COURSE GUIDE

KHE 416
EXERCISE PHYSIOLOGY

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INTRODUCTION

Exercise physiology as a course plays a pivotal role in medicine, and allied health fields.

Although some lump exercise physiology with sports performance or athletic training, that is a mere fraction to what the field of exercise physiology contributes too.

With an upsurge in diseases like obesity and diabetes rising in the general population, the increased need for medical doctors, physical and occupational therapists, scholars of exercise physiology not only provide applied and clinical research for medical doctors and other practitioners to draw upon, but they are able to teach at these schools of thought preparing waves of new physicians, physical therapists or teach undergraduates who aspire to these roles.

Also, increase in rising interest in genetics, epigenetics and molecular processes, exercise physiology goes right in, studying not only the effects of exercise on these processes but also diving into how metabolism, cardiology and patho-physiology interacts with molecular, genetic and epigenetic processes.

Exercise physiology contributes to many fields and this is increasing in more in health and allied health programmes.

COURSE AIM

The aim of this course is to train you to understand the Physical Education Methods, various teaching styles as well as advantages and disadvantages of each style.

COURSE OBJECTIVES

At the end of this course, you should be able to:

- Define Exercise Physiology, why study Exercise Physiology and career prospects.
- 2. Define Physical Activity, Physical fitness, Exercise, Exercise training, Training Detraining and adaptation to exercise
- Explain exercise response, factors involved for exercise response and exercise response pattern.
- Define the concept of energy and metabolism
- Describe the function of Adenosine Triphosphate in metabolism
- Define Cellular respiration

- Explain the participation of carbohydrate, fat, and protein metabolism
- Describe the regulation of cellular respiration and ATP Production
- State the Nutrition for Fitness and Athletics
- Explain the Body composition and weight control
- Define Anaerobic and Aerobic Metabolism
- State the measurement of Anaerobic and Aerobic measurement
- Explain the Anaerobic and Aerobic Exercise Response
- Describe the Pulmonary system
- Differentiate Conducive zone and respiratory zone
- Define breathing
- State the factors responsible in the mechanism of breathing in the pulmonary system.
- State the function of Pulmonary Circulation
- Define Minute Ventilation
- Describe the concept and structure of Pulmonary Ventilation
- Describe static Exercise
- State Male-Female Respiratory Differences
- State Lung Volumes and Capacities and Pulmonary Ventilation
- Describe External and Internal Respiration
- Describe Detraining
- Describe the Heart
- Define the Vascular System
- Differentiate between Blood and Hormonal Control of Blood Volume
- State the neural control and anatomical sensors and factors affecting control of the cardiovascular system.
- Describe the Short-Term, Light to Moderate Submaximal Aerobic Exercise
- Describe the Long-Term, Moderate to Heavy Submaximal Aerobic Exercise
- Describe the Incremental Aerobic Exercise to Maximum
- Describe the Upper-Body versus Lower-Body Aerobic Exercise
- Describe Intensity of Muscle Contraction
- Describe Blood Flow During Static Contractions
- Describe the skeletal tissue
- Describe measurement of bone health
- List the factors influencing bone health
- Describe exercise response

WORKING THROUGH THIS COURSE

You will need to study for at least 30 hours to complete this course successfully. You also needat least 2 hours on each course per week. Each

course has a stated and intended learning outcome(s) (ILOs) which must be achieved during the study of each unit. There is a self-assessment exercise in each unit to be done by the student which will help you make progress.

STUDY UNITS

Module 1

Unit 1	Meaning of Exercise Physiology and why study it
Unit 2	The Exercise Response
Unit 3	Energy and Metabolism
Unit 4	Metabolic System
Unit 5	Anaerobic & Aerobic Metabolism During Exercise

Module 2

Unit 1	Cardiovascular- Respiratory System
Unit 2	Mechanism of Breathing
Unit 3	Respiratory Circulation
Unit 4	Measurement of Lung Volumes

Module 3

Unit 1	Response of The Respiratory System to Exercise
Unit 2	The Influence of Sex and Age on Respiration at Rest and During Exercis**e**
Unit 3	Respiratory Training and Detraining Adaptations
Unit 4	Overview & Regulation of The Cardiovascular System
Unit 5	Measurement of Cardiovascular Variables & Responses to Aerobic Exercise

Module 3

Unit 1	Neuro-Muscular and Skeletal System
Unit 2	Factors Influencing Bone Health and Exercise Response
Unit 3	Application of The Training Principles & Skeletal Adaptation To training.

PRESENTATION SCHEDULE

The presentation schedule sent to you gives you the important dates for the completion of your Tutor Marked Assignments (TMAs) and participation at facilitation. You are required to forward all your assignments at the right time without any deferment or plagiarisms. Plagiarism is falsification and it is unlawful, anyone culpable will be seriously punished.

ASSESSMENT

The assessment to this course is divided into two main parts. The first part is the Tutor MarkedAssignments (TMAs) and the second part is the e-examination which is a computer based examination. In solving the problems in the assignments, you are meant to use the facts, knowledge and experience gathered in the course of the study. A link will be provided throughwhich the assignment will be submitted between the stipulated times. The assignment will be evaluated within the given guidelines and a feedback provided. The TMAs will form 30% of the total marks for the course.

Each student will be required to sit for an e-examination which will last for one hour. The e- examination will form 70% of the course mark. The computer will be programmed to open atthe beginning of the examination and end as scheduled automatically.

TUTOR-MARKED ASSIGNMENT (TMA)

There are three (3) assignments that make up the tutor-marked assignment and they must be submitted for grading at the stipulated time as no extension shall be granted to any student afterthe due date unless for exceptional cases. The three assignments form the 30% of the course which is a Tutor Marked Assignment.

TUTORS AND TUTORIALS

Each student will be assigned to a tutorial group at their various Study Centre, date and timeof the tutorials with the name and phone number of your tutor will be communicated to students through the Centre Director. 12 hours of tutorials will be provided for this course. Your assignments will be graded and correction made on them. You need to keep a close watch on the comments made by your tutor and identify any area where you are having difficulties. Your TMAs must be mailed to your tutor within the deadline stipulated and thiswill be marked and sent back to you almost immediately. Where you require assistance, do not hesitate to contact your tutor through phone, e-mail or direct discussion.

There may be situations where you will need to urgently make contact with your tutor when:

1.) You do not understand the assigned readings or any part of the study units.
2.) You find it hard to deal with your self-tests or exercises.
3.) You have a query or difficulty with your assignment, with comments made by yourtutor on your assignment or with the grading system of the assignment.

You must ensure you attend all tutorials, this is the only avenue you have for physical contactwith your tutor/facilitator and your questions will be answered immediately. You are at liberty to query any difficulty encountered during the course of the study.

There is a great advantage in getting actively involved in the group discussion and to benefit immensely from the course tutorials, you must have done your personal preparation and drawout your own questions, this helps you to be actively involved in the course tutorials.

FINAL EXAMINATION AND GRADING

KHE416 final examinations will last for two hour and will account for 70% of the total coursegrade. The examination will comprise of questions similar to the type of the tutor marked exercises you have initially practiced. Every part of the course will be assessed. You are to usethe time gap between concluding the last unit and sitting for the examination for the revision of the entire course.

Revising the tutor mark assignment exercises with the comments made by the tutorial facilitators might be useful for the final examination. The final examination will cover every information from all parts of the course materials.

HOW TO GET THE MOST FROM THE COURSE

In this course you have the opportunity of working and studying through a well-designed studymaterial at your own pace and at a time and place that suits you best. Read the material as against listening to a lecturer in the conventional school system. The content is complementedwith audios teachings as well as watching related videos. In the same way that a lecturer mightrecommend some reading materials, the study units tell you when to read recommended books or other materials and when to undertake practical activities. Note that the study unit replaces the university lecturer. Just as a lecturer might give you class exercises/activities, your study units provide exercises for you to do at the appropriate time. Each of the study units follows acommon format. The first item is an introduction to the subject matter of the unit and how a particular unit is integrated with the other ones and the course as a whole. Next is a set of learning Intended Learning Outcome(s) (ILOs) which state what you will be able to do by thetime you have completed the unit. These Intended Learning Outcome(s) are set to guide your study. When you have finished a unit, you must go back and check whether you have achievedthe Intended Learning Outcome(s). If you cultivate the habit of doing this, you will make tremendous improvement in your chances of passing the course.

The main body of the unit guides you through the required reading from other courses. This will usually be either from your recommended books or from a reading section. Self- assessment exercises are interspersed throughout the unit. You are expected to work on them as well. Working through these exercises will help you to achieve the Intended Learning Outcome(s) of the unit and prepare you for the assignments and the examination. You should attempt the self-assessment exercise as you come across it in the study unit. There will also beseveral examples given in the study units; work through these when you come across them too.

FACILITATION

You will receive online facilitation which is asynchronous. Your facilitator will summarize each unit of study and send to your mail weekly. The facilitator will also direct and coordinate your activities on the learning platform.

Do not hesitate to contact your tutor by telephone and e-mail if you:

- Do not understand any part of the study units or the assignment.
- Have difficulty with the self-assessment exercises
- Have a question or problem with an assignment or with your tutor's comments on anassignment.

Read all the comments and notes of your facilitator especially on your assignments, participatein the forums and discussions. This is the only chance you have to interact with others in the programme. You can raise any problem encountered in the course of your study. To gain the maximum benefit from course tutorials, prepare a list of questions before the discussion session. You will learn a lot from participating actively in the discussions.

SUMMARY

KHE416 intends to introduce you to Exercise Physiology with particular reference to its ability to use the body's response to exercise. It also describes the adaptation to exercise training to maximize human physical potential. You will also be able to answer the tutor marked assessment as presented in each unit.

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MODULE 1 EXERCISE PHYSIOLOGY, ENERGY AND METABOLISM & METABOLIC SYSTEM, ANAEROBIC & AEROBIC METABOLISM

INTRODUCTION

Exercise Physiology is the ability to use the body's response to exercise. It is also a part of science that describes the adaptation to exercise training to maximize human physical potential. The performance of body of an individual is enhanced when such individual has ability to perform exercise.

Unit 1 Meaning of Exercise Physiology and why study it
Unit 2 The Exercise Response
Unit 3 Energy and Metabolism
Unit 4 Metabolic System
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UNIT 1 MEANING OF EXERCISE PHYSIOLOGY AND WHY STUDY IT

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7.0 References/Further Reading

1.0 INTRODUCTION

This unit describes exercise physiology, the reason why we study exercise physiology, the physical activity, physical fitness, exercise and exercise training, training, detraining, and the concepts of adaptation to exercise.

2.0 INTENDED LEARNING OUTCOMES (ILOS)

By the end of this Unit, you will be able to;

- Define Exercise Physiology
- Explain reasons for studying Exercise Physiology.
- List the career prospects in exercise Physiology.
- Define Physical Activity, Physical fitness, Exercise, Exercise training, Training Detraining and adaptation to exercise

3.0 MAIN CONTENT

3.1 Exercise Physiology

Exercise physiology is the study of how the body responds and adapts to physical stress. It can be defined as both a basic and an applied science that describes, explains, and uses the body's response to exercise and adaptation to exercise training to maximize human physical potential. Exercise physiology is the study of the function of biological systems. In sport and exercise physiology we are interested in how the body responds to exercise and physical activity, what limits physical performance, and how training can be used to improve health and performance. For instance, we examine how the heart functions to deliver blood to muscle and how muscle functions to produce force. Other areas covered in sport and exercise physiology include the effects of diet on health and performance. The box below presents some of the key questions and issues that sport and exercise physiologists are interested in. Exercise physiologists study the effect of exercise on pathology, and the mechanisms by which exercise can reduce or reverse disease progression.

3.2 Why study exercise physiology

No single course or textbook, of course, can provide all the information a prospective professional will need. However, knowledge of exercise physiology and an appreciation for practice based on research findings help set professionals in the field apart from mere practitioners. To become a respected professional in the field you need to learn exercise physiology in order to:

1. Understand how the basic physiological functioning of the human body is modified by short and long term exercise as well as the mechanisms causing these changes. Unless one knows what responses are normal, one cannot recognize an abnormal response or adjust to it.
2. Provide quality physical education programs in schools that stimulate children and adolescents both physically and

intellectually. To become lifelong exercisers, students need to understand how physical activity can benefit them, why they take physical fitness tests, and what to do with fitness test results.

3. Apply the results of scientific research to maximize health, rehabilitation, and/or athletic performance in a variety of subpopulations.
4. Respond accurately to questions and advertising claims, as well as recognize myths and misconceptions regarding exercise. Good advice should be based on scientific evidence.

3.3 Meaning of Physical Activity, Physical Fitness, Exercise, Exercise training, training, detraining and Adaptation to exercise

Physical Activity

Physical activity refers to all movement that a person carries out as part of their day including walking, cycling, wheeling, sports, active recreation and play, which can be done at any level of skill and for enjoyment by everybody. The World Health Organization has it defined as any bodily movement produced by skeletal muscles that requires energy expenditure. Regular physical activity whether moderate or vigorous-intensity is proven to help prevent and manage non-communicable diseases such as heart disease, stroke, diabetes and several cancers. It also helps prevent hypertension, maintain healthy body weight and can improve mental health, quality of life and well-being.

Physical Fitness

Physical fitness refers to how well the human being's body is capable of performing every physical activity at work, leisure and in exercise for recreation and competition, and participation. Physical fitness is divided into two major sets of components used for describing how physically healthy people are and those indicating how well people can perform different skills as found in sports and dance.

Exercise

Exercise is a single acute bout of bodily exertion or muscular activity that requires an expenditure of energy above resting level and that in most, but not all, cases results in voluntary movement. Exercise sessions are typically planned and structured to improve or maintain one or more components of physical fitness.

Physical activity, in contrast, generally connotes movement in which the goal (often to sustain daily living or recreation) is different from that of exercise, but which also requires the expenditure of energy and often provides health benefits. For example, walking to school or work is

physical activity, while walking around a track at a predetermined heart rate is exercise.

Exercise Training

There are two main goals for exercise and they are:

1. Health-related Exercise
2. Sport-specific Exercise also called athletic fitness.

Health-Related Vs Sport-Specific

Health-related exercise refers to that portion of physical fitness directed toward the prevention of or rehabilitation from disease, the development of a high level of functional capacity for the necessary and discretionary tasks of life, and the maintenance or enhancement of physiological functions in biological systems that are not involved in performance but are influenced by habitual activity.

The individual's goal may be to participate minimally in an activity to achieve some health benefit before disease occurs. The goal may be to participate in a substantial amount of exercise to improve or maintain a high level of physical fitness. Or, a disabled individual's goal may be to participate in an activity to recover and/or attain the maximal function possible. All goals should include avoiding injury during the process. Three components of health-related physical fitness are generally recognized: cardiovascular-respiratory endurance (aerobic power), body composition, and muscular fitness (strength, muscular endurance, and flexibility) (Canadian Society for Exercise Physiology, 2004; The Cooper Institute, 2004). Figure 1.4 (inner circle) shows that these components form the core of physical fitness. The relationships between each of these fitness components and hypokinetic disease are described in appropriate later units. Hypokinetic diseases are diseases caused by and/or associated with a lack of physical activity. Health-related physical fitness is important for everyone.

Sport-specific exercise has a narrower focus. It is that portion of physical fitness directed toward optimizing athletic performance. Here, higher levels of cardiovascular-respiratory endurance and anaerobic power and capacity are generally needed for successful performance. Body composition values may be more specific than health levels in order to optimize performance. The muscular fitness attributes of power, balance, and flexibility are frequently more specific in certain athletic performances than for health.

To determine the importance of each component of fitness and develop a sport-related fitness program, you first analyze the specific sport's physiological demands.

Then, the athlete is evaluated in terms of those requirements. These elements allow for a specifically designed, individualized program. This program should:

- Work specific musculature while achieving a balance between agonistic and antagonistic muscle groups
- Incorporate all motor fitness attributes that are needed
- Use the muscles in the biomechanical patterns of the sport
- Match the cardiovascular and metabolic energy requirements of the sport
- Attend realistically to body composition issues

Training

Training is a consistent or chronic progression of exercise sessions designed to improve physiological function for better health or sport performance.

There are eight fundamental guidelines that are well established and forms the basis for the development of any exercise training.

1. Specificity: This principle is sometimes called the SAID principle, which stands for "specific adaptations to imposed demands"; i.e, what you do is what you get. When you develop an exercise training program, you first determine the goal. Fitness programs for children and adolescents, for example, differ from those for older adults. Training programs for non-athletes differ from training programs for athletes.
2. Overload: Overload is a demand placed on the body greater than that to which it is accustomed. To determine the overload, first evaluate the individual's critical physiological variables (specificity). Then, consider three factors:
   (a) Frequency: The number of training sessions daily or weekly;
   (b) Intensity: The level of work, energy expenditure, or physiological response in relation to the maximum;
   (c) Duration: The amount of time spent training per session or per day.
3. Rest/Recovery/Adaptation: This occurs during periods of rest, when the body recovers from the acute homeostatic disruptions and/or residual fatigue. This is sometimes called Super compensation. It is therefore critical for exercisers to receive sufficient rest between training sessions, after periods of increased training overload.
4. Progression: Progression is the change in overload in response to adaptation. The best progression occurs in a series of incremental

steps called Step loading, in which every third or fourth change is actually a slight decrease in training load.

5. Retrogression/Plateau/Reversibility: When an individual's adaptation or performance levels off, a plateau has been reached. If it decreases, retrogression has occurred. Reversibility is the reversal of achieved physiological adaptations that occurs after training stops (detraining). Too much time spent doing the same type of workout using the same equipment in the same environment can lead to a plateau. However, variety and rest may help the person move beyond a plateau. Then, if a plateau continues for some time or if other signs and symptoms appear, then the plateau may be an early warning signal of overreaching or overtraining. Retrogression may signal overreaching or overtraining.
6. Maintenance: It is sustaining an achieved adaptation with the most efficient use of time and effort. At this point, the individual has reached an acceptable level of training. In general, intensity is the key to maintenance. I.e. as long as exercise intensity is maintained, frequency and duration of exercise may decrease without losing positive adaptations.
7. Individualization: Individuals require personalized exercise prescriptions based on their fitness levels and goals. Individuals also adapt differently to the same training program. The same training overload may improve physiological performance in one individual, maintain physiological and performance levels in the second individual, and result in maladaptation and performance decreases in the third. Age, sex, genetics, disease, and the training modality also all affect individual exercise prescriptions and adaptations.
8. Warm-Up/Cool-Down: A warm-up prepares the body for activity by elevating the body temperature. Conversely, a cool-down allows for a gradual return to normal body temperature. The best type of warm-up is specific to the activity that will follow and is individualized to avoid fatigue.

Detraining

As noted in the retrogression/plateau/reversibility training principle, training adaptations are reversible. This is called detraining. Detraining is the partial or complete loss of training-induced adaptations as a result of a training reduction or cessation. Detraining may occur due to a lack of compliance with an exercise training program, injury, illness, or a planned periodization transition phase. Detraining should not occur during the tapering/unloading phases or cycles. The magnitude of the reversal of physiological adaptations depends on the training status of the individual when the training is decreased or ceased, the degree of reduction in the training (minimal to complete), which element of training overload is

impacted most (frequency, intensity, or duration), and how long the training is reduced or suspended. Just as all physiological variables do not adapt at the same rate (days versus months), so all physiological variables do not reverse at the same rate. Unfortunately, less information is available about detraining than training. The timeline for the loss/reversal of adaptation for all variables and in all populations is unknown. Compounding this issue, it is often difficult to distinguish among changes resulting from illness, normal aging, and detraining. What is known will be discussed in this text within each unit, following the training adaptation sections.

Adaptations to exercise

Training adaptations represent physical and physiological adjustments that promote optimal functioning. Whereas exercise responses use resting values as the baseline, training adaptations are evaluated against the same condition prior to training.

SELF-ASSESSMENT EXERCISES

i. Define Exercise physiology
ii. Explain why we study Exercise Physiology
iii. Define Physical Fitness

4.0 CONCLUSION

Having read this course and successfully completed the assessment and self-assessment test it is assumed that you have attained understanding of the introductory knowledge on exercise physiology.

5.0 SUMMARY

In this Unit, you have learnt what exercise physiology is and why we must study it. And also, definition of Physical training, Physical fitness, Exercise and Exercise Training, Training, Detraining, and the concepts of adaptation of exercise have been discussed. The assessment and self-assessment exercise have been provided to enable you understand your own rating of the understanding and learning you achieved while reading this material in this Unit. Online links have also been provided to broaden your understanding of the learning required in this Unit.

6.0 TUTOR-MARKED ASSIGNMENT

1. State the difference between Training and Detraining
2. Describe the concept of Adaptation to exercise

7.0 REFERENCES/FURTHER READING

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UNIT 2 THE EXERCISE RESPONSE

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7.0 References/Further Reading

1.0 INTRODUCTION

In this unit, we shall look at the exercise response, factors associated with exercise response, exercise response pattern and exercise and training as stressors will be considered.

2.0 INTENDED LEARNING OUTCOMES (ILOS)

By the end of this unit, you should be able to:

- Explain exercise response
- State the factors involved in exercise response
- Explain the Exercise response pattern
- Explain exercise and training as stressors

3.0 MAIN CONTENTS

3.1 The Exercise Response

An exercise response is the pattern of change in physiological variables during a single acute bout of physical exertion. A physiological *variable* is any measurable bodily function that changes or *varies* under different circumstances. For example, heart rate is a variable with which you are undoubtedly already familiar. You probably also know that heart rate increases during exercise. However, to state simply that heart rate increases during exercise does not describe the full pattern of the response. For example, the heart rate response to a 400-m sprint is different from the heart rate response to a 50-mi bike ride.

3.2 Factors involved in exercise response

To fully understand the response of heart rate or any other variable, we need more information about the exercise itself. Four factors are considered when determining the acute response to exercise:

1. the exercise modality
2. the exercise intensity
3. the exercise duration
4. the exercise categories

Exercise Modality
Exercise modality (or mode) means the type of activity or the particular sport. For example, rowing has a very different effect on the cardiovascular-respiratory system than does football. Modalities are often classified by the type of energy demand (aerobic or anaerobic), the major muscle action (continuous and rhythmical, dynamic resistance, or static), or a combination of the energy system and muscle action. Walking, cycling, and swimming are examples of continuous, rhythmical aerobic activities; jumping, sprinting, and weight lifting are anaerobic and/ or dynamic resistance activities. To determine the effects of exercise on a particular variable, you must first know what type of exercise is being performed.

Exercise Intensity
Exercise intensity is most easily described as maximal or submaximal. Maximal (max) exercise is straightforward; it simply refers to the highest intensity, greatest load, or longest duration an individual is capable of doing. Motivation plays a large part in the achievement of maximal levels of exercise. Most maximal values are reached at the endpoint of an *incremental exercise test to maximum*; that is, the exercise task begins at a level the individual is comfortable with and gradually increases until he or she can do no more. The values of the physiological variables measured at this time are labeled “max”; for example, maximum heart rate is symbolized as HRmax. Submaximal exercise may be described in one of two ways. The first involves a *set load*, which is a load that is known or is assumed to be below an individual’s maximum. This load may be established by some physiological variable such as working at a specific heart rate (perhaps 150 b.min−1), at a specific work rate (e.g., 600 kgm.min−1 on a cycle ergometer), or for a given distance (perhaps a 1-mi run). Such a load is called an absolute workload. If an absolute workload is used and the individuals being tested vary in fitness, then some individuals will be challenged more than others. Generally, those who are more fit in terms of the component being tested will be less challenged and so will score better than those who are less fit and more challenged. For example, suppose that the exercise task is to lift 80 lb in

a bench press as many times as possible, as in the YMCA bench press endurance test. As illustrated in Table 1.1, if the individuals tested were able to lift a maximum of 160, 100, and 80 lb once, respectively, it would be anticipated that the first individual could do more repetitions of the 80-lb lift than anyone else. Similarly, the second individual would be expected to do more repetitions than the third, and the third individual would be expected to do only one repetition. In this case, the load is not submaximal for all the individuals, because Terry can lift the weight only one time (making it a maximal lift for Terry). Nonetheless, the use of an absolute load allows for the ranking of individuals based on the results of a single exercise test and is therefore often used in physical fitness screenings or tests. The second way to describe submaximal exercise is as a percentage of an individual's maximum. A load may be set at a percentage of the person's maximal heart rate, maximal ability to use oxygen, or maximal workload.

This value is called a relative workload because it is prorated or relative to each individual. All individuals are therefore expected to be equally challenged by the same percentage of their maximal task. This should allow the same amount of time or number of repetitions to be completed by most, if not all, individuals. For example, for the individuals described in the previous paragraph, suppose that the task now is to lift 75% of each one's maximal load as many times as possible. The individuals will be lifting 120, 75, and 60 lb, respectively. If all three are equally motivated, they should all be able to perform the same total number of repetitions. Relative workloads are occasionally used in physical fitness testing. They are more frequently used to describe exercises that are light, moderate, or heavy in intensity or to give guidelines for exercise prescription.

There is no universal agreement about what exactly constitutes light, moderate, or heavy intensity. See examples of classification below;

Low or light:	≤54% of maximum
Moderate:	55–69% of maximum
Hard or heavy:	70–89% of maximum
Very hard or very heavy:	90–99% of maximum
Maximal:	100% of maximum
Supramaximal:	>100% of maximum

Maximum is defined variously in terms of workload or work rate, heart rate, oxygen consumption, weight lifted for a specific number of repetitions, or force exerted in a voluntary contraction. Specific studies may use percentages and definitions of maximum that vary slightly.

Exercise Duration

Exercise duration is simply a description of the length of time the muscular action continues. Duration may be as short as 1–3 seconds for an explosive action, such as a jump, or as long as 12 hours for a full triathlon (3.2-km [2-mi] swim, 160-km [100-mi] bicycle ride, and 42.2-km [26.2-mi] run). In general, the shorter the duration, the higher the intensity that can be used. Conversely, the longer the duration, the lower the intensity that can be sustained. Thus, the amount of homeostatic disruption depends on both the duration and the intensity of the exercise.

Exercise Categories

This textbook combines the descriptors of exercise modality, intensity, and duration into six primary categories of exercise. Where sufficient information is available, the exercise response patterns for each are described and discussed:

1. *Short-term, light to moderate submaximal aerobic exercise*. Exercises of this type are rhythmical and continuous in nature and utilize aerobic energy. They are performed at a constant workload for 10–15 minutes at approximately 30–69% of maximal work capacity.

2. *Long-term, moderate to heavy submaximal aerobic exercise*. Exercises in this category also utilize rhythmical and continuous muscle action. Although predominantly aerobic, anaerobic energy utilization may be involved. The duration is generally between 30 minutes and 4 hours at constant workload intensities ranging from 55% to 89% of maximum.

3. *Incremental aerobic exercise to maximum*. Incremental exercises start at light loads and continue by a predetermined sequence of progressively increasing workloads to an intensity that the exerciser cannot sustain or increase further. This point becomes the maximum (100%). The early stages are generally light and aerobic, but as the exercise bout continues, anaerobic energy involvement becomes significant. Each workload/work rate is called a stage, and each stage may last from 1 to 10 minutes, although 3 minutes is most common. Incremental exercise bouts typically last between 5 and 30 minutes for the total duration.

4. *Static exercise*. Static exercises involve muscle contractions that produce an increase in muscle tension and energy expenditure but do not result in meaningful movement. Static contractions are measured as some percentage of the muscle's maximal voluntary contraction (MVC), the maximal force that the muscle can exert. The intent is for the workload to remain constant, but fatigue

sometimes makes that impossible. The duration is inversely related to the percentage of maximal voluntary contraction (%MVC) that is being held but generally ranges from 2 to 10 minutes.

5. *Dynamic resistance exercise*. These exercises utilize muscle contractions that exert sufficient force to overcome the presented resistance so that movement occurs, as in weight lifting. Energy is supplied by both aerobic and anaerobic processes, but anaerobic is dominant. The workload is constant and is based on some percentage of the maximal weight the individual can lift (1-RM) or a resistance that can be lifted for a specified number of times. The number of repetitions, not time, is the measure of duration.

6. *Very-short-term, high-intensity anaerobic exercise*.
Activities of this type last from a few seconds to approximately 3 minutes. They depend on high power anaerobic energy and are often supramaximal.

Exercise Response Patterns

The exercise response patterns for the six categories of exercise are described verbally and depicted graphically. For ease of recognition, consistent background colors and icons represent each category of exercise. Frequent incremental exercise patterns are depicted in that is measured with its appropriate unit of measurement. Examples are heart rate (b.min−1), blood pressure (mmHg), and oxygen consumption (mL.kg.min−1). Only specific graphic patterns are applicable to any given variable. These combinations of pattern and variable are described in the exercise response sections in each unit. Although not indicated in the figure, curvilinear changes can also be described as exponential—either positive or negative. For each exercise response, the baseline, or starting point against which, the changes are compared, is the variable's resting value. Your goal here is to become familiar with the graphic patterns and the terminology used to describe each.

The patterns showing an initial increase or decrease with a plateau at steady state are the most common responses to short-term, and light to moderate submaximal aerobic exercise. Patterns that include a drift seen as the gradual curvilinear increase or decrease from a plateau despite no change in the external workload typically result from long term, moderate to heavy submaximal aerobic exercise.

Another form of gradual increase despite no change in the external workload is frequently seen during dynamic resistance exercise as a saw-tooth pattern resulting from the sequential lifting and lowering of the weight. Finally, some categories of exercise may show a smooth, gradual

increase. Minimal change during exercise with a rebound rise in recovery is almost exclusively a static exercise response.

All of these patterns of response routinely result from incremental exercise to maximum. No specific patterns are shown for very-short-term, high-intensity anaerobic exercise because these tend to be either abrupt rectilinear or curvilinear increases or decreases.

Exercise and Training as Stressors
Exercise and training are often considered only in a positive manner, but both acute exercise and chronic training are stressors.

Selye's Theory of Stress
A stressor is any activity, event, or impingement that causes stress. Stress is defined most simply as a disruption in body homeostasis and all attempts by the body to regain homeostasis. Selye defines stress more precisely as "the state manifested by a specific syndrome that consists of all the nonspecifically induced changes within a biological system." The biological system here is the human body. The specific syndrome is the General Adaptation Syndrome (GAS), a step-by-step description of the bodily reactions to a stressor. It consists of three major stages (Selye, 1956):

1. the Alarm-Reaction: shock and counter shock
2. the Stage of Resistance
3. the Stage of Exhaustion

In the *Alarm-Reaction stage*, the body responds to a stressor with a disruption of homeostasis (shock). It immediately attempts to regain homeostasis (counter shock). If the body can adjust, the response is mild and advantageous to the organism; the *Stage of Resistance* or adaptation ensues. If the stress becomes chronic or the acquired adaptation is lost, the body enters the *Stage of Exhaustion*. At this point, the nonspecifically induced changes, which are apparent during the Alarm-Reaction but disappear during the Stage of Resistance, become paramount. These changes are labeled the triad of symptoms and include enlargement of the adrenal glands, shrinkage of the thymus and lymphatic tissue, and bleeding ulcers of the digestive tract. Specifically induced changes directly related to the stressor may also occur; for example, if the stressor is cold (shock), the body may shiver to produce heat (counter shock). Ultimate exhaustion is death (Selye, 1956).

Selye's Theory of Stress Applied to Exercise and Training
In the context of Selye's theory of stress, the pattern of responses exhibited by physiological variables during a single bout of exercise results directly from the disruption of homeostasis. This is the shock

phase of the Alarm-Reaction stage. For many physiological processes (respiration, circulation, energy production, and so forth), the initial response is an elevation in function. The degree of elevation and constancy of this elevation depends on the intensity and duration of the exercise. Appropriate changes in physiological function begin in the counter shock phase of the Alarm-Reaction and stabilize in the Stage of Resistance if the same exercise intensity is maintained for at least 1–3 minutes. This is termed a physiological steady state or steady rate. The Stage of Exhaustion that results from a single bout of exercise, even incremental exercise to maximum, is typically some degree of fatigue or reduced capacity to respond to stimulation, accompanied by a feeling of tiredness. This fatigue is temporary and readily reversed with proper rest and nutrition.

Training programs are made up of a series of acute bouts of exercise organized in such a way as to provide an overload that puts the body into the Alarm-Reaction stage followed by recovery processes that not only restore homeostasis but also encourage super compensation or adaptation (Kenttä and Hassmén, 1998; Kuipers, 1998; O'Toole, 1998). This can be manifested by altered homeostatic levels at rest, dampened homeostatic disruptions to absolute submaximal exercise loads, and/or enhanced maximal performances or physiological responses. When these adaptations occur, the body has achieved a Stage of Resistance. Table 1.3 shows how the training principles previously introduced operate in the three stages of general adaptation syndrome of Selye.

The goal of a training program is to alternate the exerciser between Stages I and II and to avoid time in Stage III where recovery is not possible in a reasonable time. This process primarily proceeds by the cyclical interaction between adaptation (changes that occur in response to an overload) and progression (change in overload in response to adaptation). Each progression of the overload should allow for adaptation. However, this is not always accomplished.

SELF-ASSESSMENT EXERCISES

i. Explain the Exercise Response.
ii. State and differentiate between the factors associated with Exercise Response.

4.0 CONCLUSION

Having read this course and successfully completed the assessment and self-assessment test, it is assumed that you have attained understanding of the introductory knowledge on the exercise response and exercise and training as stressors.

5.0 SUMMARY

This Unit summarizes the exercise response, and the factors. Also, Exercise patterns and Exercise and Training as stressors were discussed. The assessment and self-assessment exercise have been provided to enable you understand your own rating of the understanding and learning you achieved while reading this material in this Unit. Online links have also been provided to broaden your understanding of the learning required in this Unit.

6.0 TUTOR-MARKED ASSIGNMENT

1. What are Exercise response pattern?
2. Explain Exercise and Training as Stressors.

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UNIT 3 ENERGY AND METABOLISM

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1.0 INTRODUCTION

This unit defines the concept of energy and metabolism, describe Adenosine Trisphosphate (ATP), and cellular respiration. The Carbohydrate, fat and protein metabolism will be discussed and the descriptive pattern of cellular respiration and ATP Production will be defined.

2.0 INTENDED LEARNING OUTCOMES (ILOS)

By the end of this unit, you will be able to:

- Define the concept of energy and metabolism
- Describe the function of Adenosine Triphosphate in metabolism
- Define Cellular respiration
- Explain the participation of carbohydrate, fat, and protein metabolism
- Describe the regulation of cellular respiration and ATP Production

3.0 MAIN CONTENT

3.1 Concept of Energy and Metabolism

Most individuals eat at least three meals a day. Eating is necessary to provide the energy that is essential for all cellular and thus bodily activity. To provide this energy, food must be transformed into chemical energy. The total of all energy transformations that occur in the body is called metabolism. When energy is used to build tissues—as when amino acids

are combined to form proteins that make up muscles—the process is called *anabolism*. When energy is produced from the breakdown of foodstuffs and stored so that it is available to do work, the process is called *catabolism*. It is catabolism that is of primary importance in exercise metabolism. Energy is needed to support muscle activity, whether a little or a lot of muscle mass is involved or the exercise is light or heavy, submaximal or maximal. In providing this needed energy, the human body is subject to the *First Law of Thermodynamics*, which states that energy is neither created nor destroyed, but only changed in form. Figure 2.1 depicts this law and the changes in form representing catabolism. Potential chemical energy—or fuel—is ingested as food. Carbohydrates, fats, and protein can all be used as fuels, although they are not used equally by the body in that capacity. The chemical energy produced from the food fuel is stored as adenosine triphosphate (ATP). The ATP then transfers its energy to energy-requiring physiological functions, such as muscle contraction during exercise, in which some energy performs the work and some is converted into heat. Thus, ATP is stored chemical energy that links the energy-yielding and the energy-requiring functions within all cells. The aim of this chapter is to fully explain ATP and how it is produced from carbohydrate, fat, and protein food sources.

Adenosine Triphosphate (ATP)

Structurally, ATP is composed of a carbon-nitrogen base called adenine, a 5-carbon sugar called ribose, and three phosphates, symbolized by P*i* (inorganic phosphate). Each phosphate group is linked by a chemical bond. When one phosphate is removed, the remaining compound is adenosine diphosphate (ADP). When two phosphates are removed, the remaining compound is adenosine monophosphate (AMP). The ATP energy reaction is reversible. When ATP is synthesized from ADP and P*i*, energy is required. The addition of P*i* is known as phosphorylation.

ADP + P*i* + energy → ATP

When ATP is broken down, energy is released. Hydrolysis is a chemical process in which a substance is split into simpler compounds by the addition of water. ATP is split by hydrolysis.

ATP → ADP + P*i* + energy for work + heat

The energy-requiring and energy-releasing reactions involving ATP are coupled reactions. Coupled reactions are linked chemical processes in which a change in one substance is accompanied by a change in another; that is, one of these reactions does not occur without the other. As the chemical agent that links the energy-yielding and energy-requiring functions in the cell, ATP is also a universal agent. It is the immediate source of energy for virtually all reactions requiring energy in all cells. ATP is often referred to as cellular energy. Actually, ATP is a high-energy molecule. The term high energy means that the probability is high that

when a phosphate is removed, energy will be transferred. To better understand how the breakdown of ATP releases energy, consider the analogy of a spring-loaded dart gun. The dart can be considered analogous to P*i*. It takes energy to "spring-load" the dart; this energy corresponds to the energy involved in the energy-requiring reaction:

ADP + P*i* + energy → ATP
Once the dart is loaded, the compressed spring has potential energy, which is released when the gun is fired. Firing corresponds to the energy-releasing reaction:

ATP → ADP + P*i* + energy
The ATP content of skeletal muscle at rest is about 6 mmol.kg−1. If not replenished, this amount could supply energy for only about 3 seconds of maximal contraction.

The total amount of ATP stored in the body at any given time is approximately 0.1 kg, which is enough energy for only a few minutes of physiological function. However, ATP is constantly being hydrolyzed and resynthesized.

The average adult produces and breaks down (turns over) approximately 40 kg (88 lb) of ATP daily, and an athlete may turn over 70 kg (154 lb) a day. The rate of hydrolysis of ATP during maximal exercise may be as high as 0.5 kg.min−1.

ATP can be resynthesized from ADP in three ways:
1. By interaction of ADP with CP (creatine phosphate, which is sometimes designated as PC, or phosphocreatine).
2. By anaerobic respiration in the cell cytoplasm.
3. By aerobic respiration in the cell mitochondria.

Phosphocreatine is another high-energy compound stored in muscles. It transfers its phosphate—and, thus, its potential energy—to ADP to form ATP, leaving creatine:
ADP + PC → C + ATP

Resting muscle contains more CP (~20 mmol.kg−1) and C (~12 mmol.kg−1) than ATP. The maximal rate of ATP re-synthesis from CP is approximately 2.6 mmol.kg.sec−1 and occurs within 1–2 seconds of the onset of maximal contraction. PC stores are used to regenerate ATP, and in a working muscle will be depleted in 15–30 seconds.

The rest of this chapter will concentrate on the more substantial production of ATP by the second and third techniques listed, namely, anaerobic cellular respiration and aerobic cellular respiration.

Cellular Respiration

The process by which cells transfer energy from food to ATP in a stepwise series of reactions is called cellular respiration. This term is used because, to produce energy, the cells rely heavily on the oxygen that the respiratory system provides. In addition, the by-product of energy production, carbon dioxide, is exhaled through the respiratory system. Cellular respiration can be either anaerobic or aerobic. Anaerobic respiration means it occurs in the absence of oxygen, does not require oxygen, or does not use oxygen. Aerobic means it occurs in the presence of oxygen, requires, or utilizes oxygen. Brain cells cannot produce energy anaerobically, and cardiac muscle cells have only a minimal capacity for anaerobic energy production.

Skeletal muscle cells, however, can produce energy aerobically and/or anaerobically as the situation demands. Figure 2.2 outlines the products and processes of cellular respiration, which are discussed in detail in following sections. Following are some basic points about these processes:

1. All three major food nutrients, fats (FAT), carbohydrates (CHO), and proteins (PRO), can serve as fuel or substrates—the substances acted upon by enzymes— for the production of ATP.
2. The most important immediate forms of the substrates utilized are glucose (GLU), free fatty acids (FFA), and amino acids (AA). Both FFA and glycerol are derived from the breakdown of triglycerides. Some cells can use glycerol directly in glycolysis, but muscle cells cannot.
3. Acetyl coenzyme A (acetyl CoA) is the central converting substance (usually called the universal or common intermediate) in the metabolism of FAT, CHO, and PRO. Although the process of glycolysis provides a small amount of ATP as well as acetyl CoA, both beta-oxidation and oxidative deamination or transamination are simply preparatory steps by which FFA and AA are converted to acetyl CoA. That is, beta-oxidation and oxidative deamination or transamination are simply the processes for converting FFA and AA, respectively, to a common substrate that allows the metabolic pathway to continue. The end result is that the primary metabolic pathways of the Krebs cycle, electron transport system (ETS), and oxidative phosphorylation (OP) are the same regardless of the type of food precursor. This is certainly more efficient than having totally different pathways for each food nutrient.
4. Each of the energy-producing processes or stages (glycolysis, Krebs cycle, ETS/OP) consists of a series of steps. Each step represents a small chemical change to a substrate resulting in a slightly different product in a precise, unvarying sequence with

designed first and last steps. This is known as a metabolic pathway. That is, a metabolic pathway is a sequence of enzyme-mediated chemical reactions resulting in a specific product. Each stage may be made up of one or more metabolic pathways. These sequences of steps are important for the body because they allow energy to be released gradually. If all of the energy contained in food nutrients were released at one time, it would be predominantly released as heat and would destroy tissues. It is easy to be intimidated by or uninterested in so many steps, each with its own chemical structure, enzymes, and long, complicated name. There is a logic to these steps, however. It is on this logic and understanding (rather than the chemical structures) that the following discussion concentrates.

Carbohydrates, Fat, Protein Metabolism

The discussion of ATP production will begin with carbohydrate metabolism for several reasons. First, many of the energy requirements of the human body are met by carbohydrate metabolism. Second, carbohydrate is the only food nutrient that can be used to produce energy anaerobically. Energy for both rest and exercise is provided primarily by aerobic metabolism. However, exercise often requires anaerobic energy production, and then carbohydrate is essential. Third, carbohydrate is the preferred fuel of the body because carbohydrate requires less oxygen in order to be metabolized than fat. Finally, once you understand carbohydrate metabolism, it is relatively simple to understand how fats and proteins are metabolized. Carbohydrates are composed of carbon, oxygen, and hydrogen. The complete metabolism of carbohydrate requires oxygen, which is supplied by the respiratory system and is transported by the circulatory systems to the muscle cells. The metabolism of carbohydrate also produces carbon dioxide, which is removed via the circulatory and respiratory systems, and water.

The form of carbohydrate that is exclusively metabolized is glucose, a 6-carbon sugar arranged in a hexagonal formation and symbolized as C6H12O6. Thus, all carbohydrates must be broken down into glucose in order to continue through the metabolic pathways. In its most simplistic form, the oxidation of glucose can be represented by the equation $C_6H_{12}O_6 + 6O_2 \rightarrow 6H_2O + 6CO_2$

or

glucose + oxygen → water + carbon dioxide

In the skeletal muscle cell, oxidation is tightly coupled with phosphorylation to produce energy in the form of ATP. Therefore, the equation becomes;

$C_6H_{12}O_6 + O_2 + (ADP + Pi) \rightarrow CO_2 + ATP + H_2O$

or

glucose + oxygen + (adenosine diphosphate + inorganic phosphate) → carbon dioxide + adenosine triphosphate + water When excess glucose is available to the cell, it can be stored as glycogen, which is a chain of glucose molecules chemically linked together, or it can be converted to and stored as fat. The formation of glycogen from glucose is called *glycogenesis*. Glycogen is stored predominantly in the liver and muscle cells. When additional glucose is needed, stored glycogen is broken down (hydrolyzed) to provide glucose, through a process called glycogenolysis. Because glycogen must first be broken down into glucose, the production of energy from glucose or glycogen is identical after that initial step.

Fat Metabolism

Although the body may prefer to use carbohydrate as fuel from the standpoint of oxygen cost, the importance of fat as an energy source should not be underestimated.

Fat is found in many common foods. Fat, in the form of triglyceride (sometimes known as triacylglycerol), is the major storage form of energy in humans. Some triglyceride is stored within muscle cells, but the vast majority is deposited in adipose cells, which comprise at least 10–15% of the body weight of average young males and 20–25% of the body weight of average young females.

Roughly half of this adipocyte storage occurs subcutaneously (under the skin). The remaining stores surround the major organs of the abdominothoracic cavity as support and protection. Triglycerides are turned over constantly in the body. Because body fat is turned over completely about every 3–4 weeks, no one is literally still carrying their "baby fat". Fat is an excellent storage fuel for several reasons. First, fat is an energy-dense fuel yielding 9.13 kcal.g−1; both carbohydrate and protein yield slightly less than 4 kcal.g−1. The difference is due to the chemical structure of the substrates specifically, the amount of oxidizable carbon and hydrogen. It is easy to appreciate the difference by looking at the chemical composition of the free fatty acid palmitate, which is C16H32O2. This fatty acid has almost three times as much C and H, but only a third as much O as glucose ($C_6H_{12}O_6$). Remember that it is H that donates the electrons (e−) and protons (H+) used during electron transport and oxidative phosphorylation.

Second, carbohydrate, in the form of glycogen, is stored in the muscles with a large amount of water: 2.7 g of water per g of dried glycogen. Triglyceride is stored dry. Thus, the energy content of fat is not diluted, and hard as it may be to believe, body bulk is less than would otherwise be necessary. If humans had to store the comparable amount of energy as carbohydrates, we would be at least twice as big. Third, glycogen stores are relatively small in comparison to fat stores. A person can deplete

stored glycogen in as little as 2 hours of heavy exercise or 1 day of bed rest, whereas fat supplies can last for weeks, even with moderate activity. Although many Americans are concerned about having too much body fat, this storage capacity is undoubtedly important for survival of the species when food is not readily available.

The triglycerides stored in adipose tissue must first be broken down into glycerol and free fatty acids before they can be used as fuel. One glycerol and three fatty acids make up a triglyceride. Seven fatty acids predominate in the body, but since three fatty acids combine with a glycerol to make up a triglyceride, 343 (7 ×7 × 7) different combinations are possible. Some common fatty acids include oleic acid, palmitic acid, stearic acid, linoleic acid, and palmitoleic acid. Fatty acids may be saturated, unsaturated, or polyunsaturated. A saturated fatty acid has a chemical bonding arrangement that allows it to hold as many hydrogen atoms as possible. Thus, the term "saturated" means "saturated with hydrogen." Unsaturated fatty acids have a chemical bonding arrangement with a reduced- hydrogen binding potential and therefore are unsaturated with respect to hydrogen atoms. Polyunsaturated means several bonds are without hydrogen. The breakdown of triglycerides into glycerol and fatty acids is catalyzed by the enzyme hormone-sensitive lipase. The glycerol is soluble in blood, but the free fatty acids (FFA) are not. Glycerol can enter glycolysis in the cytoplasm, but it is not typically utilized by muscle cells in this way. The direct role of glycerol as a fuel in the muscle cells during exercise is so minor that it need not be considered. However, glycerol can be converted to glucose by the liver. FFA must be transported in the blood bound to albumin. Specific receptor sites on the muscle cell membrane take up the FFA into the cell. The FFA must then be translocated or transported from the cytoplasm into the mitochondria. Once in the mitochondrial matrix, the FFA undergoes the process of beta-oxidation.

Protein Metabolism

Proteins are present in many food sources. Proteins are large molecules consisting of varying combinations of amino acids linked together. Approximately 20 amino acids occur naturally. Because there are so many ways these amino acids can combine the number of possible proteins is almost infinite. Like carbohydrates and fats, amino acids contain atoms of carbon, oxygen, and hydrogen. In addition, they may include sulfur, phosphorus, and iron. All amino acids have in common an amino group containing nitrogen (NH_2). Proteins are extremely important in the structure and function of the body. Among other things they are components of hemoglobin, contractile elements of the muscle, hormones, fibrin for clotting, tendons, ligaments, and portions of all cell membranes. Because proteins are so important in the body, the constituent amino acids are used predominantly as building blocks, not as a source of

energy. However, amino acids can be, and in certain instances are, used as a fuel source. When amino acids are used as a fuel source, muscles appear to preferentially, but not exclusively, utilize the group of amino acids known as branched-chain amino acids (BCAA): leucine, isoleucine, and valine. In this situation, as with carbohydrate and fat metabolism, the final common pathways of the Krebs cycle, electron transport, and oxidative phosphorylation are utilized. The site of entry into the metabolic pathways varies, as shown in Figure 2.2, with the amino acid. Six amino acids can enter metabolism at the level of pyruvic acid, 8 at acetyl CoA, 4 at a-ketoglutarate, 4 at succinate, 2 at fumarate, and 2 at oxaloacetate. All of these intermediates except acetyl CoA are, in turn, converted to pyruvate before being used to produce energy. The acetyl CoA is used directly in the Krebs cycle and electron transport, as previously described.

Regulation of Cellular Respiration and ATP Production
Intracellular Regulation
Intracellularly, the production of ATP and, hence, the flow of substrates through the various metabolic pathways is regulated predominantly by feedback mechanisms. Again, each step in each metabolic pathway is catalyzed by a specific enzyme. At least one of these enzymes in each pathway can be acted on directly by other chemicals in the cell and, as a result, increases or decreases its activity. Such an enzyme is called a *rate-limiting enzyme*, and the other factors that influence it are called *modulators*.

When the rate-limiting enzyme is inhibited, every step in the metabolic pathway beyond that point is also inhibited. When the rate-limiting enzyme is stimulated, every step in the metabolic pathway beyond that point is also stimulated.

The primary rate-limiting enzyme in glycolysis is phosphofructokinase (PFK), the enzyme that catalyzes step 3. PFK is stimulated, and subsequently the rate of glycolysis increased, by modulators such as ADP, AMP, P*i*, and a rise in pH. ADP, AMP, and P*i* modulators are the result of the breakdown of ATP. This is an example of a positive- feedback system in which the by-product of the utilization of a substance stimulates a greater production of that original substance. Conversely, PFK is inhibited and subsequently the rate of glycolysis decreased by the modulators ATP, CP, citrate (a Krebs cycle intermediate), FFA, and a drop in pH. Each of these modulators signals that sufficient substances exist to supply ATP. This is an example of a negative-feedback system in which the formation of a product or any other similarly acting product inhibits further production of the product.

The primary rate-limiting enzyme in the Krebs cycle is isocitrate dehydrogenase (ICD), which catalyzes step 3. ICD is stimulated by ADP,

P*i*, and calcium (positive feedback) and is inhibited by ATP (negative feedback).

Cytochrome oxidase—which catalyzes the transfer of electrons to molecular oxygen, resulting in the formation of water—is the rate-limiting enzyme for the electron transport system. It is stimulated by ADP and P*i* (positive feedback) and is inhibited by ATP (negative feedback).
A pattern in the modulators is readily evident, with ATP, ADP, and P*i* being universally important. When ATP is present in sufficient amounts to satisfy the needs of the cell and to provide some reserve in storage, there is no need to increase its production. Thus, key enzymes in the metabolic pathways are inhibited. However, when muscle activity begins and ATP is broken down into ADP and P*i*, these by-products stimulate all the metabolic pathways to produce more ATP so that the muscle contractions can continue.

Extracellular Regulation
During exercise, metabolic processes must provide ATP for energy and maintain blood glucose levels at near-resting values for the proper functioning of the entire organism. This is because the brain and nervous tissue must have glucose as a fuel. One of the ways of maintaining glucose levels is by the process of gluconeogenesis.

Gluconeogenesis
As defined earlier, gluconeogenesis is the creation (-genesis) of new (-neo) glucose (gluco-) in the liver from non-carbohydrate sources. The primary fuel sources for gluconeogenesis are glycerol, lactate or pyruvate, and alanine.

Neurohormonal Coordination
The regulation of blood glucose levels, including gluconeogenesis, is governed jointly by the autonomic nervous system (particularly the sympathetic division) and the endocrine system, which function in a coordinated fashion.

SELF-ASSESSMENT EXERCISES

i. What is energy and metabolism?
ii. Describe the function of Adenosine Triphosphate in metabolism
iii. Define Cellular respiration

4.0 CONCLUSION

Having read this course and successfully completed the assessment and self-assessment test, it is assumed that you have attained understanding of the introductory knowledge on the Energy and Metabolism.

5.0 SUMMARY

This Unit summarizes the concept of exercise and metabolism, and the functions of Adenosine Triphosphate (ATP). Cellular respiration, and the participation of carbohydrate, fat, and protein in metabolism were discussed. The assessment and self-assessment exercise have been provided to enable you understand your own rating of the understanding and learning you achieved while reading this material in this Unit. Online links have also been provided to broaden your understanding of the learning required in this Unit.

6.0 TUTOR-MARKED ASSIGNMENT

1. Explain the participation of carbohydrate, fat, and protein metabolism
2. Describe the regulation of cellular respiration and ATP Production

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UNIT 4 METABOLIC SYSTEM

CONTENTS

1.0 Introduction
2.0 Intended Learning Outcomes (ILOs)
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    3.2 Body Composition, Determination and Weight control
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5.0 Summary
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1.0 INTRODUCTION

This unit describe the metabolic system during exercise.

2.0 INTENDED LEARNING OUTCOME (ILOS)

By the end of this unit, you will be able to;

- State the Nutrition for Fitness and Athletics
- Explain the Body composition and weight control

3.0 MAIN CONTENT

3.1 Nutrition for Fitness and Athletics

Proper nutrition and exercise are natural partners for health, fitness, and athletic performance. Consequently, many fitness enthusiasts pursue healthy diets, and athletes try to optimize their performance by implementing appropriate diets. Although these are very positive trends, they also have the potential to be taken to an extreme, which may simply involve spending money needlessly on “nutritional supplements” or may actually be harmful, as with eating disorders. It is the responsibility of all physical educators, athletic personnel, and fitness professionals to understand what constitutes optimal nutrition for fitness and athletics.

Individuals in exercise training need to match their training regimen with an appropriate diet. This often involves consultation with a fitness professional, a complete diet analysis by a nutritionist, and, many times, a trial-and-error approach to find what works best for a given individual.

The goals of an optimal training diet are to:

1. provide caloric and nutrient requirements
2. incorporate nutritional practices that promote good health
3. achieve and maintain optimal body composition and competition weight
4. promote recovery from training sessions and physiological adaptations
5. try variations of pre-competition and competition fuel and fluid intake to determine the body's responses.

There is almost universal agreement that poor nutritional status impairs work performance. There is also considerable, although not universal, agreement that good general nutrition (the balanced diet recommended for just about everyone, as shown in Table 6.1) is adequate and probably even optimal for most active individuals as well as sedentary individuals. All the essential classes of food are important in the nutrition for fitness and athletics.

Three categories of carbohydrate sources have been developed that are specifically marketed for active individuals and athletes: sports drinks, sports bars, and sports gels.

Sports Drinks

Liquid carbohydrate sources are as effective as solid carbohydrate sources. In fact, liquids may be even more useful, because many individuals are not hungry after an intense bout of exercise but are willing to drink liquid, and fluid replacement is also important. It compares several currently available commercial sports drinks containing carbohydrates. Several drinks also include protein. Many more with different formula are available. Note that sports drinks are not the same as "energy drinks," which are marketed for their mental stimulatory effects and sudden "bursts" of energy. Energy drinks typically contain caffeine and herbal ingredients and may or may not be safe or useful for active individuals. Although some sports drinks contain low amounts of stimulant caffeine, they are designed to maximize fluid absorption and support performance or recovery by delivering water, electrolytes, carbohydrate, and in some cases protein. Sports drinks with appropriate amounts of carbohydrate and electrolytes aid in the maintenance of homeostasis, prevent injuries, delay fatigue, and optimize performance. Those manufactured by reputable companies have generally been tested and found to be both safe and effective (Seebohar, 2007).

Sports Bars

It provides the macronutrient breakdown for selected sports bars and a Snickers candy bar for comparison purposes. Sports bars provide readily available carbohydrate and fall into two generic categories: high

carbohydrate (>60% of total calories) with minimal fat and protein; and minimal to moderate carbohydrate (20–55% of total calories) with balanced fat and protein (~22–40% of each).

High-carbohydrate bars are suitable for ingestion before, during, and after exercise. Fat consumed during exercise is not readily available for energy; moreover, fat slows digestion and can lead to stomach upset. Bars with 4 g or less of fat (per 230 kcal serving size) are fi ne for workouts, but bars with higher fat content are best used as dietary supplements or snacks after exercise. Before or during exercise, it is best to select sport bars with no more than 8–10 g of protein because higher protein amounts also slow digestion. Likewise, bars with more than 5 g of fiber should not be ingested before or during exercise because fiber also slows digestion. However, high-fiber bars can be good choices for snacks because fiber delays hunger pangs. If a sports bar is to be as a meal replacement, one with high protein should be selected. Similarly, high-protein bars can be used in recovery.

Sports Gels

Another type of packaged food for active individuals is the sport gel. Sport gels are products with a consistency of syrup or pudding and come in 0.75–1.4 oz plastic or foil packets. They contain 20–28 g (80–110 kcal) of carbohydrates per serving. Some gels contain electrolytes (especially potassium) and/or caffeine, and at least one contains protein, but none contains fat (Table 6.6). As with sports bars, it is important to ingest sufficient water (~4–8 oz per ounce of gel) and to try out a variety of gels during training to determine individual reactions before ingesting any during competition. If carbohydrates are not replenished between training bouts, local muscle fatigue will occur, and work output during succeeding training sessions will decline. Severe depletion followed by a nonoptimal diet will require more than 1 day of rest, which is one reason to alternate body parts and hard-easy exercise days.

3.2 Body Composition, Determination, Importance and Weight Control

Way to directly assess body composition is by dissection of human cadavers. Few bodies are available for such studies, however, and the technique is difficult and problematic. From 1945 to 1984 (from the earliest to the latest reported studies), body composition has been analyzed in only 40 cadavers. While these studies provided much information, particularly regarding the densities of body tissues, they were not used to determine the accuracy (validity) of the commonly used laboratory techniques for body composition assessment.

Theoretically, direct cadaver analysis should be the most accurate technique, but newer cadaver studies were often inconsistent with the results from the earlier studies. Thus, when dealing with body composition analysis, keep in mind that some techniques are better than others, and none is likely to be 100% accurate.

Many methods are currently available for the laboratory assessment of body composition. Several could possibly serve as criterion measures. Techniques such as densitometry (hydrostatic weight and air displacement plethysmography [Bod Pod]), hydrometry or dilution to determine total body water (TBW), nuclear techniques such as whole-body counting of potassium 40 or neutron activation, dual-energy X-ray absorptiometry (DXA), computed tomography (CT), magnetic resonance imaging (MRI), ultrasound, and total body electrical conductivity are all accurate enough to be used. Unfortunately, at this time, these techniques are also often expensive, cumbersome, and/or time-consuming, requiring sensitive instrumentation operated by highly trained technicians. The DXA technique is pictured and described because it is considered the "gold standard," or criterion measure, for the assessment of bone mineral density. However, DXA also allows the simultaneous measurement of fat and lean soft tissue for the determination of body composition. In the near future, DXA may become the criterion standard for body composition assessment as well. The Bod Pod also is frequently used. For now, however, pending more research, the criterion measurement for body composition is still hydrostatic, or underwater, weighing.

Hydrostatic (Underwater) Weighing: Densitometry

Hydrostatic, or underwater, weighing determines body composition through the calculation of body density (Behnke and Wilmore, 1974; Going, 2005; Goldman and Buskirk, 1961). We have Archimedes, a Greek mathematician who lived in the second century BC, to thank for this technique. When King Hieron of Syracuse commissioned a new crown, he suspected that the jeweler substituted silver for pure gold inside the crown. The king asked Archimedes to determine the composition of the crown without harming it in any way. Legend has it that as Archimedes was pondering this question at the public baths, he solved the problem and went running through the streets naked, shouting, "Eureka!" ("I have found it!").

What Archimedes observed was that an amount of water was displaced from the bath equal to the volume of the body entering the bath. Archimedes reasoned that the volume was proportional to the mass of the object and that the object's loss of weight in water equaled the weight of the volume of water displaced. We now define the mass of an object divided by its loss of weight in water as the *specific gravity* of that object. Archimedes also reasoned that the body (or any other object floating or

submerged) is buoyed up by a counterforce equal to the weight of the water displaced. Thus, Archimedes' principle states that a partially or fully submerged object experiences an upward buoyant force equal to the weight or the volume of fluid displaced by the object. Based on this principle, the volume of any object, including the human body, can be measured by determining the weight lost by complete submersion underwater. In the case of the human body, dividing the mass of the body by its volume defines *body density*. When Archimedes compared the amount of water displaced by a mass of pure gold and a mass of pure silver equal to the mass of the king's crown, he found that the crown displaced more water than the gold, but less than the silver. He confirmed this result by weighing in air and underwater, masses of gold and silver equal to the weight of the crown in air, and found the crown to have an intermediate specific gravity value. Thus, the crown was not pure gold (only about 75%), and we can only speculate as to the fate of the jeweler.

Field Tests of Body Composition

Field methods to determine body composition can be classified as anthropometry (measurement of the human body) or bioelectrical impedance analysis (BIA). Anthropometric techniques include skinfolds, height and weight, BMI, diameters, and circumferences. These techniques are generally practical, require a minimum of equipment, and (if properly applied) can provide useful, reasonably accurate information. They vary in the degree of skill needed by the tester.

Skinfolds

The most widely used anthropometric estimation of body size or composition involves the measurement of skinfolds at selected sites. Skinfolds (sometimes called fatfolds) are the double thickness of the skin plus the adipose tissue between the parallel layers of the skin. Because skin thickness varies only slightly among individuals, skinfold measures generally indicate the thickness of the subcutaneous fat. Technically, however, adipose tissue (and thus the subcutaneous fatfold) has both a fat component and a fat-free component. The fat-free component is composed of water, blood vessels, and nerves. As the fat content of the adipose tissue increases (as in obesity), the water content decreases. The use of skinfold thicknesses to estimate body composition is based on two assumptions. The first is that selected skinfold sites are representatives of the total subcutaneous adipose tissue mass. In general, evidence supports this assumption. The second assumption is that the subcutaneous tissue mass has a known relationship with total body fat.

Height and Weight

In some situations, such as when large numbers of individuals are being evaluated, the only measures of body size that can easily be obtained are height and weight. These values are often used along with height and

weight charts that include standards such as acceptable body weight (<10% over the chart weight for a given height), overweight (10–20% over the chart weight), and obese (>20% over the chart weight) sometimes by frame size. Such use of height and weight charts is minimally acceptable for large group data but is not recommended as a source of what an individual "should" weigh, for the following reasons.

Body Mass Index

Body Mass Index (BMI) is a ratio of the total body weight to height. Several ratios have been proposed, but the one used most frequently is weight (in kilograms) divided by height (in meters) squared [WT ÷ HT2 (kg.m−2)]. This ratio is also known as the Quetelet index. Calculated BMI can then be compared against standard values to determine whether the individual has acceptable body weight, is overweight, or is obese. The selection of BMI values at the upper limit of acceptable is based on the data relating BMI to morbidity (disease occurrence, particularly cardiovascular disease [CVD]) and mortality (death).

As shown in this figure, for adults there is a curvilinear increase in excess mortality (a greater number of deaths than expected in a given population) with an increasing BMI. Significant increases in risk begin at a BMI of 27.3 for females and 27.8 for males. Therefore, in 1985, the National Institute of Health defined obesity as a BMI of 27.3 for females and 27.8 for males.

Waist-to-hip Ratio/Waist Circumference

Waist-to-hip (W/H) ratio is another way to estimate health risk based on the individual's pattern of fat distribution. Research has shown that the W/H ratio is a stronger predictor for diabetes, coronary artery disease, and overall death risk than body weight, BMI, or %BF in adults. In the absence of large total fat stores, the W/H can aid in the identification of certain hormonal and metabolic abnormalities associated with a relative increase in abdominal.

Bioelectrical Impedance (Impedance Plethysmography)

Determining body composition by Bioelectrical Impedance (BIA) has gained a great deal of acceptance in fitness facilities primarily because the procedure can easily be done. In BIA, four electrodes are attached to a quietly resting supine subject's hands and feet (two per limb either ipsilaterally or contralaterally). A harmless, sensationless, low- amperage (80 mA), radio frequency (50 kHz) electrical current is passed between the electrodes, and the resistance (in ohms) to the current is recorded. Body volume is assumed to be a cylinder of constant cross-sectional area with uniform density distribution. Body volume is more often defined as height squared divided by resistance (HT2 ÷ R). The ability to conduct the electrical current is directly related to the amount of water and

electrolytes in the various body tissues. Electrical current flows more easily in fat-free tissue (which offers less resistance) than in fat tissue because the fat-free tissue has a higher percentage of water and electrolytes. Therefore, individuals with more FFW have lower resistance values, and those with more fat weight have higher resistance values.

Overweight and Obesity

Although being overweight is what bothers most people, it is really the amount and location of fat (%BF, abdominal fat mass) that should be of concern. Excess weight can be caused by high levels of lean muscle mass, but additional muscle mass is beneficial. Except in rare instances, such as providing protection from the cold water for an English Channel swimmer or certain wasting diseases, excess fat is generally not beneficial. There are no universally agreed upon acceptable %BF standards. The most typically used normal values for young adults (20–29 yr) are 12–15% for males and 22–25% for females with an allowance of an additional 2% for each decade of age. Obesity is defined as +5% BF above the normal value.

SELF-ASSESSMENT EXERCISES

i. List the Nutrition necessary for fitness and athletics.
ii. Define Body Composition

4.0 CONCLUSION

Having read this course and successfully completed the assessment and self-assessment test, it is assumed that you have attained understanding of the introductory knowledge on the Anaerobic and Aerobic Metabolism.

5.0 SUMMARY

This Unit has summarized the Anaerobic and Aerobic metabolism in exercise. It also looked at the anaerobic and aerobic exercise response. The Anaerobic characteristics in exercise metabolism was discussed. The assessment and self-assessment exercise have been provided to enable you understand your own rating of the understanding and learning you achieved while reading this material in this Unit. Online links have also been provided to broaden your understanding of the learning required in this Unit.

6.0 TUTOR-MARKED ASSIGNMENT

1. State the importance of body composition and weight control

7.0 REFERENCES/FURTHER READING

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UNIT 5 ANAEROBIC & AEROBIC METABOLISM DURING EXERCISE

CONTENTS

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  3.2 The Anaerobic Exercise Response
  3.3 Anaerobic Exercise Characteristics
  3.4 Aerobic and Measurement of Aerobic Metabolism
  3.5 The Aerobic Exercise Response
4.0 Conclusion
5.0 Summary
6.0 Tutor-Marked Assignment
7.0 References/Further Reading

1.0 INTRODUCTION

This unit describe the anaerobic and aerobic metabolism during exercise.

2.0 INTENDED LEARNING OUTCOME (ILOS)

By the end of this unit, you should be able to;

- Define Anaerobic and Aerobic Metabolism
- State the measurement of Anaerobic and Aerobic measurement
- Explain the Anaerobic and Aerobic Exercise Response

3.0 MAIN CONTENT

3.1 Anaerobic and Measurement of Anaerobic Metabolism

This describes *alactic anaerobic metabolism*, sometimes called the *phosphagen* or *ATP-PC system*. Once produced, ATP is stored in the muscle. This amount is relatively small and can provide energy for only 2–3 seconds of maximal effort. However, another high-energy compound, phosphocreatine (PC), also known as creatine phosphate (CP), can be used to resynthesize ATP from ADP almost instantaneously. The amount of PC stored in muscle is about three times that of ATP. Muscles differ in the amount of stored PC by fiber type. Fibers that produce energy predominantly by anaerobic glycolysis are called glycolytic; those that produce energy predominantly aerobically are called oxidative. In terms of contraction speed, muscle fibers are either fast twitch or slow twitch.

When contractile and metabolic characteristics are combined, three fiber types are generally described: fast-twitch glycolytic (FG also known as Type IIX), fast-twitch oxidative glycolytic (FOG also known as Type IIA), and slow-twitch oxidative (SO). Fast-twitch fibers have proportionally more PC than ATP compared to slow-twitch oxidative fibers. Any time the energy demand is increased—whether the activity is simply turning a page of this book, coming out of the blocks for a sprint, or starting out on a long bicycle ride—at least part of the immediate need for energy is supplied by these stored forms (ATP, PC), which must ultimately be replenished. These sources are also used preferentially in high-intensity, very short-duration activity. Most re-synthesis of ATP from PC takes place in the first 10 seconds of maximal muscle contraction; little, if any, occurs after 20 seconds of maximal activity.

This ATP-PC system neither uses oxygen nor produces lactic acid (LA) and is thus said to be *alactic anaerobic*. When the demand for ATP exceeds the capacity of the phosphagen system and the aerobic system (at the initiation of any activity or during high- intensity, short-duration exercise), *fast* (anaerobic) glycolysis is used to produce ATP. This is rather like calling in the reserves, for glycolysis can provide the supplemental energy quickly. The rate of ATP production from glycolysis reaches its maximum about 5 seconds after initiation of contraction and is maintained at this rate for several seconds. This system predominates in activities such as a 1500-m speed skating event. Other sport activities with a heavy reliance on the LA system include middle distances (e.g., track 200–800 m, swimming 100 m, slalom and downhill skiing); gymnastic floor exercise; parallel bars; a round of boxing; and a period of wrestling. The ability to perform the events with speed and power is the benefit. The cost is that the production of lactic acid often exceeds clearance, and lactate accumulates. Because this system does not use oxygen but does result in the production of lactic acid, it is said to be *lactic anaerobic*.

The generation of ATP from *slow* (aerobic) glycolysis, the Krebs cycle, and electron transport–oxidative phosphorylation is constantly in operation at some level. In resting conditions, this system provides basically all of the energy needed. When activity begins or occurs at moderate levels of intensity, oxidation increases quickly and proceeds at a rate that supplies the needed ATP. If the workload is continuously increased, aerobic oxidation proceeds at a correspondingly higher rate until its maximal limit is reached. The highest amount of oxygen the body can consume during heavy dynamic exercise for the aerobic production of ATP is called maximal oxygen uptake, or VO_2max. Because VO_2max is primarily an index of cardiorespiratory power, and as such is used as a measure of cardiovascular-respiratory fitness, it is discussed in depth in that unit. However, because VO_2max reflects the amount of oxygen

available for the aerobic production of ATP, it is also an important metabolic measure. Both aerobic and anaerobic exercises are often described in terms of a given percentage of VO_2max (either less than or greater than 100% VO_2max). Anaerobic metabolic processes are important at the onset of all aerobic exercise, contribute significantly at submaximal levels, and increase their contribution as the exercise intensity gets progressively higher. Depending on an individual's fitness level, lactic anaerobic metabolism begins to make a significant contribution to dynamic activity at approximately 40–60% VO_2max. However, even then the ability to use oxygen is most important. Historically, the contribution of aerobic metabolism to intense exercise has probably been underestimated and the contribution of anaerobic metabolism overestimated because of the difficulty in measuring the anaerobic metabolism accurately. By between 1 and 2 minutes of maximal exercise, the relative contributions of ATP production from the aerobic and anaerobic energy systems are approximately equal. Because the aerobic system involves the use of oxygen and proceeds completely to oxidative phosphorylation, it is said to be *aerobic* or *oxidative*. Sport activities that rely predominantly on the O2 system include long distance events, such as the 5000 and 10,000 m in track; marathons; swimming 1500 m; cross-country running, skiing, and orienteering; field hockey, soccer, and lacrosse; and race walking.

These three sources of ATP; the phosphagen system (ATP-PC), the glycolytic system (LA), and the oxidative system (O_2) contribute to maximal exercise of different durations in a pattern called the *time-energy system continuum*. This continuum assumes that the individual is working at a maximal maintainable intensity for a continuous duration. This means that it is assumed that an individual can go all out for 5 minutes or less or can work at 100%VO_2max for 10 minutes, at 95% VO_2max for 30 minutes, at 85% VO_2max for 60 minutes, and at 80% VO_2max for 120 minutes. Of course, there are individual differences, but these assumptions are reasonable in general. Note that the anaerobic systems respond immediately but cannot sustain the high level of ATP production. Conversely, the aerobic energy system (O_2) is incapable of meeting the immediate energy demands but contributes to a meaningful degree surprisingly quickly. By 30 seconds almost 27% of the ATP is being supplied by oxidative phosphorylation. The point at which the aerobic and anaerobic contribution becomes approximately equal is 75 seconds.

Four basic patterns can be discerned from this continuum. Understanding these patterns is helpful when developing training programs.

1. All three energy systems (ATP-PC, LA, and O_2) are involved in providing energy for all durations of exercise.

2. The ATP-PC system predominates in activities lasting 10 seconds or less. Since the ATP-PC system is involved primarily at the onset of longer activities, it becomes a smaller portion of the total energy supply as the duration gets longer
3. Anaerobic metabolism (ATP-PC and LA) predominates in supplying energy for exercises lasting between 1 and 2 minutes. The equal contribution point for anaerobic and aerobic energy contribution to maximal exercise is probably close to 75 seconds. However, even exercises lasting as long as 10 minutes use at least 15% anaerobic sources. Within the anaerobic component the longer the duration, the greater the relative importance of the lactic acid system is in comparison to the phosphagen system.
4. By 2 minutes of exercise, the O_2 system clearly dominates. The longer the duration, the more important it becomes.

Measurement of Anaerobic Metabolism

Unfortunately, there is no generally accepted means by which to directly measure the contribution of anaerobic energy to exercise. Two general approaches are used, One approach describes changes in the chemical substances either used in alactic anaerobic metabolism (specifically, ATP and PC levels) or produced as a result of lactic anaerobic metabolism (lactic acid or lactate). The second approach quantifies the amount of work performed or the power generated during short-duration, high-intensity activity. The assumption is that such activity could not be done without anaerobic energy; therefore, measuring such work or power indirectly measures anaerobic energy utilization and provides an indication of anaerobic capacity.

ATP-PC and Lactate

Muscle ATP, PC, and lactate can be measured by chemical analysis of muscle biopsy specimens. Lactate is the most frequently measured variable, in part because it can also be measured from blood samples. The blood sample may be obtained by venipuncture or finger prick, both of which are less invasive than a muscle biopsy. Another reason for the popularity of lactate analysis is the availability of user-friendly, fast, accurate, and relatively inexpensive analyzers.

Tests of Anaerobic Power and Capacity

Energy system capacity is the total amount of energy that can be produced by an energy system. Energy system power is the maximal amount of energy that can be produced per unit of time. The lactic anaerobic glycolytic system has almost equal power and capacity, just slightly favoring capacity. The information on the aerobic (O_2) system is included here just to show how truly high in power and low in capacity the anaerobic systems are.


A B

Lactate Analysis.

A small sample of blood is obtained by a finger prick (A) and injected into a lactate analyzer (B) for determination of lactate concentration [La−].

The Wingate Anaerobic Test

The Wingate Anaerobic Test depicted in Figure 3.7 is probably the most well-known of several cycle ergometer tests used to measure anaerobic power and capacity. The test is an all-out ride for 30 seconds against a resistance based on body weight. Both arm and leg versions are available, although the leg test is most frequently used and will be the only version discussed here. Resistance values of 0.075 $kg \cdot kg^{-1}$ body weight for children, 0.086 $kg \cdot kg^{-1}$ body weight for adult females, and 0.095 $kg \cdot kg^{-1}$ body weight for adult males appear to be optimal. Athletes may need values as high as 0.10 $kg \cdot kg^{-1}$ of body weight, but the most common value used (as in the example that follows) is 0.075 $kg \cdot kg^{-1}$ body weight. The revolutions (rev) of the flywheel are counted per second during the test, and from the available information three variables are determined.

The Margaria-Kalamen Stair Climb

To perform the Margaria-Kalamen test, an individual run for 6 m on the level and then climbs a staircase, taking three steps at a time. Power in $kgm \cdot sec^{-1}$ is calculated from the weight of the subject, the vertical height between the third and ninth steps, and the time between the third and ninth steps. The use of electronic switch mats or photoelectric cells is essential for accuracy. This test is considered to be a test of alactic anaerobic power because of the short time involved usually less than 5 seconds for the entire test and close to 1 second for the measured time between the third and ninth steps.

3.2 The Anaerobic Exercise Response

When exercise begins, regardless of how light or heavy it is, there is an immediate need for additional energy. Thus, the most obvious exercise response is an increase in metabolism. All three energy systems are involved in this response, their relative contributions being proportional to the intensity and duration of the activity. Figure 3.8 shows two scenarios for going from rest to different intensities of exercise. The oxygen requirement for this exercise is 1.4 L·min−1. The individual has a VO_2max of 2.5 L·min−1. Therefore, this individual is working at 56% VO_2max. The area under the smoothed curve during both exercise and recovery represents oxygen used. Notice, however, that there is an initial lag during which the oxygen supplied and utilized is below the oxygen requirement for providing energy. This difference between the oxygen required during exercise and the oxygen supplied and utilized is called the oxygen deficit. Because of this discrepancy between supply and demand, anaerobic sources must be involved in providing energy at the onset of all activity. The O_2 deficit has traditionally been explained as the inability of the circulatory and respiratory systems to deliver oxygen quickly enough to meet the increased energy demands. Evidence now indicates, however, that the O_2 deficit is probably due to limited cellular utilization of O_2 as a result of metabolic adjustments in both the anaerobic and aerobic systems.

The relatively slow response time of aerobic ATP production is determined by the faster speed of response of the ATP-PC system and by the content of mitochondria in the muscle. The fact that the ATP-PC system responds rapidly does not indicate any intrinsic delay or inertia of activation for either the LA or O_2 system. All three pathways respond simultaneously in an integrated fashion. The metabolic response is regulated by a series of feedback control systems that are sensitive to the release of Ca_{2+} from muscle contraction and the breakdown of ATP. The metabolic systems simply respond at different speeds. Therefore, during the transition from rest to work, energy is supplied by;

1. O_2 transport and utilization;
2. utilization of O_2 stores in capillary blood and bound to myoglobin;
3. the splitting of stored ATP-PC; and
4. anaerobic glycolysis, with the concomitant production of LA.

Eventually, if the exercise intensity is low enough, the aerobic system will predominate and the oxygen supply will equal the oxygen demand. This condition is called steady-state, steady rate, or steady-level exercise. Figure 3.8B shows a smoothed plot of O_2 consumption at rest and during and after an exercise bout in which the energy requirement is greater than VO_2max, sometimes called supramaximal exercise. The initial lag period between O_2 supply and demand is once again evident, and as in the first

example, the added energy is provided by stored ATP-PC, anaerobic glycolysis, and stored O_2. However, in this case, when the O2 consumption plateaus or levels off, it is at VO_2max, and more energy is still needed if exercise is to continue.

3.3 Anaerobic Exercise Characteristics

The anaerobic characteristics of females are generally lower than those of males in the young and middle-aged adult years. Much of the difference is undoubtedly related to the smaller overall muscle mass of the average female compared with that of the average male.

The Availability and Utilization of ATP-PC
Neither the local resting stores of ATP per kilogram of muscle nor the utilization of ATP-PC during exercise varies between the sexes. However, in terms of total energy available from these phosphagen sources, males will have more than females because of muscle mass differences.

The Accumulation of Lactate
Resting levels of lactate are the same for males and females. Lactate thresholds, when expressed as a percentage of VO_2max, are also the same for both sexes, although the absolute workload at which the lactate thresholds occur is higher for males than females. Thus, at any given absolute workload that is still submaximal but above LT1 or LT2, females have a higher lactate value than males. Consequently, the workload is more stressful for females and requires a greater anaerobic contribution. However, at a given relative workload or percentage of VO2max above the lactate thresholds, lactate concentrations are equal for both sexes. Lactate values at maximal exercise from ages 16 through 50 are higher by approximately 0.5–2.0 mmol·L−1 for males than for females. Once again, females are generally doing less in terms of an absolute workload than males at maximum.

Mechanical Power and Capacity
As previously mentioned, on average males produce higher absolute work output than females. Data available from the Wingate Anaerobic Test show that values for peak power for women are approximately 65% of values for men if expressed in watts, improve to 83% if expressed in watts per kilogram of body weight, and come close to being equal at 94% when expressed in watts per kilogram of lean body mass. The corresponding comparisons for mean power are 68%, 87%, and 98%, respectively. The peak power of women (in watts per kilogram of body weight) is very similar to the mean power of men. The fatigue index does not show a significant sex difference, indicating that both sexes tire at the same rate. It has been shown that females provide a relatively higher portion of the energy for the WAT aerobically than males. This may mean that the total

power output during a WAT actually underestimates the sex difference in anaerobic capacity between males and females. The maximal accumulated oxygen deficit (MAOD) expressed relative to the active muscle mass for cycling is significantly higher in males than females.

3.4 Aerobic and Measurement of Aerobic Metabolism

Aerobic metabolism can be measured directly by calorimetry (the measurement of heat production) or indirectly by spirometry (the measurement of air breathed and the analysis of oxygen and carbon dioxide gases). Typically, open-circuit indirect spirometry/ open-circuit indirect calorimetry is used. This system has a large error and is rarely used. It is mentioned here so that the terminology of an open circuit can be understood. In *open-circuit spirometry*, the subject inhales room or outdoor air from his or her surroundings and exhales into the same surroundings. The oxygen content of the inhaled air is normally 20.93%; the carbon dioxide does not need to be absorbed but is simply exhaled into the surrounding atmosphere. A sample of the expired air is analyzed for oxygen and carbon dioxide content. Putting these factors together results in the descriptor *open-circuit indirect spirometry*. The term *open-circuit indirect calorimetry* should technically be reserved for use when calories are calculated from oxygen consumption, but, in fact, that term is often used interchangeably with open circuit indirect spirometry and we will do so in this text.

Measuring oxygen consumption by open-circuit indirect spirometry is a valid way to assess aerobic metabolism during resting and steady-state submaximal exercise, conditions when the relationship between oxygen consumption and ATP production remains linear. However, in situations also involving anaerobic energy production, the actual energy cost of the exercise will be underestimated, because the linear relationship no longer exists and there is no way to account for the anaerobic portion. Open-circuit indirect spirometry can be used to measure oxygen consumption during any physical activity. However, the size, sensitivity, and lack of portability of the equipment have, until recently, limited its use to modalities that can be performed in a laboratory or a special swimming pool setup. By far, the most popular exercise testing modalities in the laboratory are the motor-driven treadmill and the cycle ergometer. Measurements can be performed with the subject at rest, during submaximal exercise, or at maximal levels of exertion. The following sections describe in detail how these measurements are done and the aerobic responses to varying patterns of exercise.

The Aerobic Exercise Responses

The individual uses a breathing valve, which permits air to flow in only one direction at a time in from room air and out toward the sampling

chamber. The nose clip ensures that the individual breathes only through the mouth.

Oxygen Consumption and Carbon Dioxide Production

This describes schematic configurations of an open circuit system in which the volume of either inspired air or expired air is measured and the expired air is analyzed for the percentage of oxygen and carbon dioxide. Although oxygen consumption is the variable of primary interest, because of its direct relationship with ATP, determining the amount of carbon dioxide produced is also important, because that measure enables a determination about fuel utilization and caloric expenditure. Oxygen consumption (VO_2) is technically the *amount* of oxygen taken up, transported, and used at the cellular level. It equals the amount of oxygen inspired minus the amount of oxygen expired. However, as the symbol VO_2 indicates, it is commonly labeled as the *volume* of oxygen consumed. Similarly, carbon dioxide produced (VCO_2) is technically the amount of carbon dioxide generated during metabolism, primarily from aerobic cellular respiration. It equals the amount of carbon dioxide expired minus the amount of carbon dioxide inspired. As with VO_2, VCO_2 is commonly described as the volume of carbon dioxide produced. The *amount* of a gas equals the volume of air (either inhaled or exhaled) times the percentage of the gas. Therefore, to determine these amounts, the volume of air either inhaled or exhaled is measured, as are the percentages of oxygen and carbon dioxide in the exhaled air. The percentages of oxygen and carbon dioxide in inhaled air.

The Oxygen Cost of Breathing

Part of the oxygen used both at rest and during exercise goes to support the respiratory muscles. This value does not remain constant but varies with the intensity of activity. During rest, the respiratory system uses about 1–2% of the total body oxygen consumption, or 2.5 $mL \cdot min^{-1}$ of oxygen. The oxygen cost of ventilation is higher in children than in middle-aged or older adults. During light to moderate submaximal dynamic aerobic exercise, where VE is less than 60 $L \cdot min^{-1}$, the respiratory oxygen cost changes to about 25–100 $mL \cdot min^{-1}$. At heavy submaximal exercise, when VE is between 60 and 120 $L \cdot min^{-1}$, respiratory oxygen use may rise to 50 to 400 $mL \cdot min^{-1}$. During incremental exercise to maximum, the initial VE during the lower exercise stages shows a very gradual curvilinear rise, reflecting the submaximal changes described previously. At workloads above those requiring a VE greater than 120 $L \cdot min^{-1}$, a dramatic exponential curve occurs. In this curve, by the time a VE of 180 $L \cdot min^{-1}$ is achieved in a very fi t individual, 1000–1300 $L \cdot min^{-1}$ of oxygen is used simply to support respiration. Theoretically, there may be a maximal level of ventilation above which any further increase in oxygen consumption would be used entirely by the ventilatory musculature, thus limiting

maximal exercise. At what precise point this critical level of ventilation occurs is unknown. However, even if a critical ventilation level does not exist, respiration does utilize a significant portion, 3–18%, of the VO_2 during heavy exercise. Smoking increases the oxygen cost of respiration during exercise. However, an abstinence of even one day can substantially reduce this effect of cigarette smoking. In old age, the higher oxygen cost of breathing may be a significant factor in limiting exercise performance.

SELF-ASSESSMENT EXERCISES

i. List and define the variables used to describe aerobic metabolic responses to exercise.
ii. Define Anaerobic metabolism and state its measurements
iii. Define Aerobic metabolism and state its measurement

4.0 CONCLUSION

Having read this course and successfully completed the assessment and self-assessment test, it is assumed that you have attained understanding of the introductory knowledge on the Anaerobic and Aerobic Metabolism.

5.0 SUMMARY

This Unit has summarized the Anaerobic and Aerobic metabolism in exercise. It also looked at the anaerobic and aerobic exercise response. The Anaerobic characteristics in exercise metabolism was discussed. The assessment and self-assessment exercise have been provided to enable you understand your own rating of the understanding and learning you achieved while reading this material in this Unit. Online links have also been provided to broaden your understanding of the learning required in this Unit.

6.0 TUTOR-MARKED ASSIGNMENT

1. Describe the Anaerobic Exercise response
2. State the characteristics of Anaerobic metabolism in exercise

7.0 REFERENCES/FURTHER READING

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MODULE 2 CARDIOVASCULAR- RESPIRATORY SYSTEM

INTRODUCTION

Respiration consists of four separate processes. The first is pulmonary ventilation, in which air is moved into and out of the body. The second, external respiration, involves the exchange of oxygen and carbon dioxide between the lungs and the blood. The third is internal respiration, which involves the exchange of oxygen and carbon dioxide at the cellular or tissue level. Finally, cellular respiration is the utilization of oxygen to produce energy, which also produces carbon dioxide as a byproduct.

Unit 1 Cardiovascular- Respiratory System
Unit 2 Mechanism of Breathing
Unit 3 Respiratory Circulation
Unit 4 Measurement of Lung Volumes

UNIT 1 INTRODUCTION AND STRUCTURE OF PULMONARY SYSTEM

CONTENTS

1.0 Introduction
2.0 Intended Learning Outcomes (ILOs)
3.0 Main Content
  3.1 The concept and structure of Pulmonary System
  3.2 The Conducive Zone
  3.3 Respiratory Zone
4.0 Conclusion
5.0 Summary
6.0 Tutor-Marked Assignment
7.0 References/Further Reading

1.0 INTRODUCTION

This unit describes the concept and the structure of Pulmonary system.

2.0 INTENDED LEARNING OUTCOMES (ILOS)

By the end of this Unit, you will be able to;

- Describe the Pulmonary system
- Differentiate Conducive zone and respiratory zone

3.0 MAIN CONTENT

3.1 The concept and structure of Pulmonary System

Although it is typical to think of respiration as being the same as breathing and/or ventilation, technically, it is not. The volume of air flowing into the lungs from the external environment through either the nose or the mouth is called pulmonary ventilation. Ventilation is accomplished by breathing, the alternation of inspiration and expiration that causes the air to move. The actual exchange of the gases oxygen (O2) and carbon dioxide (CO2) between the lungs and the blood is known as external respiration. At the cellular level, oxygen and carbon dioxide gases are again exchanged; this exchange is called internal respiration. Cellular respiration is the utilization of oxygen by the cells to produce energy with carbon dioxide as a byproduct. Cellular respiration includes both aerobic (with O_2) and anaerobic (without O_2) energy production and is discussed in Chapter 2. This chapter concentrates on pulmonary ventilation, external respiration, and internal respiration.

Structure of the Pulmonary System
The respiratory system consists of two major portions:

(1) the conductive zone, which transports the air to the lungs, and
(2) the respiratory zone, where gas exchange takes place.

3.2 The Conductive Zone

The basic structure of the conductive zone is shown in Figure 9.2. The structures from the nose or mouth to the terminal bronchioles comprise the *conductive zone*. The primary role of the conductive zone is to transport air. Because no exchange of gases takes place here, this zone is also called *anatomical dead space*. As a general guideline, the amount of anatomical dead space can be estimated as 1 mL for each 1 lb of “ideal” body weight (Slonim and Hamilton, 1976). Hence, a 130-lb female who is at her ideal weight has an estimated 130 mL of anatomical dead space. However, if this individual were to gain 20 lb, she would still have the same anatomical dead space and that estimate would remain at 130 mL. Anatomical dead space is important in determining the alveolar ventilation, as discussed later. The second important role of the conductive zone is to warm and humidify the air. By the time the air reaches the lungs, it will be warmed to body temperature (normally ~37°C) and will be 99.5% saturated with water vapor.

This protective mechanism helps maintain core body temperature and protects the lungs from injury (Slonim and Hamilton, 1976). The warming and humidifying of air is easily accomplished over a wide range of

environmental temperatures under resting conditions, when the volume of air transported is small and the air is inhaled through the nose. During heavy exercise, however, large volumes of air are inhaled primarily through the mouth, thus bypassing the warming and moisturizing sites of the nose and nasal cavity. As a result, the mouth and the throat may feel dry. If heavy exercise takes place in cold weather (especially at subzero temperatures), dryness increases and throat pain may be felt. These uncomfortable feelings are not a symptom of freezing of the lungs; rather, they are the result of the drying and cooling of the upper airway. The lower portions of the conductive zone still moisturize and warm the air sufficiently before it reaches the lungs. A scarf worn across the mouth will trap moisture and heat from the exhaled air and thereby decrease or eliminate the uncomfortable sensations. The third role of the conductive zone is to filter the incoming air. The nasal cavity, pharynx, larynx, trachea, and bronchial system are all lined with ciliated mucous membranes (Figure 9.2). These membranes with their hair-like projections trap impurities and foreign particles (particulates) that are inhaled. Both smoke and environmental air pollutants diminish ciliary activity and can ultimately destroy the cilia.

3.3 The Respiratory Zone

The *respiratory zone* consists of the respiratory bronchioles, the alveolar ducts, alveolar sacs (grape-like clusters), and the alveoli (Figure 9.2). The alveoli are the actual site of gas exchange between the pulmonary system and the cardiovascular system. At birth, humans have about 24 million alveoli. This number increases to about 300 million by 8 years of age and remains constant until age 30, when it begins a gradual decline. Although each individual alveolus is small, only about 0.2 mm in diameter, collectively the alveoli in a young adult have a total surface area of 50–100 m2 (West, 2005). This area would cover a badminton court or even a tennis court if flattened out. Despite this large surface area, the lungs weigh only about 2.2 lb (1 kg). The volume of the respiratory zone is about 2.5–3.0 L·min−1 (West, 2005). The membrane between the alveoli and the capillaries is actually composed of five very thin layers, two of which are the endothelial cells of the alveoli and the capillaries. The endothelium of the alveoli produces a substance called surfactant that reduces surface tension and helps prevent alveoli from collapsing (Seifter et al., 2005). Despite the number of layers, the thickness is less than the paper this book is printed on, and gas exchange takes place easily (West, 2005).

In addition to the anatomical dead space where no exchange takes place, some alveoli have no capillary blood supply and therefore cannot participate in gas exchange; these alveoli make up an *alveolar dead space*. The anatomical plus the alveolar dead space combined make up the

physiological dead space. Because alveolar dead space is minimal in healthy individuals, the physiological dead space is only slightly larger than the anatomical dead space (West, 2005).

SELF-ASSESSMENT EXERCISES

i. Describe the structure of the pulmonary system
ii. Differentiate between Conducive and Respiratory zone.

4.0 CONCLUSION

Having read this course and successfully completed the assessment and self-assessment test, it is assumed that you have attained understanding of the introductory knowledge on structure of the pulmonary system.

5.0 SUMMARY

This Unit has successfully summarized the introduction to pulmonary system and then the structure.

6.0 TUTOR-MARKED ASSGINMENT

7.0 REFERENCES/FURTHER READING

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UNIT 2 MECHANISM OF BREATHING

CONTENTS

1.0 Introduction
2.0 Intended Learning Outcomes (ILOs)
3.0 Main Content
    3.1 The Mechanism of breathing
    3.2 The Application of Boyle's law in the pulmonary system
4.0 Conclusion
5.0 Summary
6.0 Tutor-Marked Assignment
7.0 References/Further Reading

1.0 INTRODUCTION

This unit describes the mechanism of breathing in Pulmonary system.

2.0 INTENDED LEARNING OUTCOMES (ILOS)

By the end of this Unit, you will be able to;

- Define breathing
- State the factors responsible in the mechanism of breathing in the pulmonary system.

3.0 MAIN CONTENT

3.1 The Mechanism of breathing

The movement of air into the lungs from the atmosphere depends on two factors: pressure gradient (ΔP) and resistance (R).

A pressure gradient is simply the difference between two pressures. Difference is represented by the Greek capital letter delta: Δ. The larger the differences in pressure, the larger the pressure gradient is. Gases—in this case, air which is a mixture of gases—move from areas of high pressure to areas of low pressure. Resistance is the sum of the forces opposing the flow of the gases. About 20% of resistance to airflow is caused by tissue friction as the lungs move during inspiration and expiration. The remaining 80% is due to the friction between the gas molecules and the walls of the airway (airway resistance) and the internal friction between the gas molecules themselves (viscosity). Airway resistance is determined by the size of the airway and the smoothness or turbulence of the airflow.

In order for air to flow, the pressure gradient must be greater than the resistance to the flow. Thus, for inspiration to take place, pressure must be higher in the atmosphere than in the lungs; for expiration, pressure in the alveoli of the lungs must be higher than in the atmosphere. It shows how the inspiratory pressure gradient is created.

3.2 Boyle's Law and Mechanism of breathing

Boyle's law states that the pressure of a gas is inversely related to its volume (or vice versa) under conditions of constant temperature: low pressure is associated with large volume, and high pressure is associated with small volume. For pulmonary ventilation, an increase in chest cavity volume is accomplished by muscle contraction for inspiration. This increase in volume leads to an internal lung pressure decrease according to Boyle's law. As a result, a negative pressure exists in the chest cavity relative to the atmosphere outside the body. Thus, a pressure gradient has been created. Air flows into the chest cavity in an attempt to equalize this pressure difference. The volume change per unit of pressure is called *compliance* (West, 2005).

The main inspiratory muscle is the dome-shaped diaphragm. With neural stimulation, the diaphragm contracts and moves downward, elongating the chest cavity (Figure 9.3B). In normal resting breathing, the diaphragm moves about 1 cm; in heavy or forced breathing, it may move as much as 10 cm (West, 2005).

During exercise, the chest cavity is further enlarged by the action of the external intercostal muscles and others, known collectively as the accessory muscles, which elevate the rib cage and cause expansion both laterally (side-to-side) and anteroposteriorly (front-to-back). The extent of accessory muscle activity and the resultant drop in pressure depends on the depth of the inspiration. These changes in the chest cavity volume transfer themselves to the lungs through the pleura. Pleurae are thin, double-layered membranes that line both the chest cavity (the inner surfaces of the thorax, sternum, ribs, vertebrae, and diaphragm) and the external lung surfaces. The portion covering the chest cavity is called the parietal pleura; the portion covering the external lung surfaces is called the visceral or pulmonary pleura. A fluid secreted by the pleura fills the space between the pleurae (the intrapleural space), allowing the lungs to glide smoothly over the chest cavity walls. It also causes the parietal and the pulmonary pleurae to adhere to each other in the same way that two pieces of glasses are held together by a thin film of water. Because of this adhesion, the lungs themselves move when muscle actions move the chest cavity (Guyton and Hall, 2006; Martin et al., 1979).

During normal resting conditions, expiration occurs simply because the diaphragm and other inspiratory muscles relax. When these muscles relax, both the lungs and the muscles, which are highly elastic, recoil to their original positions. This elastic recoil decreases lung volume and thus creates a pressure inside the chest cavity that is higher than the atmospheric pressure. As the chest cavity volume decreases, the intrathoracic pressure increases slightly above that of the atmosphere. The result is that the air moves out of the lungs into the atmosphere. The pressures equalize again, and the cycle repeats with the next inspiration. A complete respiratory cycle includes both inspiration and expiration. During heavy breathing, as in exercise, expiration is an active process. The primary expiratory muscles are the abdominals and the internal intercostals. The abdominals (rectus abdominus, the obliques, and the transverse abdominus) push the abdominal organs—and hence the diaphragm—upward; the internal intercostals pull the ribs inward and down. This decrease in chest volume increases intrathoracic pressure more quickly than passive elastic recoil alone, and the air is forced out of the lungs faster. The pleurae also serve a purpose during expiration.

Pressure in the intrapleural space fluctuates with breathing in a way that parallels pressure within the lungs. However, the intrapleural pressure is always negative (24–28 mmHg) relative to the intrapulmonary (lung) pressure. This negative pressure protects the lungs from collapsing. If the intrapleural pressure were equal to the atmospheric pressure, the lungs would collapse at the end of expiration because of the elastic recoil. Because muscle activity is involved during the respiratory cycle of inhalation and exhalation, energy is consumed. During rest, however, this energy consumption (restricted to inspiratory muscles) amounts to only 1–2% of the total body energy expenditure in nonsmokers (Pardy et al., 1984).

SELF-ASSESSMENT EXERCISE

i. Describe the structure of the pulmonary system
ii. Differentiate between Conducive and Respiratory zone.

4.0 CONCLUSION

Having read this course and successfully completed the assessment and self-assessment test, it is assumed that you have attained understanding of the introductory knowledge on mechanism of breathing.

5.0 SUMMARY

This Unit has successfully summarized the mechanism behind every form of breathing.

6.0 TUTOR-MARKED ASSIGNMENT

7.0 REFERENCES/FURTHER READING

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UNIT 3 RESPIRATORY CIRCULATION

CONTENTS

1.0 Introduction
2.0 Intended Learning Outcomes (ILOs)
3.0 Main Content
    3.1 Respiratory System
    3.2 Minute Ventilation/Alveolar Ventilation
4.0 Conclusion
5.0 Summary
6.0 Tutor-Marked Assignment
7.0 References/Further Reading

1.0 INTRODUCTION

This unit describes the mechanism of breathing in Pulmonary system.

2.0 INTENDED LEARNING OUTCOMES (ILOS)

By the end of this Unit, you will be able to;

i. State the function of Pulmonary Circulation
ii. Define Minute Ventilation

3.0 MAIN CONTENT

3.1 Respiratory Circulation

The lung has two different circulatory systems. *Pulmonary circulation* serves the external respiratory function, and *bronchial circulation* supplies the internal respiratory needs of the lung tissue (Figure 9.4). The structure of the pulmonary circulation parallels the divisions of the structures in the conductive zone, branching in a tree-like manner called *arborization* (Figure 9.5). Arborization ends in a dense alveolar capillary network, blanketing most but not all alveoli. The capillary blood flow through this network is called perfusion of the lung (Guyton and Hall, 2006; West, 2005). The pulmonary artery exits the right ventricle of the heart and gives rise to the capillary network in the lungs (Figure 9.4A). The pulmonary vein originates from this capillary network and enters the heart at the left atrium.

As with pulmonary airflow and the rest of the circulatory system, blood flows through this circuit due to differences in pressure that produce a pressure gradient large enough to overcome resistance to the flow. Normal pulmonary artery blood pressure is low, only 25/10 mmHg, but

venous pulmonary blood pressure is even lower, only 7 mmHg. Although this pressure gradient between the pulmonary artery and vein is not large, it is enough to bring about the blood flow. Because of the low gradient, however, gravity affects the pulmonary circulation more than the systemic or total body circulation. The lowest portion of the lungs, therefore, is perfused best. The portion of the lungs that is best perfused with blood is also ventilated best (Guyton and Hall, 2006; Leff and Schumacker, 1993; Martin et al., 1979). Which portion it is varies with the body posture.

The bronchial circulation consists of relatively small systemic arteries that originate from the descending portion of the aorta, called the thoracic artery, travel through the lungs, and return as veins that empty into the pulmonary venous system. Thus, not all of the pulmonary venous blood is fully oxygenated (Slonim and Hamilton, 1976).

Minute Ventilation/Alveolar Ventilation

Alveolar Ventilation is the volume of air available for gas exchange; calculated as tidal volume minus dead space volume times frequency. The amount of air inspired or the amount of air expired in 1 minute is known as minute ventilation or minute volume. The most common units of measurement are liters per minute (L·min−1) and milliliters per minute (mL·min−1). Inspired minute ventilation is symbolized as V.I , where V is volume, the “dot” indicates per unit of time, and the subscript upper case I stands for inspired. The symbol for expired ventilation, V.E, indicates expired rather than inspired air. Minute ventilation depends on tidal volume (VT), the amount of air inhaled or exhaled per breath, and the frequency (f) of breaths per minute (b·min−1). 9.2milliliters per minute are then commonly converted to liters per minute by dividing by 1000. At rest, a normal young adult breathes at a frequency of 12–15 times per minute and has a tidal volume of 400–600 mL. Children ventilate at a much faster rate but with a smaller tidal volume. Because minute ventilation represents the total amount of air moved into or out of the lungs per minute, it includes the portion of air that fills the conduction zone. Thus, minute ventilation does not represent the amount of air that is available for gas exchange. The amount of air that is available for gas exchange is termed alveolar ventilation (or anatomical effective ventilation).

SELF-ASSESSMENT EXERCISES

i. Describe the structure of the pulmonary system
ii. Differentiate between Conducive and Respiratory zone.

4.0 CONCLUSION

Having read this course and successfully completed the assessment and self-assessment test, it is assumed that you have attained understanding of the introductory knowledge on mechanism of breathing.

5.0 SUMMARY

This Unit has successfully summarized the mechanism behind every form of breathing.

6.0 TUTOR-MARKED ASSIGNMENT

7.0 REFERENCES/FURTHER READING

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UNIT 4 MEASUREMENT OF LUNG VOLUMES

CONTENTS

1.0 Introduction
2.0 Intended Learning Outcomes (ILOs)
3.0 Main Content
    3.1 Measurement of Lung Volumes
    3.2 Regulation of Pulmonary Ventilation
4.0 Conclusion
5.0 Summary
6.0 Tutor-Marked Assignment
7.0 References/Further Reading

1.0 INTRODUCTION

This unit will state the different kinds of measurement used for lung volumes.

2.0 INTENDED LEARNING OUTCOMES (ILOS)

By the end of this Unit, you will be able to;

i. Know how lung volumes can be measured.
ii. Describe the regulation of pulmonary ventilation

Measurement of Lung Volumes
Lung volumes can be measured either statically or dynamically. Static lung volumes are anatomical measures, are independent of time, and thus do not measure flow. Dynamic lung volumes depend on time and measure both airflow and air volume.

Static Lung Volumes
Take as deep a breath as you can. At this point, your lungs contain the maximum amount of air they can hold. This amount of air is called total lung capacity (TLC).

TLC can be divided into four volumes and three other capacities, as depicted in Figure 9.6. Note that the four capacities are combinations of two or more volumes. All of these volumes and capacities have clinical significance, but only those important in the study of exercise physiology are discussed briefly here.

As mentioned previously, tidal volume (VT) is the amount of air either inhaled or exhaled in a single breath. A normal VT inflates the lungs to about half of the TLC

when sitting erect or standing and only about a third when lying supine (Slonim and Hamilton, 1976). When the demand for energy increases during exercise, VT increases by expanding into both the inspiratory reserve volume (IRV) and expiratory reserve volume (ERV). Thus, the limits of vital capacity (VC = IRV + VT + ERV) represent the absolute limit of the tidal volume increase during exercise.

Residual volume (RV) is the amount of air left in the lungs following a maximal exhalation. This leftover air is important because it allows for a continuous gas exchange between the alveoli and the capillaries between breaths. If all air were forced out of the lungs, no gas would be available for exchange. During exercise, when VT expands, the functional residual capacity (FRC) helps maintain a smooth exchange, in the following way. The VT can and does expand into both the IRV and the ERV, but it expands more into the IRV than the ERV, leaving a relatively large FRC intact. This large FRC dilutes the gas changes (the decrease in oxygen and increase in carbon dioxide) caused by the increased energy production and expenditure of exercise. By reducing fluctuation, the FRC stabilizes and smoothes the gas exchange. Despite its beneficial physiological aspects, RV presents a measurement difficulty. When body composition is determined by hydrostatic (underwater) weighing, RV must be measured or estimated. Residual air makes the body more buoyant; if not accounted for, it would reduce the underwater weight. The less an individual weigh underwater, the higher the measured percentage of body fat. Thus, if RV were not accounted for, it would erroneously increase the measurement of fat. This is the most common reason for determining RV in exercise physiology. Sometimes RV is estimated from VC, however it is more accurate if measured directly either outside or inside the tank (Wilmore, 1969). RV is estimated as 24% of VC for males and 28% of VC for females' exchange.

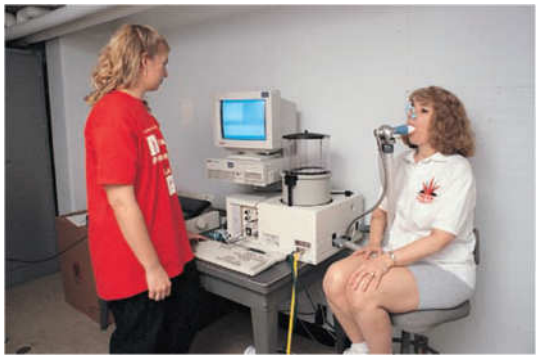
Vital capacity (VC) is simply the largest amount of air that can be exhaled following a maximal inhalation. The common way of testing VC is to ask the individual to inhale maximally and then forcefully exhale all of the air as quickly as possible. Because the exhalation is forced, the designation forced vital capacity, FVC, is used.

Dynamic Lung Volumes When volumes are measured at specified time intervals (usually 1 and 3 sec) during a forced VC test, the name is changed to *forced expiratory volume,* specifically, FEV1 and FEV3. This provides information not only about the total volume of air moved but also the rate of flow. Normal healthy individuals should be able to exhale at least 80% of their FVC in 1 second; this measurement is labeled FEV1. FEV1 values below 65–70% indicate moderate to severe restriction to airflow (Adams, 1994). The second commonly measured dynamic lung volume is a test of ventilatory capacity called *maximal voluntary*

ventilation (MVV). In this test, a timed maximal ventilation of either 12 or 15 seconds is recorded and then multiplied by 5 (if 12 sec) or 4 (if 15 sec) to extrapolate to the volume that could be ventilated in 1 minute. This value is usually higher than what can actually be achieved during exercise in untrained individuals, but it gives a rough estimate of exercise ventilation potential. Low values may reflect airway resistance and poorly conditioned or poorly functioning ventilatory muscles. Both FEV and MVV tests are often used as screening tests before maximal exercise tests of oxygen consumption.

Spirometry

All of the lung volumes and capacities described previously except for TLC, FRC, and RV—can be measured using a spirometer. Most spirometers have an inverted container, called a bell, that fits inside another container usually filled with water. When air is exhaled into the tube, the bell is pushed up; inhalation moves the bell down. In this way, the volumes of air inspired and expired can be measured, and the various volumes, capacities, and flow rates can be calculated. A spirometer that measures air volumes over water is called a wet spirometer. Newer dry spirometers do not use water.



Gas Dilution

TLC, FRC, and RV cannot be measured by simple spirometry because these involve air that cannot be exhaled voluntarily from the lungs. Thus, it is necessary to determine the volume of air that remains in the lungs. The most common technique for this measurement is gas dilution. One technique involves the dilution of an inert, insoluble, foreign gas such as helium (He). A second technique involves the dilution of medical grade oxygen and the measurement of nitrogen (N2) and is called the nitrogen washout technique. In each case, the participant inhales a known volume of either helium or oxygen and then rebreathes the gas mixture. Helium ultimately equilibrates with the gases in the lungs and spirometer and calculations are based on the dilution of the original helium mixture. Oxygen dilutes the original nitrogen concentration in the lungs and calculations are based on these values.

Standardization

The respiratory measurements detailed above are performed under ambient or atmospheric conditions. This means that the temperature measured is the temperature in the room or in the spirometer just before testing, and the pressure is the barometric pressure of the room. Because the air is exhaled from inside the human body, which is a wet environment, the air is saturated with water vapor. These measurements are therefore designated as ATPS: ambient (A) temperature (T) and pressure (P) saturated (S). Because ATPS volumes vary by environmental conditions, they must be converted to standardized conditions for purposes of comparison and assessment. Standardization is based on the known effects of pressure, temperature, and water vapor on gas volumes. Figure 9.8 illustrates these three effects, which are as follows:

1. The volume of a given quantity of gas is inversely related to the pressure exerted on it when the temperature remains constant. This effect is described by Boyle's law and was discussed previously in the section on the mechanics of breathing. As shown in Figure 9.8A, when pressure is reduced from 760 to 600 mmHg, 5 L of air expands to 6.3 L. The reverse is also true. If the pressure increases from 600 to 760 mmHg, the volume is reduced from 6.3 to 5.0 L (Slonim and Hamilton, 1976; West, 2005).

2. The volume of a given quantity of gas is directly related to the temperature of the gas when the pressure remains constant. This relationship is described by *Charles' law*. As shown in Figure 9.8B, if the temperature is reduced from 37°C to 0°C, the volume of the gas is reduced from 5 to 4.4 L. The reverse is also true. If the temperature rises from 0°C to 37°C, the volume also rises from 4.4 to 5.0 L.

3. Water molecules evaporate into a gas, such as air, and are responsible for part of the pressure of that gas. The amount of pressure accounted for by the water vapor is related exponentially to temperature. As shown in Figure 9.8C, when a volume of air is converted from saturated to dry air at a constant temperature, the volume is reduced from 5.0 to 4.7 L. Once again, the reverse is also true. If a gas volume goes from dry to wet at a given temperature, the volume increases (Slonim and Hamilton, 1976; West, 2005). The numbers for temperature and pressure in Figure 9.8 were not chosen arbitrarily. They are involved in the two standardized conditions to which ATPS lung volumes are converted: BTPS and STPD. The abbreviation BTPS means body (B) temperature (T) (37°C), ambient pressure (P), and fully saturated (S) with water vapor. Remember that in its passage through the conduction zone, the air is both warmed and

humidified to achieve these values. When converting from ATPS to BTPS, the temperature increases and the pressure, adjusted for the effects of temperature on water vapor pressure, decreases. Because of Charles' law (an increase in temperature causes an increase in volume) and Boyle's law (a decrease in pressure causes an increase in volume), the volume expressed as BTPS has to be larger than the volume originally measured as ATPS. BTPS is typically used when the anatomical space from which the volume of gas originated is of primary importance. Thus, most lung volumes and capacities are conventionally expressed as BTPS. STPD means standard (S) temperature (T) (0°) and pressure (P) (760 mmHg), dry (D). The STPD volume is smaller than the originally measured ATPS volume because under typical testing conditions, temperature decreases from ATPS (~20°C) to STPD (0°C), and by Charles' law, so does volume. Unless the testing is done at sea level, pressure increases from ATPS (variable, but around 735–745 mmHg before considering the influence of water vapor) to STPD (760 mmHg), and according to Boyle's law, volume decreases. Going from wet to dry conditions also decreases the volume. STPD volumes are used when it is necessary to know the amount of gas molecules present. Inspired or expired minute ventilation is typically converted and reported in STPD conditions, although sometimes it may be expressed as BTPS.

Regulation of Pulmonary Ventilation

Breathing, or pulmonary ventilation, results from inspiratory and expiratory muscle contraction and relaxation. The muscle action—and therefore the rate, depth, and rhythm of breathing—is controlled by the brain and nervous system and is tightly coupled to the body's overall need for oxygen and the subsequent production of energy and carbon dioxide. The coordinated control of respiratory muscles, especially during exercise, is a very complex process that is not yet fully understood (Leff and Schumacker, 1993; West, 2005).

The Respiratory Centers

There are four respiratory centers in the brain that function together to control breathing; two are located in the medulla oblongata and two are located in the pons. These four centers are schematically diagramed in Figure 9.9.

The two respiratory centers located within the medulla oblongata of the brain stem are composed of anatomically distinct neural networks. The inspiratory center (I), also called the dorsal respiratory group is the most important. The other center is the expiratory center (E), sometimes called the ventral respiratory group. The nerves of the inspiratory center depolarize spontaneously in a cyclic, rhythmical on-off pattern. During

the "on" portion of the cycle, nerve impulses traveling via motor neurons in the phrenic nerve stimulate the diaphragm and the external intercostal inspiratory muscles to contract. Inhalation occurs when the thoracic cavity is enlarged and intrathoracic pressure decreases. During the "off" portion of the cycle, the nerve impulses are interrupted, the inspiratory muscles relax, and exhalation occurs. Without outside influence and at rest, the inspiratory center causes a respiratory cycle of approximately 2 seconds for inspiration and 3 seconds for exhalation (for a rate of 12–15 br·min−1). This normal respiratory rate and oscillating rhythm is known as eupnea (Leff and Schumacker, 1993).

SELF-ASSESSMENT EXERCISES

i. Describe the structure of the pulmonary system
ii. Differentiate between Conducive and Respiratory zone.

4.0 CONCLUSION

Having read this course and successfully completed the assessment and self-assessment test, it is assumed that you have attained understanding of the introductory knowledge on mechanism of breathing.

5.0 SUMMARY

This Unit has successfully summarized the mechanism behind every form of breathing.

6.0 TUTOR-MARKED ASSIGNMENT

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MODULE 3 RESPIRATORY SYSTEM RESPONSE TO EXERCISE

INTRODUCTION

During exercise, the demand for energy increases. The demand varies, of course, with the type, intensity, and duration of the exercise. In most exercise situations, much of the body's ability to respond to the demand for more energy depends on the availability of oxygen. To provide the needed oxygen for aerobic energy production, the respiratory system including pulmonary ventilation, external respiration, and internal respiration must respond.

Unit 1 Response of The Respiratory System to Exercise
Unit 2 The Influence of Sex and Age on Respiration at Rest and During Exercise
Unit 3 Respiratory Training and Detraining Adaptations
Unit 4 Overview & Regulation of The Cardiovascular System
Unit 5 Measurement of Cardiovascular Variables & Responses to Aerobic Exercise

UNIT 1 RESPONSE OF THE RESPIRATORY SYSTEM TO EXERCISE

CONTENTS

1.0 Introduction
2.0 Intended Learning Outcomes (ILOs)
3.0 Main Content
3.1 The concept and structure of Pulmonary Ventilation
3.2 Static Exercise
4.0 Conclusion
5.0 Summary
6.0 Tutor-Marked Assignment
7.0 References/Further Reading

1.0 INTRODUCTION

This unit will explain the respiratory system response to exercise.

2.0 INTENDED LEARNING OUTCOMES (ILOS)

By the end of this Unit, you will be able to;

- Describe the concept and structure of Pulmonary Ventilation
- Describe static Exercise

3.0 MAIN CONTENT

3.1 Concept and Structure of Pulmonary Ventilation

Pulmonary ventilation increases to enhance alveolar ventilation, external respiration adjusts to maintain the relationship between ventilation and perfusion in most cases, and internal respiration responds with an increased extraction of oxygen by the muscles. These changes in respiration not only provide adequate oxygenation for the muscles but also play a major role in maintaining acid-base balance, which is, in turn, closely related to carbon dioxide levels.

In general, all levels of respiratory activity are precisely matched to the rate of work being done. Furthermore, because of this precise control and the large reserve built into the system, respiration in normal, healthy, sedentary or moderately fi t individuals is generally not a limiting factor in activity. This is true despite the perception of feeling out of breath during exercise. Only occasionally do the capacities of the cardiovascular and metabolic systems exceed that of the respiratory system such that respiration can be considered a limitation to maximal work. Of course, changes in pulmonary ventilation would be of little benefit if parallel changes in pulmonary blood volume and flow and total body systemic circulation did not also occur.

The most obvious response to an increased metabolic demand, such as exercise, is the increase in pulmonary ventilation (VE L·min−1), called hyperpnea. Note that between the onset of exercise at 0 and 2 minutes into the exercise, a triphasic response in V.E occurs. Within the first respiratory cycle at the onset of exercise, there is an initial abrupt increase in V.E, termed phase 1. This increase is maintained for approximately 10–20 seconds. Phase 2 is a slower exponential rise from the initial elevation to a steady-state leveling off. At the low to moderate workload depicted here, this exponential rise is generally completed in 2–3 minutes. At this point, phase 3, a new steady state, is achieved. The actual level of this achieved exercise steady state depends on a number of factors, including the workload, the fitness status of the individual, and the environmental conditions.

In the time span depicted in this graph, the steady-state level is maintained. The three-phase response at the onset of activity is typically not seen when V.E is reported or graphed minute by minute, rather than second by second or even breath by breath, but it does occur (Bell, 2006; Pardy et al., 1984; Whipp, 1977; Whipp and Ward, 1980; Whipp et al., 1982).

The initial rise in ventilation occurs primarily because of an increase in tidal volume (Leff and Schumacker, 1993). Theoretically, tidal volume ranges from the resting level to the limits of vital capacity (VC). In reality, rarely is more than 50–65% of VC reached before a plateau occurs. Furthermore, although tidal volume encroaches into both the inspiratory reserve volume (IRV) and the expiratory reserve volume (ERV), it encroaches much more into IRV than ERV (Koyal et al., 1976; Pearce and Milhorn, 1977; Turner et al., 1968; Younes and Kivinen, 1984).

At light to moderate workloads, the contribution of increased breathing frequency to minute ventilation is minimal and gradual. Both tidal volume and frequency level off at a steady state that satisfies the oxygen requirements of the short submaximal activity.

Airway resistance decreases because of bronchodilation as soon as exercise begins. Likewise, the ratio of dead space (VD) to tidal volume (VT) decreases, and in this case, the largest changes are evident at the lowest work rate (Wasserman et al., 1967; Whipp and Ward, 1980). This result is shown in Figure 10.1B. The depth of the drop in VD/VT is moderate at low to moderate exercise intensities. The VD itself changes minimally with bronchodilation, but with the proportionally larger increase in VT, the ratio declines (Grimby, 1969). This result is important because alveolar ventilation (V.A) thus increases from about 70% of the total pulmonary ventilation at rest to a higher percentage during exercise. Since V.A is the critical ventilation, this reduction in the VD/VT ratio means that the appropriate level of V.A can be achieved with a smaller rise in V.E than would be needed if the ratio did not change (Wasserman and Whipp, 1975).

External Respiration

The V.A response to low to moderate exercise is depicted. This curve parallels the change in V.E, except that the initial adjustments seen in V.E are not depicted for V.A. The rise in V.A is sufficient to maintain PO2 at the alveolar level (PAO2) during short-term submaximal exercise (Figure 10.2B). Maintenance of PAO2 is important because it represents the driving force for oxygen transfer across the alveolar-capillary interface (Powers et al., 1993; Wasserman, 1978).

Under resting conditions, there is an inequity in PO2 between the alveoli (PAO2) and systemic arterial blood (PaO2) owing to the dilution of the systemic arterial blood with the bronchial venous blood. During short-term, low-intensity submaximal exercise, PaO2 is maintained. The alveolar to arterial oxygen partial pressure difference, depicted as (A-a)PO2 diff in Figure 10.2C, either does not change or decreases slightly (Jones, 1975; Leff and Schumacker, 1993; Wasserman and Whipp, 1975). At moderate workloads, a slight increase may occur. The (A-a) PO2 diff

reflects the efficiency and/or adequacy of oxygen transfer in the lungs during exercise. At the steady-state submaximal levels described here, there is no noticeable change in this efficiency.

Gas exchange and blood perfusion in the lungs during low to moderate exercise are sufficient to maintain the saturation of red blood cells with oxygen (SaO2%) within a narrow range approximating resting levels (Figure 10.2D) (Gurtner et al., 1975).

Internal Respiration
Recall that internal respiration involves the dissociation of oxygen from the red blood cells so that it may diffuse down the pressure gradient into the muscles and other tissues.

Four factors are involved in increased oxygen extraction during exercise:
1. increased PO2 gradient
2. increased PCO2
3. decreased pH
4. increased temperature

3.2 Static Exercise

Static exercise involves the production of force or tension with no mechanical work being done. Therefore, gradations of static exercise are usually expressed relative to the individual's ability to produce force in a given muscle group (called the maximal voluntary contraction, or MVC) held for a specified period of time. For example, an individual might perform a 30% MVC for 5 minutes.

Entrainment of Respiration during Exercise
In some but not all individuals, the performance of rhythmical exercise (such as walking, running, cycling, and rowing) is accompanied by a synchronization of limb movement and breathing frequency called entrainment (Bechbache and Duffi n, 1977; Caretti et al., 1992; Clark et al., 1983; Hill et al., 1988; Jasinskas et al., 1980; Kay et al., 1975; Mahler et al., 1991; Sporer et al., 2007).

For example, an individual may always inhale during the recovery phase of rowing and always exhale during the drive portion of the stroke. Or a walker, runner, or cyclist may always exhale during the push-off phase of one leg or the other. Unlike swimming, in which breathing coordination is a function of head placement during the stroke as a learned response, entrainment occurs without conscious thought. Individuals who entrain naturally have a slightly improved ventilatory efficiency (Bonsignore et al., 1998) and a lower energy cost during exercise when they entrain but not when they breathe randomly. However, subjects forced to breathe in

specific entrainment patterns rather than being allowed to breathe spontaneously do not exhibit any reduction in energy cost or perceive any less breathing effort with entrained breathing (Maclennan et al., 1994).

SELF-ASSESSMENT EXERCISES

i. Describe the concept and structure of pulmonary ventilation
ii. Describe Static Exercise.

4.0 CONCLUSION

Having read this course and successfully completed the assessment and self-assessment test, it is assumed that you have attained understanding of the introductory knowledge on response of the respiratory system to exercise.

5.0 SUMMARY

This Unit has successfully summarized the mechanism response behind every form of respiratory system to exercise.

6.0 TUTOR-MARKED ASSIGNMENT

7.0 REFERENCES/FURTHER READING

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UNIT 2 THE INFLUENCE OF SEX AND AGE ON RESPIRATION AT REST AND DURING EXERCISE

CONTENTS

1.0 Introduction
2.0 Intended Learning Outcomes (ILOs)
3.0 Main Content
    3.1 The Male-Female Respiratory Differences
    3.2 The Children and Adolescents
    3.3 The Older Adults
4.0 Conclusion
5.0 Summary
6.0 Tutor-Marked Assignment
7.0 References/Further Reading

1.0 INTRODUCTION

This unit describes the influence of sex and age on respiration at rest and during exercise.

2.0 INTENDED LEARNING OUTCOMES (ILOS)

By the end of this Unit, you will be able to;

- State Male-Female Respiratory Differences
- Describe the factors in Children and Adolescents
- Describe the factors in Older Adults

3.0 MAIN CONTENT

3.1 Male-Female Respiratory Differences

Lung Volumes and Capacities
Values for total lung capacity (TLC) and each of its subdivisions are, on the average, lower for females than males across the entire age span, with the possible exception of around 12–13 years of age, when most girls have had their pubertal growth spurt but boys have not. These differences carry over into the dynamic measurements of maximal voluntary ventilation (MVV) and forced expiratory volume in one second (FEV1). Part of these differences can be attributed to the smaller size of females.

Males, for example, have larger diameter airways, more alveoli, and larger diffusion surfaces than females. However, even when values are

expressed relative to height, weight, or surface area, some differences in lung capacities remain (Åstrand, 1952; Comroe, 1965; Ferris et al., 1965; Harms, 2006).

Pulmonary Ventilation

At rest, there is no consistent difference in breathing frequency between males and females (Malina et al., 2004). However, at the same submaximal ventilation, females typically display a higher frequency and a lower VT than males. This pattern is maintained at maximal exercise (Saris et al., 1985) (Figure 10.12B). Ventilatory responsiveness during exercise may be influenced in females by levels of circulating estrogen and progesterone (Harms, 2006). Males also exhibit higher V.E at all ages than females at maximal exercise, although these differences are narrowed considerably when expressed relative to body weight (Figure 10.12A) (Åstrand, 1952, 1960). Surprisingly, experiments have shown that the inspiratory muscles of females may fatigue at a slower rate than those of males (Gonzales and Scheuermann, 2006).

External and Internal Respiration

Data to compare males and females are unavailable for most external and internal respiratory measures (Harms, 2006). The a-vO2diff has been measured at rest and during submaximal and maximal exercise, but the results show little consistency (Åstrand et al., 1964; Becklake et al., 1965; Zwiren et al., 1983). The (A-a) PO2 diff is higher and the PaO2 is lower in females compared to males at any given level of oxygen utilization. An excessive widening of the (A-a) PO2diff occurs in EIAH, and females exhibit this condition at least as often as males (Harms, 2006). V.A is equal in males and females. The PaCO2 is slightly lower in females than males at any given V.O2 (Hopkins and Harms, 2004).

3.2 Children and Adolescents

Lung Volumes and Capacities

In general, the TLC and each of its subdivisions increase in a mostly rectilinear pattern for both males and females as they progress from about 6 years of age into the late teens or early twenties. The FEV1 and MVV follow essentially the same incremental pattern in children (Åstrand, 1952; Åstrand et al., 1963; Bjure, 1963; Koyal et al., 1976; Malina et al., 2004). From birth to approximately age 10, these changes depend largely on the growth and development of the respiratory system. After that, cell proliferation ceases and hypertrophy of existing structures occurs until maturity. Thus, these changes result primarily but not exclusively from structural enlargement and are strongly related to body height (Bjure, 1963; Johnson and Dempsey, 1991). When the subdivisions of TLC are expressed as a percentage of the VT, the proportions remain the same from about age 8 to age 20. The anatomical dead space increases in

proportion to maturity (Ashley et al., 1975; Robinson, 1938) and, as in adults, is approximately 1 ml per pound of body weight (Rowland, 2005).

Pulmonary Ventilation The control of ventilation is similar in children and adults, except that there is a lower set point of PCO2 in children than adults (Rowland, 2005). However, there are some minor differences in V.E and its components across the age span (Zauner et al., 1989). Rest The V.E at rest is surprisingly consistent regardless of age (Robinson, 1938). Figure 10.13 shows that V.E in males varies less than 2 L·min−1 from ages 6 to 76. Comparable data are not available for females. When V.E is expressed relative to body weight, younger boys have a higher V.Ethan older adolescents or adults, but from adolescence to old age, there is little change in V.E.

The remarkably consistent V.E is achieved differently by children, however, than by older adolescents and adults.

Submaximal Exercise

Children's and adolescents' respiratory responses to exercise are similar to those of adults. V.E rises in response to greater oxygen needs at all ages, but it does so faster at the onset of exercise in younger individuals than adults (Rowland, 2005). The higher V.E in relation to body weight at rest is maintained, as is the variation in how V.E is obtained, that is, children exhibit a higher frequency and lower VT at any given submaximal load than adults. They also respond with a higher V.E in relation to body weight at an equal work rate. The ratio of liters of air processed per one liter of oxygen used (V.E/V.O2) is called the ventilatory equivalent. Children and adolescents have a higher ventilatory equivalent at all exercise intensities than adults, indicating that they are hyperventilating. Girls hyperventilate more than boys (Rowland, 2005). These differences are considered negative, indicating a wasteful ventilation, and gradually disappear by late adolescence (Bar-Or, 1983; Robinson, 1938; Rowland, 2005; Rowland and Green, 1988; Rowland et al., 1987). Figures 10.15A and 10.15B show differences in frequency (f), VT, and V.E for 11-year-old girls and boys in comparison to 29-year-old adults (Rowland and Green, 1988; Rowland et al., 1987). The speeds are different for the males and females, so direct comparison between the sexes cannot be made, but within the sexes, the age differences are similar.

During prolonged submaximal exercise, children and adolescents have the same ventilatory drift as adults, probably in response to a rising core temperature. Nothing in the respiratory response to prolonged exercise would indicate that children are not suited for such activity (Malina et al., 2004; Rowland, 2005).

Maximal Exercise

In general, children's responses to maximal exercise parallel the differences seen at rest and during submaximal work rates. The older the child, the higher the V.E that can be achieved in absolute terms. The V.E in relation to body weight gradually declines from about age 7 until adulthood is achieved at about 20 years of age, although in 4- to 6-year-olds, this value is comparable to that of young adults (Figure 10.12A) (Åstrand, 1952; Åstrand et al., 1963; Fahey et al., 1979; Krahenbuhl et al., 1985; Robinson, 1938; Rowland, 1990; Rowland and Green, 1988). Breathing frequency decreases consistently from the youngest children tested to approximately age 20, while VT shows a steady rise over the same time span (Figure 10.12B). The percentage of VC used as VT rises slightly from childhood to young adulthood. The V.E/V.O2 gradually declines at maximal work in both boys and girls (Rowland, 2005). Children also show ventilatory breakpoints during incremental exercise to maximum. Some inconclusive evidence suggests that VT1 and VT2 (expressed as a percentage of V.O2max) are higher in younger children than adults and gradually decline as children mature into adults (Mahon and Cheatham, 2002). The physiological mechanisms responsible for the VT1 and VT2 in children are as unclear as they are for adults (Bar-Or, 1983; Rowland and Green, 1988).

External and Internal Respiration

Little is known about the changes in gas exchange and transport that occur during normal growth and maturation. The higher frequency and lower VT of children in relation to adolescents and adults, at rest and during exercise, seem to be offset by their smaller anatomical dead space. Consequently, V.A is more than adequate at all values of V.E (Bar-Or, 1983; Malina et al.,2004; Zauner et al., 1989).

Pulmonary diffusion during exercise does not appear to differ by age. Likewise, no meaningful aging trends are apparent for PAO2, PaO2, or (A-a)PO2 diff at rest or during exercise. However, PaO2 decreases slightly and the (A-a)PO2 diff increases slightly as children mature to adulthood (Bar-Or, 1983; Eriksson, 1972; Robinson, 1938). As a consequence, SaO2% is also relatively constant across the age span (Robinson, 1938). EIAH is evident in some trained youth as in some adults, and the causes are also unclear in this age group (Nourry et al., 2004; Prefaut et al., 2000).

The a-vO2diff is also very similar at both rest and maximal exercise levels from childhood to maturity. If anything, children may be able to extract about 5% more oxygen during maximal exercise than adults (Eriksson, 1973).

3.3 Older Adults

Lung Volumes and Capacities

The effect of aging on TLC is controversial. Inconsistent evidence shows that TLC may decrease or stay the same in individuals over the age of 50 (Berglund et al., 1963; Jain and Gupta, 1974a,b; Johnson and Dempsey, 1991; Kenney, 1982; Stanescu et al., 1974; Storstein and Voll, 1974). However, research has fi rmly established that VC and inspiratory capacity (IC) decrease with age and that residual volume (RV) and functional residual capacity (FRC) increase, thus changing the percentage of total volume that each occupies (Åstrand, 1952, 1960; Ericsson and Irnell, 1974; Slonim and Hamilton, 1976; Stanescu et al., 1974; Turner et al., 1968). For example, the ratio of RV/TLC doubles from about 15–30% in the elderly (Comroe, 1965; Johnson and Dempsey, 1991). FEV1 and MVV decline steadily after approximately age 35 in both males and females (Ashley et al., 1975; Ericsson and Irnell, 1974; Shepard, 1978; Slonim and Hamilton, 1976; Stanescu et al., 1974). These declines result from a combination of structural and mechanical changes in the respiratory system. These changes include (1) decreased elastic recoil of lung tissue; (2) stiffening of the thoracic cage, which decreases chest mobility and creates a greater reliance on the diaphragm; (3) a decrease in intervertebral spaces, which in turn decreases height and changes the shape of the thoracic cavity; and (4) losses in respiratory muscle force and velocity of contraction. Of all these changes, the loss of elastic recoil appears to be the most important (Johnson and Dempsey, 1991; Turner et al., 1968).

Pulmonary Ventilation

Resting V.E and its components VT and frequency are remarkably consistent across the entire age span. However, at rest, the percentage of VC used as VT does show a very slight U-shaped curve. Both young children and older adults use slightly more of their VC for VT at rest than young adults (Robinson, 1938).

Submaximal Exercise V.E rises in older adults in response to increased energy needs. As in young adults, this increase is accomplished mainly by an increase in VT at lighter work rates and then by an increased frequency, if needed. Like children, older adults seem to have an exaggerated response in V.E compared with younger adults. That is, the absolute V.E is higher at any given work rate in older than in younger adults. Because the VC decreases with age and VT remains fairly stable, VT represents a higher percentage of the older adult's VC (Åstrand, 1952, 1960; Davies, 1972; DeVries and Adams, 1972; Robinson, 1938; Shepard, 1978).

Maximal Exercise

With aging, both the ability to exercise maximally and the ability to process air decline. The decrement in pulmonary function contributes to the decline in work capacity but probably simply parallels the changes in circulation, metabolism, and muscle function that are occurring.

The highest V.E values are typically seen in young adults, and these may decline by almost half by the seventh decade of life. This decline is evident both in absolute values (V.E in liters per minute) and in values adjusted for body weight (V.E in liters per kilogram per minute). Most of this decline is brought about by a reduction in VT, although maximal frequency does decrease slightly.

The percentage of VC used during maximal work is relatively stable with age from young adulthood on. However, the dead space to tidal volume ratio is consistently 15–20% higher in older adults than in younger adults (Robinson, 1938) because dead space increases both at rest and during exercise. The ventilatory breakpoints occur at lower absolute and relative workloads in older adults than in younger adults (Shepard, 1978). Respiratory work and the sensation of dyspnea are increased at maximal work in older adults.

External Respiration

The loss of elastic recoil in the lungs not only affects static and dynamic lung volumes but also influences the distribution of air in the lungs. Thus, ventilation may not be preferentially directed to the base of the lung in the upright posture at rest, although most blood flow is still directed there. As a result, there may be an imbalance between alveolar ventilation and pulmonary perfusion (Johnson and Dempsey, 1991). In addition, structural changes in aging lung tissue decrease the alveolar capillary surface area, which in turn means a decrease in diffusion capacity (Donevan et al., 1959; Johnson and Dempsey, 1991). Furthermore, pulmonary capillary blood volume decreases because of a stiffening of both pulmonary arteries and capillaries. The cumulative effects of these changes are a decrease in PaO2, but not PAO2, and a widening of the (A-a) PO2 diff—although these changes are neither inevitable nor large. The saturation of hemoglobin with oxygen in arterial blood declines about 2–3% from age 10 to age 70 (Robinson, 1938; Shepard, 1978).

Exercise

During exercise, the increased ventilation that is required results in a more homogeneous distribution of ventilation in the lungs. Although the decreases noted at rest in diffusion surface and pulmonary capillary blood volume remain, the matching of ventilation and perfusion improves during exercise as more of the lung is used. The available reserve is sufficient to meet the demands for oxygen transport even to maximal

exercise levels. Nevertheless, V.A is a smaller portion of minute ventilation in older adults than in younger adults. This change indicates a slightly decreased efficiency of respiration, but general arterial hypoxemia is prevented, and carbon dioxide elimination is adequate. That is, the PaO2 and PaCO2 are maintained within narrow limits and are similar to those for younger adults. The PO2 diff is more variable in older than in younger adults but, on average, is only slightly wider than the usual mean values for younger individuals (Johnson and Dempsey, 1991; Robinson, 1938; Shepard, 1978). Highly fi t older adults can, however, exhibit EIAH, as noted previously (Prefaut et al., 2000).

Internal Respiration

At rest and at any given submaximal level of exercise, the a-vO2diff is greater in older adults than in younger adults. Thus, SvO2% is lower. Conversely, at maximal exercise, the a-vO2diff is lower in older individuals than in younger ones. Maximal values average 14–15 mL·dL−1 in older adults but average 15–20 mL·dL−1 in younger adults. Some of these changes in the a-vO2diff can be attributed to a shift in the oxygen dissociation curve to the left, which makes the release of oxygen to the tissues more difficult (Kenney, 1982; Shepard, 1978).

SELF-ASSESSMENT EXERCISES

i. State the Male-Female Respiratory Differences
ii. State the factors responsible for respiratory differences in Children and Adolescents
iii. State the factors responsible for respiratory differences in Older Adults

4.0 CONCLUSION

Having read this course and successfully completed the assessment and self-assessment test, it is assumed that you have attained understanding of the introductory knowledge on the influence of sex and age on respiration at rest and during exercise.

5.0 SUMMARY

This Unit has successfully summarized the influence of sex on respiration and age on respiration during exercise.

6.0 TUTOR-MARKED ASSIGNMENT

7.0 REFERENCES/FURTHER READING

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UNIT 3 RESPIRATORY TRAINING AND DETRAINING ADAPTATIONS

CONTENTS

1.0 Introduction
2.0 Intended Learning Outcomes (ILOs)
3.0 Main Content
  3.1 The Lung Volumes and Capacities and Pulmonary Ventilation
  3.2 The External and Internal Respiration
  3.3 The Detraining
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1.0 INTRODUCTION

This unit describes the respiratory training and detraining adaptations

2.0 INTENDED LEARNING OUTCOMES (ILOS)

By the end of this Unit, you will be able to;

- State Lung Volumes and Capacities and Pulmonary Ventilation
- Describe External and Internal Respiration
- Describe Detraining

Respiratory Training Adaptations

No training principles or guidelines are included here for the respiratory system because respiratory training for healthy individuals is very rare. One exception involves exercise-induced diaphragm fatigue. During both short-and long-duration incremental or constant load exercise ≤80% V.O2 max, the diaphragm does not fatigue. However, at more than 80% V.O2 max intensity continued to exhaustion, the diaphragm does fatigue. The consequence of this fatigue is a decrease in exercise tolerance (Sheel, 2002). Because the diaphragm is a muscle, it is logical to attempt to use specific training to increase its resistance to fatigue. Two types of respiratory muscle training have been used experimentally. The first, hyperpnea training, involves maintaining a set percentage of 15-second MVV at a breathing frequency of 50–60 br·min−1, 30 min·d−1, and 3–5 d·wk−1. The second technique is termed inspiratory resistive loading training; it requires specialized pressure equipment. Both techniques have been shown to improve strength and endurance of the respiratory muscles in healthy humans. However, any positive impact on performance has

been limited at best (Sheel, 2002). One recent study (Enright et al., 2006) documented increased diaphragm thickness with increased performance and power output. A second study (Verges et al., 2007) achieved increased fatigue resistance of respiratory muscles after respiratory muscle training; however, cycling endurance did not change. More research is needed before either type of training can be recommended routinely for athletes.

Otherwise, training adaptations that have been documented in the respiratory system occur as a by-product of training for cardiovascular and/or metabolic improvement.

Applications of the training principles are presented for these systems in their respective units. The few training adaptations that do occur in the respiratory systems are documented in the following section.

3.0 MAIN CONTENT

3.1 Lung Volumes and Capacities

Whether the chronic but intermittent elevations in respiratory demand that occur with physical training actually change the lung itself is unknown. Studies of land training (running, cycling, wrestling, and the like) have found no consistent significant changes in TLC, VC, RV, FRC, or IC in males or females of any age; nor have they found any differences favoring athletes over nonathletes (Bachman and Horvath, 1968; Cordain et al., 1990; Dempsey and Fregosi, 1985; Eriksson, 1972; Kaufmann et al., 1974; Niinimaa and Shepard, 1978; Reuschlein et al., 1968; Saltin et al., 1968). Studies of water-based activities (swimming and scuba diving), however, have shown that swimmers have higher volumes and capacities than both land-based athletes and nonathletes (Cordain et al., 1990; Leith and Bradley, 1976). In addition, swim training studies have demonstrated increases in TLC and VC in both children and young adults. Similar generalizations can be made for the dynamic measures of FEV1 and MVV (Andrew et al., 1972; Bachman and Horvath, 1968; Clanton et al., 1987; Vaccaro and Clarke, 1978; Walsh and Banister, 1988).

Precisely why swimmers but not land-based athletes show improvements in static and dynamic lung volumes is not known. However, swimmers doing all strokes except the backstroke breathe against the resistance of water, using a restricted breathing pattern with repeated expansion of the lungs to total lung capacity. Swimming also takes place with the body in a horizontal position, and this posture is optimal for perfusion of the lung and diffusion of respiratory gases (Cordain and Stager, 1988; Mostyn et al., 1963).

Pulmonary Ventilation

Changes in V.E are the primary and most consistent adaptations seen in the respiratory system as a result of endurance training. Although V.E itself does not change at rest, a shift occurs in its components: VT increases, and frequency decreases. This shift is maintained during submaximal work but overall VT is lower during submaximal exercise as a result of the training. At maximal work, V.E is higher after training than before, accompanying the ability to do more work. The major component that changes is frequency, but VT increases as well (Dempsey et al., 1977; Mahler et al., 1991; Rasmussen et al., 1975; Reid and Thomson, 1985; Whipp, 1977; Wilmore et al., 1970). In addition, the capacity for sustaining high levels of voluntary ventilation is improved, reflecting increased strength and endurance of the respiratory muscles (Krahenbuhl et al., 1985; Robinson and Kjeldgaard, 1992). These adaptations occur within the first 6–10 weeks of a training program (Reid and Thomson, 1985). They result from both land- and water-based activities across the entire age span (Bar-Or, 1983; Fringer and Stull, 1974; Nourry et al., 2004; Pollock et al., 1969; Seals et al., 1984; Zauner and Benson, 1981; Zauner et al., 1989). The ventilatory thresholds shift to a higher workload and oxygen consumption as a result of training both in children and in adults indicating that a greater intensity of exercise can be maintained during endurance exercise performance across this age span (Haffor et al., 1990; Laursen et al., 2005; Loat and Rhodes, 1993; Mahon and Cheatham, 2002; Paterson et al., 1987; Pogliaghi et al.,
2006; Poole and Gaesser, 1985).

3.2 External and Internal Respiration

In a healthy individual of any age and either sex, gas exchange varies little as a result of training (Reid and Thomson, 1985). Diffusion capacity has been reported to be higher in elite swimmers (Comroe, 1965; Magel and Andersen, 1969; Mostyn et al., 1963; Vaccaro et al., 1977) and runners (Kaufmann et al., 1974), but it does not consistently increase at either submaximal or maximal work as a result of training (Saltin et al., 1968). Even in studies where diffusing capacity did increase with training, it was most likely due to circulatory changes (an increase in pulmonary capillary volume) rather than any pulmonary membrane change per se. Higher values in diffusion capacity may be an example of genetic selection for specific athletes (Comroe, 1965; Dempsey, 1986; Dempsey et al., 1977; Niinimaa and Shepard, 1978; Reuschlein et al., 1968; Vaccaro and Clarke, 1978; Wagner, 1991).

Arterial values of pH and PCO2 do not change with training, but venous pH levels increase (become less acidic) and PCO2 values decrease (Rasmussen et al., 1975) during the same submaximal exercise. The

PO2diff decreases at submaximal workloads as a result of training, indicating greater effi ciency (Saltin et al., 1968).

Training may also cause the oxygen dissociation curve to shift to the right, facilitating the release of oxygen from the blood into the muscle tissue (Rasmussen et al., 1975). In children, neither the submaximal nor the maximal a-vO2diff adapts as a result of training (Bar-Or,1983; Eriksson, 1973). In young adults, the a-vO2diff increases at rest (Clausen, 1977; Saltin et al., 1968) and at maximal exercise as a result of training (Blomqvist and Saltin, 1983; Coyle et al., 1984; Saltin et al., 1968). Both increases and decreases in the a-vO2diff have been found during submaximal exercise as a result of endurance training (Clausen, 1977; Ekelund, 1967; Ekelund and Holmgren, 1967; Saltin et al., 1968). Changes in middle-aged and elderly adults are less likely than in younger adults (Green and Crouse, 1993; Saltin, 1969). None of the other partial pressure or saturation variables changes significantly and/or consistently with training.

Why Are There So Few Respiratory: Adaptations to Exercise Training?

The most commonly accepted answer to the question of why there are so few respiratory adaptations to exercise training is that the pulmonary system is endowed with a tremendous reserve capacity that is more than sufficient to meet the demands of very heavy physical exercise. Thus, the various components of the respiratory system are not stressed to any significant limits during physical training and so do not need to change. At the same time, the cardiovascular and metabolic capacities of muscle are being stressed and do respond by adapting. The adaptations in these systems may ultimately exceed the capability of the respiratory system, as seen with EIAH and diaphragmatic fatigue. Otherwise, the generalization that the respiratory system is “overbuilt” remains accurate, so there is no need for great changes in the respiratory system in the normal healthy individual (Dempsey, 1986; Dempsey
et al., 1977; Sheel, 2002).

3.3 Detraining and the Respiratory System

All available research evidence suggests that any physiological variable that is responsive to exercise training will also respond to detraining. There is no reason to suspect that this is not also true for the respiratory system, although research evidence is sparse. The most common pattern is a rapid deterioration in maximal V.E. In addition, detraining is associated with an increase in V.E/V.O2 during standardized submaximal exercise and at maximal exercise. These changes occur rapidly and progress to reductions approximating 10–14% if training is stopped for more than 4 weeks (Mujika and Padilla, 2000a,b). Exercise Training and

Pollution Air pollution is comprised of a mixture of many different chemicals. The major components of automotive pollution include sulfur dioxide (SO2), nitrogen oxides (NOx), ozone (O3), particulates, and carbon monoxide (CO).

Cigarette smoke and by-products of the combustion of other fuels also contribute to air pollution. Sulfur dioxide is absorbed by the moist surfaces of the upper airways and can cause bronchospasm. Sulfur dioxide peaks at midday but is rarely a major problem. Nitrogen oxides are absorbed by the mucosal lining of the nose and pharynx and lead to irritation, cough, dyspnea, and diminished resistance to respiratory infection. Levels of NOx are usually relatively low. Ozone is formed naturally by the action of ultraviolet radiation (UVR) on oxygen as UVR enters the earth's atmosphere and by the action of sunlight UVR on automobile exhaust (Armstrong, 2000). Ozone causes a decrease in forced vital capacity (FVC) and FEV1 and an increase in airway resistance. Ozone levels are higher in summer, than in winter because of the greater sunlight, and in rural rather than urban areas. Particulates are solid or liquid materials produced from fuel combustion that remain suspended in air for long periods of time. They are usually under 10 μm in diameter and are thus often labeled as PM10. Particles of this size can penetrate deeply into the lungs. Particulate pollution is highest in heavy smog, which can also include ozone. Lead is associated with particulates, and a significant relationship has been shown between training duration and blood lead accumulation. The effects of particulates include airway inflammation and decreased capacity for oxygen exchange (Carlisle and Sharp, 2001). Experiments have shown that the deposit of ultrafine (<0.1 μm) particles is high during mouth breathing in healthy individuals at rest and increases more than 4.5 times as much during moderate exercise (Daigle, 2003).

As the intensity of exercise increases, both the rate and the depth of breathing also increase. To accommodate this increase, the individual switches from nose to mouth breathing and hence bypasses the cleansing filtration of the nose. As a result, a greater percentage of inhaled pollutants penetrates more deeply into the respiratory tract than at rest. Probably because of the respiratory system changes, breathing polluted air may result in decreased exercise performance (Haymes and Wells, 1986; McCafferty, 1981).

If the pollutant is CO, it reduces both the ability to carry oxygen and the ability to release oxygen already bound to red blood cells. The affinity of hemoglobin for CO is 210–230 times greater than its affinity for oxygen, and CO binds at the same site where oxygen would. Thus, when carboxyhemoglobin (COHb) is formed, the arterial percent saturation of oxygen decreases. The release of oxygen from hemoglobin is impaired by a shift in the oxygen dissociation curve to the left.

Myoglobin (Mb—the oxygen transporting and storage protein of muscles) and its role in assisting oxygen diffusion through the sarcoplasm to the mitochondria are also affected. First, the decreased release of oxygen from the red blood cells reduces the efficiency of Mb for attracting and holding oxygen within the muscle cells. Second, CO binds directly to Mb with approximately the same affinity as to Hb, thereby reducing myoglobin's ability to combine with whatever oxygen is available. The combined result of the effects of elevated CO levels on Hb and Mb is an earlier and possibly greater dependence on anaerobic metabolism.

This is manifested by a lower exercise intensity at which anaerobic metabolism becomes important, a shorter endurance time at submaximal loads, a lower maximal exercise performance, a lower maximal a-vO2diff, and a lower maximal oxygen consumption (V.O2max) (McDonough and Moffatt, 1999). As little as a 4% COHb level will have detrimental effects on exercise time and intensity. This level may result if training is done near heavy traffic (Figure 10.18). CO inhaled by smoking has additional respiratory impact, including increased pulmonary airway resistance, increased oxygen cost of ventilation, and an increased diffusion distance for oxygen and carbon dioxide across the alveolar walls because of mucosal swelling and bronchial constriction (McDonough and Moffatt, 1999). Individuals smoking 10 or fewer cigarettes per day average approximately 4% COHb, and a two-pack-a-day habit almost doubles this value. A nonsmoker riding in a car for 1 hour with a smoker can reach 3% COHb level (Haymes and Wells, 1986). Strenuous exercise near heavy traffi c for 30 minutes can increase the level of COHb as much as smoking 10 cigarettes (Carlisle and Sharp, 2001). The half-life of COHb is 3–4 hours, meaning that it takes that long for one half of the CO to become unbound to hemoglobin and be removed. Athletes are often affected by pollution levels that do not bother spectators. Individuals with cardiovascular and respiratory diseases and children are also particularly vulnerable (McCafferty, 1981). The following recommendations are suggested to minimize the impact of pollutants on an exercise training session or competition.

1. Individuals with health problems that make them particularly susceptible to the effects of pollution should not exercise outside during air quality warnings. They should seek sites where the air is filtered.

2. Everyone should avoid prolonged heavy exercise when hazardous air warnings are in effect. Individuals can adapt to breathing pollutants, but in the long term, adaptation is harmful because it suppresses normal defense mechanisms. Therefore, adaptation should not be attempted. Anyone experiencing symptoms such as coughing, wheezing, chest tightness, pain with breathing deeply,

or difficulty breathing should reduce their activity level and seek medical attention (Campbell et al., 2005).

3. Atmospheric ozone levels peak at around 1–3 p.m. and are much higher during most of the daylight hours in summer than in winter. CO peaks at approximately 7 a.m. and 8 p.m. and is higher in winter than in summer. Thus, heavy outdoor workouts might be best early in the morning and late evening during the summer and around noontime in the winter (Armstrong, 2000). Runners, cyclists, and in-line skaters should seek locations away from heavy vehicular traffic. Locations upwind with at least 50 ft between motor vehicles and exercisers are best. Avoid trailing close to a pace car or waiting at stoplights behind cars' exhaust pipes.

4. At the very least, smoking should be banned from all indoor training and competition sites. Smoking should be discouraged at all times (McCafferty, 1981).

SELF-ASSESSMENT EXERCISES

i. Define the Lung Volumes Capacities
ii. Describe the Pulmonary Ventilation
iii. Define External and Internal Respiration
iv. Describe Detraining

4.0 CONCLUSION

Having read this course and successfully completed the assessment and self-assessment test, it is assumed that you have attained understanding of the introductory knowledge on the respiratory training and detraining adaptations.

5.0 SUMMARY

This Unit has successfully summarized the impact of respiratory training and detraining adaptations.

6.0 TUTOR-MARKED ASSIGNMENT

7.0 REFERENCES/FURTHER READING

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UNIT 4 OVERVIEW & REGULATION OF THE CARDIOVASCULAR SYSTEM

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1.0 INTRODUCTION

This unit describe the ability to deliver oxygen (and other substances) depends on the proper functioning of the cardiovascular system. In many ways the cardiovascular system and the respiratory system operate together to accomplish a common mission—to deliver oxygen to working muscles—and they are driven by similar mechanisms. This chapter provides an overview of the cardiovascular system, discusses basic principles of cardiovascular dynamics, and outlines techniques currently used to assess cardiovascular function.

2.0 INTENDED LEARNING OUTCOMES (ILOS)

By the end of this Unit, you will be able to;

- Describe the Heart
- Define the Vascular System
- Differentiate between Blood and Hormonal Control of Blood Volume
- State the neural control and anatomical sensors and factors affecting control of the cardiovascular system.

Overview of the Cardiovascular System

The *cardiovascular system* includes the heart, blood vessels, and blood. Its primary functions are;

1. to transport oxygen and nutrients to the cells of the body and to transport carbon dioxide and waste products from the cells.
2. to regulate body temperature, pH levels, and fluid balance.

3. to protect the body from blood loss and infection.

The heart is a double pump that provides the force to circulate the blood throughout the vessels of the circulatory system. The blood vessels serve as conduits for the blood as it travels through the body. The blood transports gases and nutrients within the cardiovascular system.

Arteries carry blood away from the heart, and *veins* return blood to the heart. The *capillary beds* serve as the site of exchange for gases and nutrients between the blood and body tissues. Blood is ejected from the ventricles on both sides of the heart simultaneously. The right ventricle pumps blood through the pulmonary arteries to the lungs, where it is oxygenated and then returned to the left atrium via the pulmonary veins; this is called the pulmonary circulation. The left ventricle pumps oxygenated blood through the aorta, which then branches extensively into arteries to carry the blood to body cells through numerous specific circulations.

The partially deoxygenated blood returns to the right atrium. Collectively, this route from the left ventricle to the right atrium is known as the systemic circulation.

As described in the respiration chapters, external respiration is the exchange of gases (O2, CO2) between the lungs and blood. Internal respiration is the exchange of gases (O2, CO2) at the cellular level. The cardiovascular system functions primarily to move the gases (as well as nutrients from the digestive tract to the tissues) between these two exchange sites so that energy can be produced by cellular respiration. Cellular respiration is described completely in the Metabolic Unit.

3.0 MAIN CONTENT

3.1 The Heart

The heart is a hollow muscular organ located in the thoracic cavity. It weighs approximately 250–350 g and is 12–14 cm long, about the size of a clenched fist. The heart beats approximately 70 times per minute in a resting adult—or over 100,000 times per day! Macroanatomy of the Heart.

The heart has four chambers and is functionally separated into the right and left heart. The right side pumps blood to the lungs (pulmonary circulation), and the left side pumps blood to the entire body (systemic circulation).

Heart muscle is called **myocardium**. The two sides of the heart are separated by the interventricular septum. The upper chambers, called *atria* (*atrium* is the singular), receive the blood into the heart. The lower chambers, called *ventricles*, eject blood from the heart. Blood is ejected from the right ventricle to the pulmonary artery and from the left ventricle to the aorta.

One-way valves control blood flow through the heart. The *atrioventricular (AV) valves* separate the atrium and ventricle on each side of the heart. Specifically, the tricuspid valve separates the atrium and ventricle on the right side of the heart, and the bicuspid (or mitral) valve separates the atrium and ventricle on the left side of the heart. The *semilunar valves* control blood flow from the ventricles. Specifically, the aortic semilunar valve allows blood to flow from the left ventricle into the aorta, and the pulmonary semilunar valve allows blood to flow from the right ventricle into the pulmonary artery.

Microanatomy of the Heart

Cardiac muscle cells, called myocytes, are the contractile cells that produce the force that ejects blood from the ventricles. Cardiac muscle cells are both similar to and different from skeletal muscle cells. Both are striated in appearance because they contain the contractile proteins, actin and myosin. The primary difference between cardiac and skeletal muscle cells is that cardiac muscle cells are highly interconnected; that is, the cell membranes of adjacent cardiac cells are structurally and functionally linked by **intercalated discs**. The intercalated discs contain gap junctions that allow the electrical activity in one cell to pass to the next. Thus, the individual cells of the myocardium function collectively: when one cell is stimulated electrically, the stimulation spreads from cell to cell over the entire area. This electrical coupling allows the myocardium to function as a single coordinated unit, or a functional **syncytium**. Each of the two functional syncytia, the atrial and ventricular, contracts as a unit.

The Heart as Excitable Tissue

Cardiac muscle cells are excitable cells that are polarized (have an electrical charge with the inside being negative relative to the outside of the cell) in the resting state and contract when they become depolarized (the charges reverse). Repolarization (the electrical charge returns to resting value) occurs during relaxation of the muscle cells. With each contraction, blood is ejected from the chambers. Individual myocardial cells function together to produce a coordinated contraction of the entire syncytium.

Generally, when contraction of the heart is referred to, unless specified otherwise, it means contraction of the ventricles. In addition to contractile muscle cells, the heart contains specialized conducting cells. Although

there are far fewer conducting cells than contractile muscle cells, they are essential because they spread the electrical signal quickly throughout the myocardium. The conduction system cells with the fastest spontaneous rate of depolarization are called the *pacemaker* cells. These are located in the *sinoatrial (SA) node* in the right atria. As shown in Figure 11.4A, the excitation spreads from the SA node throughout the right atria by internodal tracts and to the left atria by Bachmann bundle. Because the atrial and ventricular syncytia contract separately, excitation in the atria does not lead directly to the contraction of cardiac cells in the ventricles. The signal is spread from the atria to the ventricles via the *AV node*. Once the AV node is depolarized, the electrical signal continues down the specialized conduction system consisting of the bundle of His, the left and right bundle branches, and the Purkinje fibers. The electrical excitation then spreads out from the conducting system to excite all of the cardiac muscle cells in the ventricles. Thus, the excitation is spread first by the conduction system and then by cell-to-cell contact: The excitation must be passed from muscle cell to muscle cell within the ventricles since the conduction system does not reach each individual cell.

As mentioned earlier, the cells of the SA node are considered the pacemaker cells of the heart because they normally have the fastest rate of depolarization. Cells in each area of the conduction system have their own inherent rates of depolarization. For the SA node, the intrinsic rate of depolarization is 60–100 times per minute. The AV node discharges at an intrinsic rate of 40–60 times per minute, and the Purkinje fi bers at a rate of 15–40 times per minute (Guyton and Hall, 2006). If the SA node is diseased, the AV node may take over the pace-making. While it is generally known that endurance training leads to a lower resting heart rate (HR), if the only thing you knew about an individual was that he or she had a resting HR of 40 b·min−1, you could not tell whether the person was a highly trained endurance athlete or someone in need of an artificial pacemaker implant because the SA node was not functioning properly.

Electrocardiogram

An **electrocardiogram (ECG)** provides a graphic illustration of the electrical current generated by excitation of the heart muscle. The spread of the electrical signal through the conduction system of the atria is shown in green. The P wave represents atrial depolarization, which causes atrial contraction. Repolarization of the atria, which results in a Ta wave, is normally not detectable on a resting ECG but occurs during the time period concurrent with the QRS complex and may be evident during exercise. The electrical signal reaches the AV node at the end of the P wave (shown in yellow). Excitation of the bundle of His and bundle branches (shown in red) occurs in the middle of the PR interval, followed by excitation of the

Purkinje fibers (shown in purple). Notice that excitation of the various portions of the conduction system happens very quickly and that activation of the entire conduction system precedes the QRS complex. The QRS complex reflects depolarization of the muscle fibers in the ventricles. It occurs after the electrical signal has traveled through the specialized conduction system in the ventricles and occurs simultaneously with atrial repolarization. The T wave reflects repolarization of the muscle fibers in the ventricles and is followed by relaxation in preparation to start the cycle all over again. The U wave may or may not be seen in a normal ECG but is often present in the slower cardiac cycle of trained individuals. When present, it probably represents the final phase of ventricular repolarization during which the Purkinje system recovers. Although the SA node can depolarize spontaneously, the firing of the SA node is influenced by neural and hormonal factors. Additionally, HR varies with age.

Cardiac Cycle

To function successfully as a pump, the heart must have alternating times of relaxation and contraction. The relaxation phase, called **diastole**, is the period when the heart fills with blood. The contraction phase, called **systole**, is the period when blood is ejected from the heart. The **cardiac cycle**—one complete sequence of contraction and relaxation of the heart—includes all events associated with the flow of blood through the heart. During the cardiac cycle there are dramatic changes in pressure and blood volume. This chapter summarizes the flow of blood in the heart and the position of the heart valves throughout the phases of the cardiac cycle. The ventricular-filling period (VFP) occurs when the ventricles are at rest (ventricular diastole) and the AV valves are open. The ventricles fill as blood is returned to the atria and flows down into the ventricles. Blood flow into the ventricles from the atria is assisted by gravity in an upright person. Atrial contraction also pushes a small volume of additional blood into the ventricles at the end of diastole. Blood volume in the ventricles is greatest at the end of ventricular filling, but pressure remains relatively low because the ventricles are relaxed. Systole (the contraction phase, shown on the right side of the figure) is divided into two periods, the isovolumetric contraction period (ICP) and the ventricular ejection period (VEP). During the ICP both the AV valves and the semilunar valves are closed.

Thus, blood volume in the ventricles remains constant despite the high pressure generated by the contraction of the ventricular myocardium. Once the pressure in the ventricles exceeds the pressure in the aorta, the semilunar valves are forced open. Blood is then ejected from the ventricles, initiating the VEP, and ventricular volume decreases as blood exits the ventricles through the open semilunar valves.

During the isovolumetric relaxation period (IRP) both the AV and the semilunar valves are closed. Thus, ventricular volume is unchanged, and pressure is low because the ventricles are relaxed. All these events occur within a single cardiac cycle, which repeats with every beat of the heart. The cardiac cycle graphically, shows concurrent information about the electrocardiogram; the pressure in the left atrium, the left ventricle, and aorta; the left ventricular volume; the heart phase; the period of the cardiac cycle; and the position of the heart valves. Diastole is shown in blue and systole is shown in green.

The AV valves are open, allowing blood to flow from the atria into the ventricles; therefore, ventricular volume is increasing. As the atria contract, more blood is forced into the ventricles, causing a small increase in ventricular volume and ventricular pressure. Following the QRS complex, there is an immediate and dramatic increase in ventricular pressure as the myocardium contracts. Notice, however, that the ventricular volume does not immediately change. This is the ICP.

Locate Point A on the graph of ventricular pressure—this is the point where pressure in the ventricle exceeds pressure in the aorta, and the aortic semilunar valve is forced open. Follow the dashed lines downward to the row for the semilunar valves in the chart at the bottom, and note that these valves are now opened. Also note that this dashed line coincides with the start of a rapid decrease in ventricular volume. Once the valves are open, blood is forced out of the ventricles; thus, blood volume in the ventricles decreases. This is the VEP. When the pressure in the ventricles falls below the pressure in the aorta, the semilunar valves close. Refer to Point B on the pressure curve and again follow the dashed line downward, noting ventricular volume and valve position. Ventricular pressure decreases because the myocardium is relaxed (in diastole). Ventricular volume, however, remains constant, because all the valves are closed and no blood can enter or leave the ventricles. This is known as the IRP.

Following the T wave (ventricular repolarization), the ventricles relax and begin to fill with blood: The AV valves are open, and ventricular volume increases. This is the VFP. This cycle of diastole followed by systole followed by diastole continues with each beat of the heart. Diastole provides time for the cardiac cells to relax and the ventricles to fill. The length of time spent in diastole thereby directly affects the amount of blood that will be present in the ventricles to be pumped during the subsequent systole. Furthermore, it is during diastole that the myocardium is supplied with blood.

Systole is the contraction period of the heart, first isometrically (ICP) and then dynamically (VEP). The volume of blood ejected from the ventricles directly affects the cardiovascular system’s ability to meet the demands

of the body. The volume of blood in the ventricles at the end of diastole is termed **end-diastolic volume (EDV)**. Similarly, the volume of blood in **volume (ESV).** The amount of blood ejected from the ventricles with each beat is called **stroke volume (SV)**, which is equal to EDV – ESV. Figure 11.6 depicts the volume in the left ventricle, but because both sides of the heart must pump the same amount of blood over any significant period of time, the SV is typically the same for both sides of the heart.

Before going to the next section, Be sure you understand what happens with the ECG, with ventricular, atrial, and aortic pressure, with ventricular volume, and with the heart valves at each period of the cardiac cycle. Now, find your radial pulse and begin counting: 0, 1, 2, 3, … . All the events described in this section occur every time you feel a pulse, which occurs once every 0.8 second when the HR is 75 b·min−1.

Heart rate (HR) is thus defined as the number of cardiac cycles per minute, expressed as beats per minute (b·min−1). Stroke Volume As mentioned previously, SV is the volume of blood ejected from the ventricles with each beat, expressed as milliliters per beat (mL·b−1) or simply in milliliters (mL). The amount of blood ejected from the heart is determined by three factors:

1. The volume of blood returned to the heart (preload).
2. The force of contraction (contractility).
3. The resistance presented to the contracting ventricle (afterload).

The volume of blood returned to the heart is called **preload** and is critical to the SV because the heart cannot eject blood that is not there. Under resting conditions the heart ejects approximately 50–60% of the blood that is returned; this is known as the **ejection fraction (EF)**. There are changes in the volume of blood in the ventricles throughout the cardiac cycle and defines specific volumes associated with the ventricles. The EDV is approximately 130 mL of blood which is typical for an adult male under resting conditions. Following systole the ESV is approximately 60 mL of Blood **Contractility**, the force of contraction of the heart, is determined primarily by neural innervation. Sympathetic nervous stimulation causes an increase in the contractility of the myocardium independent of the volume of blood returned to the heart. Circulating catecholamines (hormones) also reinforce the increased contractility caused by sympathetic nerve stimulation. During exercise, the Frank-Starling mechanism and increased contractility function together to enhance SV.

The **afterload**, or the resistance presented to the contracting ventricle, is determined primarily by the blood pressure (BP) in the aorta. As BP

increases, opposition to the outward flow of blood increases and less blood is ejected from the ventricles for any given force of contraction—that is, SV decreases as afterload increases. The SV decreases in this way because the increased pressure in the aorta causes the semilunar valves to remain closed longer and to close sooner. The valve is thus open for less time thereby causing a decrease in ejection time and a subsequent decrease in SV. There is a typical value for SV at rest in healthy individuals of various ages.

Coronary Circulation

The energy necessary for cardiac function is supplied through aerobic metabolism. Arterial coronary circulation supplies oxygenated blood to the myocardium through two major arteries, the right coronary artery and the left coronary artery. Both arteries originate at the root of the aorta. The left coronary artery divides into the left circumflex and anterior descending arteries. The right coronary artery divides into the marginal artery and the posterior interventricular artery. The myocardium is supplied with a dense distribution of arterioles and capillaries, approximately 3000 to 4000 capillaries per square millimeter of cardiac muscle (Rowell, 1986). The venous blood from the coronary circulation is returned to the right atrium via the coronary sinus. Coronary blood flow is affected greatly by the phase of the cardiac cycle. Because of the high intramyocardial pressure during systole, the coronary arteries are compressed and blood flow to the myocardium is decreased. Thus, the myocardium receives the largest portion of its blood flow during diastole (Guyton and Hall, 2006). The myocardial blood flow required to provide necessary oxygen at rest is about 250 mL·min−1, which represents approximately 4% of the normal resting cardiac output (Rowell, 1986). The coronary circulation very effectively extracts oxygen as the blood flows through the capillary beds. Under resting conditions, 60–70% of the available oxygen is extracted.

Myocardial Oxygen Consumption

Oxygen consumption is determined by oxygen extraction (a−vO2diff) and blood flow (Q). Because the metabolic demands of the myocardium are increased during exercise, **myocardial oxygen consumption**—the oxygen consumed by the myocardium to support contraction—increases during exercise. As mentioned previously, oxygen extraction of the coronary circulation is nearly optimal at rest (60–70%) and increases little if at all during exercise. Thus, the increased myocardial oxygen consumption during exercise occurs almost entirely by increased blood flow to the myocardium. The coronary blood flow must be regulated to meet the demands of the myocardium for oxygen. In addition to a higher HR, blood fl ow is increased by two mechanisms:

1. The greater force of myocardial contraction that results from exercise causes more blood to be forced into the coronary circulation.
2. By-products of cellular work cause vasodilation of the arterioles that supply the myocardium. Thus, as the heart works harder and produces more by-products, the arterioles dilate which decreases resistance and effectively increases blood flow.

Myocardial oxygen consumption increases as HR increases. Because HR increases with the intensity of exercise, so also does myocardial oxygen consumption (Kitamura, et al., 1972)

3.2 The Vascular System

The *vascular system* is composed of vessels that transport the blood throughout the body. Their size and structure vary throughout the vascular tree, with each portion of the vascular system having a specific structure and function related to the overall function of the cardiovascular system. Blood vessels, except for capillaries, have three layers; the adventia (outer layer), tunica media (inner layer), and tunica intima (innermost layer). The adventia is composed of connective tissue and attaches the blood vessel to surrounding tissue. The tunica media contains smooth muscle, which is critical for controlling the vessel's diameter, and connective tissue, and gives the vessel elasticity and strength. The intima consists of a single layer of endothelial cells and a thin layer of connective tissue (basal lamina). The **endothelium** serves as the barrier between blood and underlying tissue, and plays a critical role in the movement of material out of the blood, releases factors that help regulate the contraction of smooth muscle in the tunica media, helps prevent unnecessary clot formation, and interacts with immune cells in the inflammatory process. The vessels of the vascular system, along with various circulations, are illustrated.

Arteries

The arteries are thick-walled conduits that carry blood from the heart to the body's organs. They contain a large amount of elastic tissue that allows them to distend when blood is ejected during systole and pulsatile owing to the pumping action of the heart. As the left ventricle ejects blood into the aorta, the blood stretches the aorta's elastic walls. BP is the force exerted on the wall of the blood vessel by the blood. The peak pressure is referred to as **systolic blood pressure (SBP)** because it is essentially caused by the contraction of the heart (systole). During relaxation of the heart (diastole), the arterial walls recoil, maintaining pressure on the blood still in the vessels. Thus, although the BP drops during diastole, there is always some pressure in the arteries, and this lower pressure is known as **diastolic blood pressure (DBP). Mean arterial pressure**

(MAP) is a weighted average of SBP and DBP, representing the mean driving force of blood throughout the arterial system.
Typical resting BP values for males and females at different ages.

Arterioles
Arterioles, also called **resistance vessels**, are smaller than arteries and are the major site of resistance in the vascular system. Because of this increased resistance, the pulsatile arterial blood flow becomes continuous before it reaches the capillaries. Arterioles absorb the pulsatile force of blood flow because they contain a large amount of elastic tissue. Imagine bouncing a basketball on a gymnasium floor and on a wrestling mat. The ball rebounds from the gym floor at an angle and height proportional to the force imparted by your muscle action. However, the same force will not produce much (if any) rebound from the wrestling mat. The elastic tissue in the walls of the arteries absorbs the energy from the pulsatile blood flow in a similar way that the mat absorbs energy from the basketball. In an individual with reduced elasticity, or arterial stiffening (sometimes called hardening of the arteries), the arteries, like the gym floor, are not able to distend as readily, resulting in elevated BP.

The smooth muscle surrounding arterioles is able to contract and relax. Contraction of the smooth muscle around an arteriole results in *vasoconstriction*, a decrease in vessel diameter and therefore a decrease in blood flow to a given region. Relaxation of the smooth muscle results in *vasodilation*, an increased vessel diameter and therefore, an increase in blood flow to a region. The vasoconstriction and vasodilation of the smooth muscles surrounding the arterioles are primarily responsible for determining blood flow distribution to various organs.

The degree to which an arteriole vasodilates or vasoconstricts depends on the balance of extrinsic (originating outside the part on which it acts) and intrinsic (originating within the part on which it acts) mechanisms. Extrinsic mechanisms are geared toward maintaining MAP and include nervous stimulation and circulating hormones. Smooth muscle surrounding terminal arteries and arterioles is innervated by sympathetic neurons. Sympathetic stimulation causes most arterioles to vasoconstrict. However, during exercise, when the sympathetic nervous system is clearly activated, arterioles to the working muscles dilate in order to supply the working muscle with increased blood flow. The role of the sympathetic nervous system in accounting for this dilation is controversial. Although sympathetic vasodilatory fibers have been identified in several species, and postulated in humans, there is no direct evidence of vasodilatory sympathetic nerve fibers in humans (Joyner and Dietz, 2003). Intrinsic (local) mechanisms that control arteriole diameter include myogenic (originating within the muscle) and metabolic controls. Myogenic control is accomplished by mechanisms that cause the vessels

to dilate in response to decreased stretch (decreased flow) and vasoconstrict in response to increased stretch (increased flow). This reflex action helps to ensure that changes in BP do not lead to dramatic changes in blood flow to a given vascular bed. The metabolic control of vascular diameter plays a critical role in determining the degree of smooth muscle contraction and hence local blood flow. When a tissue is metabolically active, such as skeletal muscle during exercise, it produces metabolic by-products that act locally to cause vasodilation, thereby increasing blood flow to the metabolically active area. Thus, contracting skeletal muscles act locally (intrinsically) on the smooth muscle surrounding the arterioles to increase blood flow in that region. In the case of exercise, the local vasodilatory effects have a greater effect on vessel diameter in arterioles supplying the skeletal muscle than the sympathetic vasoconstrictor effects, leading to vasodilation in the skeletal muscle. On the other hand, arterioles supplying nonworking muscle and other organs (stomach, kidneys) constrict, resulting in decreased blood flow during exercise due to sympathetic nerve stimulation.

Capillaries

The *capillaries* perform the ultimate function of the cardiovascular system: transferring gases and nutrients between the blood and tissues. Some exchange of gases occurs in the smallest vessels on both sides of the capillaries (collectively termed the **exchange vessels**) but most of the gas exchange occurs across the capillary wall. The walls of the capillaries are very thin, essentially composed of a single layer of endothelial cells. Capillaries have a very small diameter, such that red blood cells often must pass through in a single file. Blood flow through capillaries also depends on the other vessels that make up the microcirculation. The **microcirculation** includes several vessels: arterioles, venules, arteriovenous anastomoses, metarterioles, and true capillaries. *Anastomoses* are wide, connecting channels that act as shunts between arterioles and venules. These vessels are not common in most tissue but are abundant in the skin and play an important role in thermoregulation (Levick, 2003). When anastomoses are open, large volumes of blood can be directed to blood vessels close to the surface of the skin, facilitating heat dissipation.

A *metarteriole* is a short vessel that connects an arteriole with a venule, creating a direct route through the capillary bed. The metarteriole gives rise to the capillaries. *True capillaries* vary in number depending on the capillary bed.

Smooth muscles around these vessels that relax or constrict in response to local chemical conditions control blood flow through the capillaries (Levick, 2003). Thus, a capillary bed can be perfused with blood or be almost entirely bypassed, depending on the needs of the tissue it supplies.

Gas Exchange
The exchange of gases and nutrients in the capillaries depends on diffusion. For a substance to diffuse from a capillary into a cell, it must cross two membranes: the capillary wall (composed primarily of endothelium) and the cell membrane. Substances pass from the capillary to the interstitial space by the process of diffusion. Movement from the interstitial space into the cell may also occur by diffusion or may require carrier-mediated transport. The movement of gases and nutrients into and out of the capillaries depends on the concentration gradient or pressure gradient of the substance or gas that diffuses. Oxygen and carbon dioxide diffuse down pressure gradients. Oxygen diffuses down its pressure gradient from systemic capillaries into muscle cells. Therefore, there is less oxygen in the veins draining skeletal muscles than in the arteries supplying them. The difference in the oxygen content of the arteries and veins is termed the *a−vO2 difference*, which reflects the oxygen taken up by the skeletal muscles.

Movement of Fluids
Fluids also pass through the capillary membrane. The movement of fluids is determined by two opposing forces: hydrostatic pressure and osmotic pressure. *Hydrostatic pressure*, created by BP, acts to "push" fluid out of the capillaries. *Osmotic pressure*, caused by the larger concentration of proteins in the capillaries, acts to "pull" water into the capillaries. The net result of these opposing force is the loss of approximately 3 L of fluid a day from the plasma into interstitial spaces (Marieb, 2007). This fluid returns to the blood via the lymphatic system.

Any change in hydrostatic pressure or osmotic pressure of the blood will alter the fluid exchange between the blood and the interstitial fluid.

Venules
The venules are small vessels on the venous side of the vascular system. These vessels contain some smooth muscle, which can influence capillary pressure. The venules and capillaries constitute the microcirculation where gas and nutrient exchange occurs. Venules empty into veins.

Veins
Veins, also called **capacitance vessels**, are low- resistance conduits that return blood to the heart. They contain smooth muscle innervated by the sympathetic nervous system, which can change their diameter.

Contraction of smooth muscle around the veins is known as *venoconstriction*; relaxation of the veins is known as *venodilation*. Because veins can expand (distensibility), they can pool large volumes of blood—up to 60% of the total blood volume at rest—and therefore are

sometimes referred to as a blood reservoir. The amount of blood in all the veins varies with posture and activity. If blood accumulates in the veins and is not returned to the heart, ventricular EDV decreases, with the result that SV decreases. Conversely, venoconstriction can significantly increase ventricular EDV and thereby lead to an increase in SV, according to the Frank-Starling law of the heart.

The skeletal muscle pump and the respiratory pump help increase venous return by "massaging" blood back toward the heart. The one-way valves in the veins also help regulate venous pressure and are particularly helpful in counteracting the effects of gravity that oppose blood flow back to the heart because they prevent the backward flow of blood. Additionally, the increased sympathetic nervous activity during exercise helps to increase venous return via venoconstriction.

Blood

Blood is the fluid that circulates through the heart and the vasculature to transport nutrients and gases. Blood contains living blood cells suspended in a nonliving fluid matrix called plasma. Blood that has been centrifuged to separate the cells and the plasma. Blood cells are classified as erythrocytes (red blood cells, RBC) or leukocytes (white blood cells, WBC). Blood cells account for 38–45% of the total blood volume in adult females and 43–48% of the total blood volume in adult males. The ratio of blood cells to total blood volume is known as **hematocrit** and is usually expressed as a percentage.

As discussed in the respiratory section, the RBCs transport oxygen from the lungs to body cells by binding oxygen to hemoglobin. Leukocytes are less numerous than erythrocytes, accounting for about 1% of total blood volume. Despite their seemingly small number, leukocytes are essential to the body's defense against disease and play a critical role in inflammation.

Plasma accounts for approximately 55% of the volume of blood. It is composed primarily of water, which accounts for approximately 90% of its volume. It also contains over 100 dissolved solutes, including proteins, nutrients, electrolytes, and respiratory gases. The composition of plasma varies greatly, depending on the needs of the body. Plasma also plays an important role in thermoregulation by helping to distribute heat throughout the body.

3.3 Hormonal Control of Blood Volume

As is discussed in the accompanying Focus on Application: Clinically Relevant Box, blood volume is decreased by blood donation. Decreased blood volume may also result from profuse sweating and/or dehydration.

Blood volume varies considerably among individuals and is affected by fitness status. Healthy adult males have an average blood volume of approximately 75 mL of blood per kg of body weight, or a total of approximately 5–6 L of blood. Healthy adult females have approximately 65 mL of blood per kg of body weight, which equals 4–4.5 L of blood for the average-size woman. Children typically have about 60 mL of blood per kg of body weight, with total volume varying depending on body size. Blood volume plays an important role in maintaining SV, cardiac output, and BP. Under normal conditions blood volume is maintained within physiological limits by homeostatic mechanisms involving the endocrine system and urinary system. The major hormones involved in maintaining blood volume are antidiuretic hormone (ADH), released from the posterior pituitary gland, and aldosterone, released from the adrenal cortex.

The hormonal mechanisms outline the respond to a reduction in blood volume. Plasma volume reduction causes a decrease in atrial and arterial pressures. The decrease in pressure is sensed by atrial baroreceptors (*baro* means “pressure”) and arterial receptors in the kidneys. Atrial baroreceptor activation leads to the release of ADH from the posterior pituitary gland, which causes the tubules of the kidneys to reabsorb water, thus increasing plasma volume. A reduction in blood volume is also associated with an increase in plasma osmolarity (solute concentration). For example, with profuse sweating more water than solutes is lost; thus, the osmolarity of the blood increases. An increase in osmolarity of the blood stimulates osmoreceptors in the hypothalamus, which signals the posterior pituitary gland to release ADH. ADH causes the kidneys to retain water, thus leading to an increase in blood volume. Simultaneously, the receptors in the kidneys respond to decreased arterial pressure by releasing the enzyme renin. Renin is necessary for the conversion of angiotensinogen to angiotensin I, which is then converted to angiotensin II. Angiotensin II signals the adrenal cortex to release aldosterone. Aldosterone causes the kidneys to retain salt and water. Angiotensin II also has a vasoconstrictor effect on arterioles, thus helping to increase blood pressure.

Cardiovascular Dynamics

The different components of the cardiovascular system function together to meet the changing demands of the body. These components are highly integrated and interdependent. Although both the heart and the vasculature respond independently to various conditions, they are interrelated because the response of the heart affects the vessels, and vice versa. To differentiate the responses of the heart and the vessels, we commonly refer to central and peripheral cardiovascular responses. **Central cardiovascular responses** are those directly related to the heart: HR, SV, cardiac output, etc. **Peripheral cardiovascular responses** are

those occurring in the vessels: vasodilation, vasoconstriction, venous return, etc.

Principles of Blood Flow

There are relationships among the cross-sectional area (CSA) of the blood vessels, BP, and blood velocity throughout the vascular system. The diameter of the various vessels, and the total CSA of the various vessels. Thus, although a single capillary is incredibly small, approximately 6m, there are so many capillaries that the total CSA far exceeds that of the other vessels. This depicts the velocity of blood in the various vessels—that is, the speed at which blood flows. The velocity of a fluid in a closed system varies inversely with the total CSA at any given point. Therefore, the velocity of blood flow decreases dramatically in the capillaries. This decreased velocity allows adequate time for the exchange of respiratory gases and nutrients. The driving force for the blood is the contraction of the myocardium. Blood flows because of a pressure gradient. Thus, blood flows through the vascular tree because pressure is highest in the aorta and major arteries and lowest in the great veins and right atrium of the heart.

Pressure continues to decrease as the blood travels further from the heart, reaching a low of approximately 4 mmHg in the right atrium. In fact, one-way venous valves and muscle and respiratory pump activity are needed to help return blood to the heart.

Regulation of the Cardiovascular System

The cardiovascular system is regulated by interrelated and overlapping mechanisms, including mechanical events, neural control, and neurohormonal control. Mechanical events, such as muscle action, influence venous return and thereby help regulate SV and cardiac output. This regulation is particularly important during exercise. Neural and neurohormonal mechanisms of cardiovascular control are more complex and are discussed in detail in the following sections.

3.4 Neural Control

Three cardiovascular centers are located within the medulla oblongata of the brain stem. The cardio accelerator and cardio-inhibitor centers innervate the heart. As the names imply, the *cardioaccelerator center* sends signals, via sympathetic accelerator nerves, that cause the HR to increase and the force of contraction to strengthen. The *cardio-inhibitor center*, also called the vagal nucleus, sends signals via the vagus nerve that cause a decreased HR and force of contraction. The *vasomotor center* innervates the smooth muscles of the arterioles via sympathetic nerves. Activation of these sympathetic fibers generally causes vasoconstriction

(sympathetic fibers to the skin are the only clear exception to this rule in humans).
In summary, activation of sympathetic nervous outflow leads to an increased HR, increased cardiac contractility, and vasoconstriction in most arterioles. During exercise, there is also vasodilation in the arterioles of skeletal muscle, but the role of the autonomic nervous system in this response is not clear. What is clear is that metabolic (intrinsic) controls lead to a wide spread vasodilation in arterioles supplying skeletal muscle in response to increased metabolic activity during exercise. Activation of the parasympathetic nervous system leads to the opposite responses in each of the above.

3.5 Anatomical Sensors and Factors Affecting Control of the Cardiovascular System

The cardiovascular centers—and therefore, the sympathetic and parasympathetic outflow from those centers—are influenced by several factors in a variety of circumstances, including exercise. This schematically presents the most important factors influencing the cardiovascular centers. These factors are described in detail in the following sections.

Higher Brain Centers
The cardiovascular medullary centers are influenced by several higher brain centers, including the cerebral cortex and the hypothalamus. Emotional influences arising from the cerebral cortex can affect cardiovascular function at rest. Input from the motor cortex, which is relayed through the hypothalamus, can influence cardiovascular function during exercise, leading to an increase in HR and vasodilation in active muscle. The influence of the cortex and hypothalamus on the cardiovascular centers during exercise is often termed “central command,” denoting that the signal to alter cardiovascular variables comes from the central nervous system.

Body temperature also affects the cardiovascular centers through the influence of the hypothalamus. An increased body temperature results in an increased HR, increased cardiac output, and vasodilation in the arterioles of the active muscles and skin.

Systemic Receptors
Systemic receptors are present in the great veins, the heart, and the arterial system. These receptors provide sensory information to the cardiovascular control centers that leads to reflex action.

Baroreceptors

Baroreceptors are located in the aorta and carotid bodies. With an increase in MAP, these receptors cause a reflex decrease in MAP through a decreased HR (and thus decreased cardiac output). The decrease in HR is mediated through an increased parasympathetic outflow and a simultaneous decrease in sympathetic outflow to the heart. This reflex control of BP is called the *baroreceptor reflex*. Because this reflex functions to maintain mean arterial blood pressure, you may wonder how someone becomes hypertensive or why mean arterial blood pressure goes up during exercise. In someone with hypertension, the action of the baroreceptors is mediated by a set point. If something causes the resting BP to be elevated (and no one knows precisely what causes this elevation), the baroreceptors fi re for about 24 hours in an effort to bring down the MAP. If this is unsuccessful, the baroreceptors appear to simply reset at a level above the previous value. The baroreceptor reflex is also reset during exercise. The resetting of the baroreflex is in direct proportion to exercise intensity (Raven et al., 2006). The baroreceptor reflex is also very important in achieving recovery to baseline values after exercise.

Stretch Receptors

Stretch receptors, located in the right atrium of the heart, are stimulated by an increase in venous return. The signal is transmitted to the cardiovascular centers in the medulla, where they cause an increase in sympathetic outflow and a decrease in parasympathetic outflow. This results in an increased HR and force of contraction, increasing cardiac output. This sequence is called the *Bainbridge reflex*.

Chemoreceptors

Chemoreceptors are located in the aortic and carotid arteries. They are sensitive to arterial blood PO2, PCO2, and H+. An increase in PCO2 and H+ or a decrease in PO2 causes a reflex vasoconstriction of arterioles.

Muscle Joint Receptors

Muscle receptors include mechanical (*mechanoreceptors*) and metabolic (*metaboreceptors*) receptors located in the joints and muscles. These receptors send impulses to the brain where the impulses synapse with the cardiovascular centers. When stimulated by muscle contraction, these receptors lead to an increased rate and force of heart contraction. Vasoconstriction occurs in inactive skeletal muscles.

Neurohormonal Control

The endocrine system also helps regulate the cardiovascular system. Considerable control is exerted by the components of the autonomic nervous system and the hormones of the adrenal medulla. The previous section discussed the influence of the sympathetic nervous system on the

heart and the blood vessels. The sympathetic nervous system also innervates the adrenal medulla, causing the adrenal glands to release the hormones epinephrine and norepinephrine. These hormones travel in the bloodstream to the heart and the blood vessels. Generally, epinephrine and norepinephrine have the same effect on these target organs as the sympathetic nerve fibers innervating them. In addition to the adrenal hormones, aldosterone and ADH help maintain blood volume and BP as described in the section on blood volume.

Measurement of Cardiovascular Variables

Cardiovascular variables are routinely measured and monitored in sports, fitness, rehabilitation, and research settings. The following variables are measured in order to assess fitness, prescribe exercise, and monitor physiological responses to exercise. Most of these variables can be assessed both at rest and during submaximal or maximal exercise.

Heart Rate

In a laboratory HR is often obtained by measuring the R-R interval in an ECG recording. Although the ECG equipment/computer may be programmed to automatically calculate the HR, it is useful to know how this measure is performed. The first step is to calculate the distance the ECG paper travels in one minute based on the speed of the paper. The distance between cycles is then measured. Because you now know the number of cycles that occurred within the distance measured, you can solve for the b·min−1 by solving for X in the following equation HR can also be recorded by wireless telemetry. Most often the transmitter is worn around the chest, and the HR signal is transmitted to a small receiver that looks very similar to a watch. Many pieces of fitness equipment also have the ability to pick up the HR signal from the transmitter, or to measure HR by having the exercisers grasp a sensor built into the exercise equipment. HR is often measured during exercise to monitor exercise intensity. In some exercise sessions, however, HR-measuring devices are not available. HR can then be assessed by counting the pulse—a method called palpation. The pulse can be felt at the carotid or radial artery. This technique requires instruction and practice.

To measure an exercise HR, the person usually pauses and finds the pulse as quickly as possible. The pulse count begins with zero and is counted for a set period of time, usually 6, 10, or 15 seconds, using a watch. This pulse count is then multiplied by 10, 6, or 4, respectively, to obtain the per-minute HR. A period less than 1 minute is used because the HR drops quickly once exercise is paused.

Factors Limiting V. O2max

At some point an individual cannot continue to increase the intensity of the exercise load or to work at maximum effort because the body cannot

provide and utilize more oxygen to support an additional workload. But what specifically limits V. O2max? There are possible limitations to oxygen consumption within the major systems involved in oxygen delivery and use during exercise.

Theoretically, maximal oxygen uptake could be limited by any system (or step) along the pathway of bringing oxygen into the body and delivering it to the mitochondria for the production of ATP. Thus, any of the following systems may limit V. O2max:

1. the respiratory system, because of inadequate ventilation, oxygen diffusion limitations, or an inability to maintain the gradient for the diffusion of O2 (a−vO2 diff)
2. the cardiovascular system, because of inadequate blood flow (Q.) or oxygen-carrying capacity (Hb)
3. the metabolic functions within skeletal muscle, such as an inability to produce additional ATP because of limited number of mitochondria, limited enzyme levels or activity, or limited substrates.

Evidence suggests that each of these systems may limit V.O2max in certain conditions (Bergh et al., 2000). For example, a reduction in the partial pressure of oxygen (PO2) at altitude or with asthma causes a reduction in V.O2max. Medications (such as beta-blockers) that limit cardiac output also cause a decrease in V.O2max, as does a reduction in hemoglobin associated with anemia.

Certain diseases in which muscle enzymes involved in metabolism are deficient can also result in reduced V.O2max. Although factors in each of these systems may limit the V.O2max, the question remains: What limits V.O2max in healthy humans performing maximal exercise? This question has energized exercise physiologists for decades, beginning with the work of A. V. Hill in the 1920s, and it continues to engender lively debate among physiologists today (Bassett and Howley, 2000; Bergh et al., 2000; Grassi, 2000; Saltin, 1985; Hale, 2008).

Current research suggests that maximal oxygen uptake is limited by the ability of the cardiorespiratory system to deliver oxygen to the muscle, rather than the ability of the muscle mitochondria to utilize oxygen (Bergh et al., 2000; Rowell, 1993; Saltin, 1985). Specifically, cardiac output appears to be the limiting factor in V.O2max (Bergh et al., 2000; di Prampero, 2003; Saltin, 1985).

Research evidence suggests that oxygen uptake is not limited by pulmonary ventilation in normal, healthy athletes without exercise-induced hypoxemia. Generally, the functional capacity of the respiratory

system is believed to exceed the demands of maximal exercise (Rowell, 1993). The only respiratory or cardiovascular variable likely to impose a limitation on oxygen transport is a−vO2diff.

Many researchers report that skeletal muscles have the ability to use more oxygen than can be supplied by the respiratory and cardiovascular systems (Richardson, 2000; Rowell, 1993; Saltin, 1985).

Not all researchers agree with this view, though, and some have proposed that failure of muscle performance may explain exhaustion during maximal exercise (Noakes, 1988). Possibly, the factors limiting V.O2max vary with the fitness level of the individual. According to this hypothesis, in an untrained individual the respiratory capacity for gas exchange exceeds the cardiovascular system's capacity to deliver oxygen. A training program results in little change in the respiratory capacity but large changes in the cardiovascular capacity. Thus, in some highly trained individuals who have exercise-induced arterial hypoxemia, the increased cardiovascular capacity may exceed the respiratory capacity (Dempsey, 1986; Legrand et al., 2005; Powers et al., 1989). In this case the respiratory system becomes the factor limiting V.O2max.

Blood Pressure

In well-equipped laboratories and hospitals, BP can be measured directly by an intra-arterial transducer. A small transducer is inserted into the artery, and SBP and DBP are recorded for every beat of the heart. Although this procedure provides valuable information, it is not practical for routine use because as an invasive procedure it involves risks. By far the most common technique for obtaining BP measurements is the auscultation method. The indirect method of auscultation uses a sphygmomanometer. For a BP measurement to be accurate and meaningful, the proper cuff size must be used to obtain the measurement. As a general rule, the BP cuff should encircle at least 80% of the arm circumference (American College of Sports Medicine, 2005). The general procedure for measuring BP is outlined in Figure 11.21. Note that SBP (Figure 11.21C) is taken as the First Korotkoff sound (the first loud sound heard through the stethoscope), but that there are two DBPs (Figure 11.21D). The first diastolic blood pressure (DBP1) occurs when the sound heard through the stethoscope is muffled (the Fourth Korotkoff sound). The second diastolic blood pressure (DBP2) occurs when sound disappears through the stethoscope (the Fifth Korotkoff sound). The pressure at the Fifth Korotkoff sound (DBP2) is considered the best measure of DBP in normal adults at rest. However, DBP1 is recommended for children, for adults during exercise, and for adults if DBP2 is lower than 40 mmHg (American Society of Hypertension, 1992). This is important for an accurate description of BP and in the calculation of MAP.

SELF-ASSESSMENT EXERCISES

i. Describe the Heart
ii. Define the Vascular System
iii. Differentiate between Blood and Hormonal Control of Blood Volume
iv. State the neural control and anatomical sensors and factors affecting control of the cardiovascular system.

4.0 CONCLUSION

Having read this course and successfully completed the assessment and self-assessment test, it is assumed that you have attained understanding of the introductory knowledge on the overview & regulation of the cardiovascular system.

5.0 SUMMARY

This Unit has successfully summarized the overview & regulation of the cardiovascular system.

6.0 TUTOR-MARKED ASSIGNMENT

7.0 REFERENCES/FURTHER READING

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UNIT 5 MEASUREMENT OF CARDIOVASCULAR VARIABLES & RESPONSES TO AEROBIC EXERCISE

CONTENTS

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1.0 INTRODUCTION

This unit describes the parallel cardiovascular responses to dynamic aerobic activity, static exercise, and dynamic resistance exercise. Minimal attention is paid here to short-term, high-intensity anaerobic exercise because this type of activity is typically performed to stress the metabolic system and is therefore discussed in detail in the metabolic unit.

2.0 INTENDED LEARNING OUTCOMES (ILOS)

By the end of this Unit, you will be able to;

- Describe the Short-Term, Light to Moderate Submaximal Aerobic Exercise
- Describe the Long-Term, Moderate to Heavy Submaximal Aerobic Exercise
- Describe the Incremental Aerobic Exercise to Maximum
- Describe the Upper-Body versus Lower-Body Aerobic Exercise
- Describe Intensity of Muscle Contraction
- Describe Blood Flow During Static Contractions

Cardiovascular Responses to Aerobic Exercise

Aerobic exercise requires more energy—and therefore more oxygen (remember the term aerobic means "with oxygen")—than either static or dynamic resistance exercise. How much oxygen is needed depends primarily on the intensity of the activity and secondarily on its duration. As in the discussion of respiration, this chapter categorizes exercises as short-term (5–10 min), light (30–49% of maximal oxygen consumption, V.O2max) to moderate (50–74% of V.O2max) submaximal exercise; long-term (>30 min), moderate to heavy (60–85% of V.O2max) submaximal exercise; or incremental exercise to maximum (increasing from ~30% to 100% V.O2max).

3.0 MAIN CONTENT

3.1 Short-Term, Light to Moderate: Submaximal Aerobic Exercise

There is generalized cardiovascular responses to short-term, light to moderate submaximal aerobic exercise. The actual magnitude of each variable's change depends on the work rate or load, environmental conditions, and the individual's genetic makeup and fitness level. At the onset of light- to moderate-intensity exercise, cardiac output (Q) initially increases to a plateau at steady state (see Figure 12.1A). Cardiac output plateaus within the first 2 minutes of exercise, reflecting the fact that cardiac output is sufficient to transport the oxygen needed to support the metabolic demands of the activity. Cardiac output increases because of an initial increase in both stroke volume (SV) and heart rate (HR); both levels off within 2 minutes.

During exercise of this intensity, the cardiorespiratory system can meet the body's metabolic demands; thus, this type of exercise is often called **steady-state** or steady-rate exercise. During steady-state exercise, energy provided aerobically is balanced with the energy required to perform the exercise. The plateau in cardiovascular variable indicates that a steady state has been achieved.

Stroke volume (SV) increases rapidly at the onset of exercise due to an increase in venous return which in turn increases the end-diastolic volume (EDV) (preload). The increased preload stretches the myocardium and causes it to contract more forcibly, as described by the Frank-Starling Law of the Heart. Contractility of the myocardium is also enhanced by the sympathetic nervous system, which is activated during physical activity. The increase in the EDV and the decrease in the endsystolic volume (ESV) both contribute to the increase in the SV during light to moderate dynamic exercise (Poliner et al., 1980). HR increases immediately at the onset of

activity as a result of parasympathetic withdrawal. As exercise continues, further increases in the HR result from the sympathetic nervous system activation (Rowell, 1986).

Systolic blood pressure (SBP) rises in a pattern very similar to that of cardiac output: an initial increase followed by a plateau once steady state is achieved. The increase in SBP results from the increased cardiac output. SBP would be even higher if not for the fact that resistance decreases, thereby partially offsetting the increase in cardiac output. When blood pressure (BP) is measured intra-arterially, diastolic blood pressure (DBP) does not change. When it is measured by auscultation, it either does not change or may go down slightly. DBP remains relatively constant because of peripheral vasodilation, which facilitates blood flow to the working muscles. The small rise in SBP and the lack of a significant change in DBP cause the mean arterial pressure (MAP) to rise only slightly, following the pattern of SBP. Total peripheral resistance (TPR) decreases because of vasodilation in the active muscles. This vasodilation results primarily from the influence of local chemical factors (lactate, K+, and so on), which reflect increased metabolism. The decrease in TPR has two important implications. First, vasodilation of the vessels supplying the active muscle causes decreased resistance that leads to an increased blood flow, thereby increasing the availability of oxygen and nutrients. Second, the decreased resistance keeps MAP from increasing dramatically. The increase in the MAP is determined by the relative changes in cardiac output and the TPR. Since cardiac output increases more than resistance decreases, the MAP increases slightly during dynamic aerobic exercise.

Myocardial oxygen consumption increases during dynamic aerobic exercise because the heart must do more work to increase cardiac output to supply the working muscles with additional oxygen. The rate- pressure product (RPP) increases in relation to increases in the HR and the SBP, reflecting the greater myocardial oxygen demand of the heart during exercise.

Blood volume decreases during submaximal aerobic exercise. Figure 12.2 shows the reduction of plasma volume during 30 minutes of moderate cycle ergometer exercise (60–70% V.O2max) in a warm environment (Fortney et al., 1981). The largest decrease occurs during the first 5 minutes of exercise, and then plasma volume stabilizes. This rapid decrease in plasma volume suggests that it is fluid shifts, rather than fluid loss, that account for the initial decrease (Wade and Freund, 1990). The magnitude of the decrease in plasma volume depends on the intensity of exercise, environmental factors, and the individual's hydration status. It shows the distribution of cardiac output at rest and during light aerobic exercise. Notice that cardiac output increases from 5.8 to 9.4 L·min−1 in this example (the increase in Q is illustrated by the larger pie chart). The

most dramatic change in cardiac output distribution with light exercise is the increased percentage (from 21% to 47%) and the increased blood flow (from 1200 to 4500 mL) to the working muscles. Skin blood flow also increases to meet the thermoregulatory demands of exercise. The absolute blood flow to the coronary muscle also increases, although its percentage of cardiac output remains relatively constant. The absolute amount of cerebral blood flow remains constant while the percentage of cardiac output distributed to the brain decreases. Both renal and splanchnic blood flow are modestly decreased during light exercise.

3.2 Long-Term, Moderate to Heavy Submaximal Aerobic Exercise

The cardiovascular responses to long-term, moderate to heavy submaximal aerobic exercise (60–85% of V.O2max). Similar to light to moderate workloads, cardiac output increases rapidly during the first minutes of exercise and then plateaus and remains relatively constant throughout the exercise. Notice, however, that the absolute cardiac output attained is higher during heavy exercise than during light to moderate exercise. The increase in cardiac output results from increased SV and HR.

SV has an initial increase, plateaus, and then has a negative (downward) drift as exercise duration continues past approximately 30 minutes. SV increases rapidly during the first minutes of exercise and then plateaus after a workload of approximately 40–50% of V.O2max is achieved (Åstrand et al., 1964) (Figure 12.4B). Thus, during work that requires more than 50% of V.O2max, the SV response does not depend on intensity. SV remains relatively constant during the first 30 minutes of heavy exercise. As with short-term, light to moderate submaximal aerobic exercise, the increase in SV is conventionally believed to result from an increased venous return, leading to the Frank-Starling mechanism, and increased contractility due to sympathetic nerve stimulation. Thus, changes in SV occur because EDV increases and ESV decreases (Poliner et al., 1980). EDV increases primarily because of the increased venous return of blood to the heart by the active muscle pump and increased venoconstriction, which decreases venous pooling. ESV decreases because of augmented contractility of the heart, which effectively ejects more blood. If the exercise continues beyond approximately 30 minutes, SV gradually drifts downward while remaining above the resting value.

This downward shift is most often attributed to thermoregulatory stress, which results in vasodilation, plasma loss, and a redirection of blood to the cutaneous vessels to dissipate heat, thus effectively reducing venous return and thus SV. This theory suggests that HR increases to compensate for a decrease in SV in order to maintain Q. An alternate viewpoint

suggests that the downward drift in SV is due to an increase in HR (due to augmented sympathetic nerve activity) that leads to a reduced filling time, thus leading to a reduced SV (Rowland, 2005b).

HR initially increases, plateaus at steady state, and then has a positive drift. HR increases sharply during the first 1–2 minutes of exercise, the magnitude of which depends on the intensity of exercise. The increase in HR is brought about by parasympathetic withdrawal and activation of the sympathetic nervous system. After approximately 30 minutes of heavy exercise, HR begins to drift upward. The increase in HR is proportional to the decrease in SV, so cardiac output is maintained during exercise.

These cardiovascular changes, notably in HR and SV, during long-term, moderate to heavy submaximal aerobic exercise without a change in workload are known as **cardiovascular drift**. Cardiovascular drift is probably associated with rising body temperature during prolonged exercise. Exercise and heat stress produce competing regulatory demands, as the skin and the muscles compete for increased blood flow. SV decreases as a result of vasodilation, a progressive increase in the fraction of blood being directed to the skin, and a loss of plasma volume (Rowell, 1974; Sjogaard et al., 1988).

SBP response to long-term, moderate to heavy submaximal aerobic exercise is characterized by an initial increase, a plateau at steady state, and a negative drift.

SBP increases rapidly during the first 1–2 minutes of exercise, the magnitude of increase depending on the intensity of the exercise. SBP then remains relatively stable or drifts slightly downward as a result of continued vasodilation and a resultant decrease in resistance (Ekelund and Holmgren, 1967).

DBP does not change, or changes so little that it has no physiological significance, during prolonged exercise in a thermoneutral environment. But it may decrease slightly in a warm environment because of increased vasodilation resulting from heat production. Because of the increased SBP and the relatively stable DBP, MAP increases modestly during prolonged activity. Again, as in light to moderate exercise, the magnitude of the increase in MAP is mediated by a large decrease in resistance.

TPR decreases rapidly, plateaus, and then has a slight negative drift during long-term heavy exercise because of vasodilation in active muscle and because of vasodilation in cutaneous vessels (Rowell, 1974). Finally, because both HR and SBP increase substantially during heavy work, the rate-pressure product increases markedly with the onset of exercise and then plateaus at steady state. An upward drift in rate-pressure product may

occur after approximately 30 minutes of exercise because the HR increases more than SBP decreases. The high rate-pressure product reflects the large amount of work the heart must perform to support heavy exercise.

During prolonged exercise, particularly in a warm environment, total body fluid is continually lost due to sweating.

This loss typically ranges from 900 to 1300 mL·hr−1, depending on work intensity and environmental conditions (Wade and Freund, 1990). If fluid is not replaced during long-duration exercise, plasma volume will continually be reduced throughout the exercise. It shows the distribution of cardiac output at rest and during heavy aerobic exercise. Notice that cardiac output increases from 5.8 L·min−1 at rest to 17.5 L·min−1 in this example. The most dramatic change here is the increased blood flow to the working muscle, which now receives 71% of cardiac output. Skin blood flow is also increased to meet the thermoregulatory demands. The absolute blood flow to the coronary muscle increases while its percentage of cardiac output remains relatively constant. The absolute cerebral blood flow remains constant, while its percentage of cardiac output decreases. Both renal and splanchnic blood flow are further decreased as exercise intensity increases. Although blood flow to the working muscle increases during aerobic exercise, blood flow to the inactive muscle decreases because of vasoconstriction. Vasoconstriction in inactive muscle is necessary to ensure that cardiac output can supply adequate blood flow to the working muscle. It presents data from a study in which participants exercised on a cycle ergometer for 8 minutes, then added an arm cranking exercise (Secher et al., 1977). The combination of leg and arm cycling increased cardiac output modestly but actually caused a decrease in leg blood flow because a portion of cardiac output now had to be distributed to the working muscles in the arm.

3.3 Incremental Aerobic Exercise to Maximum

There are cardiovascular responses to incremental aerobic exercise to maximum. Note that unlike the graphs for light to moderate and heavy exercise, the cardiovascular variables are now presented with percentage of maximum work on the x-axis. Incremental exercise to maximum (or a max test) consists of a series of work stages, each becoming progressively harder, that continue until volitional fatigue. The duration of each work stage (level of intensity) varies from 1 to 3 minutes to allow a steady state to occur, at least at the lower workloads. Max tests are performed in laboratory settings to quantify physiological responses to the maximal work that an individual can perform. During an incremental test, cardiac output has a rectilinear increase and plateaus at maximal exercise. The initial increase in cardiac output reflects an increase in the SV and the HR;

however, at workloads greater than 40–50% V.O2max, the continued increase in cardiac output in untrained individuals is achieved almost completely by an increase in the HR.

In untrained individuals, the SV increases rectilinearly initially and then plateaus at approximately 40–50% of V.O2max (Åstrand et al., 1964; Higginbotham et al., 1986). The exact SV response to incremental exercise continues to be debated (González-Alonso, 2008; Rowland, 2005a,b; Warburton and Gledhill, 2008). As indicated above, it has traditionally been believed that the SV plateaus at approximately 50% of V.O2max in untrained individuals. However, there appears to be considerable interindividual variability in this response, and many laboratories have reported an increase in the SV at maximal exercise in most endurance athletes and some untrained individuals (Ferguson et al., 2001; Gledhill et al., 1994; Warburton et al., 1999). In contrast, other researchers have documented a decrease in the SV at the maximal exercise (Mortensen et al., 2005; Stringer et al., 2005), and some researchers contend that after an initial increase (due to the skeletal muscle pump returning the pooled venous blood to the heart), the SV remains essentially unchanged during the incremental maximal exercise (Rowland, 2005b). Much of the controversy is undoubtedly associated with difficulties in measuring the SV during maximal exercise, with the use of different exercise protocols, and with individual variability. There is an indication that the changes in the EDV and the ESV that account for changes in the SV during progressively increasing exercise (Poliner et al., 1980). The EDV increases largely because of the return of blood to the heart by the active muscle pump and the increased sympathetic outflow to the veins causing venoconstriction and augmenting venous return. ESV decreases because of augmented contractility of the heart, which ejects more blood and leaves less in the ventricle.

HR increases in a rectilinear fashion throughout much of the submaximal (~120–170 b·min−1) portion of incremental exercise and plateaus at maximal exercise (Astrand and Rhyming, 1954; Hale, 2008). Myocardial cells can contract at over 300 b·min−1 but rarely exceed 210 b·min−1 because a faster HR would not allow for adequate ventricular filling. Thus, SV and ultimately cardiac output would decrease. Consider the simple analogy of a bucket brigade. Up to a point it is useful to increase the speed of passing buckets under the water source, but the maximum rate is limited because of the time required for the buckets to be filled with water. The maximal amount of oxygen an individual can take in, transport, and utilize (V.O2max) is usually measured during an incremental maximal exercise test. Although V.O2max is considered primarily.

3.4 Upper-Body versus Lower-Body Aerobic Exercise

Upper-body exercise is routinely performed in a variety of industrial, agricultural, military, recreational, and sporting activities. The cardiovascular responses to exercise -using muscles of the upper body are different in some important ways from exercise performed using muscles of the lower body. This presents data about cardiovascular responses to incremental exercise to maximum in able-bodied individuals using the upper body (arm cranking on an arm ergometer) versus lower body (cycling on a cycle ergometer). Notice that a higher peak V.O2 was achieved during lower-body exercise. Comparisons at any given level of oxygen consumption also show differences in cardiovascular responses to submaximal upper- and lower-body exercise. When the oxygen consumption required to perform a submaximal workload is the same, cardiac output is similar for upper- and lower-body exercise. However, the mechanism to achieve the required increase in cardiac output is not the same.

Upper-body exercise results in a lower SV and a higher HR at any given submaximal workload (Clausen, 1976; Miles et al., 1989; Pendergast, 1989). SBP, DBP, MAP, total peripheral resistance (Figure 12.10E), and rate pressure product (Figure 12.10F) are significantly higher in upper-body exercise than in lower-body exercise performed at the same oxygen consumption. There are several likely reasons for the differences. The higher HR observed during upper-body exercise is thought to reflect a greater sympathetic stimulation (Åstrand and Rodahl, 1986; Davies et al., 1974; Miles et al., 1989). SV is lower during upper-body exercise because of the absence of the skeletal muscle pump augmenting venous return from the legs. The greater sympathetic stimulation that occurs during upper-body exercise may also be partially responsible for the increased BP and total peripheral resistance. Upper-body exercise often involves a static component which causes an exaggerated BP response. For instance, using an arm-cranking ergometer has a static component because the individual must grasp the hand crank.

When maximal exercise is performed using upper-body muscles, V.O2max values are approximately 30% lower than when maximal exercise is performed using lower-body muscles (Miles et al., 1989; Pendergast, 1989). Maximal HR values for upper-body exercise are 90–95% of those for lower-body exercise, and SV is 30–40% less during maximal upper-body exercise. Maximal SBP and the rate-pressure product are usually similar, but DBP is typically 10–15% higher during upper-body exercise (Miles et al., 1989). The different cardiovascular responses to an absolute workload performed with the upper body versus the lower body dictate that exercise prescriptions for arm work cannot be based on data obtained from testing with leg exercises. Furthermore, the

greater cardiovascular strain associated with upper-body work must be kept in mind when one prescribes exercise for individuals with cardiovascular disease.

Cardiovascular Responses to Static Exercise

Static work occurs repeatedly during daily activities, such as lifting and carrying heavy objects. It is also a common form of activity encountered in many occupational settings, particularly manufacturing jobs where lifting is common. Additionally, many sports and recreational activities have a static component associated with their performance. For example, weight lifting, rowing, and racquet sports all involve static exercise. The magnitude of the cardiovascular response to static exercise is affected by several factors, but most noticeably by the intensity of muscle contraction.

3.5 Intensity of Muscle Contraction

The cardiovascular response to static exercise depends on the intensity of contraction, provided the contraction is held for a specified time period. The intensity of a static contraction is expressed as a percentage of maximal voluntary contraction (%MVC). It illustrates the cardiovascular response to static contractions of the forearm (handgrip) muscles at 10, 20, and 50% MVC. Notice that at 10% and 20% MVC the contraction could be held for 5 minutes, but at 50% MVC the contraction could be held for only 2 minutes. Thus, as in aerobic exercise, intensity and duration are inversely related. Also note that the data presented in this figure are from handgrip exercises. Although the pattern of response appears to be similar for different muscle groups, the actual values may vary considerably depending on the amount of active muscle involved.

Cardiac output increases during static contractions due to an increase in HR, with the magnitude of the increase dependent on the intensity of exercise. SV remains relatively constant or decreases slightly during low-intensity contractions and decreases during high intensity contractions.

There is a marked increase in SV immediately following the cessation of high-intensity contractions (Lind et al., 1964; Smith et al., 1993). This is the same rebound rise in recovery as seen in a–vO2diff, V.E, and V.O2. The reduction in SV during high-intensity contractions probably results from both a decreased preload and an increased afterload. Preload is decreased because of high intrathoracic pressure, which compresses the vena cava and thus decreases the return of venous blood to the heart. Because arterial BP is markedly elevated during static contractions (increased afterload), less blood is ejected at a given force of contraction. HR increases during static exercise. The magnitude and the rate of the increase in HR depend on the intensity of contraction. The greater the

intensity, the greater the HR response. Static exercise is characterized by a rapid increase in both systolic pressure and diastolic pressure, termed the **pressor response**, which appears to be inappropriate for the amount of work produced by the contracting muscle (Lind et al., 1964). Since both systolic and diastolic pressures increase, there is a marked increase in MAP (Donald et al., 1967; Lind et al., 1964; Seals et al., 1985; Tuttle and Horvath, 1957). As in any muscular work, static exercise increases metabolic demands of the active muscle. However, in static work, high intramuscular tension results in mechanical constriction of the blood vessels, which impedes blood flow to the muscle. The reduction in muscle blood flow during static exercise results in a buildup of local by- products of metabolism. These chemical by-products [H+, adenosine diphosphate, and others] stimulate sensory nerve endings, which leads to a pressor reflex, causing a rise in MAP (pressor response). This rise is substantially larger than the increase during aerobic exercise requiring similar energy expenditure (Asmussen, 1981; Hanson and Nagle, 1985). Notice in Figure 12.11D that holding a handgrip dynamometer at 20% MVC for 5 minutes results in an increase of 20–30 mmHg in MAP, and holding 50% MVC for 2 minutes caused a 50-mmHg increase in MAP! Total peripheral resistance, indicated by TPR, decreases during static exercise, although not to the extent seen in dynamic aerobic exercise. The smaller decrease in resistance helps to explain the higher BP response to static contractions. The high BP generated during static contractions helps overcome resistance to blood flow from mechanical occlusion. Because the SBP and the HR both increase during static exercise, there is a large increase in myocardial oxygen consumption and thus rate-pressure product.

3.6 Blood Flow During Static Contractions

Blood flow to the working muscle is impeded during static contractions because of the mechanical constriction of the blood vessel supplying the contracting muscle (Freund et al., 1979; Sjogaard et al., 1988). It depicts blood flow in the quadriceps muscle when a 5% and 25% MVC contraction were held to fatigue. The 5% MVC load could be held for 30 minutes; the 25% load could be held for only 4 minutes. Quadriceps blood flow is greater during the 5% MVC, suggesting that at 25% MVC there is considerable impedance to blood flow. In fact, blood flow during the 25% MVC load was very close to resting levels despite the metabolic work done by the muscle. The response occurring during recovery suggests that when contraction ceases, a mechanical occlusion to the muscle is released. The marked increase in blood flow during recovery compensates for the reduced flow during sustained contraction. The relative force at which blood flow is impeded varies greatly among different muscle groups (Lind and McNichol, 1967; Rowell, 1993).

Mechanical constriction also occurs during dynamic aerobic exercise. However, the alternating periods of muscular contraction and relaxation during rhythmical activity allow—and, indeed, encourage—blood flow, especially through the venous system.

Comparison of Aerobic and Static Exercise

This compares the HR and the BP responses to fatiguing handgrip (static) exercise (30% MVC held to fatigue) and a maximal treadmill (incremental aerobic) test to fatigue. The incremental aerobic exercise is characterized by a large increase in the HR, which contributes to an increased cardiac output. The treadmill exercise response also shows a modest increase in the SBP and a relatively stable or decreasing DBP. Aerobic exercise is said to impose a "volume load" on the heart. Increased venous return leads to increased SV, which contributes to an increased cardiac output. In contrast, fatiguing static exercise is characterized by a modest increase in the HR, but a dramatic increase in the BP (pressor response). Mean BP increases as a result of increased SBP and DBP. Static exercise is said to impose a "pressure load" on the heart. Increased MAP means that the heart must pump harder to overcome the pressure in the aorta.

Cardiovascular Responses to Dynamic Resistance Exercise

Weight-lifting or resistance exercise includes a combination of dynamic and static contractions (Hill and Butler, 1991; MacDougall et al., 1985). At the beginning of the lift, a static contraction exists until muscle force exceeds the load to be lifted and movement occurs, leading to a dynamic concentric (shortening) contraction as the lift continues. This is then followed by a dynamic eccentric (lengthening) contraction during the lowering phase (McCartney, 1999). A static component is always associated with gripping the barbell. During dynamic resistance exercise, cardiorespiratory system responses are dissociated from the energy demand. In contrast, during dynamic endurance activity, responses in the cardiorespiratory system are directly related to the use of oxygen for energy production. In part, the reason for this dissociation between oxygen use and cardiovascular response to resistance exercise is that much of the energy required for resistance exercise comes from anaerobic (without oxygen) sources. Another important difference between resistance exercise and aerobic exercise is the mechanical constriction of blood flow during resistance exercise because of the static nature of the contraction.

The magnitude of the cardiovascular response to resistance exercise depends on the intensity of the load (the weight lifted) and the number of repetitions performed.

Cardiovascular responses also depend on how the load and repetitions are combined.

Varying Load/Constant Repetitions

As expected, cardiovascular responses are greater when heavier loads are lifted, assuming the number of repetitions is constant (Fleck, 1988; Fleck and Dean, 1987). For example, when participants performed ten repetitions of arm curling exercises with dumbbells of three different weights (identified as light, moderate, and heavy), the SBP was highest *at the completion* of the heaviest set (Wescott and Howes, 1983). The SBP increased 16%, 22%, and 34% during the light, moderate, and heavy sets, respectively. The DBP, measured by auscultation, did not change significantly with any of the sets. There is disagreement about the DBP response to resistance exercise; some authors report an increase, while others report no change (Fleck, 1988; Fleck and Dean, 1987; Wescott and Howes, 1983). These discrepancies may refl ect differences in measurement techniques (auscultation versus intra-arterial assessment) and timing of the measurement.

Varying Load/Repetitions to Failure

A different pattern of response is seen when a given load is performed to fatigue, which lifters typically call failure. In this case, the individual performs maximal work regardless of the load. This shows the cardiovascular response at the completion of leg extension exercise performed to failure. Participants performed 50%, 80%, and 100% of their one repetition maximum (1-RM) as many times as they could, and cardiovascular variables were recorded at the end of each set (Falkel et al., 1992).

Participants could perform the 100% load only one time, of course, but they could perform the 80% and 50% loads an average of 8 and 15 times, respectively. Thus, the greatest volume of work was performed when the lightest load was lifted the greatest number of times. Cardiac output *at the completion* of the set was highest when the lightest load was lifted for the most repetitions—that is, when the total work was greatest. The SV at the end of a set was similar for each condition and was slightly below resting levels. This is in contrast to significant increases in the SV that occur during aerobic exercise. Thus, dynamic resistance exercise does not produce the SV overload of dynamic aerobic exercise (Hill and Butler, 1991; McCartney, 1999). The HR was highest after completion of the set using the lightest load and lifting it the most times. The HR was lowest when a single repetition using the heaviest weight was performed. HRs between 130 and 160 b·min−1 have been reported during resistance exercise (Hill and Butler, 1991). There is some evidence that the HR and the BP attained at fatigue are the same when loads between 60% and

100% of 1-RM are used, regardless of the number of times the load can be performed (Nau et al., 1990).

Constant Load/Repetitions to Failure

When the load is heavy, MAP and HR increase with succeeding repetitions in a set to failure (Fleck and Dean, 1987; MacDougall et al., 1985). Figure 12.16A shows the MAP, measured intra-arterially, *during a set* of leg press exercises that represented 95% of 1-RM; It shows the HR during these exercises. In this study, peak SBP averaged 320 mmHg, and peak DBP averaged 250 mmHg! The dramatic increase in BP during dynamic resistance exercise results from the mechanical compression of blood vessels and performance of the Valsalva maneuver. The TPR is higher during dynamic resistance exercise than during dynamic aerobic exercise because of the vasoconstriction caused by the pressor reflex. In fact, some studies have reported a slight increase in the TPR during resistance exercise, rather than the decrease observed with aerobic exercise (Lentini et al., 1993; McCartney, 1999; Miles et al., 1987). Myocardial oxygen consumption and thus the rate-pressure product can reach extremely high levels because of the tachycardia and the exaggerated SBP response. Dynamic resistance exercise also causes large (about 15%) but transient decreases in plasma volume (Hill and Butler, 1991). The cardiovascular response of children to resistance exercise is similar to that of adults, with the HR and the BP increasing progressively throughout a set (Nau et al., 1990).

Cardiovascular responses to resistance exercise. Resistance exercises are generally undertaken to enhance muscle size or to improve muscular health (strength or endurance). The goal is not to stress the cardiovascular system. Hence, there is insufficient evidence to adequately compare cardiovascular responses to resistance exercise among different populations (male versus female, children versus adult, and young versus older adults). Therefore, this exercise category is not included in the following sections.

Male-Female Cardiovascular Differences During Exercise

The pattern of cardiovascular responses to aerobic exercise is similar for both sexes, although the magnitude of the response may vary for some variables. Many of the differences in cardiovascular responses between the sexes are related to differences in body size and structure.

Short-term, Light to Moderate and Long-term, Moderate to Heavy Submaximal Exercise

Females have a higher cardiac output and HR, but a lower SV, than males during submaximal exercise when work is performed at the same *absolute workload* (Åstrand et al., 1964; Becklake et al., 1965; Freedson et al., 1979). The higher HR more than compensates for the lower SV in

females, resulting in the higher cardiac output seen at the same absolute workload. Thus, if a male and a female perform the same workout, the female will typically be stressing the cardiovascular system to a greater extent. This relative disadvantage to the women results from several factors. First, females typically are smaller than males; they have a smaller heart and less muscle mass. Second, they have a lower oxygen-carrying capacity than males. Finally, they typically have lower aerobic capacity (V.O2max).

When males and females perform the same *relative workload* (both working at the same percentage of their V.O2max), a different pattern emerges. The importance of distinguishing between relative and absolute workloads. This figure shows results from a study that compared the cardiovascular response of men and women to the same absolute work rate (600 kg·min−1) and the same relative work rate (50% of V.O2max). Although cardiac output was higher in women during the same absolute work rate, it is lower for women when the same relative work rate was performed. The SV was lower in women than in men whether the work was expressed on an absolute or relative basis. Notice that the values are very similar for both conditions, suggesting that the SV has plateaued as would be expected at 50% of V.O2max in both conditions. The difference in HR between the sexes was smaller when exercise was performed at the same relative work rate.

Males and females display the same pattern of response for BP; however, males tend to have a higher SBP at the same relative workloads (Malina and Bouchard, 1991; Ogawa et al., 1992). Much of the difference in the magnitude of the BP response is attributable to differences in resting SBP. The DBP response to submaximal exercise

is very similar for both sexes. Thus, MAP is slightly greater in males during submaximal work at the same relative workload. The pattern of response for resistance is similar for males and females, although males typically have a lower resistance because of their greater cardiac output. Males and females both exhibit cardiovascular drift during heavy, prolonged submaximal exercise.

Incremental Aerobic Exercise to Maximum

The cardiovascular response to incremental exercise is similar for both sexes, although again there are differences in the maximal values attained. Maximal oxygen consumption (V.O2max) is higher for males than for females.

When V.O2max is expressed in absolute values (L·min−1), males typically have values that are 40–60% higher than in females (Åstrand, 1952; Sparling, 1980). When differences in body size are considered and

V.O2max values are expressed relative to body weight (in mL·kg−1·min−1), the differences between the sexes decreases to 20–30%. If differences in body composition are considered and V.O2max is expressed relative to fat-free mass (in mL·kg−1 of fat-free mass per minute), the difference between the sexes is reduced to 0–15% (Sparling, 1980). Reporting V.O2max relative to fat-free mass is important in terms of understanding the infl uence of adiposity and fat-free mass on V.O2max. However, it is not a very practical way to express V.O2max because, in reality, consuming oxygen only in relation to fat-free mass is not an option. Individuals cannot leave their fat mass behind when exercising.

This represents the distribution of V.O2max values for males and females expressed per kilogram of weight and per kilogram of fat-free mass. This figure demonstrates the important point that there is considerable variability in V.O2max for both sexes. Thus, although males generally have a higher V.O2max, some females will have a higher V.O2max than the average men.

There is differences in V.O2max, expressed in relative terms and absolute terms, and average body weight between the sexes across the age span.

Differences in V.O2max are largely explained by the differences in the size of the heart (and thus maximal cardiac output) and the differences in the oxygen-carrying capacity of the blood. Males have approximately 6% more red blood cells and 10–15% more hemoglobin than females; thus, males have a greater oxygen-carrying capacity (Åstrand and Rodahl, 1986). Males typically have a maximal cardiac output that is 30% higher than females (Wells, 1983). Maximal SV is higher for men, but the increase in SV during maximal exercise is achieved by the same mechanisms in both sexes (Sullivan et al., 1991). Furthermore, if maximal SV is expressed relative to body weight, there is no difference between the sexes. The maximal HR is similar for both sexes.

Males and females display the same pattern of BP response; however, males attain a higher SBP than females at maximal exercise (Malina and Bouchard, 1991; Ogawa et al., 1992; Wanne and Haapoja, 1988). The DBP response to maximal exercise is similar for both sexes. Thus, MAP is slightly greater in males at the completion of maximal work. The pattern of response for resistance and rate-pressure product is the same for both sexes. Resistance is greatly reduced during maximal exercise in both sexes. Because the HR response is similar and the SBP is greater in males, males tend to have a higher rate-pressure product at maximal exercise levels than do females. The differences between the sexes in cardiovascular variables at various exercise levels.

Static Exercise

The HR response to static exercise is similar in males and females (Misner et al., 1990). However, when a group of young adult, healthy participants held maximal contractions of the handgrip muscles for 2 minutes, the BPs reported for women were significantly lower than those reported for men (Misner et al., 1990). The SV and cardiac output responses in women during maximal static contraction of the finger flexors were similar to the responses previously reported in men, but no direct comparisons between men and women were made in this study (Smith et al., 1993).

Cardiovascular Responses of Children and Adolescents to Exercise

The pattern of responses in the cardiovascular variables in children and adolescents to aerobic exercise is similar to the pattern in adults. This is not meant to imply that children are simply "little adults". Often, the actual values are higher or lower than adults, but overall, the direction and the relative degree of change are very similar across the age range. Differences can frequently be attributed to differences in body size, structure, and maturity (Rowland, 2005a,b).

Short-term, Light to Moderate and Long-term, Moderate to Heavy Submaximal Exercise

The pattern of cardiac output response to submaximal aerobic exercise is similar in children and adolescents to adults, with cardiac output increasing rapidly at the onset of exercise and plateauing at steady state. However, children have a lower cardiac output than adults at all levels of exercise, primarily because children have a lower SV at any level of exercise (Bar-Or, 1983; Rowland, 1990). As children grow and mature, cardiac output and SV increase at rest and during exercise. The lower SV in children is compensated for, to some extent, by a higher HR. The HR response to any given exercise intensity is highest in young children (Bar-Or, 1983; Cunningham et al., 1984) and declines as children grow into adolescents (Rowland, 2005a,b). Children, adolescents, and adults all exhibit the cardiovascular drift phenomenon of a slight (~15%) progressive rise in HR, simultaneous decreases in SV and MAP, and no change in cardiac output with prolonged exercise (Asano and Hirakoba, 1984; Rowland, 2005a SV in girls is less than that in boys at all levels of exercise (Bar-Or, 1983).

As always, the magnitude of the cardiovascular response depends on the intensity of the exercise. The cardiac output, SV, and HR values of children 8–12 years old during treadmill exercise at 40%, 53%, and 68% of V.O2max (Lussier and Buskirk, 1977). Both cardiac output and HR increase in response to increasing intensities of exercise. SV peaks at 40% of V.O2max and changes little with increasing exercise intensity. This is consistent with the fi nding that SV plateaus at 40–50% of V.O2max in adults (Åstrand et al., 1964). The SBP in children increases during

exercise, as it does in adults, and depends on the intensity of the exercise. Boys tend to have a higher SBP than girls (Malina and Bouchard, 1991). The magnitude of the increase in systolic pressure at submaximal exercise is less in children than in adults (James et al., 1980; Wanne and Haapoja, 1988). The failure of SBP to reach adult levels is probably the result of lower cardiac output in children. As children mature, the increases in the SBP during exercise become greater. Diastolic pressure changes little during exercise but is lower in children than adults (James et al., 1980; Wanne and Haapoja, 1988).

Similar decreases in resistance occur in children as in adults, a result of vasodilation in working muscles. Rate pressure product increases in children and adolescents during exercise. However, the work of the heart reflects the higher HR and lower SBP for these age groups than for adults. Blood flow through the exercising muscle appears to be greater in children than in adults, resulting in a higher a–vO2diff and thereby compensating partially for the lower cardiac output (Rowland, 1990; Rowland and Green, 1988).

Incremental Aerobic Exercise to Maximum

The cardiovascular responses to incremental exercise to maximum are similar for children, adolescents, and adults; however, children and adolescents achieve a lower maximal cardiac output and a lower maximal SV. HR rises in a rectilinear fashion with incremental exercise in children as in adults. However, at approximately 60% V.O2max, it begins to taper. Maximal HR is higher in children than in adults and is not age dependent until the late teens (Cunningham et al., 1984; Rowland, 1996, 2005a). The maximal oxygen consumption typically attained by youths between the ages of 6 and 18. As children grow, their ability to take in, transport, and utilize oxygen improves. This improvement represents dimensional and maturational changes—specifically, heart volume, maximal SV, maximal cardiac output, blood volume and hemoglobin concentration, and a–vO2diff increase.

The rate of improvement in absolute V.O2max (expressed in L·min−1) is similar for boys and girls until approximately 12 years of age (Figure 12.21A). Maximal oxygen uptake continues to increase in boys until the age of 18; it remains relatively constant in girls between the ages of 14 and 18.

When V.O2max is expressed relative to body weight (expressed as mL·kg−1·min−1), it remains relatively constant throughout the years between 8 and 16 for boys. However, the V.O2max (expressed as mL·kg−1·min−1) tends to decrease in girls as they enter puberty and their adiposity increases. As children mature, they also grow, and the developmental changes indicated previously are largely offset if

V.O2max is described per kilogram of body weight. The large area of overlap for reported values of V.O2max for boys and girls in Figure 12.21 reflects the large variability in V.O2max among children and adolescents. There appears to be a major difference between children/adolescents and adults in terms of the meaning of V.O2max. In adults, V.O2max reflects both physiological function (cardiorespiratory power) and cardiovascular endurance (the ability to perform strenuous, large- muscle exercise for a prolonged period of time) (Taylor et al., 1955). In children and adolescents, V.O2max is not as directly related to cardiorespiratory endurance as in adults (Bar-Or, 1983; Krahenbuhl et al., 1985; Rowland, 1990). It shows performance as determined by the number of stages or minutes completed in the PACER test (Léger et al., 1988). This progressive aerobic cardiovascular endurance run (PACER). Recall that a higher number of laps completed is positively associated with a higher V.O2max. It shows that for boys, the mean estimated value of V.O2max, expressed in mL·kg−1·min−1, changes very little from age 6–18. However, mean performance on the PACER test shows a definite linear improvement with age. The girls show the same trend as the boys before puberty, but thereafter, V.O2max declines steadily and PACER performance plateaus. Similar results have been reported for treadmill endurance times and other distance runs (Cumming et al., 1978) and measured V.O2max as well as estimated (Rowland, 2005a). Thus, in general, endurance performance improves progressively throughout childhood, at least until puberty, but V.O2max, expressed relative to body size, does not. The reason for the weak association between V.O2max and endurance performance in young people is unknown. The most frequent suggestion is that children use more aerobic energy (require greater oxygen) than adults at any submaximal pace. This phenomenon is called *running economy* and is fully discussed in the unit on metabolism. More important than the actual oxygen consumption at a set pace, however, may be the percentage of V.O2max that value represents, and more so in children than adolescents (McCormack et al., 1991). Other factors that may affect endurance running performance in children and adolescents include body composition, particularly the percentage of body fat; sprint speed, possibly as a reflection of a high percentage of muscle fibers differentiated for speed and power; and various aspects of body size (Cureton et al., 1991, 1977; Mayhew and Gifford, 1975; McVeigh et al., 1995). There is also the possibility that many children and adolescents are not motivated to perform exercise tests and therefore do not perform well despite high V.O2max capabilities. It presents the arterial BP response of children and adolescents to incremental maximal exercise. The BP response is similar for children and adults; however, there are again age- or size-related quantitative differences. For a given level of exercise, a small child responds with a lower SBP and DBP than an adolescent, and an adolescent responds with lower BP than an adult. The lower BP response in young children is consistent with their lower SV response.

Typically, boys have a higher peak SBP than girls (Riopel et al., 1979; Wade and Freund, 1990). This difference too is most likely attributable to differences in SV. Myocardial oxygen consumption at maximal exercise increases as children grow—predominantly through the influence of higher maximal SBP since maximal HR is stable until late adolescence. It reports typical cardiovascular responses to maximal exercise in prepubescent and postpubescent children.

Static Exercise

Children's and adolescents' cardiovascular responses to static exercise appear to be similar to adults' (Rowland, 2005a,b). It presents data from two studies that investigated cardiovascular responses to 3 minutes of leg extension exercise at 30% of MVC. One study tested young men between the ages of 25 and 34 (Bezucha et al., 1982), and the other young boys aged 7–12 (Rowland et al., 2006). In both studies, static exercise resulted in typical responses: an increased MAP, an elevated HR, a decreased SV, and a small rise in cardiac output. Similarly, a study that compared premenarcheal girls and young women found no differences in cardiovascular responses to 3 minutes of 30% MVC of the handgrip muscles (Smith et al., 2000).

Cardiovascular Responses of Older Adults to Exercise

Aging is associated with diminishing function in many systems of the body. Thus, aging is characterized by a decreased ability to respond to physiological stress (Skinner, 1993). There is considerable debate, though, about how much loss of function is inevitably related to age, how much is related to disease, and how much can be attributed to a sedentary lifestyle often accompanying aging. Each of these factors causes decrements in function, but for an individual, it is often difficult to know which one or which combination may cause an observed change.

Many older adults remain active into their later years and perform amazing athletic feats. For example, Mavis Lindgren began an exercise program of walking in her early 60s. She slowly increased her training volume and began jogging. At age 70, she completed her first marathon. In the next 12 years, she raced in over 50 marathons (Nieman, 1990). Many studies of physical activity suggest that by remaining active in the older years, individuals can markedly reduce loss of cardiovascular function, even if they do not run a marathon.

Short-term, Light to Moderate and Long-term, Moderate to Heavy Submaximal Exercise

At the same absolute submaximal workload, cardiac output and SV are lower in older adults, but HR is higher than in younger adults. The pattern of systolic and diastolic pressure is the same for younger and older individuals. The difference in resting BP is maintained throughout the exercise, so that older individuals have a higher SBP, DBP, and MAP at

any given level of exercise (Ogawa et al., 1992). The higher BP response is related to a higher TPR in older individuals, resulting from a loss of elasticity in the blood vessels. Because HR and SBP are higher for any given level of exercise in older adults, myocardial oxygen consumption and thus rate pressure product are also higher in older individuals than in younger adults.

Incremental Aerobic Exercise to Maximum

Maximal cardiac output is lower in older individuals than in younger adults. This results from a lower maximal HR and a lower maximal SV. Maximal SV decreases with advancing age, and the decline is of similar magnitude for both men and women, although women have a much smaller maximal SV initially. Maximal HR decreases with age but does not vary significantly between the sexes. A decrease of approximately 10% per decade, starting at approximately age 30, has been reported for V.O2max in sedentary and active adults (Åstrand, 1960; Heath et al., 1981; Wilson and Tanka, 2000). There is some indication that the rate of decline in V.O2max is greater in men than in women (Stathokostas et al., 2004; Weiss et al., 2006). It depict the change in V.O2max from childhood to 75 years of age. Like resting BP, SBP and DBP responses to maximal aerobic exercise are typically higher in older individuals than in younger individuals of similar fitness (Ogawa et al., 1992). Maximal SBP may be 20–50 mmHg higher in older individuals, and maximal DBP 15–20 mmHg higher. As a result of an elevated SBP and DBP, MAP is considerably higher at maximal exercise in older than in younger adults.
TPR decreases during aerobic exercise in older adults but not to the same extent as in younger individuals. This difference is a consequence of the loss of elasticity of the connective tissue in the vasculature that accompanies aging. Since the decrease in maximal HR for older individuals is greater than the increase in maximal SBP when compared to younger adults, older individuals have a lower rate-pressure product at maximal exercise. There is a typical cardiovascular values at maximal exercise in young and old adults of both sexes.

Static Exercise

Many studies have described the cardiovascular responses to static exercise in older adults (Goldstraw and Warren, 1985; Petrofsky and Lind, 1975; Sagiv et al., 1988; VanLoan et al., 1989). As an example, Figure 12.24 depicts the cardiovascular responses of young and old men to sustained handgrip and leg extension exercise over a range of submaximal static workloads (VanLoan et al., 1989). Note that cardiac output and SV values are lower than normally reported, because of the measurement technique. However, the relative differences between the responses of the young and the older participants show that cardiac output, SV, and HR were lower for the older men than the younger men at each intensity. In contrast, BP responses were higher for the older men

at each intensity. As with dynamic aerobic exercise, the differences in the cardiovascular responses between the two age groups are probably due to an age-related increase in resistance due to a loss of elasticity in the vasculature and a decreased ability of the myocardium to stretch and contract forcibly (VanLoan et al., 1989). The rate-pressure product was higher for the younger participants than for the older participants at 30%, 45%, and 60% MVC. The small difference in rate-pressure product reflected a higher HR in younger participants at each intensity of contraction, which was not completely offset by a lower SBP in the younger participants.

SELF-ASSESSMENT EXERCISE

i. Describe the Short-Term, Light to Moderate Submaximal Aerobic Exercise
ii. Describe the Long-Term, Moderate to Heavy Submaximal Aerobic Exercise
iii. Describe the Incremental Aerobic Exercise to Maximum

4.0 CONCLUSION

Having read this course and successfully completed the assessment and self-assessment test, it is assumed that you have attained understanding of the introductory knowledge on the measurement of cardiovascular variables & responses to aerobic exercise.

5.0 SUMMARY

This Unit has successfully summarized the measurement of cardiovascular variables & responses to aerobic exercise.

6.0 TUTOR-MARKED ASSIGNMENT

1. Describe the Upper-Body versus Lower-Body Aerobic Exercise
2. Describe Intensity of Muscle Contraction
3. Describe Blood Flow During Static Contractions

7.0 REFERENCES/FURTHER READING

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MODULE 4 SKELETAL TISSUE AND MANAGEMENT OF BONE HEALTH

INTRODUCTION

The skeletal system includes the bones and cartilage that provide the framework for the muscles and organs of the body. The skeletal system adapts to exercise training in much the same way as other body systems. A healthy skeleton is important for preventing sports-related injuries and major health problems, including osteoporosis.

Unit 1 Neuro-Muscular and Skeletal System
Unit 2 Factors Influencing Bone Health and Exercise Response
Unit 3 Application of The Training Principles & Skeletal Adaptation To training.

UNIT 1 NEURO-MUSCULAR AND SKELETAL SYSTEM

CONTENTS

1.0 Introduction
2.0 Intended Learning Outcomes (ILOs)
3.0 Main Content
  3.1 The Skeletal Tissue
  3.2 Measurement of Bone Health
4.0 Conclusion
5.0 Summary
6.0 Tutor-Marked Assignment
7.0 References/Further Reading

1.0 INTRODUCTION

This unit will explain the neuro-muscular and skeletal system.

2.0 INTENDED LEARNING OUTCOMES (ILOS)

By the end of this Unit, you will be able to;

- Describe the skeletal tissue
- Describe measurement of bone health

3.0 MAIN CONTENT

3.1 Skeletal Tissue

Bone tissue, also called osseous tissue, is a dynamic, living tissue that is constantly undergoing change. In fact, adults recycle 5–7% of their bone mass every week (Marieb, 2007).

Bone remodeling (bone turnover) refers to the continual process of bone breakdown (resorption) and the formation (deposition) of a new bone. Bone remodeling has important roles in regulating blood calcium levels and replacing an old bone with a new bone to ensure the integrity of the skeletal system. The mass and shape of bones depend largely on the stress placed on them. The more bones are stressed (by mechanical loading during physical activity), the more they increase in volume and mass, specifically at the site of mechanical loading. The concept that a bone adapts to changes in mechanical loading is described by **Wolff 's Law**.

Functions
The skeletal system provides a number of important structural and physiological functions. Structurally, the skeletal system provides rigid support and protection for vital organs and allows for locomotion. Physiologically, skeletal tissue provides a site for blood cell formation (hematopoiesis in bone marrow), plays a role in the immune function (providing the site for white blood cell formation), and serves as a dynamic storehouse for calcium and phosphate, which are essential for nerve conduction, heart and muscle contraction, blood clotting, and energy formation (Bailey and McColloch, 1990; Marieb, 2007). The ability of a bone to perform its structural functions relates directly to its role in storing calcium. Because calcium is essential for many processes in the body, bone is broken down (resorbed) as needed to maintain blood calcium levels. The body sacrifices bone mineral (calcium) when it is needed to maintain blood calcium levels.

Regulation of Blood Calcium
The skeletal system (bone), the digestive system (stomach and intestines), and the urinary system (kidneys) operate together to regulate and maintain blood calcium levels. Adequate ingestion and absorption of calcium are required through the digestive system to provide the necessary calcium to be deposited in the bone. In turn, because of the importance of calcium in so many vital processes of the body, bone mass is broken down to maintain blood calcium within normal limits (9–11 mg·dL−1). The kidneys regulate blood calcium by filtering and reabsorbing it. The primary hormones involved in regulating blood calcium levels and bone remodeling are parathyroid hormone (PTH), calcitonin, and vitamin D (calcitrol).

Levels of Organization
Understanding the structure and physiology of the skeletal system helps one understand how the skeletal system responds to exercise and training.

Bones as Organs
The human body contains over 200 different bones joined together at articulations known as joints. Joints enable movement when muscles exert force on the bones. The skeleton is typically divided into two categories: the axial (or central) skeleton that includes the bones of the skull, vertebral column, and rib cage and the appendicular (or peripheral) skeleton that includes the bones of the hips, shoulders, and extremities. Bones have several different shapes—long, short, flat, and irregular—and each shape is specific to its function. Furthermore, according to Wolff's Law, a bone's shape reflects its response to the stress placed on it.

Bone Tissue
The two types of bone tissue are cortical bone and trabecular bone. Cortical bone, also called compact, dense, or lamellar bone, is densely packed. It makes up around 80% of the skeleton. Trabecular bone, also called spongy or cancellous bone, is more porous and is surrounded by cortical bone. Individual bones are composed of both types of bone tissue in varying relative proportions. In general, bones of the axial skeleton have a much greater percentage of trabecular bone, whereas bones of the appendicular skeleton have a greater percentage of cortical bone. A typical long bone. The shaft is composed primarily of cortical bone, and the epiphyses have a greater percentage of trabecular bone. Cortical bone is composed of osteons, which are the functional units of bone (Haversian system). Osteons are organized into concentric layers of matrix called lamellae, which are surrounded by widely dispersed cells. The matrix, the intercellular space, is made up of organic and inorganic substances.

Trabecular bone is composed of branching projections or struts, called trabeculae, which form a latticelike network of interconnecting spaces. Its appearance gives rise to another of its names, spongy bone. Trabecular bone has the same cells and matrix elements as cortical bone, but with more porosity. About 80–90% of the volume of cortical bone is calcified, while only 15–25% of trabecular bone is calcified (Baron, 1993). The remaining volume is occupied by bone marrow, blood vessels, and connective tissue. Cortical bone is best suited for structural support and protection, and trabecular bone is best suited for shock absorption and physiological functions.

Because of its large surface area, trabecular bone can remodel more rapidly than cortical bone. The greatest age-related loss of bone mineral density (BMD) also occurs in trabecular bone. Therefore, most

osteoporotic fractures occur in areas composed predominantly of trabecular bone (wrist, hip, and spine).

Bone Development

Bone development involves three processes: bone growth, bone modeling, and bone remodeling. Each process occurs at different times throughout an individual's life.

Growth

Bone growth refers to size increase caused by an increasing number of bone cells (Frost, 1991a). There are two types of bone growth. Appositional growth is an increase in thickness or mass. Longitudinal growth occurs at the epiphyseal plate until a person reaches adult stature. This is an area of interest because of concerns that excessive exercise might stunt a child's growth. The longitudinal growth of bones results from the growth of a cartilage, which is later replaced by a bone. While growing, the bone is also remodeling itself—that is, changing its shape and thickness. Bone growth and remodeling are distinct but closely related processes.

In general, growth refers to the longitudinal growth of bone, and remodeling involves the balance between bone resorption and bone formation. If bone formation exceeds resorption, this process would also represent growth.

Bone Modeling

Bone modeling is the process of altering the shape of a bone and adjusting the bone strength through bone resorption and bone formation (Frost, 1991a; Khan et al., 2001c). Micro-modeling involves the microscopic level of cell organization that occurs during formation; it determines what kind of tissue will be formed (Frost, 1991a).

Macro-modeling controls if, when, and where new tissue will form or old tissue will be removed (Frost, 1991a). This process ensures that the bone's shape matches its role (Frost, 1988, 1991a,b; Khan et al., 2001b; Lanyon, 1989; Marcus, 1987). Modeling is largely responsible for bone growth during the years in which the skeleton is growing.

Remodeling

Bone remodeling involves a continual process of bone turnover, maintenance, replacement, and repair (Frost, 1991a). It reflects the balance between the coupled processes of bone resorption and bone formation. This ongoing process occurs because of the coupled actions of bone cells, with osteoclasts responsible for bone resorption and osteoblasts responsible for bone formation. Remodeling occurs in response to stress on the skeleton throughout the adult years. Physical

activity influences bone strength and mass through remodeling, which is accomplished largely because of the activity of bone cells.

Bone Cells

The three types of bone cells are osteoclasts, osteoblasts, and osteocytes. These cells are the living part of a bone. Although the cells represent a small fraction—less than 2% (Teitelbaum, 1993)—of the total composition of bone, they are responsible for its remodeling.

Osteoclasts

These are large, multinucleated bone cells that cause the resorption of bone tissue. Osteoclasts secrete enzymes that disintegrate the bone matrix. As the bone is degraded, the mineral salts (primarily calcium and phosphate) dissolve and move into the bloodstream. **Osteoblasts** are bone cells that cause the deposition of bone tissue. Also called bone-forming cells, osteoblasts produce an organic bone matrix that becomes calcified and hardens as minerals are deposited in it. Hardening of the bone matrix is known as ossification. **Osteocytes,** which are mature osteoblasts surrounded by calcified bone, help regulate the process of bone remodeling. Osteocytes appear to initiate the process of calcification. The actions of osteoclasts and osteoblasts are coupled; they work together to remodel bone. Osteoclasts must first cause bone resorption before the osteoblasts can form a new bone (Marcus, 1987; Parfitt, 1987; Teitelbaum, 1993). From the resting phase, the osteoclasts are stimulated and cause the resorption of bone, forming a cavity. Osteoblasts then appear and deposit the bone matrix where the cavity exists. The matrix is called osteoid until it is calcified.

Calcification of the new bone occurs as calcium and phosphate minerals are deposited in the osteoid. The bone then returns to the resting or quiescent phase. Bone remodeling may result in a greater bone mass, the same bone mass, or a reduction in bone mass. Through young adulthood, typically, more bone is formed than is resorbed, increasing the bone mass. This increased mass strengthens the bone and accounts for the increase in BMD that commonly occurs during this period of life. When bone remodeling is in equilibrium, the amount of bone resorbed equals the amount of bone formed; thus, BMD remains relatively constant. In older adults and those with certain diseases, the amount of bone resorbed is greater than the amount of bone formed, decreasing BMD. The remodeling of bone provides for skeletal growth and involves a constant turnover of bone throughout life. Bone remodeling is a complex process regulated by hormonal and local factors (Canalis, 1990).

Hormonal Control

Bone remodeling reflects the interrelationship between the structural and the physiological functions of bone. Calcium is necessary not only to

provide structural integrity of bone but also for the proper functioning of the heart, skeletal muscles, and nervous tissue. Only about 1 g of calcium is present in the extracellular fluid of the body, compared to approximately 1150 g of calcium present in bone tissue (Bailey and McColloch, 1990; Khan et al., 2001c).

Excess calcium in the blood leads to the release of calcitonin (from the thyroid gland), which causes deposition of calcium in the bone. This deposition decreases the blood calcium level and increases BMD. Conversely, when the blood calcium level drops below normal, PTH stimulates osteoclast activity, causing calcium to be released from its storage site, the bone. This release of calcium from the bone causes the blood calcium level to increase and BMD to decrease. Vitamin D (calcitriol) is important for the absorption of calcium from the intestines. Thus, it leads to an increased blood level of calcium. Other hormones that play an important role in skeletal health are the sex steroids (estrogen and testosterone) and growth hormone. These hormones stimulate the protein formation necessary for bone growth and are responsible for the eventual closure of the epiphyseal plate, which determines the bone length and thus a person's height (Bailey and McColloch, 1990). Estrogen promotes calcium retention and acts as an inhibiting agent of PTH. The loss of the protective role of estrogen on the skeletal system after menopause or during secondary amenorrhea has important consequences for females. Decreased estrogen causes increased bone resorption. Growth hormone and insulin-like growth factor (IGF-1) also play an important role in bone formation and remodeling in children. Hormones are themselves stimulated by other factors, including physical activity and nutritional status.

3.2 Measurement of Bone Health

Bone strength is determined by the bone mass, the external geometry, and the internal microstructure (Beck and Marcus, 1999; Frost, 1997; Heaney et al., 2000). Because it is difficult to quantify external geometry and microstructure, measures of bone mass and BMD are most often used to describe the bone strength. *Bone strength* refers to a bone's ability to withstand forces that may cause fracture. Bone strength is largely influenced by bone mass. A less dense bone will break with less force (Heaney et al., 2000). Bone mineral content (BMC) refers to the absolute amount of calcium and phosphate salts and is measured in grams. The calcium and phosphate salts are responsible for the hardness of the bone matrix. BMD is defined as the relative value of bone mineral per measured bone area, expressed as grams per centimeter squared ($g \cdot cm^{-2}$) or milligrams per centimeter cubed ($mg \cdot cm^{-3}$), depending on the technology used to measure area.

Accurate measures of BMC and BMD can be obtained only in laboratory or clinical settings, primarily because of the cost of equipment and safety considerations. Nonetheless, these measurements have dramatically increased the information available to researchers, clinicians, and those in fitness professions. At present, no field tests validly predict BMD.

Dual-Energy X-ray Absorptiometry

Dual-energy X-ray absorptiometry (DXA) is the standard method of measuring BMD for research and clinical purposes (American College of Sports Medicine, 2004). DXA uses an X-ray beam to measure regional and whole-body mineral content (Figure 16.4) and provides an areal bone mineral density (aBMD) value. Areal BMD provides a two-dimensional measure of density.

It shows a computer-generated printout of whole-body BMD and various regions of the body for a 37-year-old active female. The whole-body BMD is compared with the standard references. The dark area represents an average range across the age span of 20–100 years. Notice that the individual in the example is above average for whole-body BMD as represented by the asterisk (located at the intersection of age 37 yr and BMD of 0.197 g·cm−2).

Regional BMDs along with comparisons with young adult normative data. Notice that each region of the body has a unique BMD value because of the varying composition of bones. For example, the legs have a BMD of 1.229 g·cm−2, whereas the pelvis has a BMD of 1.217 g·cm−2. These values correspond to 106% and 110% of young adult values, respectively. In addition to these total body scans, clinicians and researchers often measure BMD at specific, clinically relevant sites, such as the hip or spine, where osteoporotic fractures are more likely. When scanning just a small area (e.g., a hip or spine), a better-quality scan result. It present a hip and spine scan of an active, older woman. These site-specific scans enable researchers to investigate differences in BMD at various sites, among various individuals, and as a result of adaptation to long-term exercise training.

BMD derived from DXA is the basis of the operational definitions of osteopenia and osteoporosis. BMD is normally distributed and is often expressed in standard deviation (SD) units related to its T or Z distribution. The T distribution has a mean score of zero (0).

Osteopenia is a condition of decreased BMD defined as a T-score of −1 to −2.5. This means a BMD value greater than 1 SD below (but not more than 2.5 SD below) values for young, normal adults (25–35 yr, sex matched). **Osteoporosis** is a condition of porosity and decreased BMD defined as a T-score greater than −2.5, indicating a BMD more than 2.5

SD below values for young, normal adults (World Health Organization, 1994). *Established osteoporosis* is the term used for the condition of osteoporosis, as defined above, plus one or more fractures (Kanis et al., 1993). Figure 16.7 indicates the relative risk of fracture at the spine, hip, and forearm based on T-scores. The International Society for Clinical Densitometry (ISCD) recommends using T-scores for evaluating postmenopausal women and Z-scores for adolescents and premenopausal women. Both T- and Z-scores represent SDs from the average score, but the comparison group for Z-scores is age matched and sex matched while the T-score compares with a single sex-matched young adult value. The ISCD defines a Z-score above −2 (< 2 SDs below average) as a BMD "within the expected range for age." Z-scores of −2 or lower (> 2 SDs below average) are defined as "below the expected range for age." Z-score comparisons with age-matched peers are especially important for individuals under the age of 20 years because they are still accumulating bone (Leib et al., 2004).

In addition to measures of BMD, DXA measures have been used to estimate bone strength (see Focus on Research box). As discussed earlier, bone strength refers to the ability of a bone to resist fracture and is determined by bone mass, physical properties of bone, and bone geometry. Variables thought to reflect bone strength include directly measured variables, such as the cross-sectional area, and calculated variables, such as a bending index and a strength index (SI).

Quantitative Computed Tomography

Quantitative computed tomography (QCT) is a newer technique that provides researchers with measures of bone health in addition to BMD (Figure 16.8). QCT can determine the volumetric bone mineral density (vBMD) of trabecular and cortical bones. Volumetric BMD is a measure of three-dimensional volume. Since QCT appears more sensitive to bone changes than DXA, QCT measures will likely be used increasingly to assess bone adaptations to exercise (Khan et al., 2001b).

SELF-ASSESSMENT EXERCISES

i. Describe the Short-Term, Light to Moderate Submaximal Aerobic Exercise
ii. Describe the Long-Term, Moderate to Heavy Submaximal Aerobic Exercise
iii. Describe the Incremental Aerobic Exercise to Maximum

4.0 CONCLUSION

Having read this course and successfully completed the assessment and self-assessment test, it is assumed that you have attained understanding of the introductory knowledge on the measurement of cardiovascular variables & responses to aerobic exercise.

5.0 SUMMARY

This Unit has successfully summarized the measurement of cardiovascular variables & responses to aerobic exercise.

6.0 TUTOR-MARKED ASSIGNMENT

1. Describe the Upper-Body versus Lower-Body Aerobic Exercise
2. Describe Intensity of Muscle Contraction
3. Describe Blood Flow During Static Contractions

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UNIT 2 FACTORS INFLUENING BONE HEALTH AND EXERCISE RESPONSE

CONTENTS

1.0 Introduction
2.0 Intended Learning Outcomes (ILOs)
3.0 Main Content
    3.1 Factors influencing Bone Health
    3.2 Exercise Response
5.0 Conclusion
6.0 Summary
6.0 Tutor-Marked Assignment
7.0 References/Further Reading

1.0 INTRODUCTION

This unit will explain the neuro-muscular and skeletal system.

2.0 INTENDED LEARNING OUTCOMES (ILOS)

By the end of this Unit, you will be able to;

- List the factors influencing bone health
- Describe exercise response

3.0 MAIN CONTENT

3.1 Factors Influencing Bone Health

Bone health is largely related to peak bone mass attainment and bone loss rate. Both processes are influenced by age and sex.

Age-Related Changes in Bone
Bones change in density throughout life. It shows the characteristic pattern between bone mass and age for males and females (Ott, 1990). The first 20 years of life are characterized by active growth in bone mass. About 25% of the final adult bone is accumulated from approximately 11.5–13.5 years (around the age of menarche) for girls and 13.0–15.0 (peripuberty to postpuberty) for boys. This approximates the amount of bone lost in females in postmenopausal years (MacKelvie et al., 2002). The skeletal consolidation phase occurs in early adulthood, and peak bone mass is generally attained by 30 years. Shortly after the attainment of peak bone mass, bone mass loss begins. After a rapid-loss phase, the rate of bone loss decreases (Teitelbaum, 1993). The theoretical curves shown in Figure 16.9 assume the attainment of full genetic potential. If

environmental factors such as exercise, nutrition, and hormonal status are inadequate, full genetic potential for bone mass may not be realized, increasing fracture risk (Heaney et al., 2000). The specific effect of aging is difficult to know precisely because bone health is affected by many factors, especially physical activity level and nutritional patterns (Tucker et al., 2002; Wohl et al., 2000).

Male-Female Differences in Bone Mineral Density

BMD varies between males and females. Total BMD changes throughout the life span for both sexes. BMD increases throughout childhood and early adult life for both sexes, but the peak BMD attained is less in females than males (Ott, 1990). In addition to differences in total BMD, BMD also varies between males and females according to measurement site (Beck and Marcus, 1999). At menopause, females lose the protective influence of estrogen, and bone loss accelerates if estrogen is not pharmacologically replaced. The loss of the protective influence of estrogen explains the prevalence of osteoporotic fractures in older postmenopausal women.

Development of Peak Bone Mass

Peak bone mass is attained during the mid-30s in both sexes, although 95% of peak bone mass is achieved by age 20 (Beck and Marcus, 1999). The individual's peak bone mass developed in young adulthood is influenced by mechanical factors, nutrition, hormonal levels, and genetics (Heaney et al., 2000; Ott, 1990). Mechanical factors include physical activity and gravity. These forces are generally considered necessary stimuli for bone formation and growth (Frost, 1997). Studies done with astronauts and with individuals confined to bed rest clearly show a loss of BMD when bone is not subjected to the force of gravity.

Although additional research is needed to specify exercise prescriptions for optimal skeletal development, children and adolescents should be encouraged to engage in physical activity to promote bone health along with other positive changes and development within the body. Specifically, for bone development, children and adolescents should be encouraged to participate in high-impact activities (Grimston et al., 1993; Khan et al., 2001c; Vicente-Rodriquez, 2006; Greene and Naughton, 2006; Macdonald et al., 2007). Some have suggested that the time period of puberty is particularly important for the development of bone mass (MacKelvie et al., 2002). For girls, peak BMC velocity occurs on the average at about age 12.7 years (the average age of menarche); for boys, it occurs about 1.5 years later. Of course, maturational age varies widely among individuals. Specific exercises for bone development are generally important from approximately 9–16 years.

Adequate nutrition is necessary for developing a strong skeletal system. Dietary calcium is essential for bone health but is often deficient in young athletes. It shows the recommended Dietary Reference Intake of calcium. Note that these are Adequate Intake (AI) values, that is, recommended amounts based on observed or experimentally determined approximations from healthy individuals when a more specific Recommended Dietary Allowance has not been determined.

Unfortunately, many individuals fall well below the recommendations, particularly young women concerned about weight control. For instance, young women may eliminate dairy products from their diet because they are high in fat. But dairy products also are an excellent source of calcium. Thus, while trying to maintain weight, these athletes may be negatively affecting the attainment of peak bone mass. Exercise professionals must consider the need for dietary calcium when counseling young athletes (particularly females) about weight management (American College of Sports Medicine, 2004; Loucks, 1988). The availability of the many low fat dairy products makes it easier to include calcium in the diet.

Adequate estrogen levels are also needed to attain peak bone mass. Finally, there are genetically determined limits to the amount of BMD that an individual can attain. The only way to achieve genetic potential, however, is to pay careful attention to modifiable factors: nutritional status, hormonal status, and activity level.

3.2 Exercise Response

Physical activity increases mechanical forces on bones. This leads to physiological changes in bone cells that allow bone to be modeled and remodeled. **Mechanotransduction** is the process by which a bone responds to a mechanical force on it. Physical activity applies a mechanical force (e.g., bending or deformation) to a bone. Bending causes both *compressive* stress and *tensile* stress that alter the hydrostatic pressure in different regions of the bone tissue, causing movement of fluid in this tissue. Fluid flows through the small canals and spaces within the bone matrix (lacunocanalicular system) and around osteocytes; this flow aids in the transport of nutrients and waste. This fluid movement also exerts a shear stress that may stimulate an osteogenic response, resulting in the formation of a new bone. Physical activity causes specific changes in bone physiology within minutes. Soon after a mechanical load is placed on bone cells, they release prostacyclin; this is followed within minutes by an increase in enzymes related to metabolism. Six to twenty-four hours after activity, RNA synthesis increases. There is evidence of increased collagen and mineral deposition on the bone surface within 3–5 days after a bout of loading (Khan et al., 2001b).

SELF-ASSESSMENT EXERCISES

i. Describe the Short-Term, Light to Moderate Submaximal Aerobic Exercise
ii. Describe the Long-Term, Moderate to Heavy Submaximal Aerobic Exercise
iii. Describe the Incremental Aerobic Exercise to Maximum

4.0 CONCLUSION

Having read this course and successfully completed the assessment and self-assessment test, it is assumed that you have attained understanding of the introductory knowledge on the measurement of cardiovascular variables & responses to aerobic exercise.

5.0 SUMMARY

This Unit has successfully summarized the measurement of cardiovascular variables & responses to aerobic exercise.

6.0 TUTOR-MARKED ASSIGNMENT

1. Describe the Upper-Body versus Lower-Body Aerobic Exercise
2. Describe Intensity of Muscle Contraction
3. Describe Blood Flow During Static Contractions

7.0 REFERENCES/FURTHER READING

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UNIT 3 APPLICATION OF THE TRAINING PRINCIPLES & SKELETAL ADAPTATION TO TRAINING

CONTENTS

1.0 Introduction
2.0 Intended Learning Outcomes (ILOs)
3.0 Main Content
    3.1 Application of the Training Principles
    3.2 Skeletal Adaptations to Exercise Training
4.0 Conclusion
5.0 Summary
6.0 Tutor-Marked Assignment
7.0 References/Further Reading

1.0 INTRODUCTION

Bone's adaptation to physical activity depends on the type of loading. In other words, the response is specific to the type of activity performed. *Stress* (or load) refers to the external force applied to a bone, whereas *strain* (deformation) refers to changes in the bone tissue. This unit will explain the neuro-muscular and skeletal system.

2.0 INTENDED LEARNING OUTCOMES (ILOS)

By the end of this Unit, you will be able to;

- List the factors influencing bone health
- Describe exercise response

3.0 MAIN CONTENT

3.1 Application of the Training Principles

Adaptations in any given physiological system are dependent on the extent to which exercise stresses that system. For instances, adaptations in the cardiovascular system depend on the intensity, duration, and frequency of predominantly aerobic exercise training. Similarly, adaptations in skeletal muscle depend on the load, number of reps, rest period, number of sets, and frequency at which load-bearing exercise is performed. The adaptation to a mechanical load (physical activity) in bones depends on the strain magnitude, strain rate, distribution of load on the bone, and number of cycles (Khan et al., 2001a).

Strain magnitude is the amount of relative change in bone length under mechanical loading. *Strain rate* is the speed at which strain develops and releases. *Distribution of load* refers to how strain occurs across a section of bone. *Strain cycles* are the number of load repetitions. The *mechanostat theory*, suggests that a bone adapts to set points of *minimal effective strain* (*MES*). This theory suggests that a control system operates in which an MES is necessary to maintain a bone and that a higher MES must be surpassed to overload a bone appropriately for positive adaptations (increased BMD and strength). Above the repair MES, bone enters a state of overuse. In the pathological overuse zone, bone suffers from microdamage, and woven (unorganized) bone is added as part of the repair process, leading to increased bone mass but not bone strength (Khan et al., 2001a).

The precise type and amount of activity for enhancing and maintaining bone health are not fully known at present. However, several recommendations can be made based on the mechanostat theory and research. The overall goals of physical activity relative to skeletal health are to (a) increase peak bone mass in adolescents, (b) minimize age-related bone loss, and (c) prevent falls and fractures (American College of Sports Medicine, 2004).

Specificity

The specificity principle applies to the specific bones being stressed, the composition of the bone being stressed (cortical versus trabecular), and the type of activity being performed. Research data suggest that the type of exercise or activity performed greatly influences skeletal adaptations.

Weight-bearing exercise refers to a movement in which the body weight is supported by muscles and bones, thereby working against gravity.

Non– weight–bearing exercise, by contrast, refers to a movement in which the body is supported or suspended, thereby not working against the pull of gravity. Weight bearing or impact-loading activities, such as running, gymnastics, stair climbing, volleyball, and resistance training, are more likely to stimulate increased bone mass than non–weight-bearing activities, such as swimming and cycling (American College of Sports Medicine, 2004; Dalsky, 1993; Duncan et al., 2002; Grimston et al., 1993; Proctor et al., 2002). The best activity is chosen based on the individual's health and preference. When considering exercise for individuals with low BMC, the risk of falling and causing a fracture is a major concern. Activities with a high risk of falls or collisions should not be recommended for certain populations, such as older adults (American College of Sports Medicine, 2004; Dalsky, 1993). Because dynamic resistance training is associated with positive adaptations in skeletal tissue as well as muscular fitness, exercise physiologists generally recommend

this type of exercise for maintaining both muscular and skeletal health (American College of Sports Medicine, 2004; Layne and Nelson, 1999). Loading seems to have a localized effect (*Wolff 's Law*); thus, specific sites can be isolated for impact. Conversely, a general dynamic resistance program that works all the major muscles of the body should benefit the total skeleton. Note that bone tissue does not appear to respond to static resistance exercise (Turner and Robling, 2003).

The Check Your Comprehension box provides you the opportunity to apply the information presented in this section.

Overload

As mentioned earlier, weight-bearing exercises result in positive skeletal adaptations. The threshold for a stimulus that initiates new bone formation is termed the MES for remodeling (Frost, 1997). A load or force that exceeds this threshold and is repeated a sufficient number of times is thought to cause osteoblasts to secrete osteoid and lead to the formation of a new bone. The MES for bone modeling, and thus the impact load necessary to induce positive skeletal adaptations in humans, is not precisely known, but the stimulus must include forces considerably greater than those of habitual activity. There is strong evidence that weightbearing, impact-loading exercises can lead to an increase in BMD in children and adolescents and also decrease the age-related loss of BMD in adulthood (American College of Sports Medicine, 2004; Borer, 2005; Ernst, 1998).

Impact loads, and thus the strain applied to bones, can be manipulated by increasing repetitions or by increasing the strain magnitude as measured by ground reaction force or joint force. For example, running loads the bones by high repetition, whereas rope jumping overloads the bones primarily by intensity (strain magnitude). For adaptations in skeletal tissue, intensity is apparently more important than repetition (Beck and Marcus, 1999). Until the amount of exercise needed to impose an overload is known, the rate of adaptation or the ideal progression necessary to induce additional gains in bone density cannot be determined. However, any type of exercise overload (intensity, duration, or frequency) must begin at a level the individual can safely tolerate and progress gradually. Skeletal adaptations are unique in terms of the slow turnover rate of a bone. Because it takes about 3–4 months for one remodeling cycle to complete the sequence of bone resorption, formation, and mineralization, a minimum of 6–8 months of exercise training are typically required to detect a measurable change in bone mass in humans, using current technology (American College of Sports Medicine, 2004).

Rest/Recovery/Adaptation
To date, little research has been done with humans to determine the optimal amount of rest and recovery for positive bone adaptations. Some researchers have used information from animal studies to develop an osteogenic index (OI) (see Focus on Application box) to guide exercise prescription for bone adaptations, but the utility of such a tool has not yet been proven. It is known that inadequate rest and recovery along with excessive repetitive loads can lead to stress fractures (discussed later in this chapter).

Individualization
The individual response principle applies to the skeletal system as well as other body systems; that is, different people respond to the same exercise stress differently depending on their genetic makeup, hormonal and nutritional status, and so on. Individuals with low BMD have the greatest potential for benefit.

Additionally, exercise interventions' goals vary for individuals across the lifespan. During childhood, the primary goal is to improve bone acquisition and attain the highest peak bone mass genetically possible. High-impact activities, such as jumping and hopping, should be incorporated into activity starting in the prepubertal years. The goal through early adulthood is to build bone, and through middle age to maintain bone. This requires weight- bearing activity with a force of impact greater than 2.5 times body weight. For older adults, the goal is to reduce bone loss and prevent falls. This means emphasizing those activities that challenge the postural system and use resistance for loading muscles and bones. Dynamic resistance programs should promote balance and upper and lower body muscle strength to reduce the risk of falling and possible resulting bone fractures. Osteoporotic individuals should not engage in jumping activities. Although walking in itself is not a strong bone stimulus (see Focus on Application box), a lifetime of walking may beneficially reduce bone loss (Beck and Snow, 2003).

Retrogression/Plateau/Reversibility
The reversibility principle suggests that if you cease exercising for a time, you lose the benefits of exercising. Studies of immobilized patients (Donaldson et al., 1970; Vogel and Whittle, 1976) and discontinued training (Dalsky et al., 1988; Iwamoto et al., 2001; Nordström et al., 2005; Winters and Snow, 2000) indicate that this principle also applies to bones.

Maintenance
The increased BMD resulting from exercise training appears to be reduced with the cessation of training. The rate of bone loss is not known, however, nor is the level of activity needed to maintain BMD or the threshold at which bone loss occurs. Intense exercise training during the

pubertal years and early adulthood may lead to greater attainment of peak bone mass, which may protect against fractures later in life because more bone mass can be lost before the bone is weakened to the point of fracture (Heaney et al., 2000; Karlsson, 2004). There is evidence that increases in BMC in the femoral neck gained during 7 months of high-impact training in prepubertal children were maintained during a 7-month detraining period (Fuchs and Snow, 2002). Clearly, activity is needed to maintain BMD, but additional research is necessary to determine the level of activity in various age groups for maintaining improvements in BMD that resulted from exercise training.

Warm-Up and Cooldown

The effect of warm-up and cooldown on bone density is not known. However, warm-up and stretching are important for ligaments and tendons, which are part of the skeletal system. In summary, the optimal exercise prescription for skeletal health is not currently known. However, this should not be used as an excuse not to exercise. Some weight-bearing or impact-loading exercise is clearly better than none. Most individuals should undertake exercise programs using weight-bearing activity and dynamic resistance exercise.

3.2 Skeletal Adaptations to Exercise Training

The adaptation of the skeletal system to exercise training. The adaptation of bone to exercise depends largely on the amount of activity and may be represented as a continuum. Measurable skeletal adaptation also depends on the type of bone being measured (trabecular or cortical) as well as the type of activity employed.

One approach to studying the effects of increased physical activity on bone density is to compare the dominant limb to the monodominant limb in sports such as tennis and baseball. These studies report that the dominant arm has greater BMD or mass than the monodominant arm (Huddleston et al., 1980; Jones et al., 1977; Kontulainen et al., 2002). This seems true for both females and males and across a wide age span. Furthermore, the difference in BMD between the dominant arm and the monodominant arm appears related to the age at which participants started playing the sport. Kontulainen et al. (2002) have reported that BMD measures of the humerus of the dominant arm are approximately 17% greater than those in the monodominant arm in racquet sport players who began playing before menarche, compared to a 9% difference between dominant and monodominant limbs in players who began playing after menarche. The control group of nonathletic individuals evidenced a 3% difference in BMD between the dominant arm and the monodominant arm (Kontulainen et al., 2002). Another approach to studying skeletal adaptations to exercise training has been to compare different athletic

groups with one another and with control groups. These studies collectively suggest that individuals involved in athletics or participating in vigorous fitness training have greater BMD than sedentary controls. Furthermore, individuals involved in weight-bearing or impact-loading sports have higher BMD than those involved in non– weight-bearing activities (American College of Sports Medicine, 2004; Duncan et al., 2002; Proctor et al., 2002; Riser et al., 1990).

Training studies have also been conducted of sedentary individuals beginning an exercise program. BMD measurements were compared before and after the exercise training. A review of 21 longitudinal studies in which participants were randomly assigned to exercise treatment or control groups strongly suggests that regular physical exercise can delay the physiological decrease in BMD that occurs with aging and reduce the risk of osteoporosis. Weight-bearing exercises, including weight lifting, jumping, and running, were associated with the greatest improvements in bone mass (Ernst, 1998).

Skeletal adaptation to exercise depends on the age of the participant. Vigorous exercise helps increase bone mass and strength in children and is thus important for the attainment of peak bone mass. Furthermore, bone mass tracks from childhood to adulthood, as shown by the following studies. One study (Barnekow-Bergkvist et al., 2006) tested female students at age 16.1 years and 20 years later. Active girls had higher BMD than inactive girls as adolescents. Those who continued to be active in weight-bearing activity had significantly higher BMD (5–19%) in adulthood than those who ceased participation or who had never been active. Membership in a sports club and site-specific physical performance in adolescence were significantly associated with higher adult BMD. Another study (Delvaux et al., 2001) tested males at ages 13 and 40 years. Static arm strength, running speed, and upper body muscular endurance as an adolescent contributed significantly to the prediction of adult bone mass. Additionally, no consistent evidence suggests that exercise training negatively affects either skeletal maturation (measured by ossification) or bone length in growing children. Although isolated studies have shown both retarded and accelerated growth in stature in young athletes, the consensus is that youngsters involved in exercise training grow at the same rate and to the same extent as their sedentary counterparts (Baxter-Jones and Maffulli, 2002; Caine, 1990; Malina, 1988; Plowman et al., 1991; Sprynarova, 1987).

A study by Welsh and Rutherford (1996) suggests that elderly men and women respond to exercise in a similar manner. High-impact aerobics, performed 2–3 $d \cdot wk^{-1}$, resulted in an increase in whole-body BMD and in hip and spine BMD. The increase in BMD was similar for the males and females who participated in the 12-month study. Overall, studies

suggest that weight-bearing and resistance exercise training play an important role in maximizing bone mass during childhood and adolescence, maintaining bone mass through adulthood, attenuating bone loss with aging, and reducing falls and fractures in the elderly (American College of Sports Medicine, 2004). However, if some is good, more is not necessarily better when it comes to exercise training and bone health. Excessive physical activity can exceed the adaptive ability of bone, resulting in overuse injuries.

Skeletal Adaptations to Detraining

Research clearly shows that a cessation of weight-bearing exercise is detrimental to the skeleton because it results in a loss of BMD. This effect has been clearly shown in astronauts and in patients confined to bed rest or immobilized in a cast. Studies have consistently indicated that weight-bearing bones are affected more and that trabecular bone (measured in the spine) is lost at a greater rate than cortical bone (Donaldson et al., 1970; Frost, 1988; Vogel and Whittle, 1976).

Research also suggests that discontinuing weight bearing exercise results in a loss of the positive adaptation that occurs with training. Detraining is associated with a reversal of the positive effects of exercise on the bones in young adult males 3 years after discontinuing intense hockey training (Nordström et al., 2005), in premenopausal women after 6 months of detraining following a year of impact and resistance training (Winters and Snow, 2000), and in postmenopausal women with osteoporosis after 1 year of detraining following a year of walking and gymnastic exercises (Iwamoto et al., 2001).

In a classic study that investigated changes in BMC with training and subsequent detraining, Dalsky et al. (1988) reported that 22 months of weight-bearing exercise caused a significant increase (6.2%) in lumbar BMC. When subjects discontinued exercise training (or trained < 3 d·wk−1), BMC returned to baseline values. After 1 year of detraining, BMC was only 1.1% above baseline values.

Collective data strongly suggest that the increased bone mineral resulting from exercise is lost if exercise is not continued; bones respond to activity and inactivity.

SELF-ASSESSMENT EXERCISEs

i. Describe the Short-Term, Light to Moderate Submaximal Aerobic Exercise
ii. Describe the Long-Term, Moderate to Heavy Submaximal Aerobic Exercise
iii. Describe the Incremental Aerobic Exercise to Maximum

4.0 CONCLUSION

Having read this course and successfully completed the assessment and self-assessment test, it is assumed that you have attained understanding of the introductory knowledge on the measurement of cardiovascular variables & responses to aerobic exercise.

5.0 SUMMARY

This Unit has successfully summarized the measurement of cardiovascular variables & responses to aerobic exercise.

6.0 TUTOR-MARKED ASSIGNMENT

1. Describe the Upper-Body versus Lower-Body Aerobic Exercise
2. Describe Intensity of Muscle Contraction
3. Describe Blood Flow During Static Contractions

7.0 REFERENCES/FURTHER READING

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