it will be focused on the two major cold stresses: air and water.

The hypothalamus has a temperature set point of about 37°C (99°F), but daily fluctuations in the body temperature can be as much as 1°C. A decrease in either skin or blood temperature provides feedback to the thermoregulatory center (hypothalamus) to activate the mechanisms that will conserve body heat and increase heat production. The primary means by which our body avoid excessive cooling are:

i. Shivering
ii. Nonshivering thermogenesis
iii. Peripheral vasoconstriction

Because these mechanisms of heat production and conservation are often inadequate, the clothing and subcutaneous fat help to insulate the deep body tissues from the environment.

Shivering

The uncontrolled muscular contraction can cause a four to five fold increase in the body’s resting rate of heat production. Nonshivering thermogenesis involves stimulation of metabolism by the sympathetic nervous system. Increasing the metabolic rate increases the amount of internal heat production.

Peripheral vasoconstriction occurs as a result of sympathetic stimulation to the smooth muscle surrounding the arterioles in the skin. This stimulation causes the muscle to contract, which constricts the arterioles and reduces the blood flow to the shell of the body and prevents unnecessary heat loss. The metabolic rate of the skin cells also decreases as the skin’s temperature falls, so the skin requires less oxygen.

Clothing

Although clothing is important in terms of insulation for the inactive person in cold weather, an individual engaged in a strenuous winter sport such as skiing or speed skating needs little clothing because of his high heat production. Since vigorous activity may cause the metabolic rate to increase by as much as 25 to 30 times basal values, and since clothing may act as a partial water vapor barrier, it is obvious that clothes worn during muscular activity in cold weather can become a hindrance in the temperature regulation process. For instance, it is known that most active people generally sweat profusely even in cold environments, and if the clothing worn is heavy and allows for no ventilation, then the sweat is condensed in the clothing and hence, does not achieve skin cooling. Eventually, however, during periods between works, the sweat evaporates and thus takes away from the body heat that it cannot afford to lose at that time. Therefore, an important consideration for individuals working in cold weather is that the clothing worn should allow for considerable ventilation as well as vapor permeability. While some people wear protective scarves or clothing over their faces to protect themselves against the possibility of chapped lips or a dry mouth and throat due to the low humidity
in the cold air, it should be pointed out that there is no evidence at this time to indicate any lung tissue damage as a result of exercising in cold weather.

**Factors Affecting Body Heat Loss**

As in the case of heat stress, the body’s ability to meet the demands of thermoregulation is limited when exposed to extreme cold. Too much heat loss can occur. Those factors (conduction, convection, radiation and evaporation) that usually perform so effectively in dissipating metabolically produced heat during exercise in warm conditions, can, in a cold environment, dissipate heat faster then the body produces it.

Pinpointing the exact conditions that permit excessive body heat loss and eventual hypothermia (low body temperature) is difficult. Thermal balance depends on a wide variety of factors that affect the gradient between body heat production and heat loss. Generally, the larger the difference between the temperatures of the skin and the cold environment, the greater the heat loss. However, a number of anatomical and environmental factors can influence the rate of heat loss.

**Body Size and Composition**

Insulating the body against the cold is the most obvious protection against hypothermia. Subcutaneous fat is an excellent source of insulation. Skinfold measurements of subcutaneous fat thickness are a good indicator of an individual’s tolerance for cold exposure. The thermal conductivity of fat (its capacity for transferring heat) is relatively low, so it impedes heat transfer from the deep tissues to the body surface. People who have more fat mass conserve heat more efficiently in the cold.

The rate of heat loss is also affected by the ratio of body surface area to body mass. Tall, heavy individuals have a small surface-area-to-body-mass ratio, which makes them less susceptible to hypothermia.

True gender differences in cold tolerance are minimal. Women tend to have more body fat than men. Some studies have shown that the added subcutaneous fat in females might give them an advantage during cold water immersion. When males and females of similar body fat mass, size, fitness are compared, little difference is noted in body temperature regulation with exposure to the cold.

**Windchill**

As with heat, the air temperature alone is not a valid index of the amount of thermal stress experienced by the individual. Wind creates a chill factor, known as the windchill, by increasing the rate of heat loss via convection and conduction. Also, the more humid the air, the greater the physiological stress. A dry, still day at 10ºC (50ºF) in the direct sun can be comfortable. Yet on a moist, windy day with complete cloud cover, the cold at this same temperature can be quite penetrating.

**Cold Acclimatization**

It is generally known that repeated exposure to cold temperatures results in greater ability to tolerate cold. However, no definite pattern of acclimatization to cold environments has been reported for humans, and some investigators question the occurrence of such a pattern. On the other hand, animals kept at 5ºC definitely indicate acclimatization. This includes an increased metabolism of 50 to 100 percent, which is linked with a decrease of shivering activity (Nonshivering thermogenesis). It appears that this nonshivering thermogenesis may originate in the muscle tissue, but not from muscle contraction. The chemical mechanism of this extra metabolism has not yet been reported, however, it is possible that the hormones, norepinephrine and thyroxine play a major role in the elevation of the metabolic rate. Following a period of 3 to 4 weeks in cold and as well as cold acclimatization, the thyroid gland gradually enlarges and thus increases the rate of thyroxine secretion. Apparently, this mechanism increases the basal metabolic rate by as much as 15–20 percent.

Although local skin cold acclimatization for the human is well defined, attempts to demonstrate whole body cold acclimatization in human populations have resulted in some contradictory as well as in unresolved problems of racial, ethnic
and dietary involvements. Nonetheless, the general trend of human cold acclimatization is hinted to be related to the following:

i. Metabolic
ii. Insulation
iii. Hypothermic
iv. Peripheral Circulation

Some experiments in which human subjects have been chronically exposed to cold strongly suggest that increased heat production without shivering can be developed. In a study comparing thermoregulation in Eskimos and European subjects, the nonshivering metabolism of the Eskimo natives was found to be about 30 to 40 percent greater than that of the European controls. While it was suggested that this difference might have been due to the Eskimo’s diet and chronic exposure to cold environment.

Information about cold acclimatization is limited, but some data suggest that chronic daily exposure to cold water increases subcutaneous body fat. Much of information regarding habituation to the cold has been obtained from observations on the aborigines, natives of the Australian outback, where they are normally exposed to low temperatures at night and high temperatures during the day. Compared to unacclimatized Europeans, the aborigines were able to sleep more comfortably in the cold with little protection and they experienced only minor changes in their metabolism and rectal temperatures. The Europeans, on the other hand, experienced significant distress and considerable difficulty in maintaining normal body temperature.

Although some data suggest that repeated exposure to the cold alters peripheral blood flow and skin temperatures, these changes are small and the findings are inconclusive. Field studies have shown that chronic exposure of some areas of the skin, such as the hands, can provide greater cold tolerance. For example, Gaspe Northern Norwegian fisherman who must work with their hands in cold water for many hours develop increased vasodilatation and local warming of this exposed skin. The rate and degree of adjustment to these conditions have not been fully explained. Thus, acclimatization to cold is not as thoroughly understood as acclimatization to environmental heat stress.

**Physiological Responses to Exercise in the Cold**

**Muscle Function**

Cooling muscle causes it to become weaker. The nervous system responds to muscle cooling by altering the normal muscle fiber recruitment patterns. Some researchers have suggested that this change in fiber selection for force development decreases the efficiency of the muscle action. If clothing insulation and exercise metabolism is sufficient to maintain the athlete’s body temperature in the cold, then exercise performance may be unimpaired. However, as fatigue sets in and muscle activity slows, body heat production gradually decreases. Long distance running, swimming and skiing in the cold can be exposed the participant to such condition.

**Metabolic Response:** Exercise triggers of the catecholamine, which increase the mobilization and use of free fatty acids for fuel. But in the cold, vasoconstriction impaired circulation to the subcutaneous fat tissues, so this process is attenuated.

**Risks during Exercise in the Cold**

If humans had retained the ability of lower animals like reptiles to tolerate low body temperatures, we could survive even extreme hypothermia. Unfortunately in humans the evolution of thermoregulation has been accompanied by a loss in the ability of vital tissues to function when they are cooled by more than a few degrees.

**Hypothermia**

If the body temperature falls below 34.5°C (94°F), the hypothalamus begins to lose its ability to regulate body temperature. This ability is completely lost when the internal temperature falls to about 29.5°C (85°F). This loss of function is associated with slowing of metabolic reaction to one-half their normal rates for each 10°C decline
in cellular temperature. As a result, cooling the body can cause drowsiness even coma.

**Cardio Respiratory Effects**

The hazards of cold exposure include potential injury to both peripheral tissues and the life supporting cardiovascular and respiratory systems. The most important effect of hypothermia is on the heart. Death for hypothermia has resulted from cardiac arrest while respiration was still functional. Cooling primarily influences the SA Node—the heart’s pacemaker.

It is questioned whether rapid, deep breathing of cold air can cause freezing or damage to the respiratory tract. In fact, the cold air that passes into the mouth and trachea is rapidly warmed even when the inhaled air is less than –25°C (–13°F). Excessive cold exposure does affect respiratory function: which decreases the respiratory rate and volume.

**Treatment of Hypothermia**

Mild hypothermia can be treated by giving a person protection from the cold and providing dry clothing and warm beverages. Moderate to severe cases of hypothermia require gentle handling avoid initiating a cardiac arrhythmia. This requires slowly rewarming the victim. Severe cases of hypothermia require hospital facilities and medical care.

**Frostbite**

The exposed skin can freeze when its temperature is lowered just a few degrees below the freezing point (0°C, 32°F). Because of the warming influence of circulation and metabolic heat production, the environmental air temperature (including windchill) required to freeze one’s exposed fingers, nose and ears is about –29°C (–20°F). Recall from our earlier discussion that peripheral vasoconstriction helps the body retain heat. Unfortunately, during exposure to extreme cold, the circulation in the skin can decrease to the point that the tissue dies from the lack of oxygen and nutrients. This is commonly called frostbite. If not treated early, frostbite injuries can be serious, leading to gangrene and loss of tissue. Frostbite parts should be left untreated until they can be thawed, preferably in a hospital, without risk of refreezing.

**Performance at Altitude**

It is an established fact that at altitude of over 5000 feet (1524 meters), the ability to perform physical work is affected—the higher the altitude, the more severe the effects. In general, one can expect a reduction in endurance capacity as measured by the maximal oxygen consumption (max VO₂) of 3 to 3 1/2 percent for every 1000 feet ascended above 5000 feet. Work performance and max VO₂ are reduced by 60 percent or more at extremely high altitudes, i.e. at around 25,000 feet. Although such reduction in physical performance are quite large as they stand.

**Altitude Acclimatization**

The longer you remain at altitude, the better becomes your performance, but it never quite reaches the values that are obtained at sea level. The improved performance during stay at altitude is brought about through acclimatization. The number of weeks to acclimatize depends on the altitude, i.e. for 9000 feet, about 7 to 10 days; for 12,000 feet, 15 to 21 days; and for 15,000 feet, 21 to 25 days respectively. These are only approximations; a great deal depends on the individual. As a matter of fact, a few people will never acclimatize and continue to suffer mountain or altitude sickness while at altitude.

**Physiology of Altitude Acclimatization**

Above sea level, the barometric pressure (Pₚ) decreases, as the weight of the atmosphere becomes less. The percentage of oxygen in the air remains 20.93 percent, but the number of oxygen molecules per unit volume decreases. This means that when at altitude, in order to receive the same number of molecules in a breath of air that receive at sea level, must breathe more air. The main reason for lessened performance at altitude is a consequence of the lowered oxygen partial pressure (PO₂). This results in hypoxia,
Table 11.1: The immediate and long-term effect on various physiological parameters at high altitude

<table>
<thead>
<tr>
<th>System</th>
<th>Immediate effect</th>
<th>Long term effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary acid-base balance</td>
<td>i. Hyperventilation</td>
<td>i. Hyperventilation</td>
</tr>
<tr>
<td></td>
<td>ii. Body fluid become more alkaline</td>
<td>ii. Excretion of base via kidneys and concomitant reduction in alkaline reserve</td>
</tr>
<tr>
<td>Cardiovascular system</td>
<td>i. Increase in submaximal heart rate</td>
<td>i. Submaximal heart rate remains elevated</td>
</tr>
<tr>
<td></td>
<td>ii. Increase in submaximal cardiac output</td>
<td>ii. Submaximal cardiac output falls to or below sea level values</td>
</tr>
<tr>
<td></td>
<td>iii. Stroke volume remains the same or slightly lowered</td>
<td>iii. Stroke volume is lowered</td>
</tr>
<tr>
<td></td>
<td>iv. Maximum cardiac output remains the same or slightly lowered</td>
<td>iv. Maximum cardiac output is lowered</td>
</tr>
<tr>
<td>Hematological value</td>
<td></td>
<td>i. Decrease plasma volume</td>
</tr>
<tr>
<td></td>
<td></td>
<td>ii. Increased hematocrit</td>
</tr>
<tr>
<td></td>
<td></td>
<td>iii. Increased Hemoglobin concentration</td>
</tr>
<tr>
<td></td>
<td></td>
<td>iv. Increased total number of RBC count.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>v. Increased capillary density of skeletal muscle</td>
</tr>
<tr>
<td></td>
<td></td>
<td>vi. Increased mitochondrial density</td>
</tr>
<tr>
<td></td>
<td></td>
<td>vii. Increased aerobic enzyme in muscles</td>
</tr>
<tr>
<td></td>
<td></td>
<td>viii. Loss of body weight and lean body mass</td>
</tr>
</tbody>
</table>

i.e. lack of adequate oxygen. Apparently, hypoxia stimulates the acclimatization mechanisms. Depending upon altitude and duration of stay, among the important physiological changes that take place during acclimatization to altitude are following:

**Immediate Responses to Altitude**

On arrival at elevations of about 2300m and higher, rapid physiological adjustments occur to compensate for the thinner air and accompanying reduced alveolar PO₂. The most important of these responses are:
- An increase in the respiratory drive that results in a relative hyperventilation (increase the breathing frequency).
- An increase in blood flow both at rest and during submaximal exercise.

**Increased Pulmonary Ventilation (Hyperventilation)**

This response is immediate (within a few hours) upon arrival at altitude, being more pronounced during the first few days and then stabilizing after about a week at altitude. The most important result of hyperventilation is an increased alveolar PO₂. This ensures a greater saturation of hemoglobin with oxygen. With hyperventilation, excessive amount of CO₂ are blown off thus decreasing both the alveolar PCO₂ and the H⁺ concentration (increased pH).

**Increased Cardiovascular Responses**

In the early stages of altitude adaptation, submaximal exercise heart rate and cardiac output may increase by 50 percent above sea level values, whereas the heart’s stroke volume remains unchanged. Because the oxygen cost of exercising at altitude is no different than at sea level, the increase in blood flow at altitude partially compensates for the reduced oxygen in arterial blood.

**Altitude Related Medical Problems**

The people who live and work at high altitude, as well as newcomers to altitude, often encounter a
variety of medical problems associated with the reduced PO2 at higher elevations. Some of these problems are transient and mild dissipate within hours or several days, depending on the rapidity of the ascent and degree of exposure; other medical complications can be severe and can significantly compromise the person’s overall safety. Three medical conditions pose potential problem to those who ascent to high altitude: (a) acute mountain sickness (b) high altitude pulmonary edema and (c) high altitude cerebral edema.

**Acute Mountain or Altitude Sickness (AMS)**

This happens even with people who were born and reside at altitude. Suddenly, for unknown reasons, they lose their acclimatization and suffer from mountain sickness. The symptoms of altitude sickness include pulmonary edema, nausea, vomiting, headache, rapid pulse and anorexia (loss of appetite). Emergency treatment of severe mountain sickness consists of administering oxygen or removal to lower altitude and both.

Appetite suppression can be severe during the early stages of a high altitude stay, resulting in an average reduction in energy intake of approximately 40 percent and an accompanying loss of body mass. Even moderate exercise can be intolerable for people suffering the effects of mountain sickness. With acclimatization, symptoms subside and many of them disappear. Concurrently, a person’s ability to exercise improves and considerably more work can be accomplished. Mountain sickness usually can be prevented by acclimatizing slowly to moderate altitude below 3,048 m followed by a slow progression to higher elevations. Physical activity also should be minimized during the first day of altitude exposure.

**High Altitude Pulmonary Edema**

The altitude above 3,048 m experience a severe complication from acute mountain sickness termed **High-Altitude Pulmonary Edema (HAPE)**. Symptoms of HAPE usually manifest within 12 to 96 hours after a rapid ascent to high altitudes. In this condition fluid accumulates in the brain and lungs. Initially the symptoms do not seem too severe (general fatigue, dyspnea upon exertion, persistent dry, irritating cough without phlegm and without pre-existing pulmonary infection, pain or pressure in the substernal area, headache, and nausea). This leads to pulmonary edema and fluid retention by the kidneys. Even in well acclimatized individuals, HAPE can develop upon severe exertion at elevations above 5,486 m and probably is related to increases in pulmonary artery pressure.

**High Altitude Cerebral Edema (HACE)**

This condition of increased intracranial pressure can lead to coma and ultimately can death. The early symptoms are similar to AMS and HAPE, but the symptoms eventually become more severe. HACE occurs in approximately 1 percent of people exposed to altitudes usually above 2700 m. In addition to debilitating headache and severe fatigue, there is disruption vision, bladder and bowel dysfunction, and loss of coordination involving the trunk muscles, paralysis on one side of the body, generally poor reflexes and mental confusion. Cerebral edema is probably due to cerebral vasodilatation and an elevated capillary hydrostatic pressure.

**Fluid Loss**

The air in mountainous region is usually cool and dry, considerable body water can be lost through evaporation as inspired air is warmed and moistened in the respiratory passages. This fluid loss often leads to a moderate dehydration and accompanying symptoms of dryness of the lips, mouth and throat.

**Long-term Adjustment to Altitude**

Hyperventilation and increased submaximal exercise cardiac output provide a rapid and relatively effectively counter to the acute challenge of altitude exposure. Concurrently, other slower-acting adjustments occur during a prolonged high altitude stay. The most important of these involve:

- Regulation of the acid base balance of body fluids that become altered by hyperventilation.
- Increased production of hemoglobin and red blood cells and accompanying changes in local circulation and cellular function.
Both of these adaptations generally reduced distress and improve tolerance to the relative hypoxia of medium and high altitudes.

Acid-Base Readjustment

Though hyperventilation at altitude increased alveolar PO$_2$, it has the opposite effect on CO$_2$. Because ambient air contains essentially no CO$_2$, the increased breathing volume at altitude tends to washout or dilute this gas in the alveoli. This creates a larger than normal gradient for the diffusion of CO$_2$ from the blood to the lungs and arterial CO$_2$ is reduced considerably. During a prolonged stay at high altitude the pressure of alveolar CO$_2$ falls to as low as 10 mm of Hg.

The loss of CO$_2$ from the body's fluids in a hypoxic environment causes a physiologic disequilibrium. A decrease in CO$_2$ as occur in hyperventilation causes the pH to rise (due to loss of carbonic acid) and the blood becomes more alkaline.

Hyper ventilation is a normal and sustained response to altitude exposure, adjustments must be made during acclimatization to minimize the side effects that disrupt the acid – base balance. This control of respiratory alkalosis is accomplished slowly as the kidneys excrete base (HCO$_3^-$) through the renal tubules. In turn, the restoration of a normal pH increases the responsiveness of the respiratory center, thus enabling ventilation to increase to even higher levels to adjust to altitude hypoxia.

The establishment of acid-base equilibrium with acclimatization occurs at the expense of a loss of absolute alkaline reserve. Thus, although the pathways of anaerobic metabolism are unaffected at high altitude, the blood’s buffering capacity for acid is gradually decreased, and the critical level for the accumulation of acid metabolites is lowered. A general depression in maximal lactate concentration is particularly apparent during maximal exercise at altitudes above 4000 m.

Decrease in Plasma Volume

During the first several days of altitude exposure the plasma volume decrease and which causes the RBC becomes more concentrated in the plasma.

After 1 week at 2300 m, for example, the plasma volume decreased by approximately 8 percent whereas the concentration of RBC increased by 4 percent and hemoglobin increased by 10 percent. This rapid reduction in plasma volume and accompanying hemoconcentration causes the oxygen content of arterial blood to increase significantly above values observed upon arrival at altitude.

Increase in Red Blood Cell

The reduced arterial PO$_2$ at high altitude also stimulates an increase in the total number of RBC, a condition termed polycythemia. This response is mediated by an erythrocyte stimulating hormone, erythropoietin, which is released from the kidneys and other tissues within 15 hours after altitude ascent. In the weeks that follow, the production of erythrocytes in the marrow of the long bones increases considerably and remains elevated during the stay at altitude.

Polycythemia directly translates into an increase in the blood’s capacity to transport oxygen. For well acclimatized mountaineers, the blood O$_2$ carrying capacity slightly more as compared to low land residents. Therefore, even with the reduced saturation of hemoglobin with high altitude, the actual quantity of O$_2$ in arterial blood approaches or even equals sea-level values. Supplementation of iron is beneficial to increase the hemoglobin concentration and hematocrit of the athletes training at high altitude as compared to the athletes are refrain from the supplementation of iron.

Tissue Level Changes

These changes include (a) increased muscle and tissue capillarization; (b) increased myoglobin concentration; (c) increased mitochondrial density and (d) enzyme changes that enhance the oxidative capacity. Unlike the previously mentioned acclimatization processes, these cellular changes take more time. In fact, they are seen most developed in the long-time resident of high altitude regions.

These are major physiological changes that greatly aid in delivering oxygen to the tissues when oxygen is hard to come by, i.e. under hypoxic
conditions. When the person returns from 3–4 week spent at altitude, he or she will lose these changes brought about by acclimatization within a period of about 2 to 4 weeks.

**Changes in Body Mass and Composition**

Long-term exposure to high altitude produces a significant loss in lean body mass and body fat; the magnitude of weight loss is directly related to the terrestrial elevation. This is because a marked increase in the basal metabolic rate upon arrival at high altitude, which facilitates weight loss. To some extent, this accelerated metabolic rate can be over hidden and weight loss can be minimized by increasing the energy intake during the high altitude stay.

**Time Required for Acclimatization**

The length of the acclimatization period depends on the altitude. Acclimatization at one altitude ensures only partial adjustment to a higher elevation. As a broad guide line, approximately 2 weeks are required to adapt to altitude up to 2300 m. Therefore, for each 610 m increase in altitude, an additional week is necessary to an fully adapt up to an altitude of approximately 4600 m. For athletes desiring to compete at altitude, intense training should commence as soon as possible during the acclimatization period. This will minimize any detraining effects because it is difficult to engage in heard training during the early days of one’s altitude stay. The benefits of acclimatization are lost within 2 or 3 weeks after returning to sea level.

**Endurance Capacity on Return to Sea Level**

Sea level exercise performance does not improve after living at altitude when VO₂-max serves as the improvement criterion. Eighteen days stay at 3100 m produce no change in the altitude-induced aerobic capacity in young runners has been reported. Also VO₂-max remained at the same as pre altitude value on return to sea level. Even in studies that reported a small improvement in VO₂-max or exercise performance at altitude and on return to sea level, the change often relates to increased physical activity during altitude exposure.

**Altitude Training and Sea Level Performance**

It is clear that altitude acclimatization improves ones capacity for aerobic metabolism for aerobic exercise at altitude. Altitude adaptations in local circulation and cellular metabolism, as well as the compensatory increase in the blood's oxygen carrying capacity, should facilitate subsequent sea level performance. Also the pulmonary adaptation
and responses during prolonged hypoxic exposure are lost immediately upon descent from altitude. Furthermore, if tissue hypoxia is an important training stimulus, altitude and training should act synergistically so that the total effects exceed that of similar training at sea level.

**Effectiveness of Altitude Training and Sea level Training**

It has been reported by the researcher that two homogenous groups were selected to determine whether altitude training is more effective than similar sea level training. Six highly trained middle distance runners trained at sea level for 3 weeks at 75 percent of the sea level VO$_2$ max at 2300 m altitude. The groups then exchanged training sites and continued training for 3 weeks at intensity similar to that of the preceding group. Initially 2 mile run times were 7.2 percent slower at altitude than at sea level. This improved approximately 2.0 percent for both groups after altitude training, but post performance at sea level was unchanged when compared to pre-altitude sea-level runs. As shown in the figure below, VO$_2$ max for both groups at altitude was reduced initially by approximately 17.4 percent. This improved only slightly after 20 days of altitude training. When the runners were then measured at sea level, aerobic capacity was 2.8 percent below pre-altitude sea level values. For these well conditioned middle-distance runners, there was no synergistic effect of hard aerobic training at medium altitude over an equivalent program of severe training pursued at sea level.

**Training and Altitude**

From a theoretical viewpoint, training at altitude could produce more rapid and even greater physiological changes than could train at sea level only. The reason for this is that altitude hypoxia is a stress that produces physiological changes (acclimatization) similar to those caused by physical training. For example, total blood volume, hemoglobin, red blood cell count, mitochondrial concentration and muscle enzyme changes have all been shown to be enhanced in both types of stress. To a certain extent, this idea has been supported experimentally. For example, in several well controlled studies using nonathletes, greater increases in maximal aerobic power and endurance time were seen when the training sessions were conducted at altitude (7400 to 11,300 feet) rather than at sea level. In addition, some effects of 8 weeks of interval training can be maintained for an additional 12 weeks by use of two 3-hour exposures to a simulated altitude of 15,000 feet (4572 meters). During the exposures, the subjects did not perform any exercise, but merely rested. Other studies have

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**Fig. 11.4:** Maximal O$_2$ uptake of two equivalent groups during training for three weeks at sea level. Group-1 trained first at sea level and then continued training for three weeks at altitude. For group-2 the procedure was reversed, as they trained first at altitude and then at sea level (From Adams, WC et.al.: Effects of equivalent sea level and altitude training on VO$_2$-max and running performance. J.Appl.Physiol. 39; 262, 1975)
Table 11.2: Time trials in running before, during & after training at an altitude of 7500 feet (2300 meters)

<table>
<thead>
<tr>
<th>Event</th>
<th>Time at sea level (min: sec)</th>
<th>Time at altitude (min: sec)</th>
<th>Time on return to sea level (min: sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Day 3</td>
<td>Day 14</td>
<td>Day 1</td>
</tr>
<tr>
<td></td>
<td>Day 21</td>
<td>Day 21</td>
<td>Day 1</td>
</tr>
<tr>
<td>880-yard run</td>
<td>2:41</td>
<td>2:48</td>
<td>2:38</td>
</tr>
<tr>
<td>1-mile run</td>
<td>6:07</td>
<td>6:30</td>
<td>—</td>
</tr>
<tr>
<td>2-mile run</td>
<td>13:08</td>
<td>13:45</td>
<td>13:09</td>
</tr>
</tbody>
</table>

Based on data from Faulkner, Daniels and Balke

shown improved in performance at sea level after training at altitude. Some of the results are given in above table. However, from this study it is possible that their performances would have been conclude with further training even at sea level.

A study on highly trained subject revealed that $V_{O_2}$ max and running performance were no different returned to sea level than in the pre-altitudes measures. Running performance after acclimatization was measured in the 440-Yard, 880 Yard, 1-mile, and 2-mile runs of the altitude natives and found that run time were slower than pre-altitude times (Fig. 11.5)

It can be seen from Figures 11.6 that in studies involving highly trained athletes performance on return from altitude was not much different from prior performance at sea level; if anything, some were poorer. This would indicate that for the highly trained athlete, training at and acclimatization to altitude does not improve performance. Also as already pointed out, maximal aerobic power and performance of these athletes do not always improve with altitude acclimatization.

This can be seen from Figure 11.7, which gives the intensity of the training workouts for six collegiate runners at various altitudes. It is clear that altitude greatly reduced their training efforts.

Training at altitude, therefore, appears to be helpful for unconditioned, nonathletic subjects but not necessarily for highly trained athletes. The following guidelines may prove to be helpful:

1. Adequate training facilities and training atmosphere must be available.
2. The bulk of time spent at altitude should be at moderate altitude (6500–7500 feet).
Environment and Physical Performance

Fig. 11.7: Percent of sea level performance of 2 mile run and Vo2 max of different duration of altitude

3. Short exposure to higher altitude should be included regularly during the general training period at moderate elevation.

4. Steady altitude exposures should be limited to the periods of 2 to 4 weeks, with intermittent sea level or lower elevation trips scheduled to assure maintenance of muscular power and normal competitive rhythm and intensity of effort.

5. Training at altitude should emphasize maintenance of muscular power yet be geared to include normal or near normal overall amounts of work.

6. Important sea level efforts should be scheduled about 2 weeks after leaving altitude.

**SUMMARY**

1. A large amount of heat is produced and lost from the body constantly, yet the body temperature remains constant within a limited range. This indicates that there is strong machinery, which keeps an exact balance between gain and loss of heat and thereby maintains a constant body temperature. Increasing heat production is obtained primarily by increasing muscular activity. In strenuous activity, heat production from metabolism has been known to increase by as much as 30 times its normal. There are three ways by which heat is lost from the body: (i) Skin (ii) lungs and (iii) excretion. Skin is the most important organ among the three and approximately 85 percent of heat is lost via this organ, although this depends on external and internal conditions. Thus, the bodily changes that regulate the exchange of heat between the body and the environment are referred to as physical heat regulation. Heat is lost from the skin by Conduction, Convection, Radiation and Evaporation.

2. The role of nervous system is also very important to control the temperature in human body. There are several centers in the spinal cord and the brain, which control such activities as vasoconstriction, vasodilatation, sweating, muscle tone and shivering. In order for body temperature to remain at or near a constant level, the integration of these various activities is essential. The effects of several hormones cause the cells to increase their metabolic rates. This affects heat balance because increased metabolism increases heat production. Cooling the body stimulates thyroxine release from the thyroid gland, which can elevate the metabolic
rate throughout the body by more than 100 percent. Also, epinephrine and norepinephrine mimic and enhances the activity of the sympathetic nervous system. Thus, they directly affect the metabolic rate of virtually all body cells.

3. Air temperature along is not an accurate index of the total physiological stress imposed on the body in a hot environment. At least four variables must be taken into account: Air temperature, Humidity, Air velocity and the amount of radiation. All these influence the degree of heat stress experience by the person. The contribution of each of these factors to the total body heat stress are not clear, because the contribution vary with changing environmental conditions.

4. Heat Stress is not accurately reflected by air temperature alone. Humidity, air velocity (or wind) and thermal radiation also contribute to the total heat stress that one experiences when exercising in the heat. Heat cramps, the least serious of three heat disorders, is characterized by severe cramping of the skeletal muscles. It primarily involves the muscles that are most heavily used during exercise. Heat exhaustion is typically accompanied by such symptoms as extreme fatigue, breathlessness, dizziness, vomiting, fainting, cold and clammy or hot and dry skin, hypotension (low blood pressure) and a weak, rapid pulse.

5. Heat syncope (fainting) is rare in a conditioned athlete. In most instances, it occurs when individuals stand for a prolonged period of time in the heat or exercise for a prolonged period in an upright position. Heat syncope is caused by a pooling of blood in the vasculature of the limbs and skin because of excessive ambient temperatures. Heat stroke is a life-threatening heat disorder that requires immediate medical attention. It is characterized by a rise in internal body temperature to values exceeding 40°C (104°F), cessation of sweating, hot and dry skin, rapid pulse and respiration, usually hypertension (high blood pressure), confusion and unconsciousness. If left untreated, heat stroke progresses to coma and death quickly follows.

6. The primary aim of fluid replacement is to maintain plasma volume so that circulation and sweating progress at optimal levels. Ingesting “extra” water before exercising in the heat provides some thermoregulatory protection. It delays the development of dehydration, increases sweating during exercise and brings about a smaller rise in body temperature compared to exercising without prior fluids.

7. Exercise in hot climates is a more serious problem than exercise in the cold. In a cold climate the increased metabolic heat production combats the increased heat loss to the environment, but in a hot climate metabolism and environment combine to increase heat gain in body tissues. The problem is further complicated by the fact that when environmental temperature approaches skin temperature (approximately 92°F), heat loss through convection and radiation gradually comes to an end, so that at temperatures above skin temperature the only means for heat loss is evaporation of sweat.

8. Repeated prolonged exercise bouts in the heat cause gradual improvement in the ability to eliminate excess body heat, which reduces the risk of heat exhaustion and heat stroke. This process, termed heat acclimatization, results in many adjustments in sweating and blood flow. This adaptation results from an increased blood volume, reduced blood flow to the skin or both. Either of these changes increases the stroke volume. Following heat acclimatization, more work can be done before the onset of fatigue or exhaustion.

9. Understanding the physiological responses and health risks associated with cold stress are important issues in exercise science. The cold stress as any environmental condition that causes a loss of body heat that threatens homeostasis. A decrease in either skin or blood temperature provides feedback to the thermoregulatory center (hypothalamus) to activate the mechanisms that will conserve body heat and increase heat production. The exact conditions that permit excessive body heat loss and eventual hypothermia (low body temperature) are difficult. Thermal balance depends on a wide variety of factors that affect the gradient between body heat production.
and heat loss. The larger the difference between the temperatures of the skin and the cold environment, the greater the heat loss.

10. It is generally known that repeated exposure to cold temperatures results in greater ability to tolerate cold. However, no definite pattern of acclimatization to cold environments has been reported for humans. Nonetheless, the general trend of human cold acclimatization is hinted to be related to the metabolic, insulation, hypothermic and peripheral circulation.

11. Physiological responses to exercise in the cold muscle become weaker. The nervous system responds to muscle cooling by altering the normal muscle fiber recruitment patterns. If the body temperature falls below 34.5°C (94°F), the hypothalamus begins to lose its ability to regulate body temperature. The hazards of cold exposure include potential injury to both peripheral tissues and the life supporting cardiovascular and respiratory systems.

12. It is an established fact that at altitude of over 5000 feet (1524 meters), the ability to perform physical work is affected—the higher the altitude, the more severe the effects. The improved performance during stay at altitude is brought about through acclimatization. However, few people will never acclimatize and continue to suffer mountain or altitude sickness while at altitude. On arrival at elevations of about 2300 m and higher, rapid physiological adjustments occur to compensate for the thinner air and accompanying reduced alveolar PO2.

13. The people who live and work at high altitude, as well as newcomers to altitude, often encounter a variety of medical problems associated with the reduced PO2 at higher elevations. Acute mountain sickness happens even with people who were born and raised at altitude. Suddenly, for unknown reasons, they lose their acclimatization and suffer from mountain sickness. The altitude above 3,048 m experience a severe complication from acute mountain sickness termed high-altitude pulmonary edema. High altitude cerebral edema is another one which increased intracranial pressure can lead to coma and ultimately can death.

14. Training at altitude could produce more rapid and even greater physiological changes than could train at sea level only. The reason for this is that altitude hypoxia is a stress that produces physiological changes (acclimatization) similar to those caused by physical training.

**Review Questions**

1. What are the mechanisms of heat production and heat loss? Which are the four pathways is most important for loss of body heat?

2. Discuss the role hypothalamus for body temperature regulation in human body.

3. What happens to the body temperature during exercise and why?

4. Why is humidity an important factor when performing in the heat? Why are wind and cloud cover important?

5. What is heat disorder? Differentiate between heat cramps, heat exhaustion, and heat stroke.

6. What measures you will take to prevention of heat illness? What is the role fluid replacement in prevention of heat disorder?

7. What physiological adaptations occur allowing one to acclimatize to exercise in the heat?

8. How does the body minimize excessive heat loss during exposure to cold?

9. What are the risks during exercise in cold? Write brief about the ‘Frost bite’.

10. Describe the conditions at altitude that limit physical activity.

11. Describe the physiological adjustments that accompany acclimatization to altitude.

12. What are the long-term adjustments takes place in human body at altitude?

13. Write notes on (a) Mountain sickness, (b) High altitude pulmonary edema and (c) High altitude cerebral edema.

14. Would an endurance athlete who trained at altitude be able to perform better during subsequent sea level performance? Why or why not?

15. What are the times required for acclimatization at an altitude? What will happen of Endurance capacity on return to sea level? Discuss the benefit of altitude training.
INTRODUCTION TO FATIGUE AND RECOVERY

Fatigue is defined as “that state following a period of mental or bodily activity characterized by a lessened capacity for work and reduce efficiency of accomplishment, usually accompanied by a feeling of weariness, sleepiness or irritability; it may also supervene when any cause energy expenditure outstrips restorative processes, e.g. lack of sleep or food.”

For the purpose of exercise physiology, the term fatigue to describe a transient decrease in working capacity that results from previous physical activity. We will further restrict our interest to dealing with neuromuscular fatigue, while fully recognizing the contribution of other systems such as the cardiovascular, pulmonary and endocrine systems to the end result. Neuromuscular fatigue can be defined as a transient decrease in muscular performance usually seen as a failure to maintain or develop a certain expected force or power.

Since fatigue has rather dramatic effects on both strength and reflexes it is to be expected that it would also affect coordination in the complex movements involved in athletics. Anyone who has ever watched the degraded performance of marathon runners at the finish line needs no further evidence. However, this effect of fatigue has been quantitatively studied, and results from many early investigations suggest that the deterioration of motor performance with fatigue and its consequent increase in oxygen demand per unit work done is probably due to irradiation of motor impulses to neighboring motor nerve centers. These nerve centers cannot make any meaningful contribution to the job at hand and may indeed
result in the coordination that can be observed in extreme fatigue states.

Definition

Fatigue is physical and/or mental exhaustion that can be triggered by stress, medication, overwork, or mental and physical illness or disease.

Description

Everyone experiences fatigue occasionally. It is the body’s way of signaling its need for rest and sleep. But when fatigue becomes a persistent feeling of tiredness or exhaustion that goes beyond normal sleepiness, it is usually a sign that something more serious is amiss.

Physically, fatigue is characterized by a profound lack of energy, feelings of muscle weakness, and slowed movements or central nervous system reactions. Fatigue can also trigger serious mental exhaustion. Persistent fatigue can cause a lack of mental clarity (or feeling of mental “fuzziness”), difficulty concentrating, and in some cases, memory loss.

Causes and Symptoms of Fatigue

Causes and Symptoms

Fatigue may be the result of one or more environmental causes such as inadequate rest, improper diet, work and home stressors, or poor physical conditioning and over training or one symptom of a chronic medical condition or disease process in the body. Heart disease, low blood pressure, diabetes, end-stage renal disease, iron-deficiency anemia, narcolepsy and cancer can cause long-term, ongoing fatigue symptoms. Acute illnesses such as viral and bacterial infections can also trigger temporary feelings of exhaustion. In addition, mental disorders such as depression can also cause of fatigue.

A number of medications, including anti-histamines, antibiotics and blood pressure medications, may cause drowsiness as a side-effect. Individuals already suffering from fatigue who are prescribed one of these medications may wish to check with their healthcare provider about alternative treatments.

Symptoms of Fatigue

The symptoms of the fatigue due to the overactivity/training/exhaustion are generally common which are as follows:

i. Color of the face: Colour of the face is looking red.
ii. Sweating: The rate of sweating is increased.
iii. Execution of movements: The movement of an individual becomes crude, staggering and lack of coordination if he is under fatigue.
iv. Concentration: Lack of concentration, nervousness and negative learning.
v. Continuity of training: The rest period of a person is prolonged if the person is in under fatigue.
vi. Behavior changes: Aggressiveness, depression, irritatedness, etc.
vii. General health symptoms: Heaviness, dizziness, breathing difficulties, nausea, vomiting, restlessness, insomnia, etc.
viii. Performance: Low physical performance will be executed by the sports person.

Extreme fatigue which persists, unabated, for at least six months, is not the result of a diagnosed disease or illness, and is characterized by flu-like symptoms such as swollen lymph nodes, sore throat, and muscle weakness and/or pain may indicate a diagnosis of chronic fatigue syndrome. Chronic fatigue syndrome (sometimes called chronic fatigue immune deficiency syndrome), is a debilitating illness that causes overwhelming exhaustion and a constellation of neurological and immunological symptoms.

Methods of Recovery

Recovery must start in the concluding part/session of the training. Aims, objectives and tasks of all aspects of the training should be clear to the athletes. Active recovery is more effective than passive one. Recovery in micro and meso cycle through change in activity and also through low intensity physical exercise. The following methods are to be followed for recovery from fatigue:

i. Rest: Adequate/proper rest is required.
ii. Living condition: Well ventilated, hygienic living condition is needed.
iii. Food: Well balanced qualitative and quantitative diet to be taken.
iv. Physical therapy: Massage and self relaxation (increased blood circulation) is recommended.
v. Hydrotherapy: Jet massage, Jaquizee, contrast bath, etc. (Stimulate blood circulation to removal of metabolites) are suggested.
vi. Thermotherapy: Wax bath, Steam packs, etc. (Increased metabolism to remove waste products, sweating to perspiration eliminates waste) are recommended.
vii. Electotherapy: Short and microwave diathermy, Ultrasound, etc. are also useful for recovery.
viii. Psychotherapy: Relaxation, spiritual, yoga, meditation, autogenic training, etc are also suggested.

**Diagnosis**

As fatigue is a symptom of a number of different disorders, diseases and lifestyle choices, diagnosis may be difficult. A thorough examination and patient history by a qualified healthcare provider is the first step in determining the cause of the fatigue. A physician can rule out physical conditions and diseases that feature fatigue as a symptom, and can also determine if prescription drugs, poor dietary habits, work environment, or other external stressors could be triggering the exhaustion. Several diagnostic tests may also be required to rule out common physical causes of exhaustion, such as blood tests to check for iron-deficiency anemia.

Diagnosis of chronic fatigue syndrome is significantly more difficult. Because there is no specific biological marker or conclusive blood test to check for the disorder, healthcare providers must rely on the patient’s presentation and severity of symptoms to make a diagnosis. In many cases, individuals with chronic fatigue syndrome go through a battery of invasive diagnostic tests and several years of consultation with medical professionals before receiving a correct diagnosis.

**TREATMENT**

Conventional medicine recommends the dietary and lifestyle changes outlined above as a first line of defense against fatigue. Individuals who experience occasional fatigue symptoms may benefit from short-term use of caffeine-containing central nervous system stimulants, which make people more alert, less drowsy and improve coordination. However, these should be prescribed with extreme caution, as overuse of the drug can lead to serious sleep disorders, like insomnia.

Another reason to avoid extended use of caffeine is its associated withdrawal symptoms. People who use large amounts of caffeine over long periods build-up a tolerance to it. When that happens, they have to use more and more caffeine to get the same effects. Heavy caffeine use can also lead to dependence. If an individual stops using caffeine abruptly, withdrawal symptoms may occur, including headache, fatigue, drowsiness, yawning, irritability, restlessness, vomiting, or runny nose. These symptoms can go on for as long as a week. Excess intake of caffeine may become under the abuse of drug for sports person.

**Alternative Treatment**

The treatment of fatigue depends on its direct cause, but there are several commonly prescribed treatments for non-specific fatigue, including dietary and lifestyle changes, the use of essential oils and herbal therapies, deep breathing exercises, traditional Chinese medicine and color therapy. These are discussed below:

**Dietary Changes**

Inadequate or inappropriate nutritional intake can cause fatigue symptoms. To maintain an adequate energy supply and promote overall physical well-being, individuals should eat a balanced diet and observe the following nutritional guidelines:

- Drinking plenty of water. Individuals should try to drink 9 to 12 glasses of water a day. Dehydration can reduce blood volume, which leads to feelings of fatigue.
- Eating iron-rich foods (i.e., liver, raisins, spinach, apricots). Iron enables the blood to transport oxygen throughout the tissues, organs, and muscles, and diminished oxygenation of the blood can result in fatigue.
- Avoiding high-fat meals and snacks. High fat foods take longer to digest, reducing blood flow to the brain, heart, and rest of the body while blood flow is increased to the stomach.
Eating unrefined carbohydrates and proteins together for sustained energy.

- Balancing proteins. Limiting protein to 15 to 20 grams per meal and two snacks of 15 grams is recommended, but not getting enough protein adds to fatigue. Pregnant or breastfeeding women, growing children, and the sportsman of power game should get more protein.

- Getting the recommended daily allowance of B complex vitamins (specifically, pantothenic acid, folic acid, thiamine and vitamin B12). Deficiencies in these vitamins can trigger fatigue.

- Getting the recommended daily allowance of selenium, riboflavin and niacin. These are all essential nutritional elements in metabolizing food energy.

- Individuals should only eat when they are hungry and stop when they are full. An overstuffed stomach can cause short-term fatigue and individuals who are overweight are much more likely to regularly experience fatigue symptoms.

**Lifestyle Changes**

Lifestyle factors such as a high-stress job, erratic work hours, lack of social or family support, or erratic sleep patterns can all cause prolonged fatigue. If stress is an issue, a number of relaxation therapies and techniques are available to help alleviate tension, including massage, yoga, aromatherapy (The therapeutic use of plant-derived, aromatic essential oils to promote physical and psychological well-being). Hydrotherapy [Hydrotherapy, or water therapy, is use of water (hot, cold, steam, or ice) to relieve discomfort and promote physical well-being]; progressive relaxation exercises, meditation and guided imagery (The use of relaxation and mental visualization to improve mood and/or physical well-being). Some individuals may also benefit from individual or family counseling or psychotherapy sessions to work through stress-related fatigue that is a result of family or social issues.

Maintaining healthy sleep patterns is critical to proper rest. Having a set “bedtime” helps to keep sleep on schedule. A calm and restful sleeping environment is also important to healthy sleep. Above all, the bedroom should be quiet and comfortable, away from loud noises and with adequate window treatments to keep sunlight and streetlights out. Removing distractions from the bedroom such as televisions and telephones can also be helpful.

**Essential Oils**

Aroma therapists, hydro therapists, and other holistic healthcare providers may recommend the use of essential oils of rosemary (Rosmarinus officinalis), eucalyptus blue gum (Eucalyptus globulus), peppermint, (Mentha x piperata), or scots pine oil (Pinus sylvestris) to stimulate the nervous system and reduce fatigue. These oils can be added to bathwater or massage oil as a tropical application. Citrus oils such as lemon, orange, grapefruit, and lime have a similar effect, and can be added to a steam bath or vaporizer for inhalation.

**Herbal Remedies**

Herbal remedies that act as circulatory stimulants can offset the symptoms of fatigue in some individuals. An herbalist may recommend an infusion of ginger (Zingiber officinale) root or treatment with cayenne (Capsicum annuum), balmony (Chelone glabra), damiana (Turnera diffusa), ginseng (Panax ginseng), or rosemary (Rosmarinus officinalis) to treat ongoing fatigue.

An infusion is prepared by mixing the herb with boiling water, steeping it for several minutes and then removing the herb from the infusion before drinking. A strainer, tea ball, or infuser can be used to immerse loose herb in the boiling water before steeping and separating it. A second method of infusion is to mix the loose herbal preparation with cold water first, bringing the mixture to a boil in a pan or teapot and then separating the tea from the infusion with a strainer before drinking.

Caffeine-containing central nervous system stimulants such as tea (Camellia senensis) and cola (Cola nitida) can provide temporary, short-term relief of fatigue symptoms. However, long-term use of caffeine can cause restlessness, irritability, and other unwanted side effects, and in some cases may actually work to increase fatigue after the stimulating effects of the caffeine wear off. To avoid
these problems, caffeine intake should be limited to 300 mg or less a day (the equivalent of 4–8 cups of brewed, hot tea).

**Traditional Chinese Medicine**

Chinese medicine regards fatigue as a blockage, or energy flow, inside the human body. The practitioner of Chinese medicine chooses acupuncture and/or herbal therapy to rebalance the entire system. The Chinese formula Minot Bupleurum soup (or Xiao Chia Hu Tang) has been used for nearly 2,000 years for the type of chronic fatigue that comes after the flu. In this condition, the person has low-grade fever, nausea and fatigue. There are other formulas that are helpful in other cases. Acupuncture involves the placement of a series of thin needles into the skin at targeted locations on the body known as acupoints in order to harmonize the energy flow within the human body.

**Deep Breathing Exercises**

Individuals under stress often experience fast, shallow breathing. This type of breathing, known as chest breathing, can lead to shortness of breath, increased muscle tension, inadequate oxygenation of blood, and fatigue. Breathing exercises can both improve respiratory function and relieve stress and fatigue.

Deep breathing exercises are best performed while laying flat on the back on a hard surface, usually the floor. The knees are bent and the body (particularly the mouth, nose and face) is relaxed. One hand should be placed on the chest and one on the abdomen to monitor breathing technique. With proper breathing techniques, the abdomen will rise further than the chest. The individual takes a series of long, deep breaths through the nose, attempting to raise the abdomen instead of the chest. Air is exhaled through the relaxed mouth. Deep breathing can be continued for up to 20 minutes. After the exercise is complete, the individual checks again for body tension and relaxation. Once deep breathing techniques have been mastered, an individual can use deep breathing at anytime or place as a quick method of relieving tension and preventing fatigue.

**Color Therapy**

Color therapy, also known as chromatherapy, is based on the premise that certain colors are infused with healing energies. The therapy uses the seven colors of the rainbow to promote balance and healing in the mind and body. Red promotes energy, empowerment, and stimulation. Physically, it is thought to improve circulation and stimulate red blood cell production. Red is associated with the seventh chakra, located at the root; or base of spine. In yoga, the chakras are specific spiritual energy centers of the body.

Therapeutic color can be administered in a number of ways. Practitioners of Ayurvedic, or traditional Indian medicine, wrap their patients in colored cloth chosen for its therapeutic hue. Individuals suffering from fatigue would be wrapped in reds and oranges chosen for their uplifting and energizing properties. Patients may also be bathed in light from a color filtered light source to enhance the healing effects of the treatment.

Individuals may also be treated with color-infused water. This is achieved by placing translucent red colored paper or colored plastic wrap over and around a glass of water and placing the glass in direct sunlight so the water can soak up the healing properties and vibrations of the color. Environmental color sources may also be used to promote feelings of stimulation and energy. Red wall and window treatments, furniture, clothing, and even food may be recommended for their energizing healing properties.

Color therapy can be used in conjunction with both hydrotherapy and aromatherapy to heighten the therapeutic effect. Spas and holistic healthcare providers may recommend red color baths or soaks, which combine the benefits of a warm or hot water soak with energizing essential oils and the fatigue-fighting effects of bright red hues used in color therapy.

**Prognosis**

Fatigue related to a chronic disease or condition may last indefinitely, but can be alleviated to a degree through some of the treatment options outlined here. Exhaustion that can be linked to
environmental stressors is usually easily alleviated when those stressors are dealt with properly.

There is no known cure for chronic fatigue syndrome, but steps can be taken to lessen symptoms and improve quality of life for these individuals while researchers continue to seek a cure.

**Prevention**

Many of the treatments outlined above are also recommended to prevent the onset of fatigue. Getting adequate rest and maintaining a consistent bedtime schedule are the most effective ways to combat fatigue. A balanced diet and moderate exercise program are also important to maintaining a consistent energy level.

### Physiological Basis of Fatigue

#### Lactic Acid

The expression “lactic acid” is used most commonly by athletes to describe the intense pain felt during exhaustive exercise, especially in events like the 400 meters and 800 meters. When energy is required to perform exercise, it is supplied from the breakdown of ATP. The body has a limited store of about 85 grams of ATP and would use it up very quickly if we did not have ways of resynthesising it. There are three systems that produce energy to resynthesise ATP: ATP-PC, lactic acid and aerobic.

The lactic acid system is capable of releasing energy to resynthesise ATP without the involvement of oxygen and is called anaerobic glycolysis. Glycolysis (breakdown of carbohydrates) results in the formation of pyruvic acid and hydrogen ions (H⁺). The pyruvic acid molecules undergo oxidation in the mitochondrion and the Krebs cycle begins. A build-up of H⁺ will make the muscle cells acidic and interfere with their operation so carrier molecules, called nicotinamide adenine dinucleotide (NAD⁺), remove the H⁺. The NAD⁺ is reduced to NADH that deposit the H⁺ at the electron transport gate (ETG) in the mitochondria to be combined with oxygen to form water (H₂O).

If there is insufficient of oxygen supply then NADH cannot release the H⁺ and they build up in the cell. To prevent the rise in acidity pyruvic acid accepts H⁺ forming lactic acid that then dissociates into lactate and H⁺. Some of the lactate diffuses into the blood stream and takes some H⁺ with it as a way of reducing the H⁺ concentration in the muscle cell. The normal pH of the muscle cell is 7.1 but if the build-up of H⁺ continues and pH is reduced to around 6.5 then muscle contraction may be impaired and the low pH will stimulate the free nerve endings in the muscle resulting in the perception of pain (the burn). This point is often measured as the lactic threshold or anaerobic threshold (AT) or onset of blood lactate accumulation (OBLA).

The process of lactic acid removal takes approximately one hour, but this can be accelerated by undertaking an appropriate cool down that ensures a rapid and continuous supply of oxygen to the muscles.

The normal amount of lactic acid circulating in the blood is about 1 to 2 millimoles/liter of blood. The onset of blood lactate accumulation (OBLA) occurs between 2 and 4 millimoles/liter of blood. In non athletes this point is about 50 to 60 of VO₂ max and in trained athletes around 70 percent to 80 percent VO₂ max and even sometime it is more.

#### Lactate Shuttle

Some of the lactate we produce is released into the blood stream and used directly as a fuel by heart muscle, and by the liver to produce blood glucose and glycogen (Cori Cycle).

The lactate shuttle involves the following series of events:

- As we exercise pyruvate is formed.
- When insufficient oxygen is available to breakdown the pyruvate then lactate is produced.
- Lactate enters the surrounding muscle cells, tissue and blood.
- The muscle cells and tissues receiving the lactate either breakdown the lactate to fuel (ATP) for immediate use or use it in the creation of glycogen.
- The glycogen then remains in the cells until energy is required.
Sixtyfive percent of lactic acid is converted to carbon dioxide and water, 20 percent into glycogen, 10 percent into protein and 5 percent into glucose. It has been estimated that about 50 percent of the lactate produced during intensive exercise is used by muscles to form glycogen which acts as a metabolic fuel to sustain exercise.

**Anaerobic Threshold**

Lactic acid starts to accumulate in the muscles once you start operating above the anaerobic threshold, i.e. high intensity short duration exercise activity. This is normally somewhere between 80 percent and 90 percent of the maximum heart rate (MHR) in trained athletes.

**Low Lactate Threshold**

If your lactate threshold (LT) is reached at low exercise intensity, it often means that the “oxidative energy systems” in your muscles are not working very well. If they were performing at a high level, they would use oxygen to break lactate down to carbon dioxide and water, preventing lactate from pouring into the blood. If the LT is low, it may mean that:
- you are not getting enough oxygen inside the muscle cells
- you do not have adequate concentrations of the enzymes necessary to oxidize pyruvate at high rates
- you do not have enough mitochondria in your muscle cells
- your muscles, heart and other tissues are not very good at extracting lactate from the blood

**Enhancement of Lactate Threshold**

The aim is to saturate the muscles in lactic acid in order to educate the body’s buffering mechanism (alkaline) to deal with it more effectively. The accumulation of lactate in working skeletal muscles is associated with fatigue of this system after 50 to 60 seconds of maximal effort. Sessions should comprise of one to five repetitions (depends on the athlete’s ability) with near to full recovery. Training continuously at about 85 to 90 percent of your maximum heart rate for 20 to 25 minutes will improve the Lactate Threshold (LT). A session should be conducted once a week and commence eight weeks before a major competition. This will help the muscle cells retain their alkaline buffering ability.

**Lactate Tolerance Training Sessions**

The following Table identifies some possible training sessions that can be used to improve your lactate tolerance:

<table>
<thead>
<tr>
<th>Distance</th>
<th>Pace</th>
<th>Recovery</th>
<th>Reps</th>
</tr>
</thead>
<tbody>
<tr>
<td>150 meters</td>
<td>400 meters</td>
<td>90 seconds</td>
<td>3 x 3</td>
</tr>
<tr>
<td>300 meters</td>
<td>800 meters</td>
<td>2 minutes</td>
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<td>800 meters</td>
<td>45 seconds</td>
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</tr>
<tr>
<td>150 meters</td>
<td>800 meters</td>
<td>20 seconds</td>
<td>2 x 4</td>
</tr>
<tr>
<td>300 meters</td>
<td>1000 meters</td>
<td>90 seconds</td>
<td>9</td>
</tr>
</tbody>
</table>

**Sodium Bicarbonate**

Energy production via anaerobic glycolysis, which is particularly important for events lasting between 30 seconds and 2 minutes, increases the acidity inside the muscle cells and very soon after does the same to the blood. It is this increase in acidity, within the muscle cells, that is a major factor in producing fatigue. If there is some way to reduce the acidity within the muscle cells, one could theoretically delay fatigue and thus continue exercising at a very high intensity for longer period. Sodium bicarbonate is an alkalinizing agent and therefore, reduces the acidity of the blood (known as a buffering action). By buffering acidity in the blood, bicarbonate may be able to draw more of the acid produced within the muscle cells out into the blood and thus reduce the level of acidity within the muscle cells themselves. This could delay the onset of fatigue.

The specific athletes who might stand to benefit from bicarbonate supplementation will typically compete in events that last between one and seven minutes, i.e. 400 meters to 1500 meters running, 100 meters to 400 meters swimming, most rowing competitions, and many team sports with their repeated nature of high intensity exercise which stresses the anaerobic glycolysis system significantly and produces a lot of acidity.

**A Practical Approach**

The most important practical point is the need to experiment with the supplement during training.
Typically, an 800 meter runner may perform a time trial on a particular day after a couple of days of light training. A further couple of days later, after only more light training, he/she can repeat the time trial in a similar environment after bicarbonate supplementation. The exact protocol would be to ingest 0.3 grams of sodium bicarbonate per kg body weight approximately one to two hours before the time trial. That is, for a 66 kg runner, consume 20 grams of sodium bicarbonate (about four teaspoons) and, the commonly found bicarbonate of soda is exactly the substance needed. This experimenting, if repeated several times, should reveal whether bicarbonate supplementation is likely to produce any benefit and whether the athlete concerned is susceptible to any side effects.

It is likely that large individual differences do exist as far as response to supplementation is concerned. It has been suggested that the more highly trained athletes are less likely to benefit from it because their body’s natural buffering systems are already so well developed, but so far, this is just speculation. It has also been shown that sprinters build-up more acidity within their muscles than endurance runners in response to the same exercise, and so may be more likely to benefit from the buffering effect. From the scientific research, it appears that the size of the dose is quite important and that taking only 0.2 grams per kg is less likely to be beneficial than 0.3 grams per kg, although no evidence exists suggesting that an even greater dose is better still.

Side Effects
As for the side effects, these may take the form of pain, cramping, diarrhea or a feeling of being bloated. Drinking up to a liter of water with the dose is often effective and should be carried out as standard. Breaking up the bicarbonate dose into, say, four equal portions taken over the course of an hour may also help. There are potential side effects to taking higher than normal levels of Sodium Bicarbonate so need consult with the physician first.

Active Recovery
After athletic competition or a hard workout, it would seem that complete rest would be the best way to encourage recovery. However, research is beginning to find some advantages in active recovery. Active recovery refers to engaging in low-intensity exercise after heavy workouts. There are two forms of active recovery. One is during the cool-down phase immediately after a hard effort or workout. The second form of active recovery includes the days following a competition or other intense workout.

It has been reported that active recovery immediately after the event encourages recovery and reduces muscle lactate levels faster than complete rest. After hard intervals, one group rested completely while a second group exercised at 30 percent intensity between intervals. The active group reduced blood lactate levels faster and could achieve a higher power output throughout the workout. Another study found that adding low intensity exercise to the rest period after competition did not decrease an athlete’s physical recovery and actually had positive effects on psychological recovery by improving relaxation. A third study found active recovery encouraged lactic acid removal and helped speed recovery. The general theory is that low-intensity activity assists blood circulation which, in turn, helps remove lactic acid from the muscle. Low-intensity active recovery appears to significantly reduce accumulated blood lactate and speed muscle recovery. However, all agree that more study is necessary to establish a clear answer regarding the best way to recover from intense exercise.

Active rest appears to allow an athlete to physically and psychologically recover from the stresses of training and competing while still maintaining fitness levels. It is becoming a common part of most training plans and appears to offer more benefit than harm.

Preventing Overtraining
Overtraining syndrome frequently occurs in athletes who are training for competition or a specific event and train beyond the body’s ability to recover. Athletes often exercise longer and harder so they can improve. But without adequate rest and recovery, these training regimens can decrease performance. Conditioning requires a balance between overload and recovery. Too much
overload and/or too little recovery may result in both physical and psychological symptoms of overtraining syndrome.

**Common Warning Signs of Overtraining**
- Washed-out feeling, tired, drained, lack of energy
- Mild leg soreness, general aches and pains
- Pain in muscles and joints
- Sudden drop in performance
- Insomnia
- Headaches
- Decreased immunity (increased number of colds and sore throats)
- Decrease in training capacity/intensity
- Moodiness and irritability
- Depression
- Loss of enthusiasm for the sport
- Decreased appetite
- Increased incidence of injuries.
- A compulsive need to exercise

It’s hard to predict overtraining since everyone’s body is different. It is important, however, to vary training through the year and schedule in significant rest time.

**Treating Overtraining Syndrome**
If you suspect you are over trained, the first thing to do is reduce or stop the exercise and allow a few days of rest. Drink plenty of fluids and alter the diet if necessary. Cross training can help to discover if you are overworking certain muscles and also help you determine if you are just mentally fatigued. A sports massage can help the recharge overused muscles.

**Measuring Overtraining**
There are several ways you can objectively measure some signs of overtraining. One is by documenting the heart rates overtime. Find out the heart rate at specific exercise intensities and speed throughout the training and note it down. If the pace starts to slow rate, the resting heart rate increases and you experience other symptoms, you may heading into overtraining syndrome. You can also record your resting heart rate at each morning. Any marked increase from the norm may indicate that you aren’t fully recovered.

Another way to test the status of recovery to use the orthostatic heart rate test, developed by Heikki Rusko while working with cross country skiers. To obtain this measurement:
1. Lay down and rest comfortably for 10 minutes the same time each day (morning is best).
2. At the end of 10 minutes, record the heart rate in beats per minute.
3. Then stand up
4. After 15 seconds, take a second heart rate in beats per minute.
5. After 90 seconds, take a third heart rate in beats per minute.
6. After 120 seconds, take a fourth heart rate in beats per minute.

Well rested athletes will show a consistent heart rate between measurements, but it has been found that a marked increase (10 beats/minutes or more) in the 120 second-post-standing measurement of athletes on the verge of overtraining. Such a change may indicate that he has not recovered from a previous workout, are fatigued, or otherwise stressed and it may be helpful to reduce training or rest another day before performing another workout.

While there are many proposed ways to objectively test for overtraining, the most accurate and sensitive measurements are psychological signs and symptoms and changes in an athlete’s mental state. Decreased positive feelings for sports and increased negative feelings, such as depression, anger, fatigue and irritability often appear after a few days of intensive overtraining. Studies have found increased ratings of perceived exertion during exercise after only three days of overload. Research on overtraining syndrome shows rest is the primary treatment plan. Some new evidence indicating that low levels of exercise (active recovery) during the rest period will accelerate recovery. Moderate exercise has also been shown to increase immunity. Total recovery can take several weeks and includes proper nutrition and stress reduction. The subjective assessments and mental state of an athlete is clearly the most reliable indicator of overtraining. Unfortunately, most athletes ignore these signs or wait too long before doing something. An important component of exercise is to objectively measure your training and modify it before damage is done.
REST AND RECOVERY AFTER EXERCISE—IMPROVE SPORTS PERFORMANCE

Most athletes know that getting enough rest after exercise is essential to high-level performance, but many still overtrain and feel guilty when they take a day off. The body repairs and strengthens itself in the time between workouts, and continuous training can actually weaken the strongest athletes.

Rest days are critical to sports performance for a variety of reasons. Some are physiological and some are psychological. Rest is physically necessary so that the muscles can repair, rebuild and strengthen. For recreational athletes, building in rest days can help maintain a better balance between home, work and fitness goals.

Building recovery time into any training program is important because this is the time that the body adapts to the stress of exercise and the real training effect takes place. Recovery also allows the body to replenish energy stores and repair damaged tissues. Exercise or any other physical work causes changes in the body such as muscle tissue breakdown and the depletion of energy stores (muscle glycogen) as well as fluid loss.

Recovery time allows these stores to be replenished and allows tissue repair to occur. Without sufficient time to repair and replenish, the body will continue to breakdown from intensive exercise. Symptoms of overtraining often occur from a lack of recovery time. Signs of overtraining include a feeling of general malaise, staleness, depression, decreased sports performance and increased risk of injury, among athletes.

SHORT AND LONG-TERM RECOVERY

There are two categories of recovery, such as immediate (short-term) recovery from a particularly intense training session or event, and is the long-term recovery that needs to be build into a year-round training schedule. Both are important for optimal sports performance.

Short-term Recovery

Sometimes called active recovery occurs in the hours immediately after intense exercise. Active recovery refers to engaging in low-intensity exercise after workouts during both the cool-down phase immediately after a hard effort or workout as well as during the days following the workout. Both types of active recovery are linked to performance benefits. Another major focus of recovery immediately following exercise has to do with replenishing energy stores and fluids lost during exercise and optimizing protein synthesis (the process of increasing the protein content of muscle cells, preventing muscle breakdown and increasing muscle size) by eating the right foods in the post-exercise meal. This is also the time for soft tissue (muscles, tendons, ligaments) repair and the removal of chemicals that build-up as a result of cell activity during intense exercise.

Long-term Recovery

The techniques refer to those that are built into a seasonal training program. Most well-designed training schedules will include recovery days and or weeks that are built into an annual training schedule. This is also the reason that athletes and coaches change their training program throughout the year, add cross training, modify workouts types, and make changes in intensity, time, distance and all the other training variables.

ADAPTATION TO EXERCISE

The Principle of Adaptation states that when we undergo the stress of physical exercise, our body adapts and becomes more efficient. It’s just like learning any new skill; at first it’s difficult, but over the time it becomes second-nature. Once you adapt to a given stress, you require additional stress to continue to make progress.

There are limits to how much stress the body can tolerate before it breaks down and risks injury. Doing too much work too quickly will result in injury or muscle damage, but doing too little, too slowly will not result in any improvement. This is why coach trainers set-up specific training programs that increase time and intensity at a planned rate and allow rest days throughout the program.

Sleep Deprivation can Hinder Sports Performance

In general, one or two nights of poor or little sleep will not have much impact on performance,
but consistently getting inadequate sleep can result in subtle changes in hormone levels, particularly those related to stress, muscle recovery and mood. While no one completely understands the complexities of sleep, some research indicates that sleep deprivation can lead to increased levels of cortisol (a stress hormone), decreased activity of human growth hormone (which is active during tissue repair) and decreased glycogen synthesis.

**Balance Exercise with Rest and Recovery**

It is this alternation of adaptation and recovery that takes the athlete to a higher level of fitness. High-level athletes need to realize that the greater the training intensity and effort, the greater the need for planned recovery. Monitoring the workouts with a training log, and paying attention to how your body feels and how motivated you are is extremely helpful in determining your recovery needs and modifying your training program accordingly.

**Ways to Recover Quickly After Exercise**

Do you know what to do after exercise to speed your recovery from a workout? The post exercise routine can have a big impact on fitness gains and sports performance but most people do not have an after exercise recovery plan. Most people exercise for the benefits they get from their workout: improved sports performance, better endurance, less body fat, added and even just feeling better. In order to maintain an exercise routine it is important to recover fully after exercise. Recovery is an essential part of any workout routine. It allows an individual to train more often and train harder so one get more out of the training.

**Recovery After Exercise**

Recovery after exercise is essential to muscle and tissue repair and strength building. This is even more critical after a heavy weight training session. A muscle needs anywhere from 24 to 48 hours to repair and rebuild and working it again too soon simply leads to tissue breakdown instead of building. For weight training routines, never work the same muscles groups two days in a row.

There are as many methods of recovery as there are athletes. The following are some of the most commonly recommended methods.

1. **Rest.** Time is one of the best ways to recover (or heal) from just about any illness or injury and this also works after a hard workout. Body has an amazing capacity to take care of itself if you allow it sometime. Resting and waiting after a hard workout allows the repair and recovery process to happen at a natural pace. It is not the only thing one can or should do to promote recovery, but sometimes doing nothing is the easiest thing to do.

2. **Stretch.** If you only do one thing after a tough workout, consider gentle stretching. This is a simple and fast way to help your muscles to recover.

3. **Cool down.** Cooling down simply means slowing down (not stopping completely) after exercise. Continuing to move around at a very low intensity for 5 to 10 minutes after a hard workout helps remove of lactic acid from your muscles and may reduce muscles stiffness. Warming up and cooling down are more helpful in cooler temperatures or when you have another exercise session or an event later the same day.

4. **Eat properly.** After depleting your energy stores with exercise, you need to refuel if you expect the body to recover, repair tissues, get stronger and be ready for the next bout of exercise. This is even more important if you are performing endurance exercise day after day or trying to build muscle. Ideally, you should try to eat within 60 minutes of the end of your workout and make sure you include some high-quality protein and complex carbohydrate along with minerals and sufficient water.

5. **Replace fluids.** You lose a lot of fluid during exercise and ideally, should be replacing it during exercise, but filling up after exercise is an easy way to boost your recovery. Water supports every metabolic function and nutrient transfer in the body and having plenty of water will improve every bodily function. Adequate fluid replacement is even more important for
endurance athletes who lose large amounts of water during hours of sweating.

6. **Active recovery.** Easy, gentle movement improves the circulation which helps promote nutrient and waste product transport throughout the body. In theory, this helps the muscles repair and refuel faster.

7. **Massage.** Massage feels good and improves circulation while allowing you to fully relax. You can also try self-massage and Foam Roller Exercises for Easing Tight Muscles and avoid the heavy sports massage.

8. **Alternate hot and cold baths or showers.** Some athletes swear by ice baths, ice massage or alternating hot and cold showers to recover faster, reduce muscle soreness and prevent injury. The theory behind this method called contrast water therapy is that by repeatedly constricting and dilating blood vessels helps remove (or flush out) waste products in the tissues. Limited research has found some benefits of contrast water therapy at reducing delayed onset muscle soreness. While taking the post-exercise shower, alternate 2 minutes of hot water with 30 seconds of cold water. Repeat four times with a minute of moderate temperatures between each hot-cold spray. If you happen to have a spa with hot and cold tubs available, you can take a plunge in each for the same time.

9. **Proper sleep.** Optimal sleep is essential for anyone who exercises regularly. During sleep, the body produces Growth Hormone (GH) which is largely responsible for tissue growth and repair.

10. **Avoid overtraining.** One simple way to recovery faster is by designing a systematic workout routine in the first place. Excessive exercise, heavy training at every session or a lack of rest days will limit the fitness gains from exercise and undermine the recovery efforts.

**SUMMARY**

i. The term fatigue to describe a transient decrease in working capacity that results from previous physical activity. We will farther restrict our interest to dealing with neuromuscular fatigue, while fully recognizing the contribution of other systems such as the cardio vascular, pulmonary and endocrine systems to the end result. Neuromuscular fatigue can be defined as a transient decrease in muscular performance usually seen as a failure to maintain or develop a certain expected force or power.

ii. Fatigue may be the result of one or more environmental causes such as inadequate rest, improper diet, work and home stressors, or poor physical conditioning, or one symptom of a chronic medical condition or disease process in the body.

iii. The symptoms of the fatigue due to the overactivity/training/exhaustion are color of the face is reddish, rate of sweating increased, movement of an individual becomes crude, staggering and lack of coordination, lack of concentration, nervousness and negative learning, rest period of a person is prolonged if the person is in under fatigue. General health symptoms are heaviness, dizziness, breathing difficulties, nausea, vomiting, restlessness, insomnia, etc. and low physical performance will be executed by the sports person.

iv. Recovery must start in the concluding part/session of the training. Active recovery is more effective than passive one. Adequate rest is also required. Well ventilated, hygienic living condition is needed. Well balanced qualitative and quantitative diet to be taken. Massage and self relaxation (increased blood circulation) is recommended. Jet massage, Jaquizee, contrast bath, etc. (Stimulate blood circulation to removal of metabolites) are also suggested. Wax bath, Steam packs, etc. are being used (Increased metabolism to remove waste products, sweating to perspiration eliminates waste).

v. Conventional medicine recommends the dietary and lifestyle changes outlined above as a first line of defense against fatigue. Individuals who experience occasional fatigue symptoms may benefit from short term use of caffeine-containing central nervous stimulants, which make people more alert, less drowsy and improve coordination. However,
these should be prescribed with extreme caution, as overuse of the drug can lead to serious sleep disorders, like insomnia.

vi. Herbal remedies that act as circulatory stimulants can offset the symptoms of fatigue in some individuals. Chinese medicine regards fatigue as a blockage, or energy flow, inside the human body. Breathing exercises can both improve respiratory function and relieve stress and fatigue. Deep breathing exercises are best performed while laying flat on the back on a hard surface, usually the floor.

vii. The expression “lactic acid” is used most commonly by athletes to describe the intense pain felt during exhaustive exercise. When energy is required to perform exercise, it is supplied from the breakdown of ATP. The process of lactic acid removal takes approximately one hour, but this can be accelerated by undertaking an appropriate cool down that ensures a rapid and continuous supply of oxygen to the muscles. It has been estimated that about 50 percent of the lactate produced during intensive exercise is used by muscles to form glycogen which acts as a metabolic fuel to sustain exercise.

viii. Sodium bicarbonate is an alkalizing agent and therefore reduces the acidity of the blood (known as a buffering action). By buffering acidity in the blood, bicarbonate may be able to draw more of the acid produced within the muscle cells out into the blood and thus reduce the level of acidity within the muscle cells themselves. This could delay the onset of fatigue. As for the side effects, these may take the form of pain, cramping, diarrhea or a feeling of being bloated.

ix. Recovery after exercise is essential to muscle and tissue repair and strength building. This is even more critical after a heavy weight training session. A muscle needs anywhere from 24 to 48 hours to repair and rebuild, and working it again too soon simply leads to tissue breakdown instead of building. For weight training routines, never work the same muscles groups two days in a row.

**REVIEW QUESTIONS**

1. Define fatigue. What are the symptoms of fatigue? Discuss the causes of fatigue.
2. What are different methods used of recovery from fatigue? Discuss with suitable example.
3. How can you diagnose the onset of fatigue? Discuss the various treatments of fatigue.
4. How can you treat the fatigue to manipulate the diet and also change the lifestyle?
5. Write notes on (i) Traditional Chinese medicine (ii) Color therapy (iii) Deep breathing exercises in relation with recovery from fatigue.
6. What do you mean by lactate shuttle? How can you improve the individuals lactate tolerance?
7. What do you mean by active recovery? How it is beneficial as compared to passive one?
8. What are the over training syndromes? How can you measure overtraining? ‘Rest and recovery after exercise - Improve Sports Performance’—Justify.
9. What is short and long-term recovery? What are the ways to recover quickly after exercise?
10. What are the causes of overtraining? How can it be identified? What is the suggested treatment of overtraining?
INTRODUCTION TO TALENT SELECTION

Talent identification refers to the process of recognizing current participants with the potential to become elite players. It entails predicting performance over various periods of time by measuring physical, physiological, psychological and sociological attributes as well as technical abilities either alone or in combination.

Talent has several properties that are genetically transmitted and, therefore, innate. Nevertheless, talent is not always evident at an early age but trained people may be able to identify its existence by using certain markers. These early indications of talent may provide a basis for predicting those individuals who have a reasonable chance of succeeding at a later stage. Very few individuals are talented in any single domain; indeed, if all children are equally gifted, there would be no means of discriminating or explaining differential success. Furthermore, talent is specific to that particular domain.

Talent selection involves the ongoing process of identifying players at various stages that demonstrates prerequisite levels of performance for inclusion in a given squad or team. Selection involves choosing the most appropriate individual or group of individuals to carry-out the task within a specific context.

For many years, scientists have attempted to identify key predictors of talent in various sports. In this type of research, particularly evident in Australia, China, Cuba, and the former Eastern bloc countries, there are attempts have been made to identify characteristics that differentiate skilled from less skilled performers and to determine the role of heredity and environment in the development of expertise. Detection and identification of talent are more difficult in team games than in individual sports such as running, cycling or rowing, where predictors of performance are more easily and scientifically prescribed. Long-term success in a team sport is dependent on a host of personal and circumstantial factors, not the least of which is the coherence of the team as a whole and the availability of good coaching. These factors make it difficult to predict ultimate performance potential in many sports at an early age with a high degree of probability.

Talent Selection

It could be argued that competition itself might be the best form of talent identification, with competition seeing the best or most talented athletes rise to the top in their chosen sport. However, many athletes that do not succeed in the particular sport they have chosen, along with many that do achieve a degree of success, may be better suited to a different sport and never realize it.
With this in mind and considering that without talent development, talent identification would be a waste of time and resources, it is easy to see why talent identification is a term that is often confused with the term talent development. Therefore, it is vital that talent identification is clearly defined before discussing the topic further. Peltola along with Thomson and Beavis define talent identification as “that process by which children are encouraged to participate in the sports at which they are most likely to succeed, based on results of testing on selected parameters. These parameters are designed to predict performance capacity, taking into account the child’s current level of fitness and maturity.”

In years gone by, and still in western countries, an individual’s participation in a particular sport might well be determined by such factors as “tradition, ideals, desire to take part in a sport according to its popularity, parental pressure, a school teacher’s specialty, the proximity of sport facilities, etc. This system can lead to the more popular sports in a particular country having a plentiful supply of athletes while the lower profile sports struggle for participants. But for many specialists involved in sport, hoping that individuals have chosen the sport that they are best suited to, and waiting for talented individuals to identify themselves through competition, is not good enough for modern sport. Instead, with the aid of sport scientists, many countries and individual sports within countries have developed specific methods to identify talented individuals and help those individuals choose the sport that is best suited to their abilities. Selection of talent in sports are of two types—Natural selection and Scientific selection.

**Natural Selection**

While talent identification through competition has, in reality, been operating ever since organized competitive sport began. It has only been relatively recently that systematic talent identification has become a part of sport around the world. Eastern bloc countries like the German Democratic Republic, the Soviet Union, Bulgaria and Romania are examples of countries that implemented state run, systematic talent identification programs as early as the 1960’s and 70’s. While western countries such as Australia and the United States have typically attempted to have systems in place to develop talented individuals after they have identified themselves through competition in their chosen sport.

In this process, the talents grow naturally and are generally spotted during sports competition. Before participating in the competition they do not receive any systematic training from a qualified coach/trainer. As a result their growth and development in performance are not enough as they possess the potentiality. Moreover, most of the talented children do not get the opportunity to participate in the competition where they come to the notice of a coach/trainer. In this system the talent may not fall under appropriate game/event as they are better suited. Sometimes they are playing a game which they are not suitable. They simply play the game because it is popular at that particular region.

**Scientific Selection**

Several authors have reported about the advantages and disadvantages of sophisticated talent identification programs. Talent identification as helping to accelerate an identified individual’s progression to an elite level and aiding them in reaching a high performance level by the time they reach international level. As well as assisting individuals to select a sport that they are suited to, thus helping to eliminate the frustration’s caused by participating in a sport that they are not suited to. They also have seen talent identification as being an advantageous to the coaches by focusing their training time on athletes with higher levels of talent and abilities for their particular sport. Talent identification also allows countries to get the best from its limited sporting resources. That scientific talent identification is a critical factor in the development of world class athletes is not in question.

In contrast other authors reported that the talent identification as having limitations due to the fact that large numbers of young athletes needs to be tested in order to produce results. Likewise some experts argue that an expert coach’s eye is still quite often the initial stage of talent identification and subsequent testing is merely reinforcing the talent potential of an already identified athlete.
Another factor limiting talent identification is the fact that it is extremely difficult to reliably predict future development of an athlete when identification is carried out at a young age. Add to this the fact that talented children generally exhibit good all-round ability, which can make it difficult to identify the particular sport a young athlete will be best suited for. This makes it extremely difficult for individual sports to pinpoint the correct age to carry-out their talent identification programs.

Some experts feel that it could be detrimental for children to specialize in a particular sport before 13 years of age, while others see it as necessary to direct young athletes to some sports before they reach 12 years of age to aid efficient skill development.

Before implementing talent identification programs, countries and individual sports need to undertake thorough examinations of the specific factors that influence performance in a particular sport. The factors that need to be examined are physiology, anthropometry and psychology. While some other scientists also include heredity and sociological factors. These factors have not been listed in order of importance. Their order of importance will differ between sports and for effective talent identification sports need to take this fact into account. For some sports anthropometric measures may be crucial in talent identification, e.g. height in basketball. While in talent identification of novices, desire to participate in a particular sport may be more important than VO2 max which can be improved, to a certain degree, through systematic training program.

Physiological factors that influence sporting performance can be assessed by administering tests such as measurement of maximum oxygen uptake (VO2 max), which correlates highly with endurance type performance in athletes, determining blood lactate concentrations by taking blood samples from the ear lobe or finger tip. As well as taking muscle biopsies in an effort to determine the type of sport an individual is most likely to be successful in by assessing the muscle fiber distribution in a particular athlete. Assessing muscle strength via tests on devices such as the Cybex dynamometer can also aid in the evaluation of physiological factors that influence sporting performance.

Weight, height and length of limbs can greatly influence sporting performance in certain sports. Therefore, during early phase of talent identification taking anthropometric measures such as height, weight, limb lengths, skinfolds and examining hip and shoulder widths, etc. and then comparing these widths can aid in talent identification. At later ages hand plates and X-ray techniques can be used to determine whether growth is complete or not. Sports such as rowing in country like Australia, the scientist have compared their elite athletes with the general population. This comparison revealed that elite Australian rowers were a tall group with proportionally long leg and arm length compared with the general population.

Research on the influence of psychological factors on sporting performance has also recognized that psychological parameters can contribute as much to elite sport performance as physiological factors.

**Genetical/Hereditary Trait of Sports Talent**

Heredity is another important factor that should be considered in talent identification process. Children tend to inherit physiological and psychological characteristics from their parents. Although some inherited characteristics such as height, limb length, speed ability and coordination are not influenced by environment while others such as weight, endurance ability (partly) and strength can be altered through training. A particular area that heredity could be useful in talent identification is in the area of training limits. If an athlete has reached the limits set by his inherited physiological characteristics, it would be pointless for that athlete to continue to attempt to reach an elite level of sporting performance even though other measures used in talent identification suggest he can.

Several authors caution that although tests that are used in talent identification give us a good indication of the future potential of an athlete and the best direction in sport for that particular athlete, they are by no means full proof. While testing does provide a measure of the factors that influence ultimate sporting performance they do not test for drive, ambition or will to win. A young athlete who
does not meet the minimum standards on the various tests may still succeed in sports due to high motivation and commitment. Similarly, the tests administered have to be sport specific.

When talent identification is undertaken, both in terms of chronological age and biological development of an athlete, is a crucial issue. There seems to be different opinions as to the precise timing of talent identification screening. It has been reported that comprehensive talent identification needs to be carried out a number of years with three main phases. The primary phase of talent identification should occur during the years 3 to 8 and needs to be dominated by a scientists/physician’s examination and is aimed at detecting body malfunctions and physical deficiencies which may restrict future sporting endeavors. The secondary phase of talent identification should be conducted between the ages of 9 to 17; however, this age range will vary between sports, e.g. 9 to 10 for gymnastics and swimming, 10 to 15 for girls and 10 to 17 for boys in other sports. This phase of talent identification needs to be conducted on athletes who have already experienced of organized training and requires a comprehensive assessment of physiological and anthropometric parameters. Psychological assessment and profiling commence in this phase. The final phase of talent identification is mainly concerned with high caliber athlete’s, e.g. national team members. Talent identification in this phase needs to be very sport specific. With particular attention paid to the athlete’s health, physiological adaptation to training and their potential for further improvement.

Contrast this with Peltola who have seen the first phase of talent identification taking place at 10 to 12 years of age and involving easy to administering field tests. The second stage of talent identification would need to be carried out between the ages of 13 to 16 years and again this stage would involve easy to administer field tests. Those athletes that are ultimately selected would then be directed to elite junior programs for their particular sport. Peltola also emphasises that all athletes that are tested need to be encouraged to participate in sport, not just those that are selected for further development. There have also been variations of these formats employed in several countries, such as the German Democratic Republic (GDR), the Soviet Union (USSR) and China, over the last three decades.

Both Peltola and Thomson and his associates have seen the most effective talent identification schemes being tied to the school system similar to aspects of the GDR program. Linking talent identification to the schooling system allows wide participation of all socioeconomic groups, aids development of a wide range of motor skills. School involvement in talent identification also ensures people with at least some professional expertise are involved in talent identification and alleviates some of the cost which can be restrictive in the 90’s.

As mentioned before there have been, over the last 30 years, several countries that have gone to great lengths to run comprehensive talent identification programs. These countries include the GDR, the USSR, Romania and China. Probably the most well known and most talked about of these programs is the one undertaken in the GDR. In the 1972 Olympics alone quite a few GDR medalists were scientifically selected, i.e. the product of system (talent identification program).

To someone living in many western countries the dimensions of the GDR talent identification program are quite staggering. The GDR program was highly organized and structured. It includes a compulsory program of physical education in schools, early identification of sports talent, a club system for talented individuals in separate sports. It also entailed continuity of selection and ruthless elimination of those that do not measure at each stage, a scientific approach to elite performers training and long range objectives. Selected schools were obliged to support talent scouting with talented individuals being trained at training centers, of which there were 2000 young athlete that catered for approximately 70,000 young sports people. There was also a system of youth sport schools that were attached to a sports club at which only specific sport disciplines were trained. Approximately 9,000 young athletes were enrolled in 20 youth sport schools. There were also about 10,000 full-time coaches involved in the training of young athletes. Competition was separated into various age groups. Substantial financial and material support was also provided.

A considerable amount of the GDR success in track and field can be attributed to the attention
Identification of Talent in Sports

that was given to the foundation and build-up phases of their identification program. The first phase, which was called the foundation training phase focused on greatly varied training in different sports and was usually completed in early childhood. The second phase known as build-up training began at 13 to 15 years and lasted between 4 to 6 years. This phase involved the participant beginning to specialize to a degree and also continuing varied training. It was hoped that the versatility encouraged in this phase would develop the ability to learn quickly and correct the faults easier.

TALENT IDENTIFICATION SYSTEM OF DIFFERENT COUNTRIES

To search talent in sports the following questions arise:

1. What makes a champion athlete? What factors in talent identification can be used to predict performance success?
2. What is the role of performance-based, long-term athlete development and assessment?
3. What is the role of sport science and technology in the development of skilled and empowered coaches who are responsible for creating environments conducive to performance excellence?

There seems to be three general categories of talent identification systems: Systematic governmental systems; systematic non-governmental systems; and nonsystematic approaches.

✦ Systematic, governmental systems—Countries who used to use this system are former soviet bloc countries; China, etc.

✦ Systematic, non-governmental systems—tennis, swimming/well-structured age-group programs; developmental infrastructure identifies and reinforces talent moving through system

✦ Non-systematic approaches—somewhat random ID systems with out particular approach.

It is not surprising, that there is no consensus of opinion, nationally or internationally, regarding the theory and practice of talent identification. Usually professional clubs depend on the subjective assessment of their experienced scouts and coaches, employing a list of key criteria. These are set out as acronyms; for example, the key phrase incorporated in the scouting process of Ajax Amsterdam is TIPS, standing for technique, intelligence, personality and speed. Alternative lists include TABS (technique, attitude, balance, speed) and SUPS (speed, understanding, personality, skill), etc.

For many years, scientists have attempted to identify key predictors of talent in various sports. In this type of research, particularly evident in Australia, China, Cuba, and the former Eastern bloc countries, and attempts were made to identify characteristics that differentiate skilled from less skilled performers and also to determine the role of heredity and environment in the development of expertise. For instance, identifying and selecting talented volleyball players are not straight forward operations. Detection and identification of talent are more difficult in team games than in individual sports such as running, cycling or rowing, where predictors of performance are more easily and scientifically prescribed. Long-term success in a team sport is dependent on a host of personal and circumstantial factors, not the least of which is the coherence of the team as a whole and the availability of good coaching. These factors make it difficult to predict ultimate performance potential in many sports at an early age with a high degree of probability.

Eastern European systems relied on the generation of a comprehensive database of personal and performance variables and formal monitoring of progress and development. The systems were most effective where clear relationships between individual characteristics were established. These were almost exclusively individual rather than team-based sports.

The most systematic talent identification model was probably the one in the former Deutsche Democratic Republic (DDR). A fundamental pillar of the country’s tremendous international success in the area of elite sport involved a talent search program. In preparation for the 2000 Summer Olympics, Australia adopted some elements of the DDR talent identification approach by implementing a talent search program. In contrast, the West German system of elite sport never developed a systematic approach. Even after the German reunification of 1990, elements of a
A Textbook of Sports and Exercise Physiology

successful system were not seriously considered as appropriate measures of talent identification in a democratic society.

Within the systems employed in the DDR, not every individual displaying characteristics of talent was selected for systematic training. Youngsters were selected for specialization, only on the provision that they were healthy and free of medical anomalies; could tolerate high training loads; had a psychological capability for training; and maintained good academic achievement levels.

More recently, the Australian Institute of Sport (AIS) created a model for some European countries, most notably Great Britain, to follow. In preparation for the 2000 Olympic Games in Sydney, the AIS paid considerable attention to its own talent identification process and development. A major effort was targeted at individual sports such as rowing, swimming, cycling and track & field. A novel approach to talent identification and development was adopted for women’s soccer as well.

The Australian system consisted of detecting individuals with athletic ability in field games and selecting them for a fast-track program of training in soccer skills. Specific to soccer in Australia, while individuals within the team achieved a limited success in the game, the Olympic Games experience did not yield convincing evidence that talent detection and identification was the perfect process in soccer.

Matsudo in 1987 described the pyramid model that was used in Brazil that embraced six tiers of performance abilities. The standards of proficiency ranged from physical education classes at the base of the pyramid to international competitors at its apex. Their test battery incorporated anthropometric, physiological and performance profiles. Its use in specific sports was limited, but phenomenal success was found in the sport of volleyball, with the direction of individuals at an early age to the sport to which participants seemed biologically most suited.

**What Does This Lead To?**

The most effective contribution from sports science to talent identification is likely to be multidisciplinary. Identifying talent for games at an early age are not likely to be mechanistic or unidisciplinary. Successful identification needs to be followed by selection onto a formal program for developing playing abilities and nurturing the individual towards realizing the potential already predicted. Eventual success is ultimately dependent upon a myriad of circumstantial factors, including opportunities to practice, staying free of injury, the type of monitoring and coaching available during the developmental years. Personal, social and cultural factors also influence ultimate performance.

For example, most games played are possession sports. American football, soccer, baseball/softball, field hockey and basketball have roles and strategies that allow each team to control the ball for extended periods of time. One statistic kept for these sports is time of possession of the ball for each team.

Using the sport of volleyball, it is a game of rebound and movement. The ball is never motionless from the moment it is served until it contacts the floor or is whistled dead by an official. The size of the court is relatively small for the number of players, creating a congested playing area. Because of this, the game has evolved into one of efficiency, accuracy and supportive movements. Each team has a maximum of three contacts with which to accomplish the game’s objective, which is to return the ball and have it contact the floor on the opponents’ side of the net within the boundaries of their court. The outcome of the rally, game and match becomes a summation of each player’s efforts. This is the ultimate in individual contribution and team effort. As a team sport, volleyball uses a net to create no intentional physical contact between opposing teams. Reaching over the net into the opponents’ court is permitted during the follow through motion of the attacker’s arm after the ball has been hit, or in the act of blocking after the hitter has contacted the ball.

The individual techniques of the game are quite different from those of most team sports. Because the essence of the game requires the body to move through all zones of movement, the ball can be played at the highest point of a jump or just inches from the floor. The forearm pass is one technique unique to the game.
No other team sport fosters ball to forearm contact as an accurate and efficient skill. Sitting volleyball is yet another example of adaptations in volleyball performance technique.

**Developing Performance Excellence**

Excellence in performance shares common roots regardless of its form of expression. The concert pianist, research neurologist, and Olympic athlete are all products of sequential, multi-stage development systems. The commonality among these pathways to excellence is surprisingly strong.

In 1985, Bloom and colleagues conducted a study to understand how world-class talent is developed. They interviewed 120 people who had achieved world-class success in the fields of art, sport, music and academics. Successful individuals had very similar learning and development stages. Bloom divided development phases into the early years, middle years and late years.

Balyi (2002; 2004), a sport scientist and coach from the Hungarian/Eastern European system, integrated much of what was involved in talent identification and long-term athlete development in the Eastern European system and adapted it to meet the needs of democratic societies, with particular focus on UK Sport and Sport Canada.

The quality of a talent identification system may influence the international success of a country’s elite sport in various ways. For example, a comparison of the results of Summer Olympic Games showed a linear decrease in Germany.

<table>
<thead>
<tr>
<th></th>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>USA</td>
<td>13.6%</td>
<td>13.3%</td>
<td>11.1%</td>
</tr>
<tr>
<td>Russia</td>
<td>15.1%</td>
<td>8.2%</td>
<td>9.8%</td>
</tr>
<tr>
<td>China</td>
<td>6.8%</td>
<td>6.3%</td>
<td>7.2%</td>
</tr>
<tr>
<td>Australia</td>
<td>3.2%</td>
<td>4.1%</td>
<td>6.3%</td>
</tr>
<tr>
<td>Germany</td>
<td>10.6%</td>
<td>7.4%</td>
<td>5.4%</td>
</tr>
</tbody>
</table>

(Rütten & Ziemainz, 2004)

At the same time, the German Olympic team was the oldest team at the last Olympic Games and it was also the team with the lowest retention rate. In particular, a high retention rate has been emphasized as a major condition of further Olympic success.

<table>
<thead>
<tr>
<th>Nation</th>
<th>Mean age of the players nationwise participating in the last Olympic game.</th>
</tr>
</thead>
<tbody>
<tr>
<td>USA</td>
<td>27.3 years</td>
</tr>
<tr>
<td>Russia</td>
<td>26.0 years</td>
</tr>
<tr>
<td>China</td>
<td>23.4 years</td>
</tr>
<tr>
<td>Australia</td>
<td>26.6 years</td>
</tr>
<tr>
<td>Germany</td>
<td>27.5 years</td>
</tr>
</tbody>
</table>

(Pfützner et al, 2001)

A high retention rate may be affected by both the quantity of potential talents available and the quality of the talent identification system. While the talent development in China can begin with approximately 120 million youngsters in the age range of 10 to 14, the base in Australia is only 1.3 million. It can be stated that countries with lesser populations seem to be depending on very systematic approaches to talent identification. Australia, with the smallest population of the top 5 recently has implemented a systematic Talent Search Program, which already has shown several achievements at national and international championships.

<table>
<thead>
<tr>
<th>Nation</th>
<th>Nationwise retention rate of selected talent.</th>
</tr>
</thead>
<tbody>
<tr>
<td>China</td>
<td>50 %</td>
</tr>
<tr>
<td>USA</td>
<td>72 %</td>
</tr>
<tr>
<td>Russia</td>
<td>40 %</td>
</tr>
<tr>
<td>Australia</td>
<td>65.8 %</td>
</tr>
<tr>
<td>Germany</td>
<td>18 %</td>
</tr>
</tbody>
</table>

(Rütten & Ziemainz, 2004)

A number of criteria that is necessary for effective talent identification tests have been identified. Here is a quick overview, as compiled by Kearney (Talent identification and development: The foundation of Olympic success—White paper presented by Sports Science and Technology Division, USOC, 1999).

**Stability**

Variable being measured is stable or unchanging overtime and is only minimally impacted by growth
Tunneling

The variable is measurable at a young age and effectively predicts the adult status on that characteristic. If height is a variable with strong tunneling, height of 7 to 8 year olds would be highly predictive. Height, however, is not a good example of tunneling because of the significant variation in rate of development across people. Early maturers are taller, younger and do not reach the adult height of some later maturers.

Performance Relevance

Variables used for talent identification should be intuitively relevant to the performance. Critical variables (Matsudo, Victor) include those that are underlying characteristics that are common among all individuals who achieve a high level of athletic success within a sport discipline, but are not necessarily capable of differentiating among elite level performers. An example might be oxygen uptake among elite 10,000 meter runners. All individuals capable of running less than 30 minutes for a 10 Km will have an oxygen uptake above 75 ml/kg/min; however, there is a weak relationship between oxygen uptake of these elite runners and performance times. In contrast, a related variable is a variable that may help differentiate among elite level performers when present in concert with critical variables. Using the same example of 10 Km runners, the velocity at lactate threshold, or velocity at VO₂ max, has a much stronger relationship to competitive performance capacity than simply oxygen uptake.

Assessment Integrity

Traditional measurement and evaluation criteria of validity, reliability and objectivity. In the area of talent identification, this can be challenging, as tests may be valid and reliable but not objective or any other combination. A test may validly assess a certain physiological, psychological or morphological characteristic, but that characteristic may not be a valid predictor of athletic talent. Test validity measures the intended variable, but that variable is not a valid predictor of talent; therefore, the test is not valid for use in talent identification process.

Applicability

Needs to be applicable to the environment in which it is going to be used. The characteristics that contribute to the applicability of a test is that it must be simple, easy to administer, and field-based. There is a continuing debate about the use of field-based test vs laboratory-based tests. The general philosophy reflected in the literature is that field-based tests should be used for initial screening and that the results of these may be further differentiated by the use of laboratory-based procedures on a more selected group of individuals. Categories of Talent identification tests proposed by various scientists which are as follows:

A. Morphological Parameters

- Body Mass
- Height
- Somatotype (physique)
- Fat mass
- Fat-free mass
- Length and interrelationships among segment lengths of the body.

---

Table 13.4: Nation wise talent development in million (10–14 and 15–19 years)

<table>
<thead>
<tr>
<th>Nation</th>
<th>10–14</th>
<th>15–19</th>
<th>Total</th>
<th>Talent pool</th>
</tr>
</thead>
<tbody>
<tr>
<td>USA</td>
<td>20 M</td>
<td>20 M</td>
<td>40 M</td>
<td>4.00 M</td>
</tr>
<tr>
<td>Russia</td>
<td>13 M</td>
<td>15 M</td>
<td>28 M</td>
<td>2.80 M</td>
</tr>
<tr>
<td>China</td>
<td>120 M</td>
<td>99 M</td>
<td>219 M</td>
<td>21.90 M</td>
</tr>
<tr>
<td>Australia</td>
<td>1.3 M</td>
<td>1.3 M</td>
<td>2.6 M</td>
<td>0.26 M</td>
</tr>
<tr>
<td>Germany</td>
<td>4.6 M</td>
<td>4.6 M</td>
<td>9.2 M</td>
<td>0.92 M</td>
</tr>
</tbody>
</table>

(Rütten & Ziemainz, 2004)
B. Physiological/Motor qualities (Balyi & Hamilton, 2003)
- Strength ability
- Speed ability
- Endurance ability
- Reaction time (reflex)
- Agility
- Flexibility
- Balance—static and dynamic

C. Psychological and Sociological (Kluka, 2003; Calder, 2000; Reilly, 2003)
- Personality Traits
- Psychological profiling
- Readiness
- Coach ability
- Self-concept
- Sociometric assessments
- Significant others
- Visual perception assessments
- Includes decision-making and “game Intelligence”

If a talent identification test is 100 percent effective, there would be a strong, linear relationship between results on the test and results in performance. The narrower the geometric distribution of the performance on the tests and the performance in competition, the more effective the talent identification test is. The false negative quadrant represents those individuals who score poorly on the test, but are able to do well in a competitive situation. The results of the talent identification test would be indicating they did not have the potential for success, but they achieved success. The false positive quadrant of individuals does well on the test, but do not achieve competitive results. There are number of factors that have an impact on this test efficacy graph. First, there are very few situations where results on a single variable can effectively predict competitive performance. Considering the traits or characteristics that are essential for success for a woman softball athlete. It is obvious that an evaluation on any single variable will not provide a strong prediction of potential success. The typical application of an efficacy graph, then, is based on the results of a test battery or assessment on a series of variables.

Talent Identification in India

The search for identification of genetically gifted talented children in sports has gained importance over the year as competition at every level has intensified. It is well known that while genetic predisposition are of importance for a successful sports carrier, nevertheless studies carried out in many countries have proved that while there are many successful athletes whose parents were good in sports, there were also many parents who had never competed in any sports in their life. In a country like India with varied climatic conditions, different food habits, wide gaps exist in socio-economic levels. Therefore, identification of talented boys and girls in younger age group assumes more significance to prepare elite sports persons. The systematic way of talent scouting in India was undertaken by Sports Authority of India (SAI) some times in middle of 1980’s. The scouting was undertaken through States Sports Department under the scheme of National Sports Talent Contest (NSTC) with the following objectives:

i. To start a movement in the country to broad base sports at grass root level, to bring about sports consciousness among the people of various age groups and create awareness that participation in sports for the children in the formative years would help in realizing the objective of a “sound mind in a sound body.”

ii. To scout the talent among school going children who are genetically gifted and are endowed natural motor qualities and physical growth.

iii. To ensure admission in adopted schools of the SAI and impart scientific and systematic training to the selected children to achieve excellence in sports in national /international competitions and become future champion.

Schemes of Sports Authority of India

Sports Authority of India has started the following schemes to select and nurture the talented children in sports for excellence. The schemes are – NSTC, SPDA, ABSC, SAG and Sports Hostel.
National Sports Talent Contest (NSTC)

The first talent scouting among the children in the age groups of 10 to 12 years for Athletics, Basketball, Badminton, Football, Hockey, Table tennis, Volleyball and Wrestling and for 8 to 10 years old children for Gymnastics and Swimming was held in 1985. Till 1988 the talent was scouted based on the performance of the children in their respective discipline by the former international sports persons of the concerned discipline.

From 1989 the talent scouting was done by applying a battery of tests to assess the physical development, motor qualities and ability in skill related to a particular game/sport. The battery of tests for this purpose were formulated in consultation with the Soviet experts and later adopted to Indian conditions.

Sports Project Development Area (SPDA)

In 1987, Govt. of India has started Sports Project Development Area (SPDA) scheme in all states/UTs which would provide in-house coaching facilities to talented youngsters in their own states. Each SPDA to cater to a maximum of four Olympic games disciplines, based on the popularity of these disciplines in that area. In addition, 1 or 2 indigenous games may also be promoted in each SPDA center. Selection of talent between 10 to 14 years of age was done on the similar grounds as for NSTC scheme. Same battery of tests were applied.

Army Boys Sports Companies (ABSC)

The introduction of another scheme by SAI was Army Boys Sports Companies started during 1991 to 1992 with the ultimate objective to reach international excellence in sports. The Army has joined hands with the SAI to nurture talented boys only in sports. The age of boys for induction is 12 to 14 years and 10 to 12 years for swimming and gymnastics in the scheme. The selection was mainly based on SAI battery of tests for identification of talent.

Special Area Games (SAG)

Special Areas Games Scheme aims at scouting and nurturing natural talent for modern competitive sports and games from inaccessible tribal, rural, and coastal areas of the country. The Scheme also envisages taping of talent from indigenous games and martial arts and also from regions, which are either genetically or geographically advantageous for excellence in a particular sports discipline. The main objective of the Scheme is to train meritorious sports persons in the age group of 14 to 21 years. The age is relaxed depending upon the merit of the case. The trainees admitted under the residential scheme are provided free Boarding and Lodging facilities, sports kit, sports equipment, competition exposure, insurance, medical expenses, etc and non-residential trainees with some monthly stipend, sports kit; sports equipment, competition exposure, insurance coverage, etc.

Disciplines Covered


Objective

1. To scout Natural Talent in their indigenous games from tribal, coastal and hilly region and nurture them scientifically for achieving excellence in modern competitive games and sports
2. To make it possible for the Central Govt. and State Govts, to work together for Sports development through integrated efforts.
3. To correct existing regional imbalance in sports infrastructure in the country and within a state.
4. To enable SAI to nurture junior sports talent scientifically who have attained excellence at sub junior level under NSTC scheme for Center of Excellence on long-term basis.
5. To provide package assistance for sports infrastructure and undertake various sports programs in that area.
6. To ensure maximum utilization of the facilities already existing/to be created in a district, proper maintenance and operation.
7. To ensure equitable distribution of the funds earmarked for various plan scheme of the Govt. of India SAI, since the concept covers linkage of quantum of assistance under various schemes.
8. To take the benefit of various plan scheme to the grass root level, i.e. up to administrative block level.

Under the scheme, Special Area Games Center are started in consultation with the State govt./UT Administration. The facilities to be provided by the State govt and SAI for establishing the center is given below:

Opening of SAG Centers
The Special Area Games centers are started in consultation with the State govt/UT Administration. The State Govt. has to provide requisite land with infrastructure. However, in case the non-availability of infrastructure the State Govt. has to provide developed land on long-term lease basis to SAI enabling it to create the required facilities. The SAI will provide the following facilities for the running of the Center:

a. The trainees are provided facilities, i.e. boarding/lodging, education, sports kit, etc as per laid down norms.
b. Furniture for hostel and catering equipment
c. Coaches
d. Sports equipment
e. Maintenance of play fields
f. Administrative and catering staff
g. Electricity, water and other obligatory charges.

Monitoring of Trainees
The performance of the trainees is thoroughly scrutinized at the Regional level and the trainees who have not performed satisfactorily during the last two years are weeded out on the recommendations of the Regional Head who chair the Monitoring Committee. The process is carried out once in a year before start of the next academic session. Immediately after the annual examination in case of student trainees so that the weeded out trainees do not have problem in seeking admission in other schools. The weeded out trainees would, however, have the option to continue studies in the associated/attached institution on their own resources but they shall not be entitled for any facilities from the SAI.

Sports Hostel (STC)
The sport hostel scheme proposed by SAI is called the SAI Training Centre (STC). This scheme has been successful to a great extent, in fulfilling SAI’s objectives of spotting and nurturing sports talents. SAI short list sports person aged between 14 and 21 years and enrolls them. out of the 58 STC’s located across India. The talent in the scheme is spotted based on their sports predisposition(category-1) and their genetical previous position as desire for particular game/sports (category-2).

Talent selection program: The talent selection process starts with the following program and takes two days time. The program includes 12 test and measurements. Out of which 7 tests are aimed on the evaluation of general physical fitness, 3 on evaluation of specific physical fitness and 2 measurements to reflect physical development. All tests and measurements are to be evaluated in 3 points systems. In this way along with the marks in physical development of a child, we are able to compare his/her general and specific fitness in the proportion of 70 and 30 percent respectively.

Specific tests are given separately for each event. All general tests and measurements should be made for all events according to the same program.

Scoring: While framing this battery of tests the emphasis was given on general physical fitness of the children. Maximum number of points which can be scored during the selection is Physical development (height and weight) = 6, general physical fitness (seven motor ability tests) = 21 and specific skill tests = 9. The number of points is enough to be selected on regional level is 13 + 5 (specific) = 18. Scoring the specified number of points in physical development and physical fitness is compulsory. No one scoring less can be considered as prospective for the top performance even if he or she scores maximum number of point in specific tests.
Test Program

**Registration for testing:** Register for registration of participants at State level should contain the following information:
- Sl. No.-ID, Card No ........................................
- Name of the participant ........................................
- Name and address of the school ............................
- Chest No. ............................................................
- Father’s name and address ....................................
- Mother tongue ....................................................
- Class studying ....................................................
- Medium of instruction ........................................
- Any formal sports training received, if so how long.
- Whether playing any game/sports on his own, if so name the game/sports.
- Height and body weight may be recorded by standard procedure followed by general medical examination and age verification to be done. After these the physical fitness tests to be conducted as per the program given below.

**Monitoring of Performance**

Progress/monitor growth and development, various physical, physiological and psychological profiles of the trainee is one of the very important

| Table 13.5: Anthropometric and Physical fitness tests (motor ability) for selection of talent |
| --- | --- | --- |
| **Day** | **Session** | **Tests conducted** | **Components measured** |
| Day - 1 | Forenoon | Height | Agility |
| | | Weight | Upper body strength & coordination |
| | Afternoon | 6 x 10 m Shuttle Run | Speed ability |
| | | Ball Throw (Mini Booketball) | Explosive power of lower limbs |
| | | Vertical Jump | 30 m Run |
| | | Balancing Test | Speed ability |
| | | Standing Broad Jump | Neuromuscular coordination |
| | | 800 m Run | Explosive power of lower limbs |
| Day - 2 | Forenoon/Afternoon | Skill Tests/ Specific Tests | Endurance capacity |

Test manual of Sports Authority of India for methods of testing and ‘norms’.

**Table 13.6: Specific skill tests of each discipline/games for selection of talent**

<table>
<thead>
<tr>
<th>Sl. No.</th>
<th>Game/ discipline</th>
<th>Test-1</th>
<th>Test-2</th>
<th>Test-3</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Athletics</td>
<td>60 m sprint</td>
<td>300 m run</td>
<td>Tennis ball throw</td>
</tr>
<tr>
<td>2.</td>
<td>Badminton</td>
<td>Badminton shuttle</td>
<td>Tennis ball throw</td>
<td>Standing backward jump</td>
</tr>
<tr>
<td>3.</td>
<td>Basketball</td>
<td>Wall pass test</td>
<td>Dribbling test</td>
<td>Jumping &amp; turning in air</td>
</tr>
<tr>
<td>4.</td>
<td>Football</td>
<td>30 m running with ball</td>
<td>Kicking for accuracy</td>
<td>Juggling with the ball</td>
</tr>
<tr>
<td>5.</td>
<td>Gymnastics</td>
<td>3 m rope climbing</td>
<td>Hip flexibility &amp; shoulder flexibility</td>
<td>Hanging on wall bars and raising legs to right angle</td>
</tr>
<tr>
<td>6.</td>
<td>Hockey</td>
<td>Shooting into the goal</td>
<td>Balance the ball on stick</td>
<td>20 m moving with ball (rolling)</td>
</tr>
<tr>
<td>7.</td>
<td>Swimming</td>
<td>Evaluation of floating ability</td>
<td>Efficiency of sliding</td>
<td>Shoulder Flexibility</td>
</tr>
<tr>
<td>8.</td>
<td>Table tennis</td>
<td>Reaction ability</td>
<td>Accuracy in striking</td>
<td>Speed of playing hand</td>
</tr>
<tr>
<td>9.</td>
<td>Volleyball</td>
<td>Accuracy of service</td>
<td>Control on volleyball pass</td>
<td>Medicine ball throw</td>
</tr>
<tr>
<td>10.</td>
<td>Wrestling</td>
<td>Aptitude test</td>
<td>Knee touching</td>
<td>Bridging</td>
</tr>
</tbody>
</table>

Test manual of Sports Authority of India for methods of testing and ‘norms’.
Identification of Talent in Sports

Aspect of improving sports performance. The scientific tests mentioned below are being conducted by the scientists of SAI generally twice in a year. Based on the result of the scientific monitoring tests, concerned coaches are advised to formulate their training schedule accordingly. These trainees who do not improve in their sports performance would be weeded out, after giving them and their parents due advice/warning. To complete the process of weeding out, three important aspects of the trainee are taken into consideration. These are (a) performance in the competition (last one year), (b) coaches assessment report towards the improvement in performance and also (c) the scientific tests results of the each an individual trainee. Considering above three factors, athletes are retain or weeded out except any disciplinary ground against the players which is generally suggested by the In-charge of the respective center for weeding out. Weeding out are generally done every end of academic year.

Periodical Evaluation and Assessment of Existing Trainees of Various SAI Schemes

Common Tests for All the Games/Sports/Events

The following tests and measurements are being conducted on all the boys/girls for all the game/sports apart form their specific fitness tests.

- Height
- Weight, LBM, Fat mass
- Relative Back strength
- Hand Grip Strength
- Trunk Flexibility Test
- Multi stage Physical Fitness Test/Direct Measurement of VO₂ max with Telemetric System in the field or in the tab.
- Measurement of Hemoglobin percent
- Tests for speed ability and explosive power
- Specific sports skill tests

Game/sports/event related specific anthropometric and physiological test

Ball Game (Football, Hockey, etc.)

- Peak Muscle Power of Hamstring, Quadriceps Gastrocnemious, Gastrocsolius, Biceps, etc.
- Semo Agility Test to measure the agility.
- Detection of Anaerobic Threshold (heart rate)
- Biochemical Test for Center of Excellence Players/national players.

Volleyball, Basketball and Racquet Sports

- Arm length and leg length
- Peak Muscle Power of Hamstring, Quadriceps, Gastrocnemious, Gastrocsolius, Biceps, etc.
- Semo Agility Test to measure the agility.
- Explosive power of lower limb
- Detection of Anaerobic Threshold (heart rate)
- Biochemical Test for Center of Excellence Players/national and advance players.

Aquatic Sports

- Arm Length/Span
- Leg Length
- Shoulder Breadth
- Trunk Length
- Palm and Foot Length (Only for Swimmers)
- Peak Muscle Power of Hamstring, Quadriceps, Gastrocnemious, Gastrocsolius, Biceps, etc.
- Detection of Anaerobic Threshold (heart rate)
- Biochemical Test for Centre of Excellence Players/national and advance players.

Combat Sports

- Shoulder Breadth
- Arm length (for Boxing)
- Peak Muscle Power of Hamstring, Quadriceps, Gastrocnemious, Gastrocsolius, Biceps, etc.
- Detection of Anaerobic Threshold (heart rate)
- Biochemical Test for Centre of Excellence Players/national and advance players.

Archery

- Arm Span, Arm Length, Shoulder Breadth
- Hamstring – Quadriceps Ratio
- Muscle endurance of Upper Limb/upper body

Athletics Middle and Long Distance Running

- Detection of Anaerobic Threshold (heart rate)
- Peak power of Leg Muscle
Biochemical Test for Center of Excellence Players/national and advance players.

**Sprinting and Jumping**
- Leg Length, Sitting Height
- Peak Muscle Power of Hamstring, Quadriceps, Gastrocnemius, Gastrocsolius
- Anaerobic Power
- Biochemical Test for Center of Excellence Players/national and advance players.

**Throwing**
- Arm Length
- Trunk Length
- Leg length
- Anaerobic Power
- Peak Muscle Power of Hamstring, Quadriceps, Gastrocnemius, Gastrocsolius, Biceps, etc.
- Biochemical Test for Center of Excellence Players/national and advance players.

**MODEL OF TALENT IDENTIFICATION AND MONITORING**

**Role of Sports Science**

**Role of Physiology and Anthropometry in Talent Identification**

Weight, height, various proportion of body and length of limbs can greatly influence sporting performance in certain sports. Therefore, during early phase of talent identification taking anthropometric measures such as height, weight, limb lengths, skinfolds and examining hip and shoulder widths and then comparing these widths can aid in talent identification. At later ages hand plates and X-ray techniques can be used to determine whether growth is proportionate and completed. Sports such as water sports, basketball, volleyball, etc. are tall group with proportionately long leg and arm length compared with the other game and also the general population.

Physiological factors that influence sporting performance can be assessed by administering tests such as measurement of maximum oxygen uptake (VO₂ max), which correlates highly with endurance type performance in athletes, determining blood lactate concentrations by taking blood samples. As well as taking muscle biopsies/other objective measures in an effort to determine the type of sport an individual is most likely to be successful in by assessing the muscle fiber distribution and other physiological qualities in a particular athlete. Assessing muscle strength by dynamometer can also aid in the evaluation of physiological factors that influence sporting performance. So, together with anthropometric variables and physiological profiles assessment is very important and useful at the time of selection of talent and as well as the selection of events.

**STEPS FOR TALENT IDENTIFICATION**

**Step - I: The Preliminary Selection Phase**

The following major tasks apply to the preliminary selection phase:

i. Pedagogical observations to evaluate interest in sport and the level of movement activity of the youngster.

ii. A preliminary selection into potential event groups (speed and explosive power events, endurance events), based on observations and an evaluation of general and specific physical performance capacities.

Young potential athletes are now divided into general and sport specific training groups. The general group is made up from youngsters with insufficient physical performance capacities. They continue to train with emphasis on general physical development. Athletes selected to the other group follow a more intensive general training program that includes some sport specific elements. Their main training means are made up from movement games, relays, gymnastics exercises, running and jumping.

The testing procedures in the preliminary selection phase are based on a small but complex physical development and physical performance test battery that includes a medical check-up. The following tests can be recommended:

- The evaluation of physical development includes height, weight, chest circumference, vital capacity, dynamometric hand strength, arm length and foot length.
- The evaluation of physical performance includes 30 m sprint (flying start), 10 sec. maximal speed
running on the spot, 6 × 10 m shuttle run or semi-agility test (for team game), 800 m run/multistage physical fitness test, standing long jump, vertical jump, medicine ball (1 kg) throw with two hands from a sitting position, pull-ups, push-ups and trunk flexibility.

A planned choice from the above listed simple tests makes it possible to evaluate speed, explosive strength, flexibility, endurance, and strength endurance potential of children in the 8 to 12 yrs. age range. This, in turn, allows one to make some predictions about their physical capacities potential, because most of the chosen indicators have stable inherited characteristics and are hardly influenced by environmental changes (training). Studies have clearly indicated that the majority of children with good physical performance indicators maintain this advantage when they grow older. Changes in the genetic structure are extremely limited and it appears likely that heredity does not only decide the potential level of physical capacities but also their trainability.

Studies have also shown that the same training processes produce different rates of improvement in the development of physical capacities in different young athletes. The rate of improvement is frequently considerably faster than the predicted values for a certain age range. It is consequently important to understand that prediction of potential capacities depend, besides the initial level, also on the rate of improvement. The initial level among younger children is considerably less than the rate of improvement.

It is not advisable to evaluate the potential of young athletes only according to the results of the various tests. The principle of many-sided physical preparation must be stressed during the whole preliminary selection phase. Nevertheless, ideal anthropometric measurements and physical capacity evaluation tables allow one to assess with some accuracy the potential suitability of youngsters for a certain group of events.

The table below represents that genetical qualities and some component which can be influenced/alter through systematic training program. This improvement also depends on the ages of the person, i.e. the training start at early age components could be improved up to maximum as mentioned above, but training start at later age the target can not be achieved. However, the hereditary traits are not influenced by the training as these are genetically determined.

Steps – II: The Basic Selection Phase

The main aim of the basic selection phase is a precise control and evaluation of the development of the young athletes who have passed the requirements of the preliminary selection, taking into consideration event specific demands.

Their potential talent for a particular event is determined by event specific capacities and the development rate during the preceding preparations. Physiological and medical controls are now increased and the psychological evaluation pays particular attention to such aspects as activeness, independence and the mental capacity to perform under competitive stresses. Laboratory evaluations, when available, provide additional information on the functioning of the nervous system, sensory motor reactions, sensory motor coordination, etc. It is important to stress here that studies have clearly shown the importance to base the directional selection mainly on the rate of improvement. A second evaluation should therefore take place about a year after the first to calculate the improvement rate of all capacities and

<table>
<thead>
<tr>
<th>Genetical/Hereditary Traits</th>
<th>Trainable Quality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body Height/ Stature</td>
<td>Explosive power</td>
</tr>
<tr>
<td>Speed Ability</td>
<td>Strength Ability</td>
</tr>
<tr>
<td>Endurance Ability</td>
<td>Endurance Ability—maximum up to 25%</td>
</tr>
<tr>
<td>Agility</td>
<td>Speed Ability—maximum up to 15%</td>
</tr>
<tr>
<td></td>
<td>Agility—maximum up to 10–15%</td>
</tr>
<tr>
<td></td>
<td>Flexibility—maximum up to 50%</td>
</tr>
</tbody>
</table>
performance indicators which are then classified as “very poor,” “poor,” “satisfactory,” “good” and “very good.”

While the first directional selection is based on the arithmetical mean, the second evaluation requires at least a “good” classification that is calculated from the arithmetical mean plus a standard deviation of ±0.5 to ±1.5 or applied norms (percentile ranking). This procedure reduces considerably the number of candidates.

It is important here to avoid narrow specialization that can have a negative influence on the development of other potential physical capacities. The best solution appears to be a balanced event specific and compulsory all-round physical development program. This approach in the basic selection phase assures that youngsters, who have not displayed their capacity for one or another group of events in the earlier part (9 to 11 years) of the selection, will be directed to their potential event in the second stage (13 to 14 years). Their many sided physical development has at the same time not suffered from narrow specialization.

The employment of the above described preliminary and basic selection phases have the following advantages:

- The method provides a reasonably reliable information system to find children with potential physical capacities and aptitudes for sport.
- An evaluation of physical and anthropometric capacities over several years makes it possible to follow individual developmental progress and to discover aptitudes towards certain event groups or events.
- The method creates from mass evaluations a data bank for future changes and corrections of training procedures, thus providing for a more rational and reliable selection system.

**Steps – III: The Profound Selection Phase**

The third phase of the selection procedures covers several years and is based on the evaluation of capacities that emerge from practical sporting participation, as well as pedagogical observations, control tests and physiological and psychological studies.

Particularly important in this selection phase are competition revealed parameters and event specific physical preparation indicators. These should correspond to potential elite performer’s requirements at the end of the profound selection phase when the biological development is practically completed and the functional capacities well defined (14 to 15 years for girls, 16 to 17 years for boys). The profound selection phase is regarded to be reliable in the determination of physical capacities and functional potential for the final selection. This begins with an evaluation of single indicators for a particular event and is followed by a complex evaluation of morphological prerequisites, physical/physiological performance capacities, functional systems and psychological assessments.

Finally, it is important to stress the fact that the development of truly talented youngsters is revealed during training processes, in which the rate of improvement plays a decisive part in the selection procedures and the correlation between the biological age and test results must be correctly interpreted. The differences between the actual and the biological ages can in extreme situations exceed three to four years and even more.

**SUMMARY**

i. Talent identification refers to the process of recognizing current participants with the potential to become elite players. It entails predicting performance over various periods of time by measuring physical, physiological, psychological and sociological attributes as well as technical abilities either alone or in combination. Talent has several properties that are genetically transmitted and, therefore, innate. Talent selection involves the ongoing process of identifying players at various stages that demonstrates prerequisite levels of performance for inclusion in a given squad or team. Selection involves choosing the most appropriate individual or group of individuals to carry-out the task within a specific context.

ii. Selection of talent in sports is of two types—Natural selection and Scientific selection. While talent identification through competition has,
Identification of Talent in Sports

in reality, been operating ever since organized competitive sport began. It has only been relatively recently that systematic talent identification has become a part of sport around the world. In contrast some authors reported that the talent identification as having limitations due to the fact that large numbers of young athletes needs to be tested in order to produce results. Likewise some other experts argue that an expert coach’s eye is still quite often the initial stage of talent identification and subsequent testing is merely reinforcing the talent potential of an already identified athlete.

iii. Weight, height and length of limbs can greatly influence sporting performance in certain sports. Therefore, during early phase of talent identification taking anthropometric measures such as height, weight, limb lengths, skinfolds and examining hip and shoulder widths, etc. and then comparing these widths can aid in talent identification. Physiological factors that influence sporting performance can be assessed by administering tests such as measurement of maximum oxygen uptake (VO2 max), which correlates highly with endurance type performance in athletes, determining blood lactate concentrations by taking blood samples from the ear lobe and figure tip. As well as taking muscle biopsies in an effort to determine the type of sport an individual is most likely to be successful in by assessing the muscle fiber distribution in a particular athlete.

iv. Heredity is another important factor that should be considered in talent identification. Children tend to inherit physiological and psychological characteristics from their parents. Although some inherited characteristics such as height, limb length, speed ability and coordination are not influenced by environment while others such as weight, endurance ability and strength can be altered through training. A particular area that heredity could be useful in talent identification is in the area of training limits. If an athlete has reached the limits set by his inherited physiological characteristics, it would be pointless for that athlete to continue to attempt to reach an elite level of sporting performance even though other measures used in talent identification suggest he can.

v. The most effective contribution from sports science to talent identification is likely to be multidisciplinary. Identifying talent for games at an early age are not likely to be mechanistic or unidisciplinary. Successful identification needs to be followed by selection onto a formal program for developing playing abilities and nurturing the individual towards realizing the potential already predicted. Eventual success is ultimately dependent upon a myriad of circumstantial factors, including opportunities to practice, staying free of injury, the type of monitoring and coaching available during the developmental years. Personal, social and cultural factors also influence ultimate performance.

vi. It is well known that while genetic predisposition are of importance for a successful sports carrier, nevertheless studies carried out in many countries have proved that while there are many successful athletes whose parents were good in sports, there were also many parents who had never competed in any sports in their life. In a country like India with varied climatic conditions, different food habits, wide gaps exist in socioeconomic levels. Therefore, identification of talented boys and girls in younger age group assumes more significance to prepare elite sports persons. The systematic way of talent scouting in India was undertaken by Sports Authority of India sometimes in middle of 1980’s.

vii. Sports Authority of India has started the following schemes to select and nurture the talented children in sports for excellence. The schemes are—NSTC, SPDA, ABSC, SAG and Sports Hostel. The performance of the trainees is thoroughly scrutinized at the Regional level and the trainees who have not performed satisfactorily during the last two years are weeded out on the recommendations of the Regional Head who chair the Monitoring Committee. The process is carried out once in a year before start of the next academic session.
viii. Progress/monitor growth and development, various physical, physiological, and psychological profiles of the trainee is one of the very important aspect of improving sports performance. The scientific tests are being conducted by the scientists of SAI twice in a year. Based on the result of the scientific monitoring tests, concerned coaches are advised to formulate their training schedule accordingly. These trainees who do not improve in their sports performance would be weeded out, after giving them and their parents due advice/warning. Weeding out are generally done every end of academic year.

**REVIEW QUESTIONS**

1. What do you mean by talent in sports? What is talent selection in sports? Discuss about natural selection and scientific selection of talents.
2. What is the role of sports science in talent selection process? Describe the role of physiology and anthropology in talent identification.
3. Discuss the talent identification process in different countries.
4. What are the basic component are to be evaluated during talent identification process? What are hereditary traits?
5. Write the different morphological, physiological and psychological parameters that are needed to be evaluated for talent identification and why?
6. Discuss about the Talent identification in India. Write about Schemes of Sports Authority of India.
8. What is the Game/sports/event related specific anthropometric & physiological test are to be conducted for monitoring the talent?
9. What are the basic steps of talent identification process? Discuss with special reference to SAI adopted process.
10. What are the aims and objectives of Special Area Games project of SAI?
INTRODUCTION TO KINANTHROPOMETRY

Kinanthropometry is a scientific specialization dealing with the measurement of human in a variety of morphological perspectives, its application to movement and those factors which influence movement, including components of body build, body measurements, proportions, composition, shape and maturation; motor abilities and cardio-respiratory capacities, physical activity including recreational activity as well as highly specialized sports performance. It provides quantitative interface between human structure and function. The Kinanthropometry is defined as the study of human size, shape, proportion, composition, maturation and gross function in order to help understand growth, exercise, performance and nutrition. It is a scientific specialization closely allied to physical education, exercise physiology, Sports Medicine, Auxology, Physical anthropology, gerontology and several medical disciplines. It is essential to examine an athlete from every possible aspect. This is in order to win in any sport or event in the olympics as well as other international competitions. Estimation of the kinanthropometric characteristics of these athletes provides a valuable reference point in human structure and functions. Analysis of the characteristics of the olympic athletes can help the scientists to understand top level performance by providing...
information useful in formulating strategies for training and for the explanation to predict the performance.

Kinanthropometric investigations have been conducted on the Olympic athletes during the Olympics more than three decades. The different characteristics examined include investigations of their size and shape, using large number of variables by sports and events. The somatotype of athletes has been examined using the latest technology. New approach has been used for the analysis of proportions and body composition. The maturity status of Olympic athletes especially in swimming and gymnastics has also been reported. Adequate consideration was given to the growth and maturity status of young Olympic athletes, in order to understand the genetic variation in physical structure and performance of athletes.

A relatively new and comprehensive approach was used for assessment of physique through Kinanthropometry which evaluates the physical structure of an individuals in relation to gross motor performance. The term Kinanthropometry is derived from morphometry, which is the measurement of shape and form of man. It is the quantitative study of size, shape, proportion, composition and maturation in relation to gross function as define by carter, in 1985.

Scope and Development

Ross, 1978 referred to kinanthropometry both as ‘an emerging scientific technology’ and as ‘an emerging scientific discipline’. However, so far development of kinanthropometry is concerned, it is known that the use of term ‘kinanthropometry’ has been gaining term popularity since its use in many international and national conferences.

Earlier an International Working group in Kinanthropometry was founded in Brazil in 1978 as a branch of research committee of the International Council of Sports Science and Physical Education. The International Seminar on Kinanthropometry was held in 1978. In the Olympic Scientific Congress of 1984 held at Eugene, USA and the third international Kinanthropometry Conference was held in 1986 at Glasgow in conjunction with VIII Commonwealth and International Conference on Sports, Physical Education, Dance, Recreation and Health. The conference at Glasgow is now history in the order of development for this new science.

The Jordan hill College of Education is of historical importance since it was the site of constitution meeting of International Society for the Advancement of Kinanthropometry (ISAK) and undertaken the following objectives:

i. To provide an international forum for the development of a scientific movement to foster basic research relating to the structural concomitants of human movements.

ii. To foster the development of instrumentation and technology, to quantify structure, understand variability and interpret individual differences in matters of growth and aging, exercise, performance and nutrition.

iii. To contribute the development of a body of knowledge with attendant philosophies and research design & methods used to explain, predict, and link empirical fact leading to new inference or have some practical value in the conduct of human behavior.

iv. To sponsor, support, and encourage the production and dissemination of scientific reports, topical bibliographies, review articles, interpretive chapters, textbooks, technical manuals, computer software and accounts in the popular press and other media.

Fig. 14.1: Derivation and content of Kinanthropometry
v. To designate an archival procedure for historical papers and documents, data assembles, and library collections.
vi. To develop and maintain a registry of referees and make the list available for grant requests, promotions and tenure, journal articles, papers in congress and symposia and other such scrutinizes which help contribute to the scientific rigor and reputation of the area.
vii. To plan, organize, sponsor and participate in certification courses, symposia, conferences, congresses and other such gatherings.

With all these background, the science of kinanthropometry has taken a strong footing at the international level. Even in India special scientific session on kinanthropometry were arranged in the year first (1985) and second (1986) National Conference of Indian Association of Sports Scientists and Physical Educationists.

**Anthropometry**

**Key Concept**

Anthropometry means the measurement of man, whether living or dead and consists primarily in the measurement of the dimensions of the body. It is the measurement of man provides scientific methods and observations on the living man and the skeleton. It represents the typical and traditional tool of human biology, physical anthropology and auxology. It has also taken a strong bonded relationship with physical education and sports sciences.

**Scope and development:** The anthropometry has developed primarily in the sports fields of physical anthropologists. Physical anthropology deals with the study of man’s biological defined behavior in time space. Physical anthropologist has been mainly concerned with the study of human origins and human evolutions as well as the varieties of mankind in different parts of the world. A new dimension has been added: namely the study of human growth and development of various human populations. The effect of environment and nutrition on the growth and development of human being is also sought to be assessed. The scientists have in recent years become increasingly concerned with the dimensions, proportions and shape of man’s immediate physical environments. Anthropometry has kept pace with these developments and provided suitable measurements and techniques. It is traditionally used for static measurements, and later on developed functional measurements also for suiting the present day needs.

The origin of anthropometry is very ancient. The scientific anthropometry, however, began with Johann Friedrich Blumenbach who laid the foundation of Craniology that is the study of human skull. He classified the human being in different races on the basis of skull form. Broca in 1875 published a paper containing instructions regarding craniometry and craniology. He defined the measurements landmarks to be used as well as the instruction required in taking them. This method was widely used by the scientists in different country. In 1935, the American association of physical Anthropologists formed an Advisory Committee on Anthropometric interest and gave a serious thought to widen the scope of the field of anthropometry.

Rudolf Martin has written a complete textbook on Anthropology and succeeded in defining more than 100 measurements on the living body and so many on the skeleton. In addition he gave a number of indices and also devised a few instruments.

Since World War I, anthropometry has been employed to provide standard sizes for different kinds of equipment in defense services and industries. Special measurements were devised suited to the particular need. It may be mentioned that Morant, Hooton, White and Hertzberg who along with a number of other anthropologists have not only added new dimensions to anthropometry, but also defined measurements, improve techniques, and devised many new instruments. The search for new avenues continued. Anthropometrists worked in different laboratories of the world on varied topics, devised new measurements and new instruments as well as improving the old ones. Simultaneously, the scientists interested to work on athletes and sportsmen also used anthropometry as a tool in
order to examine their size, shape, proportion and composition.

In 1960, Tanner examined Olympic athletes. In 1968, the Mexico Olympic athletes were also studied by him using anthropometry as a main tool. The use of anthropometry on the Indian athletes has also been extensively studied. The use of this science in sportsmen has grown so extensively that now it constitutes a new science called sports kinanthropometry or sports anthropometry.

METHODS OF MEASUREMENTS

It is understood that the two individual never be alike in their measurable characteristics. The individual undergo change in varying degrees from birth to death, in health, in disease and in physical training. Individual living under different conditions and individuals of different ethnic groups exhibit interesting differences in body size, shape and form. Therefore, it is essential to have some means of giving quantitative expressions to the variations which such traits exhibit. The anthropometry can conveniently be subdivided as follows:

**Somatometry**: The measurement of the body in the living and in the cadaver.

**Cephalometry**: It is included in somatometry; the measurement of the head and face in the living and in the cadaver and from X-ray films.

**Osteometry**: The measurement of the skeleton and its parts.

**Craniometry**: Included in Osteometry; the measurements of the skull.

In Kinanthropometry investigations, most of the research investigations referred in literature deal with the living subjects – such as athletes, sportsmen, physical education and personal sedentary people.

**Planes**

**Anatomical position**: This is the position assumed in all anatomical descriptions. In this regard the body should be in upright position with the head facing forward, the arms at the sides with the palms of the hands facing forward and the feet together.

**Midsagittal plane**: The body (in the anatomical position) is divided longitudinally into two equal parts. The dimension in this regard is through the median plane. Any structure which is described as being medial to another is, therefore, nearer the mid-line and any structure which is lateral to another is farther from the mid-lines or at the side of the body.

**Sagittal plane or Antero-posterior plane**: This plane runs parallel to the vertical dividing the body into right and left portions.

**Frontal or coronal plane**: This plane runs at right angle to the other two planes dividing the body into upper and lower positions.

**Proximal plane**: The term is used when describing bones of the limbs. It is that end of a bone which is nearest to the point of attachment of the limbs with the body.

**Distal plane**: The plane is also used in line with the proximal plane. It is that end of a bone which is farthest away from the point of attachment of the limbs with the body.

**Anterior or ventral plane**: It indicates that the part being described is nearer the front of the body.
Posterior or dorsal plane: It indicates that the part being described is nearer to the back of the body.

Superior plane: This refers to a structure nearer to the head.

 Inferior plane: This refers to a structure further away from the head.

Border: This is a ridge of bone which separates two surfaces.

Spine, Spinal process or Crest: This refers to a sharp ridge of bone.

**Anthropometric Instruments**

The use of proper equipment is most essential for various anthropometric measurements. The equipments need high precision and accuracy. The most commonly used and available equipments are as follows:

1. Anthropometric rod
2. Weighing machine
3. Stadiometer
4. Sitting height table
5. Somatotype turn table
6. Strength dynamometer (Hand grip & Back Dynamometer)
7. Goniometer
8. Steel measuring tap
9. Sliding caliper
10. Chest caliper
11. Spreading caliper
12. Skinfold caliper

**Anthropometric rod:** The anthropometric kit included two straight and two curved branches. The rod is used to measure the body height and different proportion of the body, A-P chest breadth (in lieu of chest caliper), etc. The rod is graduated in both centimeter and inches and the measurements are to be taken as nearest of the centimeter or inches respectively.

**Weighing machine:** The weighing machines are of three types—mechanical, electrical and digital. All the three types having portable one also. The machine is calibrated with accuracy up to 50 gm. Machine has got both kilogram and as well as pound scales. The machine should periodically calibrate using standard weight. The machine is used to measure the body weight.

**Stadiometer:** In laboratory operations, a wall mounted parallax correcting stadiometer or ball bearing digital wall mounted portable stadiometer can be used. However, stadiometer with foot plate is also available. The ‘Harpenden’ portable stadiometer has been designed to provide all the advantages of the wall mounted stadiometer in a portable form. It gives direct and accurate reading to the nearest millimeter. Stadiometer is being used to measure the body height in standing posture.

![Fig. 14.3: Various planes of the human body](image1)

![Fig. 14.4: Digital Stadiometer.](image2)
Sitting height table: The ‘Harpenden’ range provides a sitting height table having standard type of ball-bearing mounted, counter balance head block, giving accurate and direct reading from 320 to 1090 mm. In addition it has a secondary carriage, fitted with an anti-reverse carriage lock, in order to compensate for upper leg variation, and an adjustable foot rest in order to compensate for lower variations.

Somatotype turn table: This instrument can be used for taking Somatotype pictures of an individual. Its rounded platform is capable of rotating with the subjects’ load. It is fitted with a device which locks the turn table at each 90 degree rotation. The vertical bar on the side showing arrows will help in calculating the degree of magnification in the picture. Because of this the instrument may conveniently be used for taking some photographic measurements.

Sliding caliper: It is a simple instrument with a graduated flat bar having one fixed horizontal end at 0 mm. The other horizontal end slides with in the graduated flat bars on the right and left sides. Its horizontal bars have wider contact surfaces ensuring ease while taking measurements on subjects. The instrument can be used for taking small size measurements. In most cases it is used for measuring the bicondylar measurements, and therefore, named by some as bicondylar vernier.

Skinfold caliper: The instrument is used for assessment of subcutaneous fat tissue at specific sites. The skin is lifted with the help of thumb and index finger without holding the underlying muscle, forming a double fold. It is because of this reason that the instrument for taking this measurement is called skinfold caliper. A double layer of the fold is firmly maintained between the thumb and the forefinger through out the course of the measuring operation. The fold should be large enough to get a complete double layer, but not so large as to get so much skin and fat as may cause excessive amounts of tension beyond the fingertips. The fold of skin and fat is held some what loosely while the caliper is applied to the fold about 1 cm below the pinch point and the right had is allowed to fully relax its grip on the trigger so that the jaws can exert their full pressure. The caliper must be applied at right angles to the fold at all times. The reading on the dial is taken after permitting full spring pressure of the instrument by a complete release of the caliper trigger. The investigator should allow time for the full pressure of the caliper to take effect, but not so long that the adipose tissue becomes squeezed out of the skinfold. Considerable practice is essential in order to make judgment for skinfold of varying sizes and varying degree of compressibility. The reading is made approximately two to three seconds after application, when the riddle slows.

Fig. 14.5: Sitting height measuring table.

Fig. 14.6: Spreading caliper
**Spreading caliper:** The instrument has two curved arms with olive tips. The two arms joint together at one point or axis. The scale graduated in centimeter. On a flat bar, is loosely fixed with zero on one of the arms, passing through a socket in the other.

**Chest caliper:** This instrument has also two curved arms with olive tips, but it is bigger in size. The principle of its working and construction is just the same as that of spreading caliper.

**Goniometer:** It is used for measurement of angles of joint, indicating structure and extent of movement of different parts of the body.

**Steel measuring tape:** The anthropometric steel tape ideally—the centimeter/inches marks should be well marked for ease reading the measurements, the tape should be non extensible, and made of steel and the width should be 7 mm or less. The tape should have a slab before the zero line helping to hold the tape on the end while taking the measurements.

**LANDMARKS**

The accurate marking of the landmark is more essential for more precise anthropometric measurements. However, the technique of taking measurement by using the instrument is equally important. The technique of measurement of various anthropometric parameters must be a standard one and producing accurate results, the investigator should be also well trained and skillful.

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![Fig. 14.7: Sliding caliper with vernier scale.](image1)

![Fig. 14.8: Skin fold caliper (Harpenden).](image2)
Before listing the measurements made to determine the various dimensions are necessary to understand the landmarks from which such measurements are conventionally made. The important landmarks are as follows:

i. **Orbitale**: It is the lowest point on the inferior margin of the orbit.

ii. **Postaurale**: It is the most posterior point on the helix of the ear.

iii. **Frankfurt horizontal (F-H) plane**: The plane is determined by the lowest points on the infra-orbital margins and the tragion or tragi notch of the ear. This corresponds almost exactly to the plane of the visual axis that is obtained when the individual is looking straight ahead of him.

iv. **Tragion**: It is a point in the notch immediately above the tragus of the ear.

v. **Vertex**: It is the highest point of the head in the mid sagittal plane, when the head is held erectly in the Frankfurt plane.

vi. **Illiocristale**: It is the most lateral pointing of the mid-axillary line on the superior border of iliac crest.

vii. **Cervicale**: It is the most projecting point on the spinal process of 7th thoracic vertebrae. The point can be located while bending the neck slightly when this vertebra becomes more prominent.

viii. **Acromiale**: The point is located on the most superior and external border of the acromial process when the subject is standing erect with the arms hanging down freely.

ix. **Radiate**: The point is located on the most superior and lateral border of the head of radius. The point can be easily marked in the dimple near the lateral side of the elbow.

x. **Stylium radiate**: It is the most distal point of the styloid process of the radius. It is located in the ‘anatomical’ snuff box.
xi. *Styliol ulnare*: It is the most distal point on the styloid process of ulna.

xii. *Metacarpal radiale*: The most laterally placed point on the head of second metacarpal of the stretched hand is metacarpal radiale.

xiii. *Metacarpal ulnare*: The most medially placed point on the head of 5th metacarpal is metacarpal ulnare.

xiv. *Dactylion*: It is the most distally projecting point on the tip of the finger. The point dactylion on the thumb is dactylion 1 and that on the 5th finger, dactylion 5 respectively.

xv. *Trochanterion*: It is the most superior point on the top of greater trochanter of the femur. The point can be located by pressing the trochanter area with index and middle fingers of the individual standing with weight on the other leg.

xvi. *Tibiale mediale*: It is the most medial point on the superior boarder of the proximal end of tibia. The landmark can be located by palpatiting the quadriceps tenden with index finger at the distal end of patella.

xvii. *Tibiale laterale*: The point corresponds to the tibiale mediale above but is located on the lateral boarder of the proximal end of tibia. The tibiale mediale and laterale are almost opposite to each other in the transverse plane. To locating the landmark, the depression or dimple in the knee, bounded by a trial of prominence-epicondylar femur, anterio-lateral portion of the head of tibia and the head of fibula.

xviii. *Pternion*: It is the most backwardly projecting point on the heel of the foot when the subject is standing normally.

xix. *Acropodian*: It is the most anteriorly projecting point on the first or second toe of
the foot whichever is bigger. The subject’s toe nail may have to be chipped for taking the measurement.

xx. *Metatarsal fibiale:* It is the most medial point on the distal end of the first metatarsal when the subject is standing.

xxi. *Metatarsal tibulare:* It is the most lateral point on the distal end of 5th metatarsal when the subject is standing erectly.

xxii. *Cervicale:* It is the most backwardly projecting point on the spinal process of the seventh cervical vertebrae. To locate this landmark, the subject bends his head forwards. This helps the spinous process of the seventh cervical vertebra which moves away from the spinous processes of the usually more prominent first thoracic vertebrae. After locating the landmark, the subject assumes an erect position and the landmark is marked.

xxiii. *Gluteale:* It is the point situated at the sacrococygeal fusion in the midsagital plane. The landmark can be located by placing the thumb at the top of the gluteal furrow and palpat ing in a downward direction with the thumb. The finger should be spread over the lumbar region of the subject and the landmark can be located with minimal adjustment of clothing.

**ANTHROPOMETRIC MEASUREMENT**

i. *Body weight:* The body weight is ideally taken on a standard weighing machine having the accuracy recorded to the nearest 50 gm. The subject should stand erectly on the machine and hanging both the arms freely. The individual should wear minimum cloths at the time of recording the body weight.

ii. *Stature:* The subject to stand in a definite position in order to obtain accurate
measurements. Stature is taken as the maximum distance from the point vertex on the head to the ground. The vertex is the highest point on the head when it is in F-H plane. The subject should be barefooted, stand erect with heels together and the arms hanging naturally by the sides. The subject is instructed to look straight ahead and take deep breath. The measurement is recorded to nearest 0.1 centimeter.

iii. Sitting height: It is the vertical distance from the point vertex to the sitting plane. The subject should be instructed to sit stretched upwards with the arms downwards and hands on the thighs and also the lower legs hanging downwards at an angle of 90 degree. The height of the sitting table should be such that the subject’s feet are above the ground. Hold the position of head in F-H plane, and asked the subject to stretch up and sit straight and as tall as possible, and bring down the moving arm of anthropometer on the top of head, crushing the hair and making firm contact with the vertex.

iv. Bi-acromial breadth: It is the distance between the two acromion points when the subjects stand erect with the arms hanging down freely on the sides. The subject erect position and the anthropometric compasses is used for measuring biacromial diameter. Apply the fixed end of the horizontal bar of anthropometer on the left acromion from the back side of the subject. Bring the other horizontal bar on the right acromion in such a way that both bars and branches of caliper face upwards at an angle of 45 degrees from horizontal, measuring the largest diameter, putting firm pressure on both the landmarks.

v. Bi-iliocristal breadth: It is the maximum distance between the two iliocristal points. The subject stands erect and the measurement is taken from front. The landmarks on either side are located by using index or middle fingers with anthropometer in hand. The horizontal bars of anthropometer are applied with pressure on the superior boarder of the illiac crest at an angle of 45 degrees from the horizontal, measuring the largest diameter between two points.

vi. Chest breadth: It measures the maximum distance of the thorax at the level of the most lateral aspects of the forth ribs. The subject is made to sit erect facing the investigator. Locate the most lateral point of the forth ribs on right and left side, apply the horizontal bars of the anthropometer at an angle of 30 degrees downwards from the horizontal avoiding both pectoral and latissimus dorsi muscle.

vii. Anterio-posterior chest depth: It is the anterio-posterior diameter of the chest at the level of point mesostrenale. The subject sits on a stool or table and the measurement taken from the right side of the body. The measurements can be obtain by using the anthropometer with curved branches or the chest caliper. The anthropometer rests on forearms of the investigators. The olive tip of the instrument is kept on the point mesosternale and posteriorly on the spinal process of vertebra in the axis of the point mesosternale.

viii. Trunk length: Subtract the value of symphysal height from suprasternal height.

ix. Upper arm length: It measures the vertical distance from the landmark acromion to radiale or subtract the value of height radiale from height acromion.

x. Fore arm length: It measures the vertical distance from the landmark radiale to styliion radiale; or subtract the value of height styliion radiale from that of height radiale.

xi. Total arm length: Subtract the value of height styliion radiale from height acromion.

xii. Upper leg length: Subtract the value of height tibiale from that of height symphysion.

xiii. Lower leg length: It measures the straight distance from the landmark tibiale mediale to sphyrion.

xiv. Total leg length: Subtract the value of height sphyrion from that of height symphysion.

xv. Foot length: It measures the straight distance between acropodion and pternion.

xvi. Humerus bicondylar diameter: It measures the maximum distance between the lateral and
medial epicondyles of the humerus at right angles to the long axis of the upper arm. The subject sits on a chair with the upper arm extended straight forwards and the forearm flexed at an angle of 90 degree to the elbow. The sliding caliper is applied as in case of above measurement.

xvii. **Femur bicondylar diameter:** It measures the maximum distance between the lateral and medial epicondyles of the femur. The individual is asked to sit on a chair with the knee bent at a right angle. The caliper is applied bisecting the angle of knee, with the firm pressure on the crossbars and the measurement is recorded to the nearest 0.5 cm.

xviii. **Wrist diameter:** It measures the width between the most medial and lateral points of the distal epiphyses of radius and ulna. The subject while sitting extends hand towards the investigator with palm facing downwards. The measurement is taken to the nearest 0.5 mm while keeping the caliper at right angles from the axis of the forearm, with firm pressure on the crossbars of sliding caliper.

xix. **Arm circumference (relaxed):** It is the perimeter distance of the right arm parallel to the long axis of the humerus when the subject stands erect and the relaxed arm hangs by the sides. The tape is held at the measured and marked mid-acromiale-radiale distance.

xx. **Forearm circumference:** It is the maximum girth of the forearm when the hand is held palm up and relaxed. The measurement is taken to the nearest 0.5 mm while keeping the caliper at right angles from the axis of the forearm, with firm pressure on the crossbars of sliding caliper.

xxi. **Chest circumference:** The subject slightly abducts his arms to permit the investigator facing him to pass the tape around his chest; the tape is held in the right hand while the investigator’s left hand adjusts the tape at the subject’s back to the horizontal level of the marked mesosternale. The cross handed technique is used to put the tape scale in juxtaposition with the zero on the stub end of the tape. The reading is obtained at the end tidal of a normal expiration.

xxii. **Thigh circumference:** It is the perimeter of the thigh with the subject standing erect, legs slightly apart with the body weight equally distributed on both feet. The tape is raised to a level one to two centimeters below the gluteal line. A cross-handed technique is used to raise the tape to this level on the inner thigh, and then the tape is read when the stub end is brought in juxtaposition to the housing end.

xxiii. **Calf circumference:** It is also measured with the subject in the same position as above. The tape is maneuvered to obtain the maximum perimeter of the calf. This measure is obtained by manipulation of the tape taking a series of girth measurements to assure the largest value.

xxiv. **Skin folds:** (a) **Biceps**—The skinfold is measured by raising a vertical fold at the marked mid acromeale-radiale line on the anterior surface of the arm. The subject stands with the arms hanging down freely. (b) **Triceps**—This measurement is taken on the posterior surface of the arm at the level of biceps skinfold with the subject in the same position. (c) **Forearm**—The skinfold is raised beneath the inferior angle of the left scapula in the direction running obliquely downwards at an angle of about 45 degree from the horizontal. (d) **Subscapular**—The skinfold is measured vertically on the planter surface of the forearm. The subject stands with both arms hanging down so that the palms face anteriorly. (d) **Subscapular**—The skinfold is measured vertically on the medial portion of the calf muscle. At the time of measurements the calf muscle should be in relaxed position and the subject will stand freely with the leg is slightly forwarded. (f) **Thigh**—The skinfold is taken on the anterior surface mid-way between the mid-inguinal point and the superior border of patella. The knee should be flexed at 90 degree. (g) **Abdomen**—The fold is taken at the level of umbilicus about two inches to its left.
HUMAN PHYSIQUE

Somatotyping is a form of body classification, i.e. it is a shorthand method of describing an individual’s physique according to certain physical characteristics. Somatotyping was originally developed by an American psychologist—William Sheldon in 1940s, after he studied over 4,000 photographs of college age men. These photographs have since been termed the ‘Ivy League posture photographs’. Over the years other scientists and professionals have also been instrumental in further developing body classification systems (Naccarati, Kretschemer, Heath-Carter, etc.).

The experience of Dr Barbara Honeyman Heath, Roll with somatotyping provide a background suitable for empirical modification of constitution Laboratory Somatotype Methodology. She worked on over 7000 somatotype photograph. From 1940 to 1960 the system of somatotyping aroused more controversies. There have been questions about Somatotype techniques, methodology and findings. Keeping all small factors in mind, Heath (1963) suggested that the seven point rating scale and relationship of Somatotype to the criterion of height/√weight reported by Sheldon.

A new and improved Somatotype method with universal application to both sexes, for all ages and which is reproducible was innovated after requisite validation. This method is known as “A Modified Somatotype Method” and is designed by Barbara Honeyman Heath, now known as Heath-Roll, and JE Lindsay Carter in 1967. The new method retained the well—accepted terminology of Somatotype, endomorphy, mesomorphy and ectomorphy.

Extreme Somatotypes

Sheldon divided physique into three basic categories or somatotypes, known as ‘extreme somatotypes’ by dividing body into five areas: such as (i) head, face and neck; (ii) thoracic trunk; (iii) arms, shoulders, hands; (iv) abdominal trunk; (v) legs and feet. He then secured a seven point scale for each component in each region, by approximating the mid point of each range and subdividing into three equal intervals on both sides of the mid-point. Thus each component in each region was scored from a minimum of 1 to a maximum of 7, and the somatotype of the subject was determined from the average of each of three components in the five regions combined. Each somatotype was then presented by a three digit combination: 711, Extreme endomorph, 171, Extreme Mesomorph; 117 Extreme Ectomorph and 444, a balanced physique falling at the mid-point of all three scales.

The general characteristics of endomorph mesomorph and ectomorph are as follows:

Endomorph
- Pear-shaped body
- Rounded head
- Wide hips and shoulders
- Wider front to back rather than side-to-side
- A lot of fat on the body, upper arms and thighs

Mesomorph
- Wedge-shaped body
- Cubical head
- Wide broad shoulders
- Muscled arms and legs
- Narrow hips
- Narrow from front to back rather than side-to-side
- A minimum amount of fat

Ectomorph
- High forehead
- Receding chin
- Narrow shoulders and hips

Fig. 14.13: Diagram representing the somatochart
Narrow chest and abdomen
Thin arms and legs
Little muscle and fat

An extreme or true endomorphic individual is generally considered to be an individual who is overweight, an extreme mesomorph is generally a smaller, stocky person, and an extreme or true ectomorph is a taller, leaner individual. Obviously the heredity predominantly determines which somatotype you are, however, through a combination of diet and exercise, you can move closer to one particular somatotype as well.

From Sheldon’s perspective it was not suggested that we were one category or the other; we generally have combination of all three elements in our bodily make-up. For example, it would be unusual to meet someone who is a pure ectomorph, with no endomorphic or mesomorphic tendencies. You may meet an Ecto-meso-endomorph (someone who has more ectomorph tendencies than mesomorph, and more mesomorph tendencies than endomorph).

To assist in the body classification, Sheldon evaluated the degree of a somatotype component which is present on a scale from one to seven, with one corresponding to the minimum and seven to the maximum tendency to have that component. Using a score of one to seven we can grade our bodies on each of the extreme body types. For example, 2-6-3 indicates lowest endomorphy-high mesomorphy-low ectomorphy.

**HEATH-CARTER SOMATOTYPE METHOD**

A new improved somatotype method with universal application to both sexes, for all ages and which is reproducible was innovated after requisite, validation. This method is known as modified somatotype method and is designed by Barbara Honeyman Heath and JE Lindsey Carter in 1967. The new method retained the well-accepted terminology—somatotype, endomorphy, mesomorphy and ectomorphy. Basically a somatotype is a description of the present morphological confirmation of an individual. It is expressed in a three numeral rating, consisting of three sequential numerals, always recorded in the same order. Each numeral represents evaluation of one of the three primary components of physique which describe individual variations in human morphology and composition. The three components – 1st, 2nd and 3rd components which are as follows:

**First component (endomorphy):** It refers to relative fitness in individual physique. That is the first component ratings are evaluations of degrees of fitness which lie on a continuum from the lowest recorded values to the highest recorded values.

**Second component (mesomorphy):** It refers to relative musculo-skeletal development per unit of height. The second component ratings are evaluations of musculo-skeletal development which lie on a continuum from lowest to highest degree recorded. The second component can be thought of as lean body mass relative to height.

**Third component (ectomorphy):** It refers to relative linearity of individual physique. The third component ratings are based largely, but not entirely on height/√weight ratios. This ratio and third component is closely related, so that at the low ends of their distributions both connote relative shortness of the several body segments and the high ends connote elongation or linearity of the several body segments.

**Classification of Somatotypes**

Somatotype if studied in large population, a variety of somatotypes with varying strengths of different components will be observed. These somatotypes can be classified in the following generalized category:

a. Balanced endomorphy: The first component is dominant and the second and third components are equal (or do not differ by more than one-half unit), example: 5-3-3, 6-2-2, etc.

b. Mesomorphic endomorph: Endomorphy is dominant and the second component is greater than the third. Example: 5-4-2, 6-3-1, etc.

c. Mesomorph-endomorph: The first and second components are equal (or do not differ by more than one-half unit) and the third component is smaller: Example: 5-5-2, 4-4-2, etc.

d. Endomorphic mesomorph: The second component is dominant and the first component is greater than the third component. Example: 3-6-1, 3-4-2, etc.
c. Balanced mesomorph: The second component is dominant and the first and third components are less and equal. Example: 2-5-2, 3-6-3, etc.
f. Ectomorphic mesomorph: The second component is dominant and the third component is greater than the first component. Example: 2-5-3, 2-6-4, etc.
g. Mesomorphic ectomorph: The second and third components are equal and the first component is lower. Example: 2-4-4, 1-5-5, etc.
h. Mesomorphic endomorph: The third component is dominant and the second component is greater than the first component. Example: 1-3-6, 2-4-5, etc.
i. Balanced ectomorph: The third component is dominant and the first and second components are equal and lower. Example: 2-2-6, 3-3-5, etc.
j. Endomorphic ectomorph: The third component is dominant and the first component is greater than the second component. Example: 3-2-6, 3-1-5, etc.
k. Endomorph ectomorph: The first and third components are equal and the second component is lower. Example: 5-1-5, 4-2-4, etc.
l. Ectomorphic endomorph: The first component is dominant and the third component is greater than the second component. Example: 6-2-3, 5-1-4, etc.
m. Central: No components differ by more than one unit from the other two, and consist of ratings of 2, 3 or 4, 3-3-3, etc.

**THE ANTHROPOMETRIC SOMATOTYPE METHOD**

**Measurement Techniques**

Ten anthropometric dimensions are needed to calculate the anthropometric somatotype: stature, body mass, four skinfolds (triceps, subscapular, supraspinale, medial calf), two bone breadths (bicipital and humerus and femur), and two limb girths (arm flexed and tensed, calf). The following descriptions are adapted from Carter and

i. Stature (height).
ii. Body mass (weight).
iii. Skinfolds:
   - Triceps skinfold.
   - Subscapular skinfold.
   - Supraspinale skinfold.
   - Medial calf skinfold.
iv. Biepicondylar breadth of the humerus.
v. Biepicondylar breadth of the femur.
vi. Upper arm girth.
vii. Calf girth.

Read stature and girths to the nearest mm, biepicondylar diameters to the nearest 0.5 mm, and skinfolds to the nearest 0.1 mm (Harpenden caliper) or 0.5 mm on other calipers. Traditionally, for the anthropometric somatotype, the larger of the right and left breadths and girths have been used. When possible this should be done for individual assessment. However, in large surveys it is recommended that all measures (including skinfolds) be taken on the right side of the body. The investigators should mark the sites and repeat the complete sequence a second time. For further calculations, the duplicated measurements should be averaged. For more reliable values, relatively inexperienced measurers should take triplicate measurements and use the median value.

Calculating the Anthropometric Somatotype

There are two ways to calculate the anthropometric somatotype.

A. Enter the data onto a somatotype rating form.
B. Enter the data into equations derived from the rating form.

The use of the rating form will be described first. Figures 14.14 and 14.15 are examples of calculations using the rating form. It is assumed that the measurements have been recorded on an appropriate Somatotype Instruction Manual 5 recording form and average or median values calculated before transfer to the rating form. A blank rating form is provided in Figure 14.16.

The Heath-Carter Somatotype Rating Form

1. Record pertinent identification data in top section of rating form.

   **Endomorphy rating** (steps 2–5)

   2. Record the measurements for each of the four skinfolds.

   3. Sum the triceps, subscapular, and supraspinale skinfolds; record the sum in the box opposite sum of three skin folds. Correct for height by multiplying this sum by \((170.18/\text{height in cm})\).

   4. Circle the closest value in the sum of three skin folds table to the right. The table is read vertically from low to high in columns and horizontally from left to right in rows. “Lower limit” and “upper limit” on the rows provide exact boundaries for each column. These values are circled only when sum of three skin folds are within 1 mm of the limit. In most cases circle the value in the row “midpoint”.

   5. In the row for endomorphy circle the value directly under the column for the value circled in number (4) above.

   **Mesomorphy rating** (steps 6–10)

   6. Record height and breadths of humerus and femur in the appropriate boxes. Make the corrections for skinfolds before recording girths of biceps and calf. (Skinfold correction: Convert triceps skinfold to cm by dividing by 10. Subtract converted triceps skinfold from biceps girth. Convert calf skinfold to cm, subtract from calf girth.)

   7. In the height row directly to the right of the recorded value, circle the height value nearest to the measured height of the subject. (Note: Regard the height row as a continuous scale.)

   8. For each bone breadth and girth circle the number nearest to the measured value in the appropriate row. (Note: Circle the lower value if the measurement falls midway between two values. This conservative procedure is used because the largest girths and breadths are recorded.)

   9. Deal only with columns, not numerical values for the two procedures below. Find the average deviation of the circled values for
breadths and girths from the circled value in the height column as follows:

a. Column deviations to the right of the height column are positive deviations. Deviations to the left are negative deviations. (Circled values directly under the height column have deviations of zero and are ignored.)

b. Calculate the algebraic sum of the ± deviations (D). Use this formula: mesomorphy = (D/8) + 4.0. Round the obtained value of mesomorphy to the nearest one-half (½) rating unit.

10. In the row for mesomorphy circle the closest value for mesomorphy obtained in number 9 above. (If the point is exactly midway between two rating points, circle the value closest to 4 in the row. This conservative regression toward 4 guards against spuriously extreme ratings.)

**Ectomorphy rating** (steps 11–14).

11. Record weight (kg).

12. Obtain height divided by cube root of weight i.e. Hight-weight Ratio (HWR). Record HWR in the appropriate box.

13. Circle the closest value in the HWR table to the right. (See number 4 above.)

14. In the row for ectomorphy circle the ectomorphy value directly below the circled HWR.

15. Move to the bottom section of the rating form. In the row for Anthropometric Somatotype, record the circled ratings for Endomorphy, Mesomorphy and Ectomorphy.

16. Sign your name to the right of the recorded rating.

The identification data in the upper section of the rating form are somewhat arbitrary. Investigators may change these to suit their purposes.

**Principles of the calculations:**

Two principles are important in understanding the calculation of mesomorphy on the rating form. (i) When the measurements of bone breadths and limb girths lie to the right of the circled height column, the subject has greater musculo-skeletal robustness relative to height (i.e. higher mesomorphy) than a subject whose values lie to the left of the height column. The average deviation of the circled values for breadths and girths is the best index of average musculo-skeletal development relative to height. (ii) The table is constructed so that the subject is rated 4 in mesomorphy when the average deviation falls in the column under the subject’s height, or when the four circled values fall in the subject’s height column. That is, the average deviation (±) to the left or right of the height column is added to or subtracted from 4.0 in mesomorphy.

**Height-Weight Ratio Calculation**

The height-weight ratio (HWR), or height divided by the cube root of weight (stature/mass^1/3) as it is used in somatotyping, may be determined by using a hand calculator. A calculator with a y to the x power (yx) key is needed. To get the cube root, enter mass, i.e. base (y), press yx, enter.3333, and press ‘equals’. If there is an INV yx function, this may be used instead by entering 3 (for the cube root).

**Limitations of the Rating Form**

Although the rating form provides a simple method of calculating the anthropometric somatotype, especially in the field, it has some limitations. First, the mesomorphy table at the low and high ends does not include some values for small subjects, e.g. children, or for large subjects, e.g. heavy weightlifters. The mesomorphy table can be extrapolated at the lower and upper ends for these subjects. Second, some rounding errors may occur in calculating the mesomorphy rating, because the subject’s height often is not the same as the column height. If the anthropometric somatotype is regarded as an estimate this second limitation is not a serious problem. Nevertheless, the following procedures described by Carter (1980) and Carter and Heath (1990) can correct these problems.

**EQUATIONS FOR A DECIMAL ANTHROPOMETRIC SOMATOTYPE**

The second method of obtaining the anthropometric somatotype is by means of equations into which the data are entered.
Endomorphy \( = -0.7182 + 0.1451 (X) - 0.00068 (X^2) + 0.0000014 (X^3) \)

where \( X \) = (sum of triceps, subscapular and supraspinale skinfolds) multiplied by \((170.18/\text{height in cm})\). This is called height-corrected endomorphy and is the preferred method for calculating endomorphy.

The equation to calculate mesomorphy is:

\[
\text{Mesomorphy} = 0.858 \times \text{humerus breadth} + 0.601 \times \text{femur breadth} + 0.188 \times \text{corrected arm girth} + 0.161 \times \text{corrected calf girth} - \text{height} 0.131 + 4.5
\]

Three different equations are used to calculate ectomorphy according to the height-weight ratio:

If HWR is greater than or equal to 40.75 then

\[
\text{Ectomorphy} = 0.732 \text{HWR} - 28.58
\]

If HWR is less than 40.75 but greater than 38.25 then

\[
\text{Ectomorphy} = 0.463 \text{HWR} - 17.63
\]

If HWR is equal to or less than 38.25 then

\[
\text{Ectomorphy} = 0.1
\]

For subjects A and B respectively (Figures 14, 15, 14, 16), the resulting somatotypes (using height corrected endomorphy) are 1.5-5.5-3.0, and 3.0-2.0-5.0. The preceding equations, derived from data used by Heath and Carter (1967), use metric units. The equation for endomorphy is a third degree polynomial. The equations for mesomorphy and ectomorphy are linear (When the HWR is below 40.75 a different equation is used for ectomorphy.). If the equation calculation for any component is zero or negative, a value of 0.1 is assigned as the component rating, because by definition ratings cannot be zero or negative.

Checking the results

Now that the anthropometric somatotype has been calculated, is the result is logical? There are several ways to check the results for measurement or calculation errors. Using the rating form examples in above Figures the resulting somatotypes rounded to the nearest half-unit, are 1½-5½-3 and 3-2-5 for subjects A and B respectively. Are these reasonable somatotypes? Certain somatotype ratings are not biologically possible, although our examples are not among them. For example, a 2-2-2 or a 7-8-7 are impossible somatotypes. Generally, somatotypes high in endomorphy and/or mesomorphy cannot also be high in ectomorphy. Conversely, those high in ectomorphy cannot be high in endomorphy and/or mesomorphy; and those low in endomorphy and mesomorphy must be high in ectomorphy. Next, look at the pattern of circled values in the endomorphy and mesomorphy sections of the rating form. Are there inconsistencies in the data? For endomorphy, are the skinfold values reasonable? For mesomorphy, is there one measure (excluding height) that is quite far apart from the others? In Figure 14.15, upper limb circled values are slightly to the right of, and larger relative to height, than the lower limb measures. However, this pattern is not unusual and is quite acceptable in this case. On the other hand, if the femur width was 7.95 cm instead of 9.75 cm, or corrected calf girth was 44.9 cm instead of 37.1 cm; such large deviations would suggest errors. Check for errors in recording and re-measure the subject if possible. Also, check to see that the correct skinfolds in cm have been subtracted for the corrected girth values. In Figure 14.16, the small corrected biceps girth (23.4 cm) looks suspiciously low, but in this subject it truly represented her his small muscular development in the upper limb.

If the calculation for any component is zero or negative, a value of 0.1 is assigned as the component rating, because by definition ratings cannot be zero or negative. The photoscopic rating would be one-half (½). If such low values occur the raw data should be checked. Values less than 1.0 are highly unlikely to occur for endomorphy and mesomorphy, but are not unusual for ectomorphy. Component ratings should be rounded to nearest 0.1 of a unit, or nearest half-unit depending on their subsequent use. After the values are entered into the equations (either by calculator or computer program) rather than onto the rating form, it is impossible to check the pattern of values in either the endomorphy or mesomorphy section as in the rating form, although the raw values can be examined for errors. This is a limitation of using the equations. Further checking can be done for either method by using the HWR and by plotting the somatotype.
### Fig. 14.15: Calculations of the anthropometric somatotype using the rating form
### Fig. 14.16: Calculations of the anthropometric somatotype using the rating form

<table>
<thead>
<tr>
<th>Name</th>
<th>Age</th>
<th>Sex</th>
<th>M</th>
<th>F</th>
<th>No</th>
<th>Project</th>
<th>Measured by</th>
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</thead>
<tbody>
<tr>
<td>Skinfolds mm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Sum of three skinfolds (mm)</td>
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</tr>
<tr>
<td>Triceps</td>
<td>4.4</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(Height corrected skinfold)</td>
<td></td>
</tr>
<tr>
<td>Subscapular</td>
<td>1.9</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Supraepigastrica</td>
<td>4.6</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
</tbody>
</table>

#### Anthropometric somatotype

<table>
<thead>
<tr>
<th>Anthropometric somatotype</th>
<th>Antropometric plus</th>
<th>Photometric somatotype</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endomorphy</td>
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<td>1.5</td>
</tr>
<tr>
<td>Mesomorphy</td>
<td>5.5</td>
<td>5.5</td>
</tr>
<tr>
<td>Ectomorphy</td>
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<td>3.0</td>
</tr>
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</table>

**BY:**

**RATER:**

---

#### Calculations of the anthropometric somatotype using the rating form

<table>
<thead>
<tr>
<th>Height(cm)</th>
<th>176.3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biceps(girth)(cm)</td>
<td>33.9</td>
</tr>
<tr>
<td>- Triceps (skinfolds)</td>
<td>33.3</td>
</tr>
<tr>
<td>- Calf (skinfolds)</td>
<td>37.5</td>
</tr>
<tr>
<td>Mesomorphy</td>
<td>4.5</td>
</tr>
<tr>
<td>Weight(kg)</td>
<td>89.2</td>
</tr>
<tr>
<td>SB(1/3)</td>
<td>43.4</td>
</tr>
<tr>
<td>Lower limit</td>
<td>Below 48.6</td>
</tr>
</tbody>
</table>

---

#### Endomorphy Calculations

<table>
<thead>
<tr>
<th>Endomorphy</th>
<th>1.5</th>
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</thead>
<tbody>
<tr>
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</tbody>
</table>

---

#### Mesomorphy Calculations

<table>
<thead>
<tr>
<th>Mesomorphy</th>
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#### Ectomorphy Calculations

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</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

---

#### Sum of three skinfolds (mm)

<table>
<thead>
<tr>
<th>Sum of three skinfolds (mm)</th>
<th>188.0</th>
</tr>
</thead>
</table>

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#### Height corrected skinfold

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<thead>
<tr>
<th>Height corrected skinfold</th>
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</thead>
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---

#### Calculations of the anthropometric somatotype using the rating form

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<thead>
<tr>
<th>Name</th>
<th>Age</th>
<th>Sex</th>
<th>M</th>
<th>F</th>
<th>No</th>
<th>Project</th>
<th>Measured by</th>
<th>Sum of three skinfolds (mm)</th>
<th>Height corrected skinfold</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skinfolds mm</td>
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<td></td>
<td></td>
<td>(Height corrected skinfold)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Triceps</td>
<td>4.4</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subscapular</td>
<td>1.9</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Supraepigastrica</td>
<td>4.6</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Name</td>
<td>Age</td>
<td>Sex</td>
<td>M</td>
<td>F</td>
<td>No</td>
<td>Occupation</td>
<td>Ethnic group</td>
<td>Date</td>
<td></td>
</tr>
<tr>
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<td>---</td>
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<td>------</td>
<td></td>
</tr>
<tr>
<td>Project</td>
<td>Measured by</td>
<td>Sum of three skinfolds (mm)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Skinfolds mm</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Triiceps</td>
<td>=</td>
<td>Upper Limit</td>
<td>109.9</td>
<td>14.8</td>
<td>18.9</td>
<td>22.9</td>
<td>26.9</td>
<td>31.2</td>
<td>35.3</td>
</tr>
<tr>
<td>Subscapular</td>
<td>=</td>
<td>Mid-point</td>
<td>9.0</td>
<td>13.0</td>
<td>17.0</td>
<td>21.0</td>
<td>25.0</td>
<td>29.0</td>
<td>33.3</td>
</tr>
<tr>
<td>Suprapectoral</td>
<td>=</td>
<td>Lower limit</td>
<td>7.0</td>
<td>11.0</td>
<td>15.0</td>
<td>19.0</td>
<td>23.0</td>
<td>27.0</td>
<td>31.3</td>
</tr>
<tr>
<td>Sum 3 skinfolds</td>
<td>=</td>
<td>$X_{3sum}^{1.8-1.5}$</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calf</td>
<td>=</td>
<td>(Height corrected skinfold)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height(cm)</td>
<td>=</td>
<td>Endomorphy</td>
<td>1</td>
<td>1.5</td>
<td>2</td>
<td>2.5</td>
<td>3</td>
<td>3.5</td>
<td>4</td>
</tr>
<tr>
<td>Biceps girth(cm)</td>
<td>=</td>
<td>=</td>
<td>1.19</td>
<td>1.34</td>
<td>1.49</td>
<td>1.64</td>
<td>1.78</td>
<td>1.93</td>
<td>2.08</td>
</tr>
<tr>
<td>Biiceps girth(cm)</td>
<td>=</td>
<td>=</td>
<td>23.7</td>
<td>24.4</td>
<td>25.0</td>
<td>25.7</td>
<td>26.3</td>
<td>27.0</td>
<td>27.7</td>
</tr>
<tr>
<td>Calf girth(cm)</td>
<td>=</td>
<td>Endomorphy</td>
<td>27.7</td>
<td>28.5</td>
<td>29.3</td>
<td>30.1</td>
<td>30.8</td>
<td>31.6</td>
<td>32.4</td>
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<td>1.5</td>
<td>2</td>
<td>2.5</td>
<td>3</td>
<td>3.5</td>
<td>4</td>
</tr>
<tr>
<td>Weight(kg)</td>
<td>=</td>
<td>Upper Limit</td>
<td>39.05</td>
<td>40.74</td>
<td>42.43</td>
<td>44.12</td>
<td>45.81</td>
<td>47.50</td>
<td>49.19</td>
</tr>
<tr>
<td>Ht Wt</td>
<td>=</td>
<td>Mid-point</td>
<td>Acid</td>
<td>40.28</td>
<td>42.08</td>
<td>43.89</td>
<td>45.68</td>
<td>47.51</td>
<td>49.34</td>
</tr>
<tr>
<td>Lower limit</td>
<td>=</td>
<td>Below</td>
<td>39.80</td>
<td>40.73</td>
<td>41.66</td>
<td>42.59</td>
<td>43.54</td>
<td>44.48</td>
<td>45.43</td>
</tr>
<tr>
<td>Ectomorphy</td>
<td>=</td>
<td>0.5</td>
<td>1</td>
<td>1.5</td>
<td>2</td>
<td>2.5</td>
<td>3</td>
<td>3.5</td>
<td>4</td>
</tr>
</tbody>
</table>

**Fig. 14.17:** Blank Somatotype Rating Form
There is a relationship between the HWR and the likely somatotypes (Fig. 14.20). The somatotypes in the rows are those most likely to occur for the given HWR. For example, given a HWR of approximately 49.6, the most likely somatotypes are 1-1-8, 1-2-9 or 2-1-9. (The hyphens are left out of the somatotypes to conserve space.) The next most likely somatotypes are those in the rows directly above and below the row for 49.6. If none of these somatotypes match or are not close when interpolating for half-unit ratings, there may be errors in the data or calculations. However, other factors such as heavy meals or dehydration can affect body weight sufficiently to alter the “normal” HWR. For subject A, HWR = 43.4, and Figure 14.20 shows that in the row for a HWR of 43.6 the somatotypes 1-6-3 and 2-5-3 occur. His 1.5 - 5.5 -3 is a combination of these two ratings, therefore, his anthropometric rating agrees with that expected from the HWR table.

For subject B, HWR = 45.6, his 3-2-5 somatotype appears in the row above that for his HWR. His ectomorphy is borderline between 4½ and 5, which suggests that he might be a 3-2-4.5, i.e. half way between the two rows. The somatotypes for both subjects appear to be reasonable.

**Plotting the Somatotype**

Traditionally, the three-number somatotype rating is plotted on a two-dimensional somatochart using X,Y coordinates derived from the rating. The coordinates are calculated as follows:

\[
X = \text{ectomorphy} - \text{endomorphy} \\
Y = 2 \times \text{mesomorphy} - (\text{endomorphy} + \text{ectomorphy})
\]

For subject A, X = 1.5, and Y = 6.5. For subject B, X = 2.0 and Y = -4.0. These points on the somatochart are called somatoplots. If the somatoplots for the subject is far from that expected...
when compared to a suitable reference group, check the data and calculations. Because Figure 14.17 is quite crowded with numbers, the final somatoplots could be projected onto a somatochart without the numbers. Figures 14.18a,b are two blank somatocharts, one with printed somatotypes and one without. These may be copied for use.

**The Three-dimensional Somatotype**

As somatotype is a three-number expression meaningful analyses can be conducted only with special techniques. Somatotype data can be analyzed by both traditional and non-traditional descriptive and comparative statistical methods although descriptive statistics are used for each of the components, comparative statistics should be made in the first instance using the whole (or global) somatotype rating. This is followed by analysis of separate components. Here are some useful definitions:

*Somatopoint* ($S$). A point in three-dimensional space determined from the somatotype which is represented by a triad of $x$, $y$ and $z$ coordinates for the three components. The scales on the coordinate axes are component units with the hypothetical somatotype 0-0-0 at the origin of the three axes.

*Somatotype attitudinal distance* (SAD). The distance in three dimensions between any two somatopoints. Calculated in component units.

*Somatotype attitudinal means* (SAM). The average of the SADs of each somatopoint from the mean somatopoint ($S$) of a sample. The SAD represents the “true” distance between two somato-points (A and B). The SAD is calculated as follows:

$$SAD_{A,B} = \sqrt{((endomorphy_A - endomorphy_B)^2 + (mesomorphy_A - mesomorphy_B)^2 + (ectomorphy_A - ectomorphy_B)^2)}$$

Where A and B are two individuals, two different times for one individual, or two means. The SAM is calculated by dividing the sum of the SADs from their mean somatopoint by the number of subjects.

**Equations for Somatotype Analysis**

The following equations are used in calculation and analysis of somatotype data. Items 1 and 2 are
for calculating and plotting the somatotype. Items 3 to 6 are for analysis of the whole somatotype. Item 7 suggests methods for comparisons by components.

1. The anthropometric somatotype.

Endomorphy = – 0.7182 + 0.1451 (X) – 0.00068 (X2) + 0.0000014 (X3)

Mesomorphy = (0.858 HB + 0.601 FB +0.188 CAG + 0.161 CCG) – (0.131 H) + 4.5

Ectomorphy:

If HWR ≥ 40.75, then Ectomorphy = 0.732 HWR – 28.58
If HWR < 40.75 and > 38.25, then Ectomorphy = 0.463 HWR – 17.63
If HWR > 38.25, then Ectomorphy = 0.1 (or recorded as ½)

Where: X = (sum of triceps, subscapular and supraspinale skinfolds) multiplied by (170.18/height in cm); HB = humerus breadth; FB = femur breadth; CAG = corrected arm girth; CCG = corrected calf girth; H = height; HWR = height/cube root of weight. CAG and CCG are the girths corrected for the triceps or calf skinfolds respectively as follows: CAG = flexed arm girth – triceps skinfold/10; CCG = maximal calf girth – calf skinfold/10.

2. Plotting somatotypes on the 2-D somatochart.

X-coordinate = ectomorphy - endomorphy
Y-coordinate = 2 x mesomorphy - (endomorphy + ectomorphy)

Applications in Sports Science

All human being including athletes are made up of the three extreme body types so we are all part of endomorph, part mesomorph and part ectomorph. By classifying our own body physiques using somatotyping, we can compare our body type with that of other athletes. Graphs and tables have been developed to decide which sports suit which somatotypes better (as already been discussed above).

These tables and graphs can be quite helpful for physical education teachers, coaches, trainer, etc. to ascertain where on the pitch someone should or could be playing, or which sport that particular individual may be good at. Obviously, there will always be the exception to the rule, but generally, certain somatotypes are more suited to certain activities than others. For example, an extreme ectomorph will generally make a better long distance runner than they will a prop in rugby, and a mesomorph will generally make a better wrestler than they will a ballet dancer. Obviously, the somatotype is not the only factor determining how good the player is at a particular sport; each sport required specific skills, such as good hand-eye coordination, awareness, concentration, timing, speed, endurance, agility etc.

It has been seen that javelin throwers and gymnasts have practically identical somatotypes, although the javelin throwers, (179.5cm and 76.7kg) are much bigger than the gymnasts (167.4cm and 67.1kg). This shows that the use of somatotyping on its own has limits, but used with

Table 14.1: Dominant somatotype rating and respective sports/ events

<table>
<thead>
<tr>
<th>Somatotype</th>
<th>Examples of suggested suitable sports</th>
</tr>
</thead>
<tbody>
<tr>
<td>Predominantly Endomorph</td>
<td>i. Darts</td>
</tr>
<tr>
<td></td>
<td>ii. Sumo wrestling, Shot put thrower etc.</td>
</tr>
<tr>
<td>Predominantly Mesomorph</td>
<td>iii. Power lifting, Weight lifting, Wrestling, Boxing, etc.</td>
</tr>
<tr>
<td></td>
<td>iv. Canoeing, Javelin &amp; Discuss Throw, Sprinting, etc.</td>
</tr>
<tr>
<td></td>
<td>v. Judo, Cycling, Gymnastics etc.</td>
</tr>
<tr>
<td>Predominantly Ectomorph</td>
<td>vi. Marathon, Middle &amp; long distance running, Skiing, etc.</td>
</tr>
<tr>
<td></td>
<td>vii. Orienteering</td>
</tr>
<tr>
<td></td>
<td>viii. Dancing, Rhythmic Gymnastics</td>
</tr>
</tbody>
</table>

Table 14.1: Dominant somatotype rating and respective sports/ events
other data on body size and shape it may be of prime importance in Physiological and biomechanical interpretation of various sports performances.

**Body Size**

Body size refers to the person’s height and weight. The ideal size for an athlete depends on their sport, and sometimes on the position they play in their sport (consider the various body sizes in a soccer/hockey/rugby team). There are standard ideal weight charts based on an individual’s height, however, these tables do not help athletes because they do not allow for body composition, i.e. muscle is heavier than fat and therefore a person may seems overweight when they are not.

**Body Composition**

Theoretically, the body can be divided into several compartments according to definable tissues. The two component model used commonly divides the body to a fat portion, and further divides the fat free mass to skeleton, muscle and the remainder. The fluids, etc. can also be studied in terms of total body water, extra-cellular water, intra-cellular water, total body potassium, calcium, sodium and so on. However, the athletes have mostly been studied considering the body either as two or four compartment model.

Body composition refers to the athlete’s body fat. In most sports the athlete will try to keep his levels of body fat to a minimum. In general, the higher the percentage of body fat the poorer the performance. The examination of body fat and skinfolds at selected sites is most important for them. It has been found that the athletes who were lean or less fatty but heavy because of a well developed musculature were superior in performance in certain competitive sports. On the other hand the athletes who had substantial amount of adipose tissue have permanently increased energy demands owing to the inert weight of fat thus making the work more difficult to perform in such activities where the body has to be projected as in jumping movements, or propelled against gravity over long distances as in distance running. The science of body composition is an important morpho-physiological characteristic.

The proportion of these components is different in males and females. Its relative development is dependent on the environmental influences, sex, socioeconomic conditions, occupation, genetics, nutrition and exercise.

**Estimation of Body Composition**

The estimation of body composition has been attempted using various techniques. These are cadaver studies, fat cell size and number, skinfolds, body density, anthropometric methods, roentgenogrammetry, fat soluble gases, creatinine excretion, total body water, total body potassium, total body nitrogen, total body carbon, nuclear resonance scattering, ultrasound, computed tomography, nuclear magnetic resonance imaging, radioactive krypton, total body electrical conductivity, electrical impedance and infra-red interactance, etc.

For the estimation of body fat from the body density can be calculated with the help of equations derived by scientists of many method. Scientists have also devised different formulae for different populations, sexes and different age groups. The formulae used for the prediction of body fat are as follows:

a. **Pascale et al.(1956):**
   \[
   \text{Body density} = 1.088468 - 0.007123 \text{ Midaxillary} - 0.004834 \times \text{Juxta} \text{Nipple} - 0.005513 \text{ Triceps skinfold.}
   \]

b. **Sloan (1967):**
   \[
   \text{Body density} = 1.1043 - 0.001327 \text{ Thigh} - 0.001310 \text{ Subscapular}
   \]

c. **Chinn and Allen (1960):**
   \[
   \text{Body fat} = \left[ (0.00285 \times (\text{Subscapular} + \text{Triceps})/2 - 0.0114)^{1/2} - 0.061 \right] \times \text{Weight (kg) + 1.1 height}^3 \text{ meters} + 0.234 \text{ Age (yrs)} - 6.4\% \text{ fat} = \text{Body fat (kg)}/ \text{Body weight} \times 100
   \]

b. **Jackson and Pollock (1978):**
   \[
   \text{Density (kg/m}^3) = 1.10938 - 0.8267 \text{ (Chest + Abdominal + Thigh skf)} - 0.0016 \text{ (Chest + Abdominal + Thigh skf)}^2 - 0.2574 \text{ (Age)}
   \]

c. **Weltman and Katch (1978):**
   \[
   \text{Density (kg/m}^3) = [\text{Body Weight}/0.8719 \text{ Weight} + 0.2629 \text{ Thigh circumference - 7.795}] \times 103
   \]

f. **Lohman (1981):**
### A Textbook of Sports and Exercise Physiology

**Fig. 14.20:** Distribution of somatotypes according to the HWR (height/weight\(^{1/3}\))

<table>
<thead>
<tr>
<th>A</th>
<th>B</th>
</tr>
</thead>
<tbody>
<tr>
<td>15.40</td>
<td>119</td>
</tr>
<tr>
<td>15.20</td>
<td>118</td>
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A = Height in inches/cube root of weight in pound
B = Height in centimeter/cube root of mass in kilogram
Density (kg/m³) = 1098.2 − 0.815 (Triceps + Subscapular + Abdominal skinfold) + 0.0084 (Triceps + Subscapular + Suprailliac skinfold)²

g. Norgan and Ferro-Luzzi (1985):
Density (kg/m³) = 1145.5 − 59.69 (log sum of Thorax + Triceps skf) − 0.529 (Age).

h. Durnin and Womersley (1974):

Males:
(17 – 19 years): Body density = 1.1620 − 0.0630 log (Biceps + Triceps + Subscapular + Suprailliac skinfold)
(20 – 29 years): Body density = 1.1631 − 0.0632 log (Biceps + Triceps + Subscapular + Suprailliac skinfold)
(30 – 39 years): Body density = 1.1422 − 0.0544 log (Biceps + Triceps + Subscapular + Suprailliac skinfold)
(40 – 39 years): Body density = 1.1620 − 0.0700 log (Biceps + Triceps + Subscapular + Suprailliac skinfold)
(50 years and above): Body density = 1.1715 − 0.0779 log (Biceps + Triceps + Subscapular + Suprailliac skinfold)

Females:
(16 – 19 years): Body density = 1.1549 − 0.0678 log (Biceps + Triceps + Subscapular + Suprailliac skinfold)
(20 – 29 years): Body density = 1.1599 − 0.0717 log (Biceps + Triceps + Subscapular + Suprailliac skinfold)
(30 – 39 years): Body density = 1.1423 − 0.0632 log (Biceps + Triceps + Subscapular + Suprailliac skinfold)
(40 – 49 years): Body density = 1.333 − 0.0612 log (Biceps + Triceps + Subscapular + Suprailliac skinfold)
(50 years and above): Body density = 1.1339 − 0.0645 log (Biceps + Triceps + Subscapular + Suprailliac skinfold)

Body density calculated with the help of these formulae is converted to percent body fat by the formula devised by Brozek et al. (1963) or Siri (1956) respectively.

\[
\text{Percent body fat} = \left(\frac{4.57}{\text{Body density}} \right) - 4.142 \times 100
\]

\[
\text{Percent body fat} = \left(\frac{495}{\text{Body density}} \right) - 450
\]

**Body Fat Analyzing—Comparing Methods for Measuring Body Fat**

Most of the people now understand that to have a healthier body means to have a leaner body. These people are ready for Body Composition Analysis (BCA.) This text will attempt to acquaint you with the most popular methods of BCA used in laboratories, gyms, homes and professional practices.

**Hydrodensitometry Weighing (Underwater Weighing)**

This method measures whole body density by determining body volume. There is a variety of equipment available to do underwater weighing ranging in sophistication from the standard stainless steel/glass tank with a chair or cot mounted on underwater scales, to a chair and scale suspended from a diving board over a pool or hot tub.

This technique first requires weighing a person outside the tank, then immersing them totally in water and weighing them again. The densities of bone and muscles are higher than water, and fat is less dense than water. So a person with more bone and muscle will weigh more in water than a person with less bone and muscle, meaning they have a higher body density and lower percentage of body fat. The volume of the body is calculated and the individual’s body density is determined by using standard formulas. Then body fat percentage is calculated from body density using standard equations (either Siri or Brozek).

The underlying assumption with this method is that densities of fat mass and fat-free mass are constant. However, underwater weighing may not be the appropriate gold standard for everyone. For example, athletes tend to have denser bones and muscles than non-athletes, which may lead to an underestimation of body fat percentage. While the body fat of elderly patients suffering from osteoporosis may be overestimated. To date, specific equations have not been developed to accommodate these different population groups.
An important consideration in this method is the amount of air left in a person’s lungs after breathing out. This residual lung volume can be estimated or measured, but it is established that a direct measure is desirable and it should be taken in the tank whenever possible. Another consideration is that the water in the tank must be completely still; there can be no wind or movement.

Although this method has long been considered the laboratory “gold standard”, many people find it difficult, cumbersome, and uncomfortable, and others are afraid of total submersion or cannot expel all the air in their lungs. Clinical studies often require subjects to be measured three to five times and an average taken of the results.

**Calipers (Anthropometry- Skinfold Measurements)**

Using hand-held calipers that exert a standard pressure, the skinfold thickness is measured at various body locations (3–7 test sites are common). Then a calculation is used to derive a body fat percentage based on the sum of the measurements. Different prediction equations are needed for children and specific ethnic groups. This approach usually uses underwater weighing as a reference method. The caliper method is based upon the assumption that the thickness of the subcutaneous fat (found just under the skin) reflects a constant proportion of the total body fat (contained in the body cavities) and that the sites selected for measurement represent the average thickness of the subcutaneous fat.

Skinfold measurement is made by grasping the skin and underlying tissue, shaking it to exclude any muscle and pinching it between the jaws of the caliper. Duplicate readings are often made at each site to improve the accuracy and reproducibility of the measurements. Often to save time in large population studies, a single skinfold site measurement is also made to reduce the time involved. Such a test should be used only for a rough estimate of obesity.

Generally skinfold measurements are easy to do, inexpensive and the method is portable. Overall, results can be very subjective as precision ultimately depends on the skill of the technician and the site measured. The quality of the calipers is also a factor; they should be accurately calibrated and have a constant specified pressure. Inexpensive models sold for home use are usually less accurate than those used by an accredited caliper technician. The more obese the subject, the more difficult to
“pinch” the skinfold correctly, requiring even more skill to obtain an accurate measurement.

**DEXA (Dual Energy X-ray Absorptiometry)**

A relatively new technology that is very accurate and precise, DEXA is based on a three-compartment model that divides the body into total body mineral, fat-free soft (lean) mass, and fat tissue mass. This technique is based on the assumption that bone mineral content is directly proportional to the amount of photon energy absorbed by the bone being studied.

DEXA uses a whole body scanner that has two low dose X-rays at different sources that read bone and soft tissue mass simultaneously. The sources are mounted beneath a table with a detector overhead. The scanner passes across a person’s reclining body with data collected at 0.5 cm intervals. A scan takes between 10 to 20 minutes. It is safe and noninvasive with little burden to the individual, although a person must lie still throughout the procedure.

DEXA is fast becoming the new “gold standard” because it provides a higher degree of precision in only one measurement and has the ability to show exactly where fat is distributed throughout the body. It is very reliable and its results extremely acceptable; in addition, the method is safe and presents little burden to the subject. Although this method is not as accurate in measuring the extremely obese and the cost of equipment is high, DEXA is quickly moving from the laboratory setting into clinical studies.

**NIR (Near Infrared Interactance)**

A fiber optic probe is connected to a digital analyzer that indirectly measures the tissue composition (fat and water) at various sites on the body. This method is based on studies that show optical densities are linearly related to subcutaneous and total body fat. The biceps is the most often used single site for estimating body fat using the NIR method. The NIR light penetrates the tissues and is reflected off the bone back to the detector. The NIR data is entered into a prediction equation with the person’s height, weight, frame size, and level of activity to estimate the percent body fat.

This method has become popular outside of the laboratory because it is simple, fast, noninvasive, and the equipment is relatively inexpensive. However, the amount of pressure applied to the fiber optic probe during measurement may affect the values of optical densities, and skin color and hydration level may be potential sources of error. To date, studies conducted with this method have produced mixed results; a high degree of error has occurred with very lean and very obese people; and the validity

![Fig. 14.23: Measurement of fat mass of whole body by DEXA method](image-url)
of a single-site measurement at the biceps is questionable. Numerous sources report that more research is needed to substantiate the validity, accuracy and applicability of this method.

**Magnetic Resonance Imaging (MRI)**

An x-ray based method in which a magnetic field “excites” water and fat molecules in the body, producing a measurable signal. A person lies within the magnet as a computer scans the body. High-quality images show the amount of fat and where it is distributed. MRI takes about 30 minutes and is very safe as it uses no ionizing radiation, but use is limited due to the high cost of equipment and analysis.

**Total Body Electrical Conductivity (TOBEC)**

This method is based on lean tissue being a better conductor of electricity than fat. A person lies in a cylinder that generates a very weak electromagnetic field. The strength of the field depends on the electrolytes found in the person’s body water. In about 10 seconds, TOBEC makes 10 conductivity readings that estimate lean body mass. Although very accurate, its use is limited due to the high cost of the equipment.

**Computed Tomography (CT)**

CT produces cross-sectional scans of the body. An X-ray tube sends a beam of photons toward a detector. As the beam rotates around a person, data is collected, stored, and applied to complex algorithms to build images that determine body composition. CT is particularly useful in giving a ratio of intra-abdominal fat to extra-abdominal fat. It is noninvasive, but potential is limited by exposure to radiation and high equipment cost.

**BOD POD (Air Displacement)**

Based on the same principle as underwater weighing, the BOD POD uses computerized sensors to measure how much air is displaced while a person sits for 20 seconds in a capsule. It uses a calculation to determine body density, then estimated body fat. The equipment is very expensive and limited in availability.

**BIA (Bioelectrical Impedance)**

The only method that is based on measuring something, not estimating anything, is Bio-Impedance measurement. Bio-Impedance is a means of measuring electrical signals as they pass through the fat, lean mass, and water in the body. Through laboratory research we know the actual impedance or conductivity of various tissues in the body, and we know that by measuring current between two electrodes and applying this information to complex proven scientific formulas accurate body composition can be determined. The fact that the measurement is based on a reading of lean mass and not an estimate of fat mass, lends to a much more comprehensive testing method and results.

**Ideal Body Fat Percentage**

The absolute perfect body fat percentage does not exist. Age and gender make a big contribution to the ideal value. Some people might feel and perform better at a higher or lower body fat percentage than others of the same age and sex. The following table consist of body fat percentage for the average population.

The first table gives the ideal body fat percentage ranges for the general population. The second table is the average body fat percentage for different athletes. Anywhere within the range is good. Staying below the upper limit should be your target but as you’ll soon see lower is not necessarily better.

**Weight Loss**

Weight loss is simple, burn more calories than one consume. If one can fully understand that then you are on your way to weight loss. There are four simple steps to weight loss. These are as follows:

1. Count how many calories you eat in a normal day. Don’t change anything, just eat like your normally do and count how many calories. Also, weigh yourself.
2. Starting the day after you counted calories, for example, eat 300 calories less then you normally do. So pretend the day you counted calories you counted 2500 Cal. For the rest
of the week, eat 2200 Cal. Instead of eating 3 big meals a day or eating all day all the time, spread those 2200 calories out over 5 small meals. Eat one every 2 and a half to 3 hours. Doing this will speed the metabolism.

3. Increase daily physical activity for weight loss. Jogging, walking, swimming, etc., i.e. the activity should be sub-maximal level (aerobic type) and should at least for 30 minutes a day, 5 days a week.

4. At the end of that week, weigh yourself. You’ll notice a difference just after one week. Anymore then ½ or 1 kilogram lost a week is unhealthy. So look for ½ or 1 kilogram loss at the end of the week. You can lose 2 to 4 kilogram a month! So if you have a lot of weight to lose, you can lose it.

Avoid more fat. Get rid of the chips, candy, fast food, fried, etc. Stay away from high carbohydrate foods which will eventually turn into fat. Foods like bread, rice, potatoes, are high in carbohydrates. However, can be eaten, but don’t eat bread and potatoes all the time. High protein and low carbohydrate/low fat foods like lean fish (and other seafood), chicken breast, fruits, and vegetables, etc. are suggested. Drink water around 2 to 3 liter a day, more if you can. This will maintain fluid balance in the body. Strength training increase muscle and fat loss. Muscles burn calories. Get at least 8 hours of sleep a night.

### How to Gain Weight

To gain weight, you must consume more calories then you burn. This is the reverse process as weight loss. There are four simple steps to weight gain. These are as follows:

1. Count how many calories you eat in a normal day. Don’t change anything, just eat like your normally do and count how many calories you ate. Also, weigh yourself.

2. Starting the day after you counted calories, for example eat 300 calories more then you normally do. So pretend the day you counted calories you counted 2500 Cal. For the rest of the week, eat 2800 Cal. Instead of eating 3 big meals a day or eating all day all the time, spread those 2800 calories out over 5 small meals. Eat one every 2 and a half to 3 hours.

3. Decrease physical activity. At the end of that week, weigh yourself. Anymore then ½ or 1 kilogram gained a week is unhealthy and means you’re putting on fat, not muscle. So look for ½ or 1 kilogram gains at the end of the week. In this way you can gain 2 or 3 kilogram a month.
4. At some point, you will stop seeing weight gains. At this point, you will have to eat even more. So, when you see weight gain stops for at least 2 weeks, eat an extra 200 calories a day. Every time you see you haven’t gained weight for at least 2 weeks, add an extra 200 calories.

Fat should be avoided as you don’t want to gain fat. High protein and low fat foods like lean fish (and other seafood), chicken breast, lean meats, fruits and vegetables, etc are suggested. Drink water around 2 liter a day, more if you can. Strength training increase muscle and fat loss. Muscles burn calories. Get at least 8 hours of sleep a night.

**Reversibility of Body Composition in Sportsmen**

The reversibility process starts operating immediately after the cessation of physical activity, no matter how long the athlete remained under strenuous work loads. It seems the adaptations of body composition in the organism are gradually wiped out with the passage of increased inactive phase of an athlete. The regular physical exercise in top-athletes is so essential that even in case of injury; they need quick relief under active conditions. The advantage in physiotherapy has made it possible. This is essential in order to avoid reversibility of the adaptive changes.

Otherwise an athlete following a program of training for a competition expected to reach his peak performance at the time of pre-fixed date is likely to be left behind by his competitors in other countries.

**Summary**

i. The Kinanthropometry is defined as the study of human size, shape, proportion, composition, maturation and gross function in order to help understand growth, exercise, performance and nutrition. Kinanthropometric investigations have been conducted on the Olympic athletes during the Olympics more than three decades. The different characteristics examined include investigations of their size and shape, using large number of variables by sports and events.

ii. Anthropometry means the measurement of man, whether living or dead and consists primarily in the measurement of the dimensions of the body. It is the measurement of man provides scientific methods and observations on the living man and the skeleton. The anthropometry has developed primarily in the sports fields by physical anthropologists. Physical anthropology deals
with the study of man’s biological defined behavior in time space.

iii. The use of proper equipment is most essential for anthropometric measurements. The equipments need high precision and accuracy. The most commonly used and available equipments are Anthropometric rod, Weighing machine, Stadiometer, Sitting height table, Somatotype turn table, Strength dynamometer (Hand grip & Back Dynamometer), Goniometer, Steel measuring tap, Sliding caliper, Chest caliper, Spreading caliper, Skinfold caliper, etc.

iv. The accurate marking of the landmark is more essential for more precise anthropometric measurements. However, the technique of taking measurement by using the instrument is equally important. The technique of measurement of various anthropometric parameters must be a standard one and producing accurate results, the investigator should be well trained and skillful. Before listing the measurements made to determine the various dimensions are necessary to understand the landmarks from which such measurements are conventionally made.

v. Somatotyping is a form of body classification, i.e. it is a shorthand method of describing an individual’s physique according to certain physical characteristics. Somatotyping was originally developed by an American psychologist - William Sheldon in 1940s, after he studied over 4,000 photographs of college age men. Sheldon divided physique into three basic categories or somatotypes, known as ‘extreme somatotypes’ by dividing body into five areas: such as (i) head, face and neck; (ii) thoracic trunk; (iii) arms, shoulders, heads; (iv) abdominal trunk; (v) legs and feet.

vi. A new improved somatotype method with universal application to both sexes, for all ages and which is reproducible was innovated after requisite, validation. This method is known as modified somatotype method and is designed by Barbara Honeyman Heath and JE Lindsey Carter in 1967. The new method retained the well accepted terminology of somatotype; endomorphy, mesomorphy and ectomorphy. Somatotype if studied in large population, a variety of somatotypes with varying strengths of different components will be observed. These somatotypes can be classified into 13 classifications.

vii. As somatotype is a three-number expression and meaningful analysis can be conducted only with special techniques. Somatotype data can be analyzed by both traditional and non-traditional descriptive and comparative statistical methods although descriptive statistics are used for each of the components, comparative statistics should be made in the first instance using the whole (or global) somatotype rating. All human being including athletes are made up of the three extreme body types so we are all part endomorph, part of mesomorph and part of ectomorph. By classifying our own body physiques using somatotyping, we can compare our body type with that of other athletes. Graphs and tables have been developed to decide which sports suit which somatotypes better.

viii. Body size refers to the person’s height and weight. The ideal size for an athlete depends on their sport and sometimes on the position they play in. Theoretically, the body can be divided into several compartments according to definable tissues. The two component model used commonly divides the body to a fat portion, and further divides the fat free mass to skeleton, muscle and the remainder. The fluids, etc can also be studied in terms of total body water, extra-cellular water, intra-cellular water, total body potassium, calcium, sodium and so on. However, the athletes have mostly been studied considering the body either as two or four compartment model.

ix. The estimation of body composition has been attempted using various techniques. These are cadaver studies, fat cell size and number, skinfolds, body density, anthropometric methods, roentgenogrammetry, fat soluble gases, creatinine excretion, total body water, total body potassium, total body nitrogen, total body carbon, nuclear resonance scattering, ultrasound, computed tomo-
graphy, nuclear magnetic resonance imaging, radioactive krypton, total body electrical conductivity, electrical impedance and infrared interactance, etc.

The absolute perfect body fat percentage does not exist. Age and gender make a big contribution to the ideal value. Some people might feel and perform better at a higher or lower body fat percentage than others of the same age and sex. Weight loss process is simple, burn more calories than one consume. If you can fully understand that then you are on your way to weight loss. To gain weight, you must consume more calories one usually you burn. This is the reverse process as weight loss.

**Review Questions**

1. Define Anthropometry. What do you mean by Kinanthropometry? Write in brief about the scope and development of anthropometry.

2. What are the different planes of human body? What do you mean by the F-H planes? Discuss Mid-Sagittal planes.

3. Name some important anthropometric equipment. Write the procedure of measurement of height by the stadiometer.

4. Write some important landmarks for anthropometric measurements and write the procedure of measuring (i) Biacromial diameter and (ii) sitting height of an individual.

5. Define Physique. What are the different components of somatotype? Write classification of somatotype with example.

6. Discuss Heath-Carter somatotype. What are the measurement techniques of anthropometric somatotype?

7. What do you mean by extreme somatotype? Discuss somatochart with suitable example. How can you plot a elite shot put thrower in somatochart?

8. What is somatopoint? Distinguish between somatotype attitudinal distance and somatotype attitudinal means.

9. Differentiate between body built, body size and body composition.

10. What is densitometry? How is it used to assess the body composition of the athlete? What is the major weakness of densitometry with respect to its accuracy?

11. What are several field techniques for estimating body composition? What are their strength and weaknesses?

12. What is the relationship of relative leanness and fatness to performance in sports?

13. What is more important to sports performance, body fat or body weight? Why?

14. What is the principal of weight loss and weight gain? Discuss with example.
The desire to take medicine to improve physical and mental health is perhaps the greatest feature which distinguishes man and animals. The word originated from a primitive South-East African alcoholic drink called ‘DOP’ that was used as stimulant in ceremonial dances. This word reached the English language via the Bores “Doping”, first appeared in an English Dictionary in 1889. Over the years, literally hundreds of ingredients have been used as “the magic potion”: Camphor, ephedrine, gelatin, iron, oxygen, aspartic acid, amphetamines, tranquilizers, vitamins, steroids and red blood cells. The current is to use varying mixtures of these substances, vitamins, and the male hormone testosterone. Polypharmacy (the use of multiple drugs) has reached its most absurd proportions among athletes.

**HISTORICAL PERSPECTIVES**

Since the dawn of recorded history, a great variety of drugs have been taken by athletes hoping to improve performance, and few—if any—have stood the time. Even in the first Olympic Games, held in Greece in 300 BC, doping methods were used, competitors sought to improve their performance by eating certain mushrooms.

The first documented case of illicit drug use in sports occurred in 1865 among the channel swimmers of Amsterdam. This spread rapidly, and by 1879 the six days bicycle racers of several nations were using caffeine, alcohol, nitroglycerine, ether dropped on sugar cubes, strychnine pills, cocaine and opium. According to an article in the Journal of American Veterinary Medical Association, the problem of doping was evident even in ancient
The Romans gave their horse’s hydromel, a mixture of honey and water that increased their endurance and speed during cart races. For hundreds of perhaps thousands of years, Indian tribes in Central America have chewed coca leaves to help them in long and arduous mountain journeys, a stimulant contained in the leaves increases endurance and decreases weariness. In modern times, especially in America and Western Europe, sports are so commercial that there is an exaggerated emphasis on winning. This social pressure forces many athletes and even some trainer to seek shortcuts to the extra degree of superior performance.

In 1920s and 1930s, doping was already a serious problem in sports such as horse racing and dog racing. But in human sports the doping has only become an important issue in 1960s. The first recorded death from the use of doping in sport was in 1886 when a cyclist died from an overdose of Trimethyl. In 1904 Olympics, marathon runner Thomas Hicks was using a mixture of brandy and strychnine and nearly died. Heroin, cocaine and caffeine were widely used until heroin and cocaine became available only on prescription. During the 1930s it was amphetamines that replaced strychnine. In the 1950s the Soviet Olympic team used male hormones to increase strength and power. The American’s developed steroids as a response.

In the 1952 Winter Olympics, several speed skaters became ill and needed medical attention after taking amphetamines. In 1960, during an Olympic road race, a young Danish cyclist collapsed and later died as a result of an excessive dose of amphetamine. During the 1970s anabolic steroids became the form of doping.

In 1988 Seoul Olympics, a Canadian sprinter Ben Johnson was stripped of his Olympic gold medal for taking stanozolol, a banned substance. ‘Doping contravenes the fundamental principle of Olympism, Sports and Medical ethics’.

**Definition**

A commission of experts convened by the Council of Europe in 1963 gave the following definition of doping: “Doping is defined as the administering or use substances in any form alien to the body, or of physiological substances in abnormal amounts and with abnormal methods, by health personnel with the exclusive aim of attaining an artificial and unfair increase of performance in competition.”

**Prohibited Classes of Substances and Prohibited Methods**

Adopted by the union Cycliste International (UCI) President on the proposal of the Anti-Doping Commission of the UCI.

**Doping Classes**

**Class-I**

(i) **Stimulants:** The physiologic changes that prepare a body for strenuous exercise are sometimes called the “fight or flight” response. These changes persist during exercise and are necessary for maximal performance. Blood flow through working muscles increases as do alertness and the focusing of attention. Activity of the sympathetic nervous system results in these changes, during which release of epinephrine into circulation by the adrenal glands, adrenal medulla plays a key role. Amphetamines constitute a class of central nervous stimulants that are related in structure to epinephrine. It soon became obvious that some people were taking the drugs in excessively high doses simply to derive a feeling of well-being (euphoria). This misuse led to addiction, and the drugs are thus now classified as narcotics in many countries. The only correct medical use of these drugs today is in the treatment of narcolepsy, an abnormally increased need for sleep.

The reason for the inclusion of stimulants in the doping register is that they conceal the feeling of exhaustion. Most athletes try to get themselves “psyched up” before an important contest. That is, they deliberately stimulate the sympathetic nervous system. The use of amphetamines in sports in which maximum force or aggression is desired; it is also used by jockeys and wrestlers for appetite suppression during attempts to lose weight. Amphetamine use is associated with increased time to exhaustion while cycling and with increased knee extension strength.
The four most notorious examples of stimulants that are used in sports are amphetamine, cocaine, ephedrine and caffeine etc.

(ii) **Cocaine**: It is an alkaloid related to caffeine and nicotine. It is used medically as a topical anesthetic and vasoconstrictor, particularly in the upper respiratory tract. It is used to increase endurance and promote a sense of well-being.

*Side effects*: Deleterious effects of cocaine include psycho-social problems, psychological dependency, and sometimes damage to the tissue through which the drug is absorbed, such as the nasal mucosa.

(iii) **Caffeine**: Caffeine is a central nervous system stimulant found in coffee, tea and many soft drinks as well as in tablets to combat drowsiness. It has been used by runners, cyclists, swimmers, and other athletes to diminish feelings of fatigue. Caffeine can improve work production by promoting metabolism of lipids during exercise, and thus sparing glycogen in muscle, the loss of which can limit exercise.

*Side effects*: The toxic effects of caffeine include restlessness, tremor and irritability. At very high doses seizures may occur.

(iv) **Nicotine**: It is an alkaloid used for its property as a mild stimulant of the central nervous system (CNS). It causes facilitation of attention but relaxation of skeletal muscle. Nicotine is vasoconstrictor and tends to decrease the perfusion of tissues by blood as well as elevate blood pressure and heart rate.

*Side effects*: Tobacco contact with the oral mucosa is associated with damage to the gums and increased cancer of the mouth. Smoking is associated with a decrease in maximal O₂ uptake capacity. It may impair performance by causing airway constriction. Smoking is habituating and is also associated with the cancer, heart disease and other health problems.

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### Banned substances

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Adrenergic Stimulants

The adrenergic stimulants (sympathetic amines, also known as alpha or beta_2_ stimulants)— ephedrine and ephedrine derivatives are related chemically to the body’s own ‘stress hormones’, adrenaline and nor-adrenaline. High doses of ephedrine given intravenously will give an amphetamine like effect. The chief medical use of these substances is in the treatment of asthma and nasal congestion. Ephedrine is an ingredient of many cough medicines. The banning of ephedrine and phenylpropanolamine and related compounds has caused considerable problems in how to interpret the IOC regulations. In pharmacological terms, all alpha stimulants can be considered as ‘related compounds’. This means that all nasal decongestants are banned, although all nasal decongestant applied locally are permitted.

**Side effects:** All preparations containing ephedrine and its derivatives can cause troublesome palpitations, agitation, tremors, insomnia and effects on the blood pressure even at normal dose. Large doses of the preparation may even result in damage to the heart muscle.

**Other potential side effects are:**
- Loss of appetite
- Loss of sleep (insomnia)
- Increased body temperature (hyperthermia)
- Euphoria
- Hallucination (psychosis)
- Trembling
- Restlessness, agitation and tenseness
- High blood pressure (hypertension)
- Palpitations and heart rhythm disorders
- Diminished coordination, judgmental ability and self-criticism.

Narcotic Analgesics

Morphine like analgesics is the most effective of all analgesics, and is indispensable to the practice of medicine. In addition, they are powerful anxiolytics and sedatives. They are derived from opium, which in turn is derived from the poppy plant. They act on CNS and reduce feelings of pain. Narcotic analgesics may be used to mask the sensation of pain. Besides reducing pain, narcotic analgesics are used to depress coughs. Their use, however, is associated with the potent euphoric properties of the drugs. The euphoria produced by morphine and morphine like preparation differs from that evoked by cocaine or amphetamine in that the individual withdraws into his or her own dreamworld and becomes drowsy and inactive. This state is hardly conducive to heightened performance.

The doping effects of morphine like analgesics are thought to modify fear in marked nervousness and anxiety states and to have analgesic effect on injuries.

**Side effects:** The greatest risk associated with the use of morphine like analgesics is the risk of addiction. The drugs are classified as string narcotics. Combined with alcohol, even small doses of the weakest of the preparations may result in respiratory arrest and death. The use of narcotics induces a false sense of security that may cause an athlete to ignore a potentially serious injury, risking further damage.

**Other potential dangerous side effects are:**
- Addiction
- Loss of balance and coordination
- Nausea and vomiting
- Loss of sleep and depression
- Breathing become slower
- Decreased heart rate
- Decreased ability to concentrate.

Anabolic Agents

Adrenergic anabolic steroids:

Anabolic adrenergic steroids are man-made substances related to male sex hormones. Anabolic “refers to muscle building”, and “adrenergic” refers to increased masculine characteristics. “Steroids” refers to the class of drugs. These drugs are available legally only by prescription, to treat conditions that occur when the body produces abnormally low amounts of testosterone, such as delayed puberty and some types of impotence. They are also used to treat body wasting in patients with AIDS and other diseases that result in loss of lean muscle mass. Abuse of anabolic steroids, however, can lead to serious health problems, some are irreversible.

Athletes and body builders abuse anabolic steroids to enhance performance and improve physical
appearance. Anabolic steroids are taken orally or injected, typically in cycles of weeks or months (referred to as “cycling”), rather than continuously. Cycling involves taking multiple doses of steroids over a specific period of time, stopping for a period, and starting again. In addition, users often combine several different types of steroids to maximize their effectiveness while minimizing negative effects (referred to as “stacking”). Higher doses also have psychological effects. Studies have shown that the intake of large doses of anabolic steroids combined with intensive training increases muscle strength.

The effects of anabolic steroids on women are relatively clear. Muscle mass and strength appear to increase when these agents are used in conjunction with strength training. This means that, with large doses of anabolic steroids, a woman’s athletic performance could approach that of a man.

**Side Effects**

The major side effects from abusing anabolic steroids can improve liver tumors and cancers, jaundice (yellowish pigmentation of skin, tissues and body fluids), fluid retention, and high blood pressure; in addition there are some gender specific side effects:

i. **For Men**: Shrinking of the testicles, reduced sperm count, infertility, baldness, development of breast, and increased risk of prostate cancer.

ii. **For Women**: Growth of facial hair, male patterned baldness, changes in or cessation of the menstrual cycle, enlargement of the clitoris, deepened voice.

iii. **For Adolescents**: Growth halted prematurely through premature skeletal maturation and accelerated puberty changes. This means that adolescents risk remaining short the remainder of their lives if they take anabolic steroids before the typical adolescent growth spurts.

Research has also shown that aggression and other psychiatric side effects may result from abuse of anabolic steroids. Many users report feeling good about themselves while on anabolic steroids, but extreme swings also can occur, including maniac-like symptoms leading to violence, leading to the coining of the phrase “Road rage”. Steroids commonly abused by athletes include nandrolone, oxandrolone and stanozolol, the structures of nandrolone and dextosterone, and its natural counterparts of the common steroids.

**Other Anabolic Agents**

Other anabolic agents are substances which are pharmacologically not related to the androgenic anabolic steroids, but which might have a similar anabolic effect. This doping class has been established because of the abuse of the beta-2 agonist clenbuterol. In veterinary medicine clenbuterol is used to treat calves for colds and coughs, beta-2 agonists or beta-2 adreno-receptor agonist actually belong to the class of stimulants and have been developed for the treatment of chronic bronchitis, and emphysema and asthma.

People discovered that while using clenbuterol in large quantities with calves the percentage of fat was reduced and the muscular tissue increased. It is assumed that athletes also started to use clenbuterol on the basis of these findings. Scientifically the alleged anabolic effects on humans are dubious.

**Side Effects**

- Trembling
- Restlessness, agitation
- Feeling of anxiety
- Heart rhythm abnormalities (arrhythmias)
- Muscle cramps

Not only clenbuterol has been banned, but also all other substances that belong to the class of beta-2 agonist are also banned. Most sports governing bodies made an exception for salbutamol and terbutaline when administered by inhalation for respiratory congestion.

**Diuretics**

Diuretics are drugs which increase the amount of fluid excreted by the kidneys. They do this by inhibiting the re-absorption of water in the kidney.

Diuretics are used by sportsmen and women to decrease body weight, to increase power to weight ratios and to help qualify for certain weight categories, e.g. boxing and judo. By taking diuretics,
the athletes run the risk of severe dehydration and even gout. Other side effects include potassium deficiency, leading to muscle cramps, if a potassium sparing diuretic isn’t used.

**Side Effects**
- Dehydration
- Decreased circulation of blood volume (hypovolemia)
- Muscle cramps
- Renal disorders
- Dizziness when standing up (orthostatic hypotension)
- Heart rhythm abnormalities (arrhythmias)

**Important Note**
The use of diuretics in sports is forbidden. Non medical use of diuretics can be very dangerous. Because of dehydration one will not be able to perform at his best. Losing too much water may cause heart and kidney failure which could lead to death.

**Peptide Hormones and Analogues**
Since 1989 the IOC has classified the Human chorionic gonadotropins (hCG), Adrenocorticotropicin (ACTH), and Human growth hormone (hGH) as doping agents. Peptide and glycoprotein hormones are natural substances that act as messengers within the body and cause the production of other endogenous hormones like testosterone and corticosteroids. Human Chorionic Gonadotropins can increase the production of testosterone in male subjects, that is why it is banned. ACTH has been misused to considerably increase the blood level of endogenous corticosteroids and as corticosteroids injected either intravenously or intramuscularly have been banned, it was only logical to add ACTH to the list of forbidden substances. Human Chorionic Gonadotropins has been used to bring about similar effects to anabolic steroids, and its misuse was considered both unethical and unsafe due to several dangerous side effects.

**Human Chorionic Gonadotropin (hCG)**
This hormone increases the production of endogenous steroids and the effect is similar to using testosterone. Human Chorionic Gonadotropins may be used because it seems to increase muscle size and strength when the individual taking it is experienced in strength training, is performing strenuous strength training concurrently and is well nourished.

**Side Effects**
- Breast development—gynecomastia (in males)
- Menstrual disorders (in females)

**Human Growth Hormone (hGH)**
Human growth hormone (hGH) is a hormone that is synthesized and secreted by cells in the anterior pituitary gland located at the base of the brain. Human Chorionic Gonadotropins is known to act on many aspects of cellular metabolism and is also necessary for skeletal growth in humans. The major role of hGH in body growth is to stimulate the liver and other tissues to secrete insulin like growth factor (IGF-1). IGF-1 stimulates production of cartilage cells, resulting in bone growth and also plays a key role in muscle and organ growth. hGH is prohibited both in- and out-of-competition under the World Anti-Doping Agency’s (WADA) list of Prohibited Substances and Methods.

**Side effects:** Commonly reported side effects for hGH abuse are: diabetes in prone individuals; worsening of cardiovascular diseases; muscle, joint and bone pain; hypertension and cardiac deficiency; abnormal growth of organs; accelerated osteoarthritis. In untreated acromegalic individuals (over-production of hGH), many of the symptoms described above are observed and life expectancy is known to be significantly reduced. Because of the role that hGH plays in stimulating IGF-1 secretion, excessive use of hGH may also lead to metabolic dysfunction, including glucose intolerance and other side effects associated with excess levels of IGF-1.

A test for hGH was first introduced at the 2004 Summer Olympic Games in Athens, Greece. The test to detect hGH abuse is a blood test. The current test is reliable. Another test, in its final development stage, will be combined with the current test to further enhance the detection window for hGH abuse. The concepts and development of both hGH tests have been systematically reviewed by
international independent experts in such fields as hGH, endocrinology, immunoassay, analytical chemistry, etc. In addition, these tests are the outcome of nearly US$6 million in research over the course of more than 10 years. Research was initiated by the International Olympic Committee (IOC) and the European Union, and then taken over by WADA when it was created and had adopted scientific research as one of its priority activities.

The current test is based on the blood matrix and was initially implemented on a limited scale to a number of the WADA-accredited Anti-Doping laboratories worldwide. The antibodies used for the current tests were initially produced in a research environment. The production of antibodies in a research environment is characteristically small. Industrial production of the antibodies was the following step for the widespread implementation of the hGH test.

Efforts made by WADA for widespread production of antibodies needed for hGH detection were slowed following the take-over if the company with which WADA had an agreement for the development of these antibodies, and the decision made by the company’s new management in 2006 to stop its cooperation with WADA. WADA subsequently found a new partner for the large-scale production and distribution of antibodies kits. The kits have now been validated, and mass production of the kits has started.

The test was introduced at the Athens Olympic Games in 2004 and other major sport events. However, because hGH is often taken by doping athletes in the off-season to optimize performance, the test is most effective when implemented in a no-advance-notice out-of-competition strategy. Widespread implementation of the test may change these statistics.

According to the majority of international experts, the blood matrix is the most suitable matrix for the detection of hGH. hGH in urine is found in extremely small quantities (less than 1% than that found in blood). WADA is collaborating with research teams to explore the development of urine-based detection methods for hGH.

Adreno-corticotrophic Hormone (ACTH)

This hormone increases the level of endogenous corticosteroids. ACTH may be used to repair damaged tissues and muscles. If used for long periods of time it would cause muscle wasting.

Side Effects

- Sleeping problems—insomnia
- High blood pressure—hypertension
- Diabetes mellitus
- Stomach ulcer
- Poor healing of wounds
- Loss of bone mass—osteoporosis

Erythropoietin (EPO)

Erythropoietin is a peptide hormone that is produced naturally by the human body. EPO is released from the kidneys and acts on the bone marrow to stimulate red blood cell production. An increase in red blood cells improves the amount of oxygen that the blood can carry to the body’s muscles. It may also increase the body’s capacity to buffer lactic acid.

Normal levels of EPO are 0 to 19 (some say up to 24 milli units per milliliter). Higher levels might indicate that an athlete has been abusing EPO for a competitive advantage. EPO has been misused as a performance enhancing drug in aerobic sports such as cycling (in the Tour de France), long distance running, speed skating, and Nordic (cross country) skiing etc. when misused in such situations, EPO is thought to be especially dangerous (perhaps because dehydration due to vigorous exercise can further increase the thickness (viscosity) of the blood, raising the risk for heart attacks and strokes. EPO has been banned by the Tour, the Olympics, and other sports organizations.

Side Effects

While proper use of EPO has an enormous therapeutic benefit in the treatment of anemia related to cancer or kidney disease, its misuse can lead to serious health risks for athletes who use this substance simply to gain a competitive edge. It is well known that EPO, by thickening the blood, leads to an increased risk of several deadly diseases, such as heart disease, stroke, and cerebral or pulmonary embolism. The misuse of recombinant human EPO may also lead to autoimmune diseases with serious health consequences.
Implementation of Test to Detect EPO

A test for EPO was introduced at the 2000 Summer Olympic Games in Sydney (Australia). The test, validated by the International Olympic Committee (IOC), was based on the blood and urine matrix. A blood screening was performed first, and a urine test was then used to confirm possible use of EPO. In June 2003, WADA’s Executive Committee accepted the results of an independent report stating that urine tests alone can be used to detect the presence of recombinant EPO. This report, requested by WADA’s stakeholders and commissioned by the Agency to evaluate the validity of urinary and blood tests for detecting the presence of recombinant EPO, concluded that urinary testing is the only scientifically validated method for direct detection of recombinant EPO. This report also recommended that urine testing be used in conjunction with blood screening for a variety of reasons, including the cost savings of performing blood screening prior to testing urine. Some international sports federations still use both urine and blood matrix for the detection of EPO.

Beta Blockers

Beta blockers act to reduce tremors and palpitation and have now been classified as doping drugs. These drugs are widely used in the treatment of patients with angina pectoris, systemic hypertension, arrhythmias, and also in migraine. Beta blockers are mainly used to steady and slow the heart rate.

These are not depressants but sometimes are used to blunt the “stage fright” or flight or fight response associated with sports especially like archery, shooting, modern pentathlon, diving etc. might benefit from using it. Beta blockers can be used to control anxiety. Beta blockers can cause a decreased in maximal cardiac output and work capacity and therefore are contraindicated when maximal work is required.

Side Effects

Exercise performance is adversely affected by the use of beta blockers, as the perception about exertion is increased and reduction in endurance time during progressive exercise. Furthermore, they have adverse effects like:

- Low blood pressure (hypotension)
- Slow heart rate (bradycardia)
- Cardiac failure
- Impaired circulation
- Impotence

Important Note

The ban has created many problems, particularly in shooting, as even top class participant in these sports may be in their late middle age and may suffer from high blood pressure or symptoms due to arteriosclerosis of the coronary arteries (angina pectoris).

Prohibited Method—Class-II

Blood Doping

Blood doping is the misuse of certain techniques and/or substances to increase one’s red blood cell mass, which allows the body to transport more oxygen to muscles and therefore increase stamina/endurance and performance.

There are three widely known substances or methods used for blood doping: erythropoietin (EPO), synthetic oxygen carriers, and blood transfusions. Each is prohibited under the World Anti-Doping Agency’s (WADA) List of Prohibited Substances and Methods.

Blood doping is based on physiological principles. In sports that require muscles to work over an extended period of time, the oxygen carried to muscle by blood is an important—and possibly limiting—factor. Several studies have shown that people who breathed oxygen while exercising could maintain high work levels for longer time than while breathing air. However, this is not possible during competitions. Another way of improving the amount of oxygen carried by blood is to increase the hemoglobin concentration. Clinical procedure is done as follows: Approximately one liter of blood is drawing from the athlete and deep frozen. In response to this blood loss, athlete’s blood forming organs increase their production of red blood cells. After 3 to 4 weeks the amount of blood and the hemoglobin level return to normal. A day or so before to competition/exercise tests, the drawn blood is re-infused into the athlete,
Doping

thereby increasing the hemoglobin concentration. Several controlled studies found that aerobic performance was increased by this method.

Blood doping procedure is more complicated and usually requires the support of people with medical knowledge. There are many practical problems, such as storing of blood, and then transportation to the competition site, how to arrange transfusion and so on.

Side Effects
- Blood clots which cause strokes (embolism)
- Life threatening hypersensitivity reaction (anaphylactic shock) and other transfusion reactions.
- Infections (hepatitis, AIDS)
- Allergic reactions (fever, rash, etc.)

Pharmacological, Chemical and Physical Manipulation
Pharmacological, chemical and physical manipulation consists of the use of substances and/or methods which may alter the integrity and/or validity of urine samples obtained in doping controls. In this class, catheterization, urine substitution and/or tampering, probenecid, epi-testosterone application and related compounds are banned. Probenecid inhibits the transport of organic acids across some tissue barriers. This is most important in the kidney, where many drugs and drug metabolites are excreted. The effect of probenecid is twofold. It increases the blood level of any drug, which gives a more pronounced effect with the usual dosage (or the usual effect with a lower dosage). Secondly, probenecid reduces the amount of trace substances (for example, anabolic steroid) released in the urine, thus making such substances much more difficult to detect in a urine sample.

Side Effects
All the methods and substances stated above have harmful side effects such as:

Probenecid
- Headache
- Intestinal problems
- Dizziness and hot flushes
- Kidney stones

Catheterization
- Cystitis (bladder infection)
- Inflammation of lower urinary tract.

Gene Doping
Gene of cell doping is defined as the non-therapeutic use of genes, genetic elements and/or cell that have the capacity of enhance athletic performance.

Classes of Prohibited Substances Subject to Certain Conditions—Class-III

Alcohol
Alcohol has two entirely different effects on the brain. One is an anxiolytics (anxiety relieving) or tranquilizing effect, the other a stimulatory effect. There were reports of cyclists in 24 hours races drinking rum and champagne throughout the race, marathon runners and walkers consuming considerable quantities of cognac or beer during competition. Supposedly alcohol had a refreshing effect and the ability to restore the athlete’s strength.

Anxiolytics Effect
The anxiolytics, or tranquilizing, effects of alcohol are often exploited by people who tend to worry unnecessarily under normal circumstances and who become even more anxious when faced with demanding situations, for example public appearances. The relief offered in these situations by the anxiolytic effects of alcohol may lead to habitual drinking, with an increasing need for larger quantities of alcohol. In a relatively short time, these people are unable to cope without alcohol.

Stimulatory Effect
Research has shown that alcohol stimulates certain brain functions. It helps a person to relax and promotes a feeling of well-being (euphoria). Alcohols are a group of toxic chemicals; the most common is ethanol or ethyl alcohol obtained by
the fermentation of sugar. Alcohol consumed in small quantities reduces trembling, improve self-confidence and relaxes.

**Side Effects**

Depending on the amount, alcohol can have many adverse effects such as:
- Long-term use of alcohol leads to addiction
- Alcohol decreases balance, reaction time, hand-eye coordination
- Alcohol induces violent and aggressive behavior
- Excessive long-term use causes liver problems

**Marijuana**

Marijuana and Hashish come from the complex Indian Hemp Plant Cannabis Sativa. Over 400 compounds have been isolated from this plant, of which 61 have been identified as cannabinoids. Marijuana is a relaxant and impairs judgment. In high doses, it causes hallucinations. Marijuana is the general term of crude preparations of the dried leaves, seeds, stems and sometimes flowers of the cannabis plant.

**Side Effects**

There can be no doubt that the immediate and long-term effect of cannabis use are detrimental to sporting performance due to effect on the heart, lungs and CNS. Furthermore, marijuana can have an effect on the reproductive system.

**Heart**
- Increased heart rate
- Increased blood pressure

**Lungs**
- Inflammation of lung tissue and cancer

**Central Nervous System**
- Impaired balance and coordination
- Loss of memory (short-term)
- Loss of concentration
- Hallucination (psychosis)
- Abnormal body temperature

**Reproductive System**
- Decreased sperm count and motility
- Disturbances with ovulation (in female).

**Important Notes**

Marijuana is prohibited in some sports. However, tests may be carried out at the request of an international sports governing body.

**Local Anesthetics**

Local anesthetics are used to block the sensation of pain from any injured part of the body. The use of local anesthetics induce a risk of concealing the severity of injury or disease, therefore, aggravating the problem. Administration of local anesthetics aims to make parts of the body insensitive to pain but to leave the central nervous system functions unaffected.

**Side Effects**

The use of local anesthetics may aggravate an injury. In case of hypersensitive or an overdose the following may happen:
- Fear
- Confusion
- Disturbed speech
- Visual and hearing problem
- Muscle spasm in the face
  - In case of accidental intravascular injection the following may happen:
    - Tremors
    - Muscle cramps
    - Convulsions
    - Heart failure
    - Apnea because of respiratory depression.

**Important Notes**

The use of local anesthetics is only permitted in the following conditions:
- It must be justified on medical grounds,
- Its application must be local,
- In case injection, it must be local, e.g. intra-articular,
iv. The use of cocaine is prohibited: a different anesthetic must be used, such as procaine, xylocaine, carbocaine, bupivacaine, liodocane, mepivacaine and related substances. Justification on medical grounds and the method of application must be proved by the rider in the form of a medical certificate; for riders of Trade Teams I and II, only the registration on the health record booklet will be taken into consideration.

Apart from the conditions cited above, the use of local anesthetics is prohibited. Thus systemic use and for example, intramuscular injections are prohibited.

**Corticosteroids**

The corticosteroids are mainly used as anti-inflammatory drugs. They influence the concentrations of natural corticosteroids hormones released in the body from adrenal cortex. The therapeutic use of corticosteroids is to treat inflammation, asthma and pain. In high doses they also produce euphoria. In sports, they carry the risk of impaired healing of wounds and fractures. The IOC medical commission has therefore, banned the use of corticosteroids except for topical use (aural, ophthalmologic, and dermatological), inhalation therapy in asthma and allergic rhinitis, and local or intra-articular injections.

**Side Effects**

- Loss of sleep
- High blood pressure
- Poor healings of wounds
- Heartburn
- Diabetes mellitus
- Loss of bone mass

**Important Notes**

a. If the application is local:
   i. It must be justified on medical grounds.
   ii. Justification on medical grounds and the method of application must be proved by the rider in the form of a medical certificate; for riders of Trade Teams I and II, only the registration on the health record booklet will be taken into consideration.

b. If the application is systemic:
   i. It must be justified on medical ground.
   ii. A document must be presented to the UCI Anti-Doping Commission.
   iii. The consent of the Anti-Doping Commission must be obtained; except if the medical urgency is incompatible with the consent, this written consent must be obtained before the application.
   iv. The application must be registered in the health record booklet.
   v. The above procedure must be followed in case of treatment renewal except for the conditions cited above and the use of Glucocorticosteroids is prohibited.

**LIST OF DOPING SUBSTANCES**

Adopted by the UCI President on the proposal of the Anti-Doping Commission of the UCI Table: 15.1.

**PROHIBITED CLASSES OF SUBSTANCES**

**Stimulants**
- Amfepramone ethamivan nikhetamide
- Amineptine ethylamphetamine orciprenaline
- Amiphenazole etilefrine pemoline
- Amphetamine fencamfamine pentetrazol
- Amphetaminil fenethylline phendimetrazine
- Bambuterol fenoterol phenmetrazine
- Benzhetamine fenproporex phentermine
- Bromatane furfenorex pipadrl
- Carphedone hepatimol procaterol
- Chlorphentermine isoprenaline prolintane
- Clobenzorex mefenorex propylhexedrine
- Clorpeenaline mesocarbe pyrovalerone
- Cocaine metaraminol reprotoerol
- Cropropamide (constituent of “Micorene”) methoxyphenamine selegiline
- Crothetamide (constituent of “Micorene”) methylamphetamine strychnine (nux vbmica)
- Dimethylamphetamine methylphenidate
- Etaphedrine morazone and related substances as well as -

**Caffeine:** For caffeine, a sample shall be considered as positive if the concentration in the urine exceeds 12 micrograms/ml.
Cathine: (normephrine): For cathine (normephrine), a sample shall be considered as positive if the concentration in the urine exceeds 5 micrograms/ml.

Ephedrine

Methyl ephedrine: For ephedrine and methyl ephedrine, a sample shall be considered as positive if the concentration in the urine exceeds 10 micrograms/ml.

Phenylpropanolamine and Pseudoephedrine: For Phenylpropanolamine and Pseudoephedrine a sample shall be considered as positive if the concentration in the urine exceeds 25 micrograms/ml.

Formoterol
Salbutamol
Salmeterol
Terbutaline

Formoterol, salbutamol, salmeterol and terbutaline are permitted in the form of aerosols for the purpose of preventing and/or treating asthma and stress related asthma. Asthma and/or stress related asthma must be confirmed by a lung specialist or a team doctor. The rider must produce a medical certificate and a prescription issued by a lung specialist or a team doctor during the drug tests (for riders of Trade Teams I and II only the registration of the health record booklet will be taken into consideration). If he does not comply with this, and the laboratory finds traces of one of those substances, he will be considered positive. For salbutamol a concentration of less than 100 nanograms per milliliter will not be taken into consideration.

Narcotic Analgesics

Examples:

i. Alphaprodine methadone
ii. Anileridine morphine
iii. Buprenorphine nalbuphine
iv. Dextromoramide pentazocine
v. Diamorphine (heroin) pethidine
vi. Dipipanone phenazocine
vii. Ethoheptazine trimeperidine
viii. Levorphanol and related substances
ix. Morphine
x. Pentazocine

For morphine, a sample shall be deemed positive if the concentration in the urine exceeds 1 microgram/ml.

Note: Codeine, dextromethorphan, dextropropoxyphene, dihydrocodeine, diphenoxylate, ethylmorphine, propoxyphene, tramadol and pholcodine are permitted.

Anabolic Agents

Androgenic Anabolic Steroids

Examples:

i. Androstenediol methandienone
ii. Androstenedione methyl testosterone
iii. Bolasterone nandrolone
iv. Boldenone 19-norandrostenediol
v. Clostebol 19-norandrostenedione
vi. Dehydrochloromethyltestosterone norethandrolone
vii. Dehydroepiandrosterone oxandrolone
viii. Dehydrotestosterone oxymesterone
ix. Fluoxymesterone oxymetholone
x. Gestrinone stanozolol
xi. Mesterolone testosterone
xii. Methemoneolate and related substances

The Anti-Doping Commission can have any sample or part of a sample analyzed by a Gas-Chromatography/Combustion/Isotope Ratio Mass Spectrometry (GC/IRMS) in any laboratory which has this apparatus. It will inform the rider about this; if this analysis shows an exogenous application, the rider will be considered positive. In other cases, the Anti-Doping Commission can ask for further tests. If the rider refuses to undergo these tests, he will be considered positive.

For dehydrotestosterone, a sample will be considered positive if the concentration of dehydrotestosterone and its metabolites and/or their ratio of non-5 alpha steroid exceed the normal level of a human being in such a way that they cannot be attributed to endogenous production.

For nandrolone and its derivatives, a sample will be considered positive if the nandrosterone concentration found in the urine after hydrolysis exceeds 5 ng/ml. If the concentration is in between 2 and 5 ng/ml or is equal to 5 ng/ml, Anti-Doping Commission can request further analysis. If the rider
refuses to undergo the tests, he will be considered as positive.

For analysis other than those performed with a GC/C/IRMS apparatus, a sample will be considered positive for testosterone and if the level of testosterone/epitesterone is higher than 6 inicrogram. Nevertheless, the rider can request an endocrinological examination to determine if the level is due to a physiological or pathological state. The request for an endocrinological examination must be made to the UCI. Anti-Doping Commission at the latest 5 working days after receipt of the registered letter to the rider’s national federation informing it of the positive result, i.e. within the deadline for the request of a counter analysis (article 64 AER). The Anti-Doping Commission will decide which laboratory and date will be chosen for the analysis. The cost of the analysis must be paid in advance by the rider. The date of the analysis can not be postponed. If the result of the endocrinological examination show that the level of T/E is due to a physiological or pathological state, the UCI will refund to rider the cost of the examination must be paid by the rider. If the rider requests a counter-analysis, he must do so within the deadline stipulated in article 64 AER, even if he requests an endocrinological examination. If the counter analysis does not confirm a positive result, the rider will not be considered positive and the endocrinological examination will not be conducted.

Instead of an endocrinological examination, the rider can request, within the same deadline, that the Anti-Doping Commission carries out a retrospective analysis of previous results, which should be addressed to the commission within 3 working days of the request. If the Anti-Doping Commission judges that the request is not valid it may oblige the rider to have an endocrinological examination. If the rider refuses, he is considered positive. If the circumstances justify it, the Anti-Doping Commission may immediately propose a retrospective analysis.

The Anti-Doping Commission may also demand a long-term examination consisting of random tests conducted during a period which it will determine.

Nonsteroidal Anabolic Agents

Examples:
  i. Bambuterol salbutamol
  ii. Clenbuterol salmeterol
  iii. Fenoterol terbutaline
  iv. Formoterol zeranol
  v. Reproterol and related substances

Except with a therapeutic justification for use by inhaling as described above, for use by inhaling as described under point I.A.1; for salbutamol a sample shall be considered as positive in the class of anabolic agents if the concentration in the urine exceeds 1000 nanograms per milliliter.

Diuretics

Examples:
  i. Acetazolamide mannitol
  ii. Ethacrynic acid mersalyl
  iii. Bumetanide spironolactone
  iv. Chlorthalidone triamterene
  v. Furosemide
  vi. Hydrochlorothiazide

Hydrochlorothiazide and related substances-1 prohibited by intravenous injection

Peptide Hormones, Mimetic and Analogues

1. Chorionic Gonadotrophin (hCG- Human chorionic gonadotrophin – prohibited in males only): It is well known that the administration of human chorionic gonadotrophin or other related compounds leads to an increase in the production of natural androgenic steroids and is considered equivalent to the exogenous administration of testosterone. A sample would be considered positive if the concentration is above 20 ImU/ml. During the analysis, two different immunoassay methods must be used in this case.

2. Pituitary and Synthetic Gonadotrophin (LH- prohibited in males only): Pituitary hormones are secreted from the pituitary gland (such as LH) which stimulate the testes and ovary. In males it act as hCG and stimulate ovulation in female. LH is a potential drug of abuse, hence banned by IOC in 1999. It is prohibited for male only.
3. Corticotrophin (ACTH, tetracosactide): Corticotrophin has been misused to increase the levels of endogenous corticosteroids in the blood, particularly to obtain the mood-elevating effects of corticosteroids. The administration of corticotrophin is regarded as equivalent to the oral, intramuscular or intravenous administration of corticosteroids.

4. Growth Hormones (hGH, Somatotrophin): The use of growth hormone in sports is regarded as amoral and dangerous by reason of its various side effects such as allergic reactions, diabetogenic effects, and acromegaly when administered in large doses.

5. Insulin-like Growth Factors (IGF-1): Insulin-like growth factors is one of the combinations of about seventy amino acid, polypeptide. In fact in muscle fiber all biological actions of growth hormone appeared to be mediated by IGF. So, it is an important stimulator of a number of biological activities involving anabolic process in skeletal muscle. Some athletes use IGF-1 to take advantage of its anabolic effects.

6. Erythropoietin (EPO): A glycoproteinic hormone produced in the human kidney which regulates the synthesis of RBC, apparently by retroaction, the rate of synthesis of erythrocytes.

7. Insulin: Permitted only to treat insulin-dependent diabetes. The rider must produce a medical certificate issued by an endocrinologist or team doctor, certifying his condition as an insulin-dependent diabetic (for riders of Trade Teams I and II only the registration on the health record booklet will be taken into consideration).

8. Also prohibited for males are clomiphene, cyclofenil, tarryoxifen and aromatase inhibitors.

Without prejudice to the concentration stipulated for hCG, the presence of an abnormal concentration of an endogenous hormone in class (E) or its diagnostic markers in the urine of a competitor constitutes an offence unless it has been proven to be due to a physiological or pathological condition.

Notes:
1. This list is based on the list contained in the appendix to the Anti-Doping Code of the Olympic movement, but is not wholly identical. It stays in effect until a new list is published.
2. It is recalled that under the Anti-Doping Examination Regulations, all products, regardless of denomination, that contain even a minor amount of the above mentioned substances are prohibited.
3. Warning: The listings of examples in this document are not exhaustive! Numerous substances that are not itemized in this list are considered prohibited under the designation of ‘related substances’. Riders and other permit-holders must make sure that every drug, supplement, preparation, particularly if sold over the counter, as well every other substance they use does not contain any prohibited substance.

4. Warning: This list relates to a sports regulation. The use of, and traffic in, a number of substances on this list is prohibited or regulated in the national laws of many countries. Provisions for criminal penalties may be laid down. A substance or method that is not prohibited under the present list may be prohibited under national law, as for example with regard to cannabinoids.

Drug
A drug, broadly speaking, is any chemical substance that, when absorbed into the body of a living organism, alters normal bodily function. There is no single, precise definition, as there are different meanings in medicine, government regulations, and colloquial usage. In pharmacology, defines a drug as “A chemical substance used in the treatment, cure, prevention, or diagnosis of disease or used to otherwise enhance physical or mental well-being.” Drugs may be prescribed for a limited duration, or on a regular basis for chronic disorders.

Recreational drugs are chemical substances that affect the central nervous system, such as opioids
or hallucinogens. They may be used for perceived beneficial effects on perception, consciousness, personality, and behavior. Some drugs can cause addiction and habituation. Drugs are usually distinguished from endogenous biochemicals by being introduced from outside the organism. For example, insulin is a hormone that is synthesized in the body; it is called a hormone when it is synthesized by the pancreas inside the body, but if it is introduced into the body from outside, it is called a drug. Many natural substances such as beers, wines, and some mushrooms, blur the line between food and drugs, as when ingested they affect the functioning of both mind and body.

**Drug Abuse**

Comparison of the perceived harm for various psychoactive drugs from a poll among medical psychiatrists specialized in addiction treatment. Drug abuse has a wide range of definitions related to taking a psychoactive drug or performance enhancing drug for a non-therapeutic or non-medical effect. All of these definitions imply a negative judgment of the drug use in question (compare with the term responsible drug use for alternative views). Some of the drugs most often associated with this term include alcohol, amphetamines, barbiturates, benzodiazepines, cocaine, methaqualone, and opium alkaloids. Use of these drugs may lead to criminal penalty in addition to possible physical, social, and psychological harm, both strongly depending on local jurisdiction. Other definitions of drug abuse fall into four main categories: public health definitions, mass communication and vernacular usage, medical definitions, and political and criminal justice definitions.

**Drug Testing**

Drug testing is an integral part of the system of dope control. It has become a way of life for many athletes. Its aim is to provide correct and dependable analytical results which are based on scientific data. Drug testing in sports is a very complex area and comprises not only the methods of modern analytical chemistry but also the know-how of many other scientific disciplines. For example, biochemistry explains the metabolism of doping agents and pharmacokinetics describes the time course of the blood and urine concentration of doping agents and their metabolites. In under sport, the biological fluid available for analysis is urine. There are two types of drug testing which are commonly used: (a) **Competition testing** (b) **Out of competition Testing**.

**Competition Testing**

Even though there is slight variation in the policy of law to select the athletes, in most cases of individual events, it is the three medal winners, 4th and 5th places and one picked up at random, thus totaling six athletes for each event. For team sports such as football or hockey, one or two players per team are randomly selected. As soon as each event is over, selected athletes are notified and he/she, under supervision of the doping control escort is asked to come to the doping station within an hour, where the sample for testing is collected as per laid down procedure.

**Out of Competition Testing**

It is mainly for anabolic agents, peptide hormones, mimetic and analogues and prohibited methods. Random samples are taken during training seasons without prior notification.

The urine sample has been chosen as the body fluid for evidence of drug abuse. The American Medical Association (AMA), Council on Scientific Affairs have laid down/enumerated the following reasons for this selection:

i. The collection of urine is not invasive.

ii. Large volumes can be collected easily.

iii. Drugs and their metabolites are generally present in higher concentration in urine than in other tissues or fluids because of the concentrating function of kidneys.

iv. Urine is easier to analyze than blood and other things because of usual absence of protein and cellular constituent.

v. Drugs and their metabolites are usually very stable in frozen urine allowing long-term storage of positive samples.
On the contrary, for some substances like Human Growth Hormones, blood assay is the only method available to detect its abuse. Similarly, methods of detecting blood doping require blood specimens.

Why Athletes Use AAS?

The goals of individuals who use anabolic steroids depend upon the activities in which they participate, e.g.

◆ Bodybuilders desire more lean mass and less body fat.
◆ Weightlifters desire to lift maximum amount of weight as possible.
◆ Field athletes want to put the shot or throw the hammer, discus or javelin farther than other competitors or record holders.
◆ Swimmers and runners hope to perform their frequent high intensity, long duration workouts without physical breakdown.
◆ Football players want to increase their lean body mass and strength.

Plan and Procedure for Sample Collection

The main purpose of International Standard for Testing is to plan for effective testing and to maintain the integrity and identity of the samples, from notifying the athlete to transporting the sample for analysis.

The International Standard for Testing includes standard for test distribution planning, notification of athletes, preparing for and conducting sample collection, security/post-test administration and transport of samples.

◆ Planning
◆ Notification of athletes
◆ Preparing for sample collection
◆ Conduction of sample collection session
◆ Security/post-test administration
◆ Transports of sample and documentation

Planning

The objective of the planning is to plan and implement an effective distribution of athlete's test. Planning starts with establishing criteria for athletes to be included in a registered testing tool and ends with selecting athletes for sample collection. The main activities are information gathering, risk evaluation and developing, monitoring, evaluating and modifying the test distribution plan.

The Anti-Doping organization (ADO) shall define and document the criteria for athlete to be included in a registered testing tool. This shall include as a minimum for international federations and for national Anti-Doping organizations.

Requirements for collecting athlete’s information for the purposes of out of competition testing. The collection, maintaining and monitoring sufficient information to ensure that sample collection can be planned and conducted without prior advanced notice for all athletes included in the registered testing tools, and when athletes failed to provide accurate and timely information, taking appropriate action to ensure the information stage up to date and complete in all respects. As a minimum of the following information regarding athletes shall be collected.

1. Name of the candidate
2. Sports/game/discipline
3. Home address
4. Contact/telephone numbers
5. Training times and venues
6. Training camps
7. Traveling plans
8. Competition schedule
9. Disability if applicable, including the requirement for 3rd party involvement with notification.

For this purpose a format of sample collection to be used which is prepared by WADA.

The ADO shall evaluate the potential risk of doping and possible doping pattern for which sports and/or discipline based on:

a. Physical demands of the sports and possible performance enhancing effects that doping may elicit
b. Available doping analysis statistics
c. Available research on doping trends
d. Training periods and competition season.

The ADO shall also establish a system for maintaining test distribution planning data, such data shall be used to assist with determining modification to the plan, if necessary. This information shall include as a minimum for each test:
Table 15.1: List of various agents banned by WADA (Chemical Name)

<table>
<thead>
<tr>
<th>Anabolic agents</th>
<th>Hormones and related substance</th>
<th>Beta-2 agonists</th>
<th>Diuretics</th>
<th>Stimulants*</th>
<th>Narcotic analgesics</th>
<th>Beta blocker</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. <strong>Exogenous AAS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>i. Boldione</td>
<td>Erythropoetin (EPO)</td>
<td>Bitolterol</td>
<td>Acetazolamide</td>
<td>Adrafinil</td>
<td>Alphaprodine</td>
<td>Acebutalol</td>
</tr>
<tr>
<td>ii. Bolasterone</td>
<td>Growth Hormone (hGH &amp; Insulin-like growth factor (IGF-I))</td>
<td>Orciprenaline</td>
<td>Amiloride</td>
<td>Aminetepine</td>
<td>Dextromoramide</td>
<td>Bisoprolol</td>
</tr>
<tr>
<td>iii. Boldenone</td>
<td>Gonadotrophins (hCG &amp; LH)</td>
<td>Reproterol</td>
<td>Bendroflumethiazide</td>
<td>Amphetamine</td>
<td>Diamorphine (Heroin)</td>
<td>Bunolol</td>
</tr>
<tr>
<td>iv. Calusterone</td>
<td>Insulin</td>
<td>Rimiterol</td>
<td>Benzthiazide</td>
<td>Aminetepine</td>
<td>Levorphanol</td>
<td>Labetolol</td>
</tr>
<tr>
<td>v. Clostebol</td>
<td>Corticotrophins (ACTH, tetacosactide)</td>
<td>Salbutamol</td>
<td>Bumetanide</td>
<td>Amphetamine</td>
<td>Levobunolol</td>
<td>Levobunolol</td>
</tr>
<tr>
<td>vi. Danazol</td>
<td></td>
<td>Salmeterol</td>
<td>Canrenone</td>
<td>Andrafinil</td>
<td>Dipipanone</td>
<td>Carteolol</td>
</tr>
<tr>
<td>vii. Delta-1</td>
<td>Androstenediol</td>
<td>Terbutaline</td>
<td>Chlormerodrin</td>
<td>Benzphetamine</td>
<td>Ethoheptazine</td>
<td>Carvedilol</td>
</tr>
<tr>
<td>viii. Dehydroclormethyl</td>
<td></td>
<td>Formoterol</td>
<td>Chlorthalidone</td>
<td>Bromantane</td>
<td>Fentanyl</td>
<td>Celiprolol</td>
</tr>
<tr>
<td>ix. Ethylestrenol</td>
<td></td>
<td>Diclofenamide</td>
<td>Carphedone</td>
<td>Hydrocodone</td>
<td>Esmolol</td>
<td></td>
</tr>
<tr>
<td>x. Testosterone</td>
<td>Ethacrynic acid</td>
<td>Carthine</td>
<td>Levorphanol</td>
<td>Levobunolol</td>
<td></td>
<td></td>
</tr>
<tr>
<td>xi. Drostanolone</td>
<td>Hydrochloorthiazide</td>
<td>Indapamide</td>
<td>Cocaine</td>
<td>Morphine</td>
<td>Metipranolol</td>
<td></td>
</tr>
<tr>
<td>xii. Fenoterole</td>
<td></td>
<td>Mannitol</td>
<td>Dimetamfetamine</td>
<td>Nalbuphine</td>
<td>Metoprolol</td>
<td></td>
</tr>
<tr>
<td>xiii. Formebolon</td>
<td></td>
<td>Metolazone</td>
<td>Ephedrine</td>
<td>Pentazocine</td>
<td>Nadolol</td>
<td></td>
</tr>
<tr>
<td>xiv. Furazabol</td>
<td></td>
<td>Spironolactone</td>
<td>Etamivan</td>
<td>Pethidine</td>
<td>Oxprenolol</td>
<td></td>
</tr>
<tr>
<td>xv. Gestrinone</td>
<td></td>
<td>Triamterene</td>
<td>Fenacine</td>
<td>Phenazocine</td>
<td>Propanolol</td>
<td></td>
</tr>
<tr>
<td>xvi. Mestanolone</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

Contd...
### Anabolic agents (continued)

<table>
<thead>
<tr>
<th>Anabolic agents</th>
<th>Hormones and related substance</th>
<th>Beta-2 agonists</th>
<th>Diuretics</th>
<th>Stimulants*</th>
<th>Narcotic analgesics</th>
<th>Beta blocker</th>
</tr>
</thead>
<tbody>
<tr>
<td>xvii. Mesterolone</td>
<td></td>
<td></td>
<td></td>
<td>Fentanyl</td>
<td>Trimpridine</td>
<td>Pindololol</td>
</tr>
<tr>
<td>xviii. Methyltestosterone</td>
<td></td>
<td></td>
<td></td>
<td>Fenproporex</td>
<td></td>
<td>Sotalol</td>
</tr>
<tr>
<td>xix. Methyltrienolone</td>
<td></td>
<td></td>
<td></td>
<td>Furfenorex</td>
<td>Heptaminol</td>
<td>Timolol</td>
</tr>
<tr>
<td>xx. Nandrolone</td>
<td></td>
<td></td>
<td></td>
<td>Metenorex</td>
<td></td>
<td></td>
</tr>
<tr>
<td>xxi. Norbolethone</td>
<td></td>
<td></td>
<td></td>
<td>Mesocarbe</td>
<td></td>
<td></td>
</tr>
<tr>
<td>xxii. Norethandrolone</td>
<td></td>
<td></td>
<td></td>
<td>Methylphenadrine</td>
<td></td>
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</tr>
<tr>
<td>xxiii. Orabolin</td>
<td></td>
<td></td>
<td></td>
<td>Morazone</td>
<td>Metraminol</td>
<td></td>
</tr>
<tr>
<td>xxiv. Oxandrolone</td>
<td></td>
<td></td>
<td></td>
<td>Nikethamide</td>
<td></td>
<td></td>
</tr>
<tr>
<td>xxv. Quinbolone</td>
<td></td>
<td></td>
<td></td>
<td>Pemoline</td>
<td></td>
<td></td>
</tr>
<tr>
<td>xxvi. Stanozolol</td>
<td></td>
<td></td>
<td></td>
<td>Pentetrazol</td>
<td></td>
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</tr>
<tr>
<td>xxvii. Delta-1 dehydro testosterone</td>
<td></td>
<td></td>
<td></td>
<td>Phentermine</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>Pyrovaledrine</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>Selegiline</td>
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<td></td>
</tr>
</tbody>
</table>

#### b. Endogenous AAS

- i. Androstenediol
- ii. Androstenedion
- iii. Dehydroepiandrosterone
- iv. Dehydrotestosterone

*Note:*

- a. Cathine is prohibited when its concentration in urine is greater than 5 micrograms/ml.
- b. Ephedrine is prohibited when its concentration in urine is greater than 10 µg/ml.
- c. For methyl ephedrine, the definition of positive is a concentration in urine greater than 10 microgram/ml.
1. The sports/game/discipline
2. The country represented by the athlete (if applicable)
3. The type of sample collection
4. The date of sample collection
5. The country in which the sample collection was conducted.

In addition, for each advanced analytical finding:

a. Dates of sample collection and analysis
b. Classes of substance found
c. Actual substances detected
d. Sections of Anti-Doping rules if violation any

For selection of athletes the ADO shall select athletes for sample collection using target testing and random selection methods. As minimum target testing athletes should be considered based on the following information:

a. Injury
b. Withdrawal or absence from expected competition
c. Way into or coming out of retirement
d. Behavior indicating doping
e. Sudden major improvements in performance
f. Changes in athlete’s information that can indicate a potential increase in the risk of doping, including moving to a remote location.
g. Athlete’s sport performance history
h. Details of first doping controls
i. Reliable information from a third party.

Following the selection of an athlete for sample collection and prior to notification it should be ensured that selection decisions were disclosed only to those who need to know in order to ensure the athlete can be notified and tested on a no advance notice basis.

**Notification of Athletes**

The objective is to ensure that the selected athletes is notified, the rights of the athlete are maintained, there are no opportunities to manipulate the sample to be provided and the notification is documented. It starts when the ADO initiates the notification of the selected athlete and ends when the athlete arrives at the Dope Control Center or when the athlete’s possible failure to comply is brought to the ADO’s attention.

**Preparation for Sample Collection**

Preparing for the sample collection session starts with the establishment of a system for obtaining relevant information for effective conduct of the session and ends when it is confirmed that the sample collection equipment confirms to the specified criteria.

The DCO shall only use sample collection equipment systems that are authorized by the ADO, which at a minimum, shall meet the following criteria. They shall:

i. Have a unique numbering system incorporated into all bottles, containers, tubes or any other item used to seal the athlete’s sample.
ii. Have a sealing system that is temper evident.
iii. Ensure the identity of the athlete is not evident from the equipment itself.
iv. Ensure that all equipment is clean and sealed prior to use by the athlete.

**Conduction of Sample Collection Session**

The sample collecting session starts with defining overall responsibility for the conduction of the sample collection session and once the sample collection documentation is complete. The main activities are:

i. Preparing for collecting the sample
ii. Collecting the sample and
iii. Documenting the sample collection.

In conducting the sample collection session the following information should be recorded as a minimum:

i. Date, time and type of notification.
ii. Date and time of sample provision.
iii. The name of the athlete.
iv. The date of birth of the athlete.
v. The gender of the athlete.
vi. The athlete’s home address and telephone number.
vii. The athlete’s sports and disciplines.
viii. The sample code number.
ix. The name and signature of the person who witnessed the urine sample provision.
x. The name and signature of the blood collection official who collected the blood sample, where applicable.
xi. Required laboratory information on the sample.

xii. Medications and supplements taken and recent blood transfusion details if applicable within the timeframe specified by the laboratory as declared by the athlete.

xiii. Any irregularities in procedures.

xiv. Athlete comments or concerns regarding the conduct of the session, if provided.

xv. The name and signature of the athlete;

xvi. The name and signature of the athlete’s representative if required; and

xvii. The name and signature of the DCO.

The athlete and DCO shall sign appropriate documentation to indicate their satisfaction that the documentation accurately reflects the details of the athlete’s sample collection session including any concerns recorded by the athlete. The athlete’s representative shall sign on behalf of the athlete if the athlete is a minor. Other persons present who had a formal role during the athlete’s sample collection session may sign the documentation as a witness of the proceedings. The DCO shall provide the athlete with a copy of the records of the sample collection session that have been sign by the athlete.

**Security/Post-test Administration**

All the samples collected at the Doping Control Station and sample collection documentation are securely stored prior to their departure from the Doping Control Station. Post-test administration begins when the athlete has left the Doping Control Station after providing his/her sample/s, and ends with preparation of all the collected samples and documentation of transport.

**Transports of Sample and Documentation**

All the collected samples and related documentation arrived at the WADA accredited laboratory or as otherwise approved by WADA in proper condition to do the necessary analysis. Transport starts when the scaled samples and documentation leave the Doping Control Station and ends with the confirmed receipt of the samples and sample collection documentation at their intended destinations.

The main activities are arranging for the secure transport of samples and related documentation to the WADA-accredited laboratory or as otherwise approved by WADA, and arranging for the secure transport of sample collection documentation to the ADO.

**Collection of Urine Samples**

To collect an athlete’s urine sample in a manner that endures: (i) Consistency with relevant principles of internationally recognized standard precautions in health care settings so that the health and safety of the athlete and sample collection personnel are not compromised. (ii) The sample is of a quality and quantity that meets laboratory guidelines. (iii) The sample is clearly and accurately identified, and (iv) The sample is securely sealed.

The collection of a urine sample begins with ensuring the athlete is informed of the sample collection requirements and ends with discarding any residual urine remaining at the end of the athlete’s sample collection session. The DCO has the responsibility for ensuring that each sample is properly collected, identified and sealed. The DCO has the responsibility for directly witnessing the passing of the urine sample.

**Procedure:** At the beginning, the DCO shall ensure that the athlete is informed of the sample collection requirements and ends with discarding any residual urine remaining at the end of the athlete’s sample collection session. The DCO shall inspect the equipment to ensure that it will not affect the identity or integrity of the sample.

The DCO shall ensure that the athlete is offered a choice of appropriate equipment for collecting the sample. If the nature of athlete’s disability requires that he/she use additional or other equipment and the DCO shall inspect the equipment to ensure that it will not affect the identity or integrity of the sample.

The DCO shall instruct the athlete to select a collection vessel. When the athlete selects a collection vessel and for selection of all other sample collection equipments that directly holds the urine samples, the DCO will instruct the athlete to check that all seals on the selected equipment is intact and the equipment has not been tampered with. If the athlete is not satisfied with the selected equipment, he/she may select another one. If the athlete is not satisfied with any of the equipment
available for the selection, this shall be recorded by the DCO. If the DCO does not agree with the athlete’s opinion that all of the equipment available for the selection is unsatisfactory and the DCO shall instruct the athlete to proceed with the sample collection. If the DCO agrees with the reasons put forward by the athlete that all of the equipments available for the selection is unsatisfactory, the DCO shall terminate the collection of the athlete’s urine sample and same should be recorded by the DCO.

The athlete shall retain control of the collection vessels and any sample provided until the sample is sealed, unless assistance is required by an athlete disability. The DCO/Chaperone who witnesses the passing of the sample shall be of the same gender as the athlete providing the sample. And thereafter, the officials and athlete shall proceed to an area of privacy to collect the sample. The officials shall witness the sample leaving the athlete’s body and record the witnessing in writing.

The DCO shall use the relevant laboratory’s specifications to verify, in full view of the athlete that the volume of the urine sample satisfies the laboratory’s requirements for analysis. If the volume of urine is insufficient, the DCO shall conduct a partial sample collection procedure.

The DCO shall instruct the athlete to select a sample collection kit containing/marked A & B bottles. Once a sample collection kit has been selected, the DCO and the athlete shall check that all code numbers match and that these code numbers are recorded accurately by the DCO. If the athlete or the DCO finds the numbers are not the same, the DCO shall instruct the athlete to choose another kit and in this case the DCO shall record the matter.

The athlete shall pour the relevant laboratory’s prescribed minimum volume of urine into the B bottle and then fill the A bottle as much as possible. The athlete shall then fill the B bottle as much as possible with the remaining urine. The athlete shall ensure that a small amount of urine is left in the collection vessel. The athlete shall seal the bottles as directed by the DCO. The DCO shall check, in full view of the athlete that the bottles have been properly sealed.

Finally, the DCO shall use the relevant laboratory’s guidelines for pH for specific gravity to test the residual urine in the collection vessel to determine if the sample is likely to meet the laboratory’s guidelines. If it is not then the DCO should record that the sample do not meet laboratory pH and specific gravity guidelines.

Collection of Blood Samples

To collect an athlete’s blood sample in a manner that ensures: (i) The health and safety of the athlete and sample collection personnel are not compromised. (ii) The sample is of a quality and quantity that meets the relevant analytical guidelines. (iii) The sample is clearly and accurately identified and (vi) The sample is securely sealed.

The collection of blood sample begins with ensuring the athlete is informed of the sample collection requirements and ends with properly storing the sample prior to dispatch for analysis at the WADA-accredited laboratory or as otherwise approved by WADA.

The DCO has the responsibility for ensuring that: each sample is properly collected, identified and sealed; and all samples have been properly stored and dispatch in accordance with the relevant analytical guidelines. The blood collection officials has the responsibility for collecting the blood sample, answering related questions during the provision of the sample, and proper disposal of used blood sampling equipment not required for completing the sample collection session.

Procedure: Procedures involving blood shall be consistent with relevant principles of internationally recognized standard precautions in health care settings. Blood sample collection equipment shall consist of either an A sample tube, or a B sample tube. If the sample collection consists solely of blood then a B sample shall be collected and used as a confirmation if required.

The DCO shall ensure that the athlete is informed of the requirements of the sample collection, including any modifications for athletes with disabilities. The DCO/Chaperone and athlete shall proceed to the area where the sample will be provided. The DCO shall ensure that the athlete is offered comfortable
conditions including being in a relaxed position for at least 10 minutes prior to providing a sample.

The DCO shall instruct the athlete to select the sample collection kits required for collecting the sample to check that the selected equipment has not been tampered with and the seals are intact. If the athlete is not satisfied with a selected kit, he/she may select another one. If the athlete is not satisfied with any kits and others are available, this shall be recorded by the DCO. If the DCO agrees with the reasons put forward by the athlete that all available kits are unsatisfactory, the DCO shall instruct the athlete to proceed with the sample collection session. But if the DCO agrees with the reasons put forward by the athlete that all available kits are unsatisfactory, the DCO shall terminate the collection of the athlete’s blood sample and this shall be recorded by the DCO.

When a sample collection kit has been selected, the DCO and the athlete shall check that all code number matches and that this code number is recorded accurately by the DCO. If the athlete or DCO finds that the numbers are not the same, the DCO shall instruct the athlete to choose another kit and the DCO shall record the matter.

The blood collection official shall clean the skin with a sterile disinfectant wipe or swab in a location unlikely to adversely affect the athlete or his/her performance and if required, apply a tourniquet. The blood collection official shall take the blood sample from a superficial vein into the final collection container. The tourniquet, if applied, shall be immediately removed after the venipuncture has been made. The amount of blood removed shall be adequate to satisfy the relevant analytical requirements for the sample analysis to be performed.

However, if the amount of blood that can be removed from the athlete at the first attempt is insufficient, the blood collection official shall repeat the procedure. Maximum attempts shall be three. Should all attempts fail, then the blood collection officials shall inform the DCO. The DCO shall terminate the collection of the blood sample and record this and the reasons for terminating the collections. The blood collection official shall apply a dressing to the puncture site/s and dispose of used blood sampling equipment not required for completing the sample collection session. The athlete shall seal his/her sample into the sample collection kit as directed by the DCO. In full view of the athlete, the DCO shall check that the sealing is satisfactory.

Finally, the sealed sample shall be kept at a cool, but not freezing, temperature prior to analysis at the doping control station or dispatch for analysis at the WADA-accredited laboratory or as otherwise approved by WADA.

Detection Time

The elimination time for drugs that appear on the banned list is a major factor in the testing program. There are significant biological and physiological variances between individuals. Only a brand generalization can be made regarding elimination of particular drug from the system. Individual metabolism, amount of substance used, frequency of use, length of time used, and normal biodegradation process in any individual varies.

Detection Limit

After taking a pharmaceutical dose of a drug, the following range of drug concentration should be achieved in urine:

<table>
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<th>Substance</th>
<th>Concentration</th>
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<tr>
<td>Stimulants</td>
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<tr>
<td>Narcotic analgesics</td>
<td>0.5 – 50 µg/ml</td>
</tr>
<tr>
<td>Anabolic steroids</td>
<td>0.1 – 2 µg/ml</td>
</tr>
<tr>
<td>Diuretics</td>
<td>0.1 – 2 µg/ml</td>
</tr>
<tr>
<td>Beta blockers</td>
<td>0.5 – 50 µg/ml</td>
</tr>
</tbody>
</table>

WADA Sanctions for Doping Offence

i. Disqualification of results in event during which an Anti-Doping rule violation occurs.

ii. Imposition of ineligibility for prohibited substances and prohibited methods except for the specific substances:
   a. First violation: Two years ineligibility
   b. Second violation: Lifetime ineligibility

iii. Specified substances:
   The period of ineligibility for the use of specified substance will be as follows:
   a. First violation: At a minimum, a warning and reprimand and no period of ineligibility from future events, and at a maximum, one years ineligibility.
   b. Second violation: Two years ineligibility
   c. Third violation: Lifetime ineligibility.
World Anti-Doping Agency (WADA)

World Anti-Doping Agency (WADA) was established on November 10, 1999 in Lausanne. It is the first worldwide agency to try to combat use of drugs in sports. World Anti-Doping Agency (WADA) promotes and coordinates the fight against doping in sports internationally. The WADA was set up as a foundation under the initiative of the WADA with the support and participation of intergovernmental organizations, governments and other public and private bodies to eradicate doping from sports. It was set up in 1999 following a series of high profile doping scandals. The mission of WADA is to promote and coordinate at international level to fight against doping in all forms. World Anti-Doping Agency coordinates and comprehensive Anti-Doping program at international level with the support of the following:

i. International Olympic Committee (IOC)
ii. International Sports Federation (ISF)
iii. National Olympic Committee (NOC)
iv. The athletes

The following (Table 15.2) table reveals the approximate detection periods for each substance by test type i.e after this period the below mentioned banned substances will not be present in the sample. The detection depend upon multiple factors - drug class amount and frequency of use, metabolic rate, body mass, age, overall health, and urine pH. For ease of use, the detection times of metabolites have been incorporated into each parent drug. For example, heroin and cocaine can only be detected for a few hours after use, but their metabolites can be detected for several days in urine.

World Anti-Doping Agency is to establish list of banned substances coordinate unannounced out-of-competition drug testing, develop standards for collecting and analyzing samples, set unified drug sanction and promote research. It selected six cities as finalists, to become WADA’s permanent headquarters. Bonn-Germany, Lausanne-Switzerland, Lille-France, Montreal-Canada, Stockholm-Sweden and Vienna-Austria.

The purposes of the world Anti-Doping program and the code are:

◆ To protect the athlete’s fundamental right to participate in doping-free sports and thus promote health fairness and equality for athletes worldwide; and

◆ To ensure harmonized coordinated and effective Anti-Doping programs at the international and national level with regard to detection deterrence and prevention of doping.

The main elements of world Anti-Doping programs are:

Level 1: The code
Level 2: International standards
Level 3: Models of the best practice

The Code

The code is the fundamental and universal document upon which the world Anti-Doping program in sports is based. The purpose of the code is to advance the

<table>
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<th>Elimination time</th>
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<td></td>
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<td>ii. Cocaine—occasional use</td>
<td>6-12 Hours</td>
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<td>3–5 Days</td>
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<td>24–48 Hours</td>
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<td>3. Marijuana (Tetrahydrocannabinol)</td>
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<td>4. Anabolic steroids:</td>
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<tr>
<td>i. Fat—soluble injectable type</td>
<td>6–18 Months</td>
</tr>
<tr>
<td>ii. Oral of water—soluble types</td>
<td>4–6 Weeks</td>
</tr>
<tr>
<td>5. Beta blockers and Diuretics</td>
<td>24–72 Hours</td>
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</table>
Anti-Doping effort through universal harmonization of core Anti-Doping elements.

International Standards

The following international standards for different technical and operational areas within the Anti-Doping program have been developed in consultation with the signatories and governments and approved by WADA:
- International standard for laboratories
- International standard for testing
- International standard for therapeutic use exemptions.

Models of Best Practice

A model of best practice based on the code has been developing to provide state of the art-solution in different areas of Anti-Doping. Recently WADA has issued the following guidelines/forms:
- a. Guidelines for athlete information
- b. Guidelines for result management
- d. Guidelines for bodies operating certification of quality systems for doping control program
- e. International standard for laboratories version 4.0 August, 2004
- f. Chain of custody form
- g. Requirement for Anti-Doping analysis of whole blood, plasma, serum or other blood fractions
- h. Reporting Norandrostone findings
- i. Reporting and evaluation guidance for testosterone, epitestosterone T/E ratio and other endogenous steroids.

SUMMARY

1. Doping is defined as the administering or use of substances in any form alien to the body, or of physiological substances in abnormal amounts and with abnormal methods, by health personnel with the exclusive aim of attaining an artificial and unfair increase of performance in competition. In sports, the use of performance-enhancing drugs is commonly referred to by the disparaging term “doping”, particularly by those organizations that regulate competitions. The use of performance enhancing drugs is mostly done to improve athletic performance. This is why many sports ban the use of performance-enhancing drugs.

2. The World Anti-Doping Agency’s (WADA) mission is to promote, coordinate and monitor the fight against doping in sport in all its forms. Its key activities include scientific research, education, development of Anti-Doping capacities, and monitoring of the World Anti-Doping Code (Code)—the document harmonizing Anti-Doping policies in all sports and all countries. WADA is a Swiss private law foundation. Its seat is in Lausanne, Switzerland, and its headquarters are in Montreal, Canada. WADA works towards a vision of the world that values and fosters a doping-free culture in sport. This section provides information about WADA’s constitution, structure and governance.

3. Prohibited classes of substances are stimulants, opioid analgesics, anabolic agents, diuretics, peptide hormones, mimetics and analogues. Prohibited methods are blood doping, pharmacological, chemical and physical manipulation. Classes of drugs subject to restrictions are alcohol, cannabinoids, local anesthetics, corticosteroids and beta blockers.

4. The adrenergic stimulants—ephedrine and ephedrine derivatives are related chemically to the body’s own ‘stress hormones’, adrenaline and noradrenaline. The physiologic changes that prepare a body for strenuous exercise are sometimes called the “fight or flight” response. These changes persist during exercise and are necessary for maximal performance. The reason for the inclusion of stimulants in the doping register is that they conceal the feeling of exhaustion.

5. Morphine like analgesics are the most effective of all analgesics and they are powerful anxiolytics and sedatives. They act on CNS and reduce feelings of pain. Narcotic analgesics may be used to mask the sensation of pain. The greatest risk associated with the use of morphine like analgesics is the risk of addiction. The drugs are classified as string narcotics. Combined with alcohol, even small
doses of the weakest of the preparations may result in respiratory arrest and death.

6. Anabolic adrenergic steroids are man-made substances related to male sex hormones. Athletes and bodybuilders abuse anabolic steroids to enhance performance and improve physical appearance. The effects of anabolic steroids on women are relatively clear. Muscle mass and strength appear to increase when these agents are used in conjunction with strength training. This means that, with large doses of anabolic steroids, a woman's athletic performance could approach that of a man. The major side effects from abusing anabolic steroids can improve liver tumors and cancers, jaundice, fluid retention, and high blood pressure.

7. Diuretics are drugs which increase the amount of fluid excreted by the kidneys. Diuretics are used by sportsmen and women to decrease body weight, to increase power to weight ratios and to help qualify for certain weight categories, e.g. boxing and judo. Human growth hormone (hGH) is known to act on many aspects of cellular metabolism and is also necessary for skeletal growth in humans. The major role of hGH in body growth that is muscle and organ growth. hGH is prohibited both in- and out-of-competition under the WADA's List of Prohibited Substances and Methods. The common side effects for hGH abuse are: diabetes in prone individuals; worsening of cardiovascular diseases; muscle, joint and bone pain; hypertension and cardiac deficiency; abnormal growth of organs; accelerated osteoarthritis.

8. Blood doping is the misuse of certain techniques and/or substances to increase one's red blood cell mass, which allows the body to transport more oxygen to muscles and therefore increase stamina/endurance and performance. The common side effects are blood clots which cause strokes (embolism), life threatening hypersensitivity reaction (anaphylactic shock), and other transfusion reactions. Alcohol has two entirely different effects on the brain. One is an anxiolytics (anxiety relieving) or tranquilizing effect, the other a stimulatory effect. Probably alcohol has a refreshing effect and the ability to restore the athlete's strength. Marijuana is a relaxant and impairs judgment. In high doses, it causes hallucinations. There can be no doubt that the immediate and long-term effect of cannabis use are detrimental to sporting performance due to effect on the heart, lungs and CNS.

9. Drug is defined as “A chemical substance used in the treatment, cure, prevention, or diagnosis of disease or used to otherwise enhance physical or mental well-being.” Drugs may be prescribed for a limited duration, or on a regular basis for chronic disorders. Drug abuse has a wide range of definitions related to taking a psychoactive drug or performance-enhancing drug for a non-therapeutic or non-medical effect. Drug testing is an integral part of the system of dope control. It has become a way of life for many athletes. Its aim is to provide correct and dependable analytical results which are based on scientific data.

**REVIEW QUESTIONS**

1. Define ‘Doping’. What is the historical perspective of doping?
2. What are the prohibited classes of substances and prohibited methods of doping?
3. What do you mean by drug? What is drug abuse? Discuss the type of drug testing.
4. What is presently known about the use of amphetamines in athletics competition? What are the potential risks of using amphetamines?
5. What are the adrenergic stimulants? What are the side effects of this group of stimulant?
6. Under what circumstances might beta blockers be ergogenic aids? Write some of their ergolytic properties.
7. What is known about Cocaine and Marijuana as an ergogenic aids? How might caffeine improve athletic performance?
8. What is anabolic agent and Diuretics? Are diuretics ergogenic? What are the potential risks associated with the use of diuretics?
9. What is known about human growth hormone as potential ergogenic aid? What are the risks associated with its use?

10. What is blood doping? Does blood doping improve athletic performance? Discuss with suitable example.

11. How does the use of alcohol in moderate or large doses affect athletic performance?

12. Write the systematic plan and procedure of sample collection for drug testing.

13. Discuss about the Collection of Urine and Blood Samples. What do you mean by the detection time and limit?

14. What do you mean by World Anti-Doping Agency (WADA)? Write down about the WADA sanctions for doping offence.
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