Indira Gandhi National Open University
School of Social Sciences

v

\

# BANE-141

# PHYSIOLOGICAL ANTHROPOLOGY

**BANE-141**

# PHYSIOLOGICAL ANTHROPOLOGY

**School of Social Sciences**
**Indira Gandhi National Open University**

## EXPERT COMMITTEE


Prof. A. K. Kapoor (Retired)
Department of Anthropology
University of Delhi, Delhi

Prof. Satwanti Kapoor (Retired)
Department of Anthropology
University of Delhi, Delhi

Prof. Gautam, K Kshatriya(Retired)
Department of Anthropology
University of Delhi, Delhi

Prof. Rashmi Sinha
Faculty of Anthropology
School of Social Sciences
IGNOU

Dr. Rukshana Zaman
Faculty of Anthropology
School of Social Sciences
IGNOU

Dr. P. Venkatramana
Faculty of Anthropology
School of Social Sciences
IGNOU

Dr Mitoo Das
Faculty of Anthropology
School of Social Sciences
IGNOU

Dr. K. Anil Kumar
Faculty of Anthropology
School of Social Sciences
IGNOU


## COURSE PREPARATION TEAM

| Block | Unit Writers |
|---|---|
| **Block 1: Fundamentals of Work Physiology** | |
| Unit 1 Energy Intake, Metabolism and Homeostatis | Dr. Deepali Verma, Research Consultant, Freelance |
| Unit 2 Exercise Physiology | Dr. B S Yadav, MD, Associate Professor, Department of Physiology, Institute of Medical Sciences, Banaras Hindu University, Varanasi. |
| Unit 3 Haemodynamics | Dr. Deepali Verma, Research Consultant, Freelance |
| **Block 2 Cardio-vascular and Respiratory Endurance** | |
| Unit 4 Physical Working Capacity and Physical Fitness | Dr. R R M Singh, Department of Physical Education and Sports, Pondicherry University, Puducherry |
| Unit 5 Chronic Physiological Adaptations to Exercise Training | Dr. R R M Singh, Department of Physical Education and Sports, Pondicherry University, Puducherry |
| Unit 6 Exercises and Age | Dr.Shumayala, Regional Manager, Research Mamta, HIMS, Delhi |
| Unit 7 Anthropometry and CVD and Respiratory Functions | Dr. Heemanshu Aurora, Public Health Researcher, Foundation of Healthcare Technologies Society, Delhi |
| Unit 8 Gender and Ethnicity | Dr. N.K. Mungreiphy, Assistant Professor, Amity Institute of Anthropology, Amity University, Noida |
| **BLOCK 3: Factors Affecting Physical Performance** | |
| Unit 9 Principles of Physical Conditioning Techniques | Dr Suman Dua, Guest Faculty, Department of Anthropology, University of Delhi, Delhi. |
| Unit10 Effect of Diverse Conditions | Dr. Sandip Sinha, Assistant Professor, Department of Human Physiology with Community Health, Vidyasagar University, Paschim Medinipur |
| Unit 11 Physique, Nutrition and Performance | Dr Meenal Dhall, Department of Anthropology, University of Delhi, Delhi. |
| Practical Manual | Prof. Rashmi Sinha and Ms Kaneeka Joshi, PhD Scholar, Faculty of Anthropology, School of Social Sciences, Indira Gandhi National Open University, New Delhi(Portions adopted from Practical Manual of the Course Physical Anthropology (MANI002) of the Programme Master in Anthropology (MAAN). |


**Course Coordinator:** Professor Rashmi Sinha, Discipline of Anthropology, School of Social Sciences, Indira Gandhi National Open University, New Delhi.

**Editor and Language Editor:** Professor Rashmi Sinha, Faculty of Anthropology, School of Social Sciences, Indira Gandhi National Open University, New Delhi.

**Academic Consultant:** Dr. Smarika Awasthi Sharma, Dr. Pankaj Upadhyay, Faculty of Anthropology, School of Social Sciences, Indira Gandhi National Open University, New Delhi

## PRINT PRODUCTION

Mr. Rajiv Girdher
Assistant Registrat
MPDD, IGNOU
New Delhi

**Secretarial Assistant**
Mr Suresh, Mr. Rampal Singh and Mr. Naresh Kumar

**Cover Page Design :**
Ms Kaneeka Joshi, Faculty of Anthropology, School of Social Sciences, Indira Gandhi National Open University, New Delhi

*November, 2021*

*Indira Gandhi National Open University, July 2021*

*ISBN:*



*Further Information on Indira Gandhi National Open University courses may be obtained from the University's office at MaidanGarhi, New Delhi-110068 or visit University website http://www.ignou.ac.in.*

*Printed and published on behalf of the Indira Gandhi National Open University, New Delhi by Director, School of Social Sciences.*

Laser Composed by : Tessa Media & Computers, C-206, Shaheen Bagh, New Delhi-25

ignou
THE PEOPLE'S
UNIVERSITY

# Content

ignou
THE PEOPLE'S
UNIVERSITY

# PHYSIOLOGICAL ANTHROPOLOGY

Physiological anthropology mainly focuses on the capacity for the adaptation of the environment observed in the physiological function of human. The areas of study under this discipline include cultural and physical aspects associated with the living environments as parameters that affect the capacity for environmental adaptation. It will be very interesting to learn the interactions with all these parameters such as sex, age, and geographical location on the adaptability of humans with the common triggers which are based on the human functional and physical resources. The physical approach in physiological anthropology examines the environmental difference between the early human to which humans are adapted biologically and the existing environment in which we seek out the comforts of a high-advanced society.

The first block (Unit 1 through Unit 3) offers an introduction to the fundamentals of work physiology. Nowadays, environmental factors in which the human lives are more varied and work is often done in critical environmental conditions i.e., at high altitudes in extreme climates. Unit 1 explores that the energy substances i.e., fats and carbohydrates are the basis of any life activity, specifically the oxidative phosphorylation for the supply of energy. However, excessive absorption and assimilation of these components results in metabolic ailments such as diabetes, hyperlipidemia, obesity, and cancers. Unit 2 deals with Exercise physiology that analyses the various perspectives during exercise including nutrient, training, respiratory and cardiovascular system among others. Unit 3 talks about Hemodynamics that is defined as the parameters that govern the flow of blood and depends upon the relationship between blood pressure, resistance, and cardiac output.

Block 2 (Unit 4 to Unit 8) elucidates Cardiovascular and Respiratory endurance that is the level at which an individual's lungs, heart, and muscles work in combination while exercising for a long time period. This indicates how efficiently your cardiorespiratory system is functioning and how physically fit you are. Unit 4 begins with defining physical fitness and then the impact of exercise on our body system. In Unit 5, training adaptations are discussed which are the long-term physiological changes that allow the body to meet new demands. The kind of training determines the adaptations. Unit 6 will make you understand that exercise performed at any age is beneficial. Staying fit and active helps you to continue being independent. The right type of regular exercise decreases the risk of diabetes, heart diseases, etc. Unit 7 explores the pulmonary function test that is performed using the measurement of various cardiovascular risk parameters. Anthropometric measurements, spirometry, and cardiac cycle are discussed in this unit. Unit 8 discusses the potential scientific and clinical usefulness of the contextual approach as a framework for learning and interpreting the group difference socio-demographically in cardiorespiratory fitness.

Block 3 (Unit 9 through Unit11) discusses a few factors such as skills, training, body proportions, endurance, and flexibility that influence the type of sport you play, what kind of position you play, and how good you are in both of them. In Unit 9, the principle of physical conditioning techniques is to expose the body safely to the stimuli that cause structural and physiological adaptations to take place. Unit 10 is the effects of diverse conditions like alcohol, drugs, smoking, environmental stress, pollution, malnutrition, etc. on physical performance or physical conditioning techniques. Unit 11 discusses physique, body composition, nutrition vis-a-vis performance of an athlete. Regular exercise and an active lifestyle regimen, along with good eating habits are the best and advised ways to stay healthy and fit.

The Practical manual gives us a glimpse of cardiovascular functions: blood pressure, heart and pulse rate; respiratory functions: tidal volume, vital capacity, forced vital capacity and minute ventilation along with familiar hemoglobin estimation, treadmill test and step test. This manual is of immense significance as it facilitates you in understanding important parameters of your healthy life.

# BLOCK 1

# FUNDAMENTALS OF WORK PHYSIOLOGY

ignou
THE PEOPLE'S
UNIVERSITY

# UNIT 1 ENERGY INTAKE, METABOLISM AND HOMEOSTASIS*

**Contents**



**Learning Objectives**

After reading this unit, you will be able to:

- understand the physiological role of homeostasis;
- learn about the feedback mechanism involved in maintaining homeostasis;
- know about the different energy systems that synthesise ATP; and
- explore the components of energy balance.

## 1.0 INTRODUCTION

Humans are complex organisms made up of trillions of cells which are specialised to carry out particular function. These cells organise into tissues, various tissues together make up an organ and organs organise into organ systems. They work in an integrated manner to perform life's biological processes (Modell et al., 2015). While cells, tissues, and organs may perform very different functions, but they are similar in their metabolic needs (oxygen, nutrients, and removal of waste). Maintaining a constant internal environment by providing the cells with what they need to is necessary for the well-being of individual cells and of the entire body (Cooper, 2008).


* **Contributor**: Dr Deepali Verma, Research Consultant, Freelance.

Here, internal environment refers to the extra cellular fluid surrounding the cells and external environment is the immediate environment in which a living organism lives (Tortora & Anagnostakos, 2003; Modell, 2015).

## 1.1 HOMEOSTASIS

In the late nineteenth century, French Physiologist, Claude Bernard (1865) asserted, "It is the fixity of the internal environment that is the condition of free and independent life..... All the vital mechanisms, however varied they may be, have only one object, that of preserving the constant conditions of life in the internal environment" (Cannon, 1929; Widmaier et al., 2008). In other words, organisms can live a free and independent life by maintaining a stable internal environment and within a narrow range in response to internal and external stimuli. It ensures the vitality of human body and survival (Cooper, 2008; Damasio & Damasio, 2016). Though the concept was first proposed by Claude Bernard who described the processes of physiologic control as the *milieu interieur* (internal milieu), the term 'homeostasis' was coined by Walter Cannon, an American physiologist, half a century later (1929).

*Thus, homeostasis can be described as the physiological ability of human body to maintain a constant internal environment. The stability is attained by constantly adjusting the physiological processes to the conditions caused by external and internal stimuli and maintaining the variables within a range, optimal for survival (Widmaier et al., 2008; Damasio & Damasio, 2016; Torday, 2015). Homeostasis can be considered as dynamic equilibrium because continuous adjustments that are occurring in response to altering stimuli enable optimum conditions within the body (Clancy & McVicar, 1995).*

The body is said to be in homeostasis if internal environment has optimal

- temperature,
- levels of ions, water, gases and nutrients, and
- pressure. (Doherty & Foudy, 2006; Standfield & Germann, 2007)

However, it can be modified by the following factors:

- oxygen ($O_2$) and carbon dioxide ($CO_2$) concentrations
- the pH of the internal environment
- concentrations of nutrients and waste products
- concentration of salt and other electrolytes (osmoregulation)
- volume and pressure of extracellular fluid
- body temperature and
- blood glucose level

(Doherty & Foudy, 2006; Standfield & Germann, 2007)

Any alterations in body's internal environment constitute, may lead to a series of pathological changes and threaten life. Thus, these need to be regulated within the normal range despite the constant change in internal (such as pH, electrolytes) and external environment (such as diet, physical activity) for optimal functioning of the body and for sustenance of life (Tortora & Anagnostakos, 2003).

### 1.1.1 Homeostasis Regulation

Whenever an imbalance occurs, the homeostatic control mechanisms or regulatory systems become active to restore the optimum conditions. Each of the above factors may be regulated by one or more homeostatic mechanisms (Tortora & Anagnostakos, 2003; Doherty & Foudy 2006). All homeostatic control mechanisms have at least three interdependent components:

- A sensor, also known as receptor monitors that detects changes in the internal or external environment.
- The integrating centre or control centre receives information from the sensors and initiates the response to maintain homeostasis.
- An effector can be any organ or tissue that receives signal from the integrating center and acts to bring about the changes needed to maintain homeostasis (Figure 1) (Modell et al., 2015; Abozenadah et al., 2018; Biga et al., 2021a).

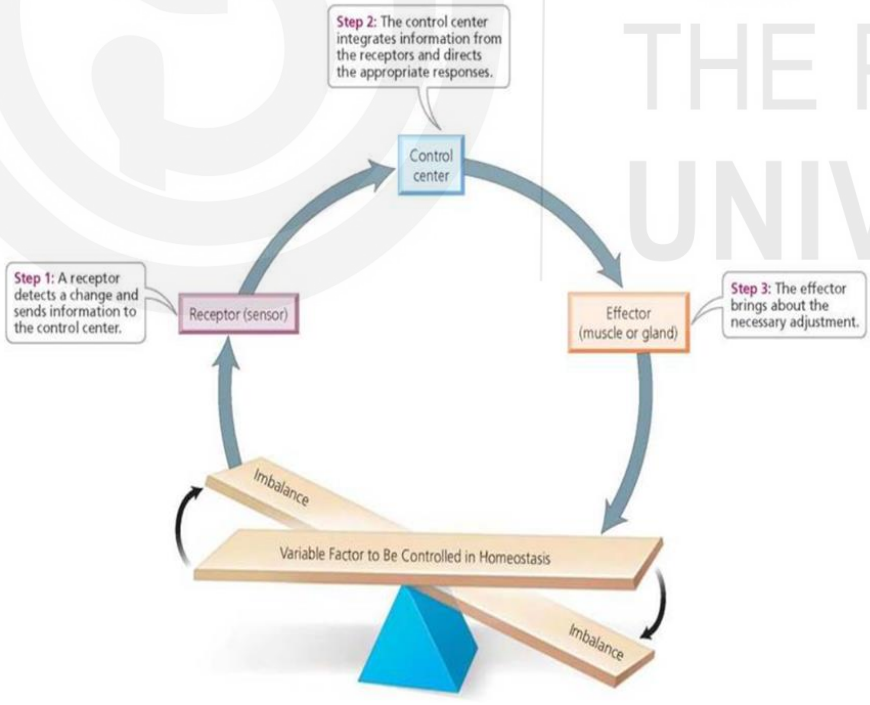



**Figure 1: Homeostasis control mechanism**

***Source:*** https://schoolbag.info/biology/humans/5.html

These regulatory systems consist of coordinated physiological processes controlled by endocrine and nervous systems (particularly hypothalamus-pituitary complex) in response to deviation from the equilibrium (Doherty &

Foudy, 2006; Widmaier et al., 2008). The nervous system (i.e. integrating centre) receives and interprets the signal from the internal and external stimuli, and thereafter directs the endocrine gland of the affected organ (i.e. effector) to release hormones so as to enable adaptive changes in prevailing physiological condition. These can have either stimulatory or inhibitory effect (Fink, 2007).

In fact, the activity of endocrine system depends on the hormone level in the blood and relies on a series of feedback loops mediated by the hypothalamus in interaction with the pituitary gland. Continuous feedback from the target gland to the nervous system ensures optimal endocrine function to maintain homeostasis (Hiller-Sturmhöfel & Bartke, 1998; Standfiel & Germann, 2007). Thus, both these systems work parallelly as well as in conjunction with each other to maintain the homeostasis condition of the body (Tortora & Anagnostakos, 2003; Doherty & Foudy, 2006; Widmaier et al., 2008).

## 1.1.2 Regulatory Feedback Loop

The feedback loops facilitating homeostasis equilibrium can be: negative and positive.

### 1.1.2.1 Negative Feedback Loop

This mechanism counteracts the change by negating the effect of stimulus and returning the variable to the normal range. The feedback pathway either shuts down the original stimulus or reduces its intensity. This is the most common feedback mechanism used by the body to maintain homeostasis (Betts et al., 2013; Biga et al., 2021a).

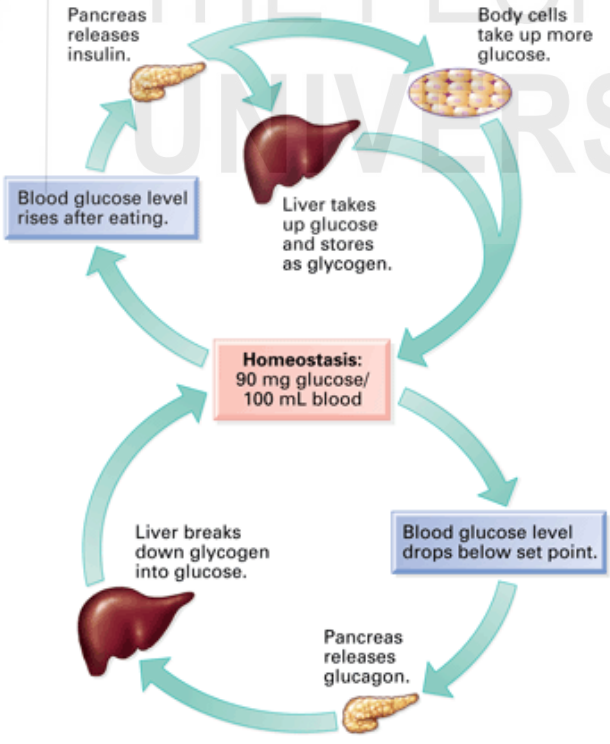



**Figure 2: Negative feedback loop - Glucose**

***Source:*** http://amandahsieh-sbi4u.blogspot.com/

For example, when the blood glucose levels rise in the blood post meal, the beta-cell of pancreas (integrating centre) are stimulated to release insulin hormone. Insulin facilitates the absorption of glucose into the cell either for further metabolism or storage, thereby decreasing the blood sugar level to normal. If blood glucose gets too low, alpha-cell of pancreas releases glucagon which catabolises the glycogen (stored in liver and muscle) into glucose and restores the blood glucose level. This metabolic process of breakdown of glycogen into glucose is known as gluconeogenesis (Figure 2) (Doherty & Foudy, 2006; Standfield & Germann, 2007).

The regulation of human body temperature is another example of negative feedback mechanism. Normally, body temperature fluctuates around 37°C but can be affected by various metabolic, physiological or environmental conditions. The thermoreceptors in the brain and skin detect the changes and send signal to the hypothalamus. In response to low body temperature, hypothalamus communicates to the effector organs to stimulate vasoconstriction and shivering, but when body temperature is high, it initiates vasodilation and sweating (Figure 3a & b) (Standfield & Germann, 2007; Kurz, 2008; Osilla et al., 2021).

a) Increased body temperature

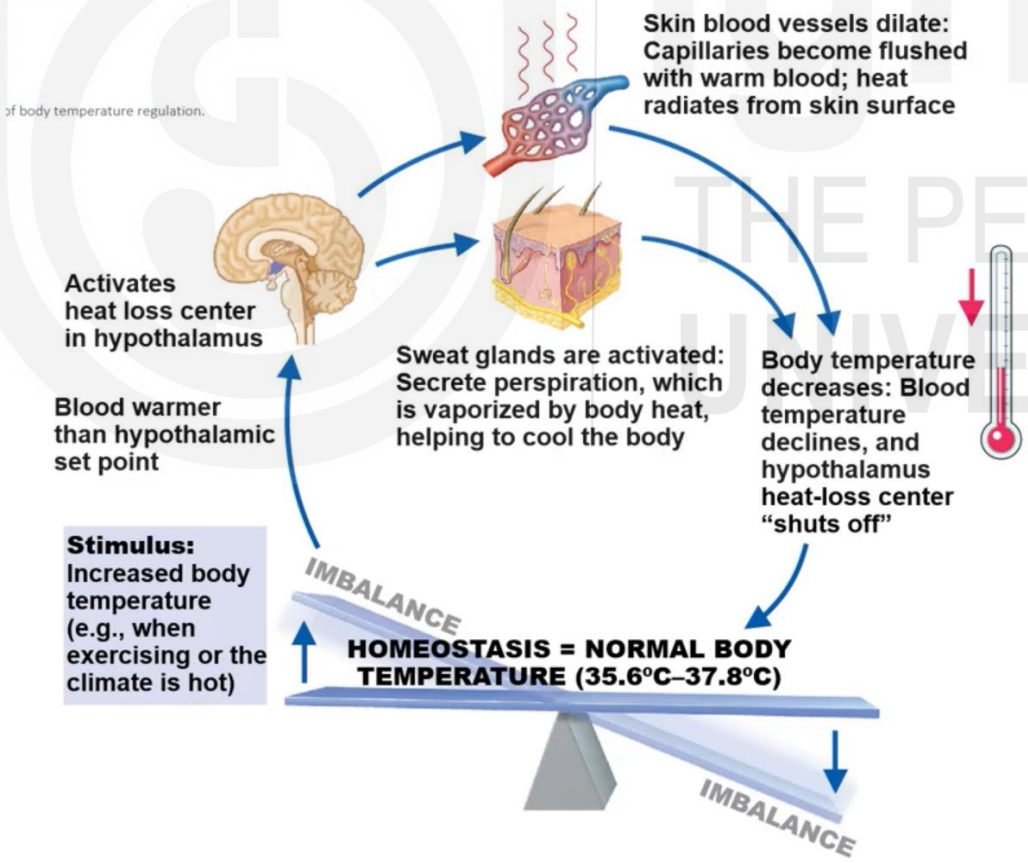



**Figure 3a**

b) Decreased body temperature

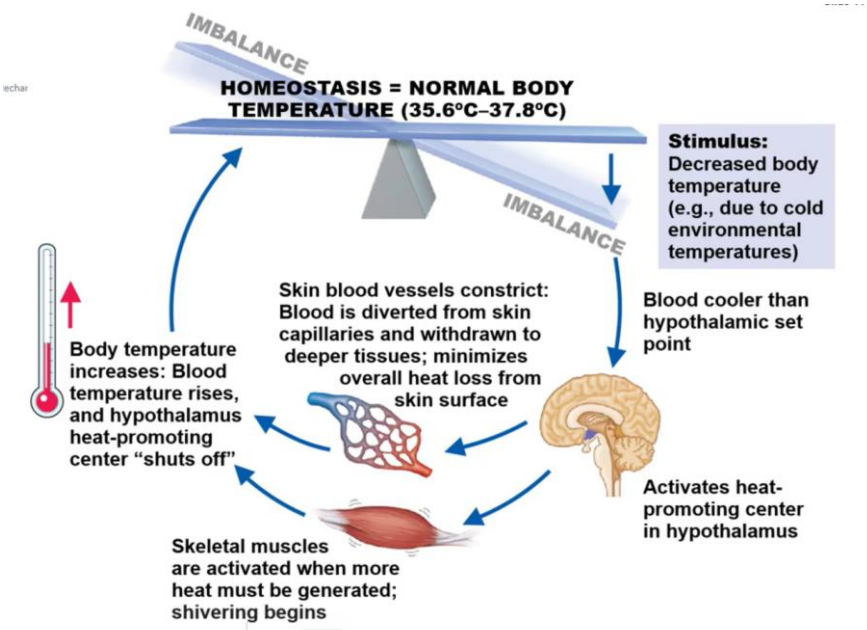



**Figure 3b**

**Figure 3a & 3b: Body temperature - Negative feedback loop**

***Source:*** https://www.youtube.com/watch?v=lnqXxghfB7Y

### 1.1.2.2 Positive Feedback Loop

It intensifies a change in the body's physiological condition in response to a stimulus (Biga, 2021a). Platelet clotting demonstrates the positive feedback. It is initiated by the vasoconstriction of the damaged blood vessel and accumulation of platelets. These activated platelets release some chemical that stimulates platelet coagulation and eventually clotting (Figure 4) (Jesty & Beltrami, 2005; Periayah et al., 2017).

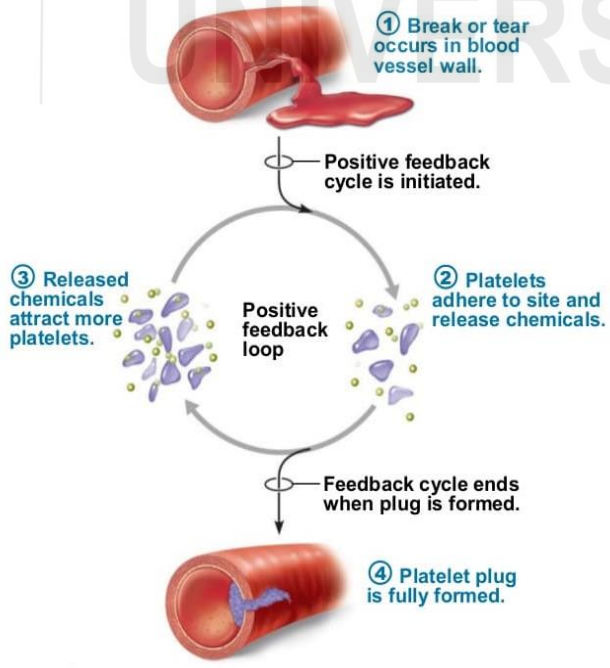



**Figure 4: Positive Feedback loop- Formation of platelet plug and clotting**

***Source:*** https://www.slideshare.net/mstish38/ch-01lecturepresentationa

During lactation, stimulation of the nipple and removal of milk triggers the release of prolactin hormone from the anterior pituitary gland and oxytocin from posterior pituitary gland. This feedback loop induces continuous production and ejection of milk during lactation (Pillay, 2021).

**Check Your Progress 1**

1) Describe the significance of homeostasis.

……

……

……

……

……

2) In the negative feedback loop:

   a) The effect or response enhances the original stimulus.
   b) The effect opposes the original stimulus or reduces its intensity.
   c) The variable deviates farther and farther from its normal range.
   d) The variable remains constant.

3) Explain what homeostasis regulatory system would your body use if you were trapped in a snowstorm in an unheated and un-insulated cabin in the hilly region?

……

……

……

……

……

## 1.2 ENERGY INTAKE AND METABOLISM

Humans need energy for their essential body functions and physical activity. They obtain this energy from the food they eat which mainly consists of carbohydrates, fat, and proteins. When consumed, digestion processes in the gastrointestinal tract breakdown these nutrients into their simplest forms. Carbohydrates degrade to simple sugars (including glucose), fat to fatty acids and proteins to amino acids. These breakdown products are then absorbed into blood stream and transported to the cells either to be used for energy or stored as glycogen (in muscle and liver) and triglycerides (in fat cells, known as adipocytes) for later use (Rubinstein-Litwak, 2003; Standfield & Germann, 2007). The energy can be utilised for:

- Cellular activities such as active transport of biomolecules, muscle contraction, etc.,

- Synthesis of building block molecules for growth, and repair (such as proteins, lipids, nucleic acids), and
- Elimination of waste products (Rubinstein-Litwak, 2003; Standfield & Germann, 2007).

These processes underlying utilisation and storage of energy to sustain living condition of an organism are called metabolism. It typically consists of a set of inter-connected biochemical reactions organised as 'metabolic pathways' and are catalysed by the enzymes. The activation and inhibition of these enzymes in response to the change in cell's environment or signals from other cells regulate the rate of metabolic reaction (Standfield & Germann, 2007; Fordham & Harmey, 2016; Abozenadah et al., 2018). During a metabolic pathway, the substrate is converted into a series of metabolic intermediates before yielding the final usable product. It means the products of one reaction act as a substrate for subsequent reactions and so on. Each step is being facilitated by a specific enzyme that act as catalysts and allow a reaction to proceed more rapidly (Cooper, 2000; Abozenadah et al., 2018; Boundless, 2021).

The metabolic reactions can be:

**Anabolic**: It synthesises complex molecules from simpler ones and is an endergonic process requiring energy; for example, building up of protein from amino acid.

**Catabolic**: It breaks down the complex molecules into simpler ones and is an exogenic process releasing energy; for example, synthesis of compounds such as protein, nucleic acid and others (Figure 5) (Standfield & Germann, 2007; Evans & Heather, 2019).

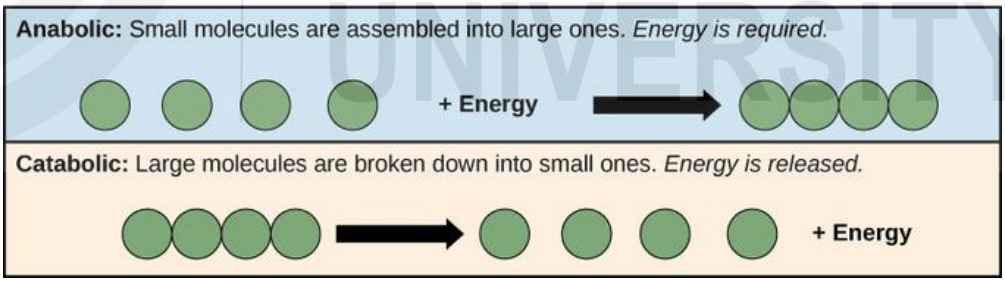



**Figure 5: Anabolic and catabolic metabolism**

***Source:*** https://courses.lumenlearning.com/bio1/chapter/reading-anabolic-and-catabolic-pathways-2/

## 1.2.1 Adenosine Triphosphate (ATP) as Energy Carrier

Energy from food cannot be used directly and must be converted into adenosine triphosphate (ATP), also known as 'energy currency of the cell'. An ATP molecule comprises of the nitrogenous base i.e., adenine, the sugar ribose; and a chain of three phosphate groups bound to ribose. The phosphate groups are attached to one another by high-energy bonds (release large amount of energy when split) known as phosphoanhydride bonds (Figure 6).

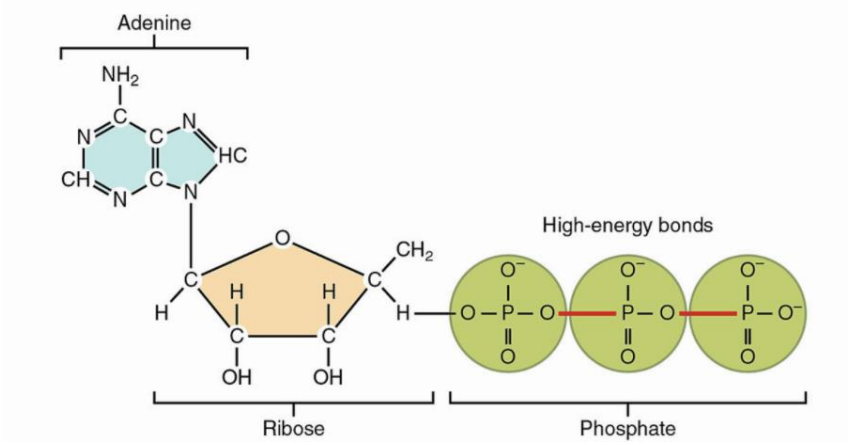



**Figure 6: Structure of Adenosine Triphosphate (ATP)**

***Source:*** https://wasfa-hd.blogspot.com/2019/04/where-is-energy-stored-in-atp-molecule.html

When a cell needs energy, the terminal phosphate bond of ATP hydrolyses (i.e. water-mediated breakdown reaction) to adenosine diphosphate (ADP) and a free phosphate molecule (Figure 7). The reaction is catalysed by an enzyme called adenosine triphosphatase (ATPase).

$$ATP + H_2O \xrightarrow{ATPase} ADP + Pi + Energy\ (7.3\ kcal/mol)$$

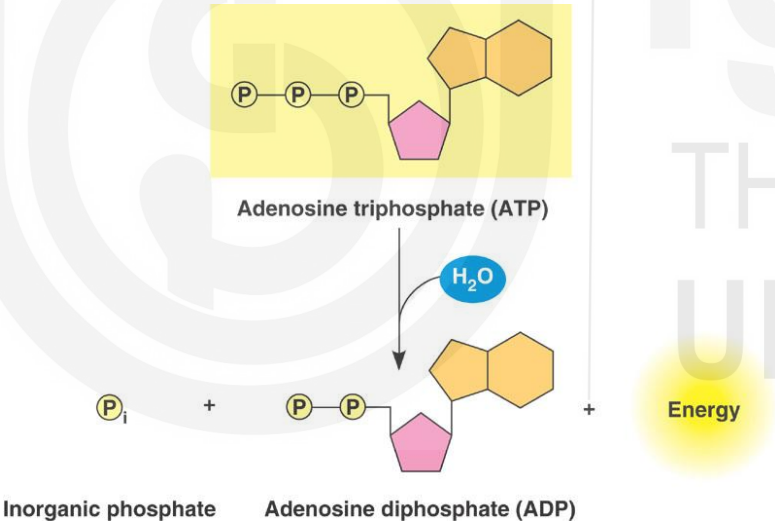



**Figure 7: The hydrolysis of ATP**

***Source:*** https://biology.stackexchange.com/questions/34413/what-is-the-difference-between-electrons-and-energy

Additional energy can be produced by the hydrolysis of the second phosphate bond, that is ADP breakdown into adenosine monophosphate (AMP) and another phosphate (PPi). But the second bond releases less energy compared to the first bond (Bergman, 1999; Karp, 2009; Dunn & Grider, 2021).

$$ADP + H_2O \rightarrow AMP + PPi + Energy$$

Simultaneously, reduced ATP level stimulates the pathway that convert ADP molecule into ATP through transfer of free phosphate i.e., phosphorylation (Dunn & Grider, 2021).

$$ADP + Pi + Energy \rightarrow ATP + H_2O$$

During this ATP-ADP cycle (Figure 8), ATP synthesis utilises the energy from nutrients (the exergonic reaction, energy releasing process) and at the same time, the energy released from the hydrolysis of ATP is used for cellular work (endergonic reactions, energy consuming process). Such a set of simultaneous reactions in which one reaction induces another reaction is known as coupling reaction (Botham & Mayes, 2012).

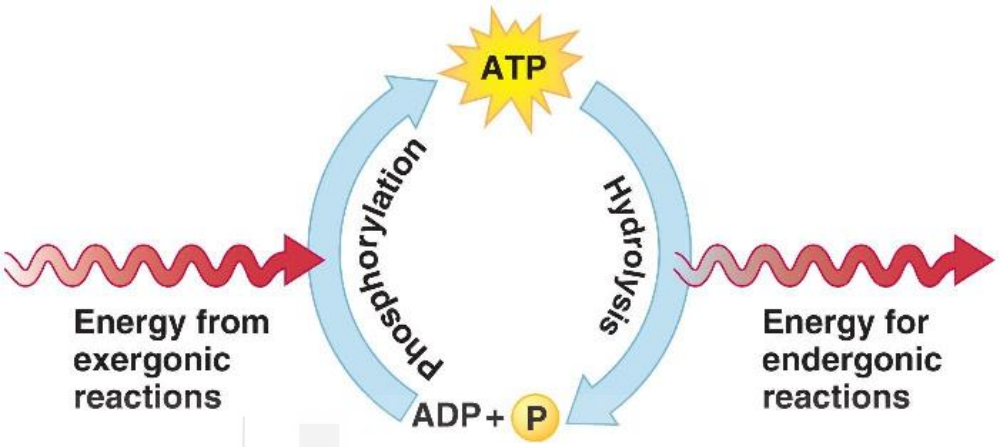



**Figure 8: ATP-ADP cycle**

***Source:***https://www.chegg.com/flashcards/test-2-biology

## Check Your Progress 2

4) What is the difference between hydrolysis and phosphorylation?

……

……

……

……

……

5) Breakdown of the complex molecules into simpler ones is a ..... and ..... process.

6) Which of the following statement describes 'high-energy bond'?

a) It is formed when ATP is hydrolysed to ADP and one phosphate group.

b) It absorbs a large amount of free energy when the phosphate group attaches to ADP during synthesis process.

c) It releases a large amount of usable energy when the phosphate group is split off during ATP hydrolysis.

d) It is usually found in the nutrient molecules which release energy when metabolised.

## 1.3 ENERGY SYSTEMS

Since human body doesn't store ATP in significant amount, it needs to be continually re-synthesised. There are three energy systems that contribute to the ATP re synthesis either aerobically (in the presence of oxygen) or anaerobically (in the absence of oxygen) that are discussed.

### 1.3.1 The Phosphagen System

When an individual performs an activity, regardless of intensity, the body initially utilises the existing store of ATP for immediate source of energy but it lasts only for about three seconds. To replenish the ATP level, creatine phosphate (or phosphocreatine, PCr) stored in muscle cell is utilised. The phosphate group of creatine phosphate is broken down by an enzyme called creatine kinase, and is transferred to ADP (produced as a result of ATP hydrolysis) to form additional ATP (Figure 9). Together, the ATP and creatine phosphate form the phosphagen system, also known as ATP-PC (Adenosine Triphosphate Phosphocreatine) system. It is an anaerobic process (without oxygen) and occurs in cell cytoplasm (Mortan, 2008; Da Poian et al., 2010).

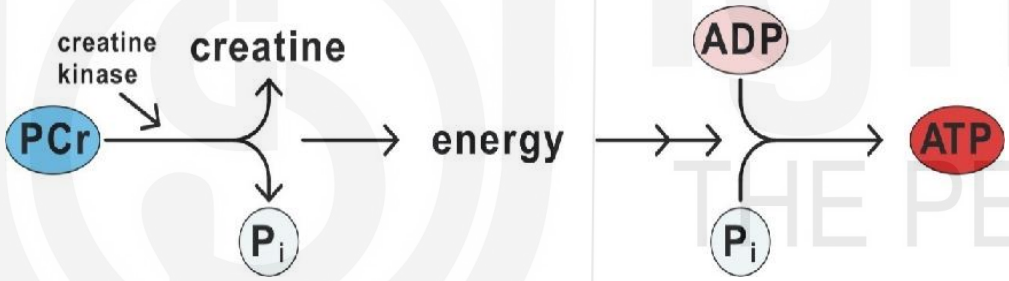



**Figure 9: The phosphagen energy system**

***Source:*** https://zona-fit.com/metabolism.php

Though this system generates ATP quickly, it supports activity for only 8-10 seconds. These include short and intensive exercises such as weight-lifting, sprinting, jump off the step, punch a bag and others. Beyond this period, ATP and creatine phosphate stores are fully exhausted and will need time to restore. If the activity continues for a longer period, the energy demand shifts to other energy systems for ATP synthesis (Freudenrich, 2008; Karp, 2009; El Bacha et al., 2010).

### 1.3.2 Glycolysis

Glycolysis (or Lactic acid system) is the sequence of biochemical reactions that metabolises glucose into pyruvate. For each molecule of glucose broken down, two ATPs are consumed but four ATPs are generated, yielding a net gain of two ATPs (Brooks et al., 2000; Da Poian, 2010). Like the ATP-PC system, glycolysis is an anaerobic process that occurs in cell cytoplasm. In the absence of oxygen, pyruvate is metabolised into lactate. However, the enzyme lactate dehydrogenase quickly oxidises it back to pyruvate which can then be used to fuel further anaerobic metabolism (Farhana & Lappin, 2021).

During high intensity efforts, lactate may build up in the muscle and is often considered to cause fatigue. But it should be noted that the change in cellular environment due to other metabolic by-products is also the contributing factor. This system plays its role for the activities that require high intensity efforts over a period longer than 10 seconds and up to around 2 minutes (Freudenrich, 2008; The Sustainable Training Method, 2018).

When adequate oxygen is available, pyruvate enters into mitochondria and converts into a metabolic intermediary molecule called acetyl coenzyme A (acetyl-CoA) for further metabolism and production of ATP (Karp, 2009). We will talk about this more in aerobic system.

### 1.3.3 Aerobic System

Aerobic system is the most complex of the three metabolic energy systems. Since it depends on oxygen, it is the slowest way to re-synthesise ATP but offers a sustainable flow of energy over longer durations. Thus, it is the primary source of energy at rest or during low/moderate intensity activities. It not only uses carbohydrates, but also proteins and fats as fuel for ATP synthesis. Carbohydrates are broken down into pyruvate (end product of glycolysis), proteins into amino acids and fats into fatty acids. Then these nutrient products via distinct metabolic pathways form acetyl-coenzyme A (acetyl-CoA) which enters the tricarboxylic acid (TCA) or Kreb's cycle in mitochondria (Figure 10) (El Bacha et al., 2010; Biga et al., 2021a).

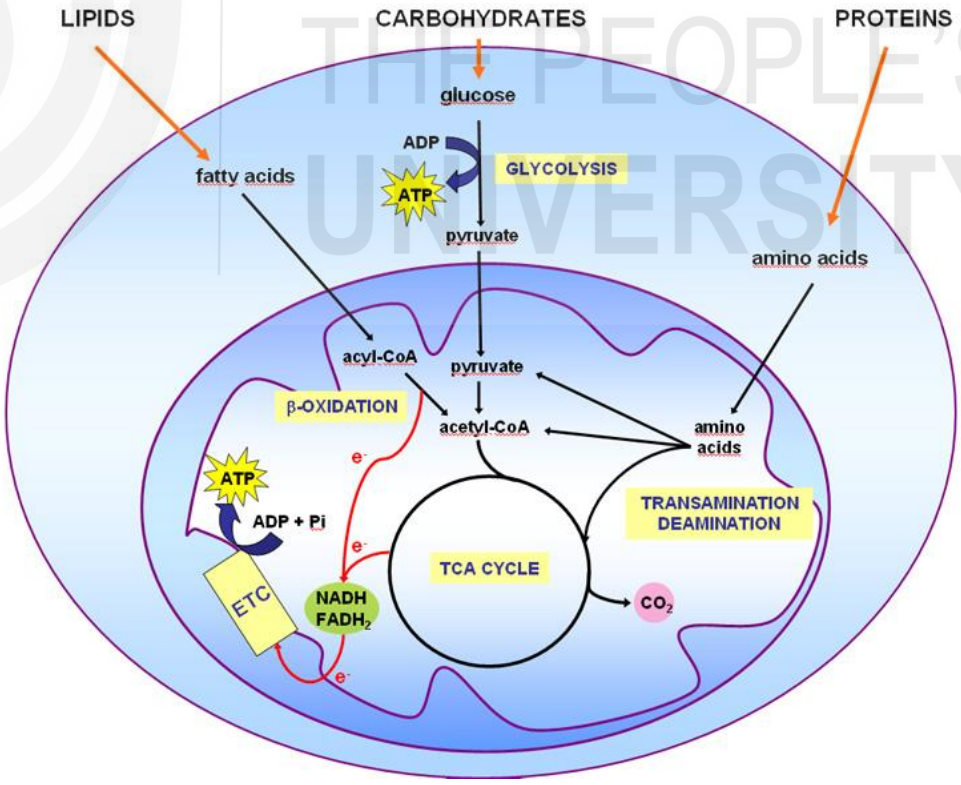



**Figure 10: The energy systems in cell cytoplasm and mitochondria**

***Source:*** https://zona-fit.com/metabolism.php

In this pathway, the acetyl-CoA is completely oxidised to $CO_2$ with concomitant production of ATPs and reduction (a chemical process involving addition of electron) of electron carrier coenzymes NAD+ and FAD to NADH and FADH2 respectively. These reduced electron carriers themselves are oxidised (a chemical reaction involving loss of electron) via the electron transport chain, resulting in production of ATP and water. This process is known as oxidative phosphorylation (Figure 11) (Da Poian et al., 2010; El Bacha et al., 2010; Biga et al. 2021b).

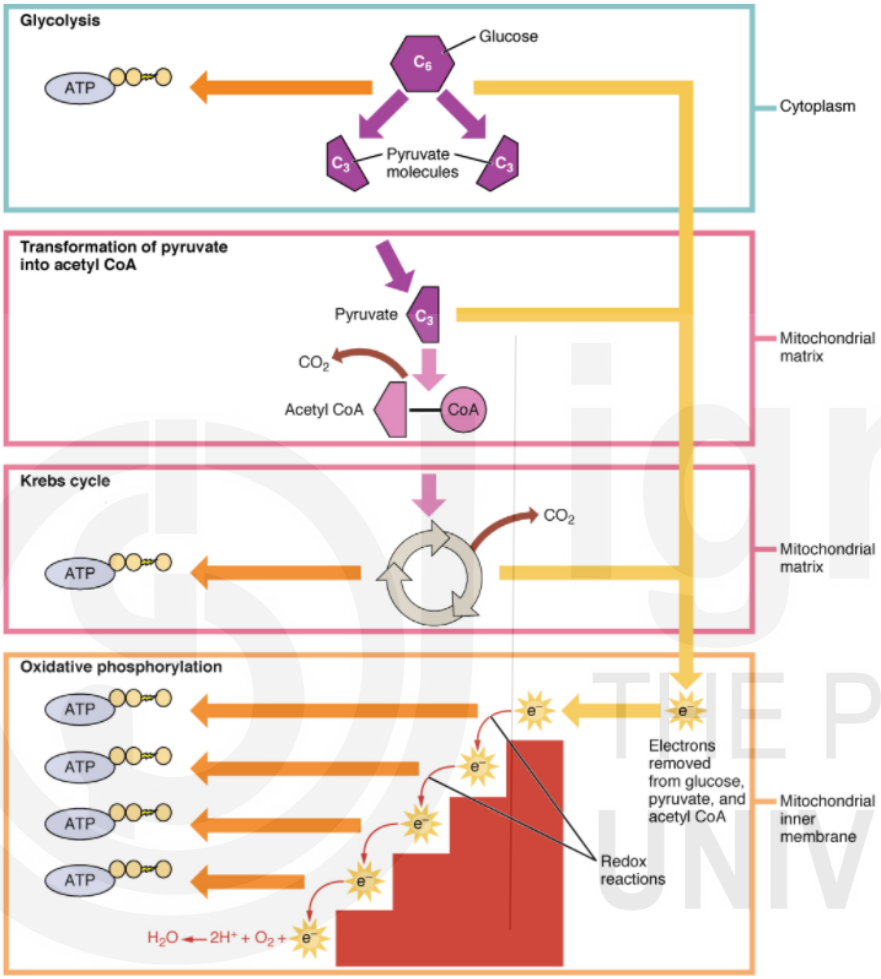



**Figure 11: Glycolysis and aerobic energy system**

***Source***: https://open.oregonstate.education/aandp/chapter/24-2-carbohydrate-metabolism/

The electron transport chain is a series of proteins embedded in the inner wall of mitochondria. These proteins transport the electron released by NADH and FADH2 from matrix to inter membrane space. This develops a gradient of hydrogen ion and movement of hydrogen ion from area of low concentration to areas of high concentration facilitates production of ATP (Figure 12) (Da Poian et al., 2010; Nolfi-Donegan et al., 2020). The aerobic system, including Kreb's or TCA cycle and electron transport chain has potential to resynthesise more ATP than anaerobic metabolic system. In fact, the ATPs gained from fat metabolism are over twice the amount obtained from carbohydrates and protein. A single molecule of free fatty acid produces over a hundred ATP molecules in contrast to 30-50 ATPs obtained from amino acid and pyruvate (Karp, 2009; El Bacha et al., 2010; Hewitt, 2014; Barclay, 2017).

Though, these systems rely on different metabolic pathways, they work together to resynthesise ATP. Their relative contribution is determined by the duration, intensity and frequency of the exercise (Karp, 2009).

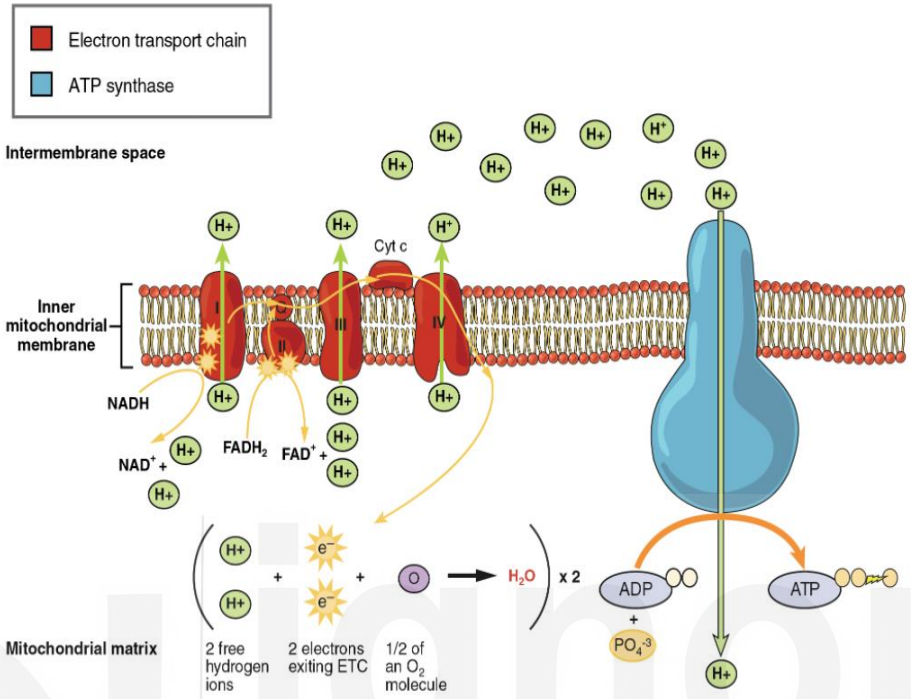



**Figure 12: Electron transport chain**

***Source***: https://open.oregonstate.education/aandp/chapter/24-2-carbohydrate-metabolism/

## Energy Needs of Different Cells

Human being, a multicellular organism, has different kinds of cells specialised to perform particular functions. The energy requirement of each of these cells may vary depending on their role. As already mentioned, humans obtain their energy by metabolising three major nutrients: carbohydrate, protein and fat. They undergo different metabolic pathways, but converge onto to a common pathway, tricarboxylic acid (TCA) or Krebs cycle for ATP synthesis.

Though mitochondria are the main site for ATP synthesis (aerobic system), some ATP is also synthesised in the cytoplasm (anaerobic ATP-CP system and glycolysis). But some cells don't have mitochondria or enzymes to use all three nutrients as fuel for ATP synthesis. For example, blood cells don't contain mitochondria and so are unable to metabolise fatty acid and amino acid. They rely completely on the constant supply of glucose for energy (Wijk & Solinge, 2005; El Bacha et al. 2010).

For some cells, source of energy depends on the nutritional status of the body and intensity of exercise. For example, skeletal muscle cells mainly consume fatty acid for energy at rest or low-mild intensity exercise. During high intensity exercise, the energy system shifts to anaerobic glucose metabolism (ATP-CP or glycolysis) (Barclay, 2017). Even, cardiac muscles mainly meet their ATP demands by breaking down fatty acids (Grynberg & Demaison, 1996). Even organs such as kidney and liver primarily depend on fatty acid

for a constant supply of energy (Grynberg & Demaison, 1996; El Bacha et al., 2010; Barclay, 2017).

Let's summarise what we learnt in tabular form.

**Table 1: Comparison of different energy systems**

| Energy system | Phosphagen system | Glycolysis | Aerobic system |
|---|---|---|---|
| **Alternative term** | ATP-PCr system | Lactic acid system | Kreb's cycle and Oxidative phosphorylation |
| **Metabolic reaction** | Anaerobic (without oxygen) | Anaerobic (without oxygen) | Aerobic (with oxygen) |
| **Site of occurrence** | Cytoplasm | Cytoplasm | Mitochondria |
| **Fuel source** | Creatine phosphate (CP), also known as Phosphocreatine (PCr) | Glucose | Glucose, Fatty acid and Amino acid |
| **Duration and intensity of activity** | High intensity, 10s | High intensity, 10-90s | Rest and low-mid intensity, >90s |
| **ATP synthesised** | 1 ATP | 2 ATPs | Glucose and amino acid: 30-50 ATPs<br>Fatty acid: 100 ATPs |
| **By products** | Inorganic phosphate (Pi), ADP | Lactic acid, Hydrogen ion (H+), ADP | $CO_2$, $H_2O$ |

**Check Your Progress 3**

7) Describe the energy system your body will use while swimming for leisure.

…….....

…….....

…….....

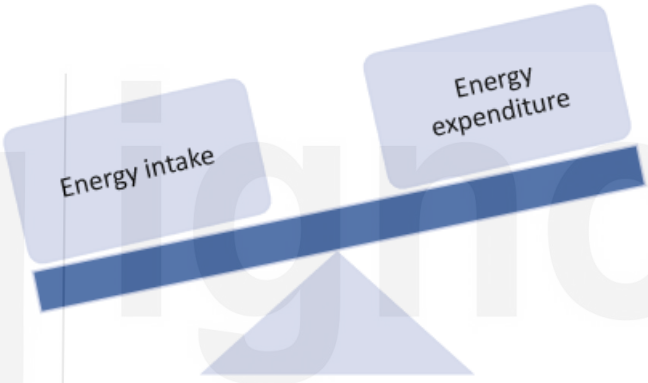
…….....

…….....

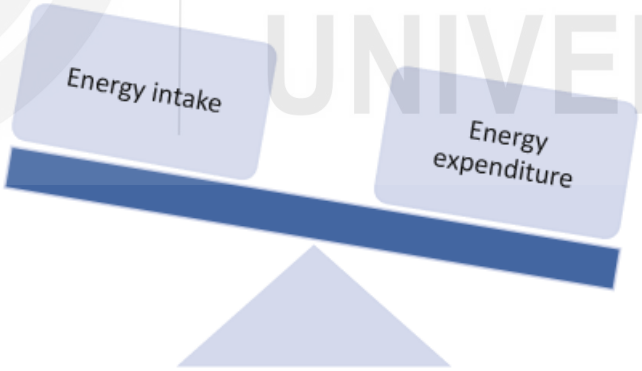
8) Glycolysis results in the production of two ……. molecules from a single molecule of glucose. In the absence of ……., the end product of glycolysis is ……...

## 1.4 ENERGY BALANCE

The body is said to be in energy balance or energy equilibrium if energy intake (amount of food consumed) is equal to energy expenditure (energy utilised in body function and physical activity). When energy intake exceeds energy expenditure, a state of positive energy balance is achieved resulting into weight gain. Conversely, more energy expenditure than energy intake indicates negative energy balance and consequently causes weight loss (Figure 13) (Wouters & Baarends, 2006; Standfield & Germann, 2007; Lam & Ravussin, 2016).




**Negative Energy Balance**




**Positive Energy Balance**

**Figure 13: Energy balance – Positive and Negative**

A typical example of energy imbalance is obesity. It is a state of excess body weight as a result of sustained positive energy balance over time (Romieu et al., 2017). Body weight is regulated by the interaction of genetic, homeostasis, environmental, and behavioral factors. Homeostatically, hypothalamus integrates signal regarding food intake and body weight and in response stimulates endocrine system to achieve energy balance. However, in current time, consumption of energy-dense food and reduced physical

activity that is obesogenic environment has allowed obesity to reach epidemic proportions (Spiegelman & Flier, 2001; Galgani & Ravussin, 2008; Hall et al., 2012; Greenway, 2015).

Thus, the two components of energy balance are:

### 1.4.1 Energy Intake

Carbohydrate, protein and fat constitute the major portion of energy intake. It is quantified by the amount, frequency and caloric value of food consumed. Energy intake is regulated by the hormones- leptin and ghrelin. When activated in response to a stimulus, they initiate different signaling cascade which eventually brings change in energy intake. Leptin mediates long-term regulation of energy balance and reduces food intake while ghrelin is a fast-acting hormone and stimulates food intake (Klok, 2007). However, the 'obesogenic environment' and change in individual's behavioral pattern seems to have shadowed the result of homeostatic functioning.

### 1.4.2 Energy Expenditure

The factors contributing to the energy expenditure are:

- **Resting Metabolic Rate (RMR)**: It is the amount of energy needed to perform vital body function at rest such as cellular metabolism, cardiac function, respiratory function and so on. It accounts for approximately 60-75% of the daily energy expenditure of an individual. It may vary with age, gender, body composition and physiological condition of the individual (Westerterp, 2004; Pelley, 2012).
- **Thermic Effect of Food (TEF)**, also called diet-induced thermogenesis: It refers to the increase in energy expenditure (or increase in the metabolic rate above basal metabolic rate) after consumption of meal. It accounts for the energy consumed during digestion and metabolism of nutrients (Westerterp, 2004; Pesta & Samuel, 2014).
- **Physical activity**: The energy expenditure due to physical activity is usually quantified by its intensity, duration and frequency. It accounts for the energy spent in addition to resting metabolic rate and thermic effect of food (Westerterp, 2004; Hills et al., 2014).

**Check Your Progress 4**

9) Match the following:

| A | B |
|---|---|
| i) Energy balance<br>ii) Basal metabolic rate<br>iii) Positive energy balance<br>iv) Hermic effect of food | a) Energy expenditure is more than energy intake<br>b) Increase in energy expenditure post meal<br>c) Energy expenditure is equal to energy intake<br>d) Amount of energy spent during rest |

## 1.5 SUMMARY

Optimal functioning and survival of human depends on a stable internal environment that is, homeostasis equilibrium. It consists of plethora of metabolic and physiological processes that are regulated by the neuro-endocrine system. Any significant deviation from the normal range is resisted and homeostasis is restored through feedback loops. Positive feedback promotes changes such as clotting, lactation while negative feedback inhibits or slows down the process such as glucose level and body temperature. Maintaining homeostasis requires that the body continuously monitors its internal conditions and this is achieved through a system of sensor, control centre and effector.

Metabolism provides the energy required by an individual for their body functions and to maintain homeostasis. However, the energy obtained from food first needs to be converted into a usable form that is ATP to be utilised at cellular level. It either occurs anaerobically in the cytoplasm or aerobically in the mitochondria through three different energy systems – phosphagen, glycolysis and aerobic. The relative contribution of these systems depends on the intensity and duration of the activity. An energy balance is achieved when energy intake is equal to energy expended. Though it is regulated by the endocrine system, changes in eating habits and reduced physical activity has resulted into positive energy balance and obesity epidemic.

## 1.6 REFERENCES

Abozenadah, H., Bishop, A., Bittner, S., & Flatt, P.M. (2018). Chemical reactions in biological systems. *Allied Health Chemistry*. CC. BY-NC-SA. Retrieved from: https://wou.edu/chemistry/courses/online-chemistry-textbooks/ch103-allied-health-chemistry/

Barclay, C.J. (2017). Energy demand and supply in human skeletal muscle. *Journal of Muscle Research and Cell Motility, 38(2),* 143–155

Bergman, J. (1999). ATP: The perfect energy currency for the cell. *Creation Research Society Quarterly, 36 (1)*

Bernard, C. (1865). *Introduction à L'étude de la Médecine Expérimentale.* Flammarion; Paris, France.

Betts et al (2013). *Introduction to the human body, anatomy & physiology*. OpenStax. Houstan: Texas.

Biga et al (2021a). An introduction to the human body: Homeostasis. *Anatomy & Physiology* (1st ed.). Oregon State University: Pressbooks. Retrieved from https://open.oregonstate.education/aandp/chapter/1-3-homeostasis/

(2021b). Metabolism and nutrition: Carbohydrate metabolism. *Anatomy & Physiology* (1st ed.). Oregon State University: Press books. Retrieved from https://open.oregonstate.education/aandp/chapter/24-2-carbohydrate-metabolism/

Botham, K.M., & Mayes, P.A. (2012). Bioenergetics: The role of ATP. In R.K. Murray et al (Eds.). *Harper's illustrated biochemistry* (29th ed., pp. 109-114). McGraw Hill.

Boundless. Metabolic Pathways. (2021) *The Libre texts projects: Biology library*. Retrieved on 27 September 2021, from https://bio.libretexts.org/Bookshelves/Introductory_and_General_Biology/Book%3A_General_Biology_(Boundless)/6%3A_Metabolism/6.1%3A_Energy_and_Metabolism/6.1C%3A_Metabolic_Pathways

Brooks, G.A., Fahay, T.H., & White, T.P. (2000). Glycogenolysis and glycolysis in muscle: The cellular degradation of sugar and carbohydrate. *Exercise physiology: Human bioenergetics and its applications* (2nd ed., pp. 54-77). Mountain View, CA: Mayfield.

Cannon, W.B. (1929). Organization for physiological homeostasis. *Physiolog ical Reviews, 9,* 399-431

Clancy, J., & McVicar, A. (1995). *Physiology and anatomy: A homeostatic approach*. London: Edward Arnold Publishers.

Cooper, G.M. (2000). The central role of enzymes as biological catalysts. *The Cell: A Molecular Approach* (2nd ed.). Sunderland (MA): Sinauer Assoc iates.

Cooper, S.J. (2008). From Claude Bernard to Walter Cannon. Emergence of the concept of homeostasis. *Appetite, 51(3),* 419–427

Da Poian, A.T., El-Bacha, T., & Luz, M.R.M.P. (2010). Nutrient utilization in humans: Metabolism pathways. *Nature Education, 3(9),* 11

Damasio, A., & Damasio, H. (2016). Exploring the concept of homeostasis and considering its implications for economics. *Journal of Economic Behavior & Organization, 126(B),* 125-129

Doherty, B., & Foudy, C. (2006). Homeostasis: Anatomy and physiology. *Nursing Times, 102(14),* 26–27

Dunn, J., & Grider, M.H. (2021). *Physiology, adenosine triphosphate*. StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing. Retrieved from: https://www.ncbi.nlm.nih.gov/books/NBK553175/

El Bacha, E.T., Luz, M. & Da Poian, A. (2010). Dynamic adaptation of nutrient utilization in humans. *Nature Education 3(9),*8

Evans, R.D., & Heather, L.C. (2019). Human metabolism: Pathways and clinical aspects. *Surgery (Oxford), 37 (6),* 302-309

Farhana, A., & Lappin, S.L. (2021). *Biochemistry, lactate dehydrogenase*. StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing. Retrieved from: https://www.ncbi.nlm.nih.gov/books/NBK557536/

Fink, G. (2007). Neuroendocrine systems. *Encyclopedia of Stress* (2nd ed., pp. 851-564). Academic Press.

Fordham, D., & Harmey, J. (2016). Metabolic pathways, nutrition, and abnormalities. In W. E. G., Thomas et al. (Eds.). *Oxford textbook of fundamentals of surgery* (1st ed.). UK: Oxford University Press.

Freudenrich, C. (2008). *How exercise works*. Retrieved on 29 September 2021, from: https://health.howstuffworks.com/wellness/diet-fitness/exercise/sports-physiology.htm

Galgani, J., & Ravussin, E. (2008). Energy metabolism, fuel selection and body weight regulation. *International Journal of Obesity, 32 (7),* S109–S119

Greenway F.L. (2015). Physiological adaptations to weight loss and factors favouring weight regain. *International Journal of Obesity, 39(8),* 1188–1196

Grynberg, A., & Demaison, L. (1996). Fatty acid oxidation in the heart. *Journal of Cardiovascular Pharmacology, 28 (1),* S11–S17

Hall et al (2012). Energy balance and its components: Implications for body weight regulation. *The American Journal of Clinical Nutrition, 95(4),* 989–994

Hewitt, J. (2014). *How understanding energy systems can make you a faster cyclist*. Retrieved on 28 Sep 2021, from: https://jameshewitt.net/2014/07/17/physiology-fuel-tanks-understanding-energy-systems/

Hiller-Sturmhöfel, S., & Bartke, A. (1998). The endocrine system: An overview. *Alcohol Health and Research World, 22(3),* 153–164

Hills, A.P., Mokhtar, N., & Byrne, N.M. (2014). Assessment of physical activity and energy expenditure: An overview of objective measures. *Frontiers in Nutrition, 1,* 5

Jesty, J., & Beltrami, E. (2005). Positive feedbacks of coagulation: Their role in threshold regulation. *Arteriosclerosis, Thrombosis, and Vascular Biology, 25(12),* 2463–2469

Karp, J. (2009). *The three metabolic energy systems: How you get metabolic energy and how you use it.* Retrieved from: https://www.ideafit.com/personal- training/the-three-metabolic-energy-systems/

Klok, M.D., Jakobsdottir, S., & Drent, M.L. (2007). The role of leptin and ghrelin in the regulation of food intake and body weight in humans: A review. *Obesity Reviews: An Official Journal of the International Association for the Study of Obesity, 8(1),* 21–34

Kurz, A. (2008). Physiology of thermoregulation. *Best Practice & Research Clinical Anaesthesiology, 22(4),* 627–644

Lam, Y.Y., & Ravussin, E. (2016). Analysis of energy metabolism in humans: A review of methodologies. *Molecular Metabolism, 5(11),* 1057–1071

Modell et al (2015). A physiologist's view of homeostasis. *Advances in Physiology Education, 39(4),* 259–266

Morton, A.R. (2008). Exercise physiology: The phosphagen system. In L. M. Taussig & L. I. Landau (Eds.). *Pediatric Respiratory Medicine* (2nd ed., pp. 89-99). Mosby.

Nolfi-Donegan, D., Braganza, A., & Shiva, S. (2020). Mitochondrial electron transport chain: Oxidative phosphorylation, oxidant production, and methods of measurement. *Redox Biology*, *37*, 101674

Osilla, E.V., Marsidi, J.L., & Sharma, S. (2021). *Physiology, temperature regulation*. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing.

Pelley, J.W. (2012). Nutrition. *Elsevier's integrated review biochemistry* (2nd ed., pp. 171-179). Saunders.

Periayah, M.H., Halim, A.S., & Mat Saad, A.Z. (2017). Mechanism action of platelets and crucial blood coagulation pathways in hemostasis. *International Journal of Hematology-Oncology and Stem Cell Research, 11(4)*, 319–327

Pesta, D.H., & Samuel, V.T. (2014). A high-protein diet for reducing body fat: Mechanisms and possible caveats. *Nutrition & Metabolism, 11(1*), 53

Pillay, J., & Davis, T.J. (2021) *Physiology, lactation*. StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing.

Romieu et al (2017). Energy balance and obesity: What are the main drivers? *Cancer Causes & Control: CCC, 28(3)*, 247–258

Rubinstein-Litwak, S. (2003). Energy metabolism. *Encyclopedia of Food Sciences and Nutrition* (2nd ed., pp. 2108-2114). Academic Press.

Spiegelman, B.M., & Flier, J.S. (2001). Obesity and the regulation of energy balance. *Cell, 104(4)*:531-43

Standfield, C.L & Germann, W.L. (2007). The endocrine system: Regulation of energy metabolism and growth. *Principles of human physiology* (3rd ed., pp 603.629). Pearson.

The sustainable training method. (2018). *Energy Systems: Anaerobic (Lactic-Glycolytic*). Retrieved on 29 Sep 2021, from: https://www.thesustainabletrainingmethod.com/tstm-blog/2018/9/6/energy-systems-part-3anaerobic-lactic-glycolytic

Torday, J.S. (2015). Homeostasis as the mechanism of evolution. *Biology, 4 (3)*, 573-90

Tortora, G.J., & Anagnostakos, N.P. (2003). *Principles of anatomy and physiology*. New York, NY: John Wiley and Sons.

Westerterp, K.R. (2004). Diet induced thermogenesis. *Nutrition & Metabolism, 1*, 5

Widmaier, E.P., Raff, H., Strang, K.T., & Vander, A.J. (2008). A framework for human physiology. *Vander's human physiology: Mechanism of body function* (11th ed.). New York: McGraw-Hill.

Wijk, R.V., & Solinge, W.W.V. (2005). The energy-less red blood cell is lost: Erythrocyte enzyme abnormalities of glycolysis. *Blood, 106 (13)*: 4034–4042

Wouters, E.F.M., & Baarends, E.M. (2006). Energy metabolism. *Encyclope dia of Respiratory Medicine, 86-90*

## 1.7 HINTS/ANSWERS TO CHECK YOUR PROGRESS

1) Homeostasis maintains a constant internal environment and allows optimal function of the body despite any change in internal or external environment. It acts either through positive feedback or negative feedback depending on the stimuli and physiological processes involved.

2) The effect opposes the original stimulus or reduces its intensity.

3) Prolonged exposure to cold would activate negative feedback process. Consequently, the blood vessel will constrict and prevent loss of body while shivering will provide additional warmth.

4) Hydrolysis is the breakdown of a molecule due to reaction with water while phosphorylation is the attachment of phosphoryl group.

5) Catabolic, exogenic

6) c) It releases a large amount of usable energy when the phosphate group is split off during ATP hydrolysis.

7) Swimming for leisure is a low intensity exercise, thus the body will use aerobic cycle for energy.

8) ATP, oxygen, lactate

9) i - c, ii – d, iii – a, iv - b

# UNIT 2 EXERCISE PHYSIOLOGY*

**Contents**



**Learning Objectives**

After going through this unit, you will be able to:

- define exercise and sports physiology;
- appreciate the importance of studying exercise physiology;


* **Contributor**: Dr. B.S.Yadav, MD, Associate Professor, Department of Physiology, Institute of Medical Sciences, Banaras Hindu University, Varanasi.

- illustrate the physiological basis of differences between male and female body structure, composition, and their relevance to exercise physiology;
- express muscle metabolism;
- explain temporal sequence of energy sources during prolonged activity;
- depict the effect of athletic training on muscles and their performance;
- express heat exchange mechanisms of the body; and
- describe the role of body water in maintaining homeostasis.

# 2.0 INTRODUCTION

**What is Exercise Physiology?**

Let us first understand what is Exercise Physiology? Exercise physiology is the study of response and adaptations of body during physical stress. The application of its principles in training of athletes and sportspersons to enhance their performance is Sports physiology. The considerable overlap in exercise and sports physiology due to same basic principle makes use of both terms interchangeably (Plowman and Smeth, 2013).

*Exercise is an intentional physical activity to generate force by activated skeletal muscles. The exercise poses severe physical stress and brings about acute (sudden and immediate) and chronic (gradual and for long term) changes in body to cope with this stress. For example, when a person starts running there will be an acute or immediate rise in heart rate and breathing rate to meet extra demands of body to sustain the activity. But when he/she keeps running regularly the physiological system of body will adapt to the effects of physical stress and the rise in heart and breathing rate will be less as compared to initial days. Due to these chronic adaptations the body will now feel lesser stress and work efficiently during exercise, resulting in improved performance.*

**Why Study Exercise Physiology?**

Studying exercise physiology will make you understand the extremes to which body's systems can withstand the stress during physical activity. It helps physical education teachers, athletes, coaches, dance trainers, fitness coaches, and other sports and exercise science professionals to enhance physical performance and health through application of its principle. Exercise physiology includes the study of effects of any kind of physical activity on the body and not merely exercise and sports.

# 2.1 GENDER SPECIFIC VARIATIONS

The basic physiological principle for both the genders remains same except for few differences which may be explained due to differences in their body built, composition and abundance of sex hormones, androgens in male. Increased protein deposition in muscles due to anabolic action of testosterone results on an average 40% larger muscles in males compared to females

whereas, females have 12% more body fat than males. Due to larger body, more muscle mass and less fat, most overall quantitative values like muscle strength, pulmonary ventilation, cardiac output are higher in male athletes as compared to female athletes. However, in terms of strength per square centimetre of muscle area, there is no difference in maximum force of contraction by muscle in male and female i.e. 3-4 kg/cm$^2$ (Hall, 2015).

## 2.2 STRENGTH, POWER AND WORK

The outcome of athletic events depends on determinants like the strength your muscle can generate in need, the power they can achieve and duration they can work for. For example, relative running speed for marathon race is about 11% higher in top male performers compared to top female performers. While for events like long distance swimming, females seem to have advantage due to higher body fat content providing heat insulation, buoyancy, and extra long-term energy (Hall, 2015).

Strength of a muscle is the maximum amount of force that can be generated on muscle contraction. The maximum muscle contractile force that can be generated per unit area of muscle is same; the strength of muscle mainly depends on its size. The amount of force applied by the muscle, multiplied to the distance over which it is applied is termed as work done by the muscle; whereas, power of the muscle is defined as amount of work done per unit time.

All the muscles in the body can generate a maximum power of about 7000 kg/min for a very short period of 8-10 seconds which is reduced to less than half for next one minute and remains only about one fifth of initial after 30 minutes of activity. This means, the efficiency of translation of muscle power into performance decreases with the activity. Physical capability to sustain exercise for an extended period, called endurance, depends mainly on nutritive support to muscle especially the amount of stored glycogen in muscles and liver (Hall, 2015).

## 2.3 METABOLIC SYSTEM IN MUSCLE DURING EXERCISE

The basic system of metabolism is same in all the muscles of body (Figure 1)

These metabolic systems are grouped in three categories:

- ATP and creatine phosphate.
- glycogen-lactate system.
- aerobic metabolism.

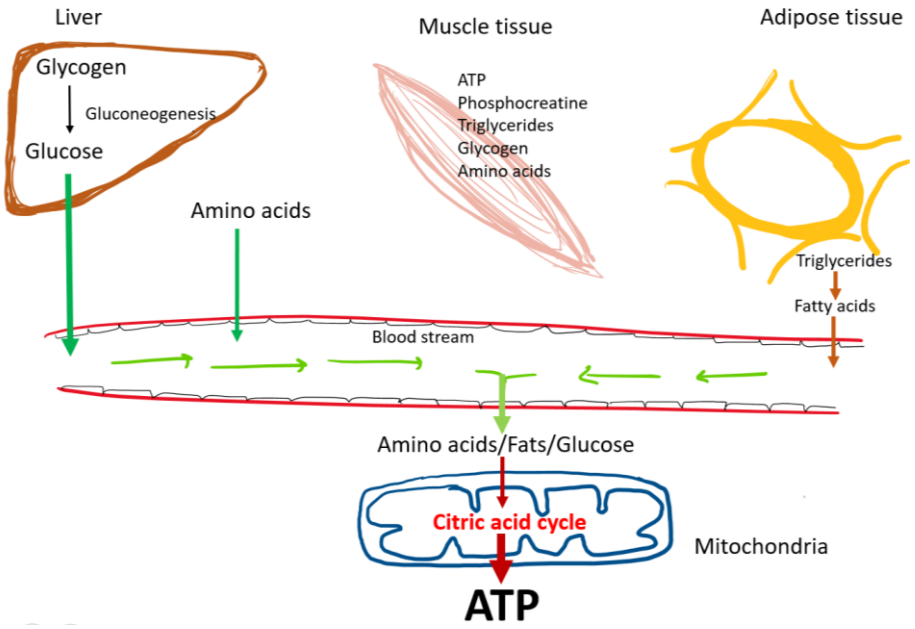



**Figure 1: Macronutrient fuel sources that supply substrates to regenerate adenosine triphosphate (ATP)**

***Source***: Adapted from:McArdle WD, Katch FI, Katch VL. Essentials of exercise physiology.4. Lippincott Williams & Wilkins; 2006.

The end metabolic product of all the three systems is adenosine triphosphate (ATP) which is the primary carrier of energy in cells thus, also referred to as currency of muscle energetics. It is used as end source of energy for muscle contraction.

## 2.2.1 The ATP and Creatine Phosphate

**ATP: Adinosine-$PO_3$~$PO_3$~$PO_3$**

The phosphate radicals and adenosine molecules are attached chemically by high energy phosphate bonds and each of these bonds store 7300 calories of energy per mole. Thus 14600 calories are released by conversion (hydrolysis) of ATP to AMP. The muscle's stored ATP can sustain maximal muscle power for 3-4 second only, to continue thereafter new ATP must be formed during the performance (Hall, 2015).

**Creatine-PO3**

This phosphate bond when broken, release about 10300 calories per mole which is greater than that of ATP. Muscle's storage of phosphocreatine is 2-4 times that of ATP hence it can provide maximal power for 8-10 seconds which is just enough for a 100 meter race. Phosphocreatine can provide enough energy to reconstitute the ATP currency in muscle, then within few milliseconds the energy transfer from phosphocreatine to ATP starts, thus almost instantaneously supplying energy for muscle contraction. The ATP and creatine-phosphate together form phosphagen energy system (Hall, 2015).

## 2.3.2 Glycogen-Lactate System

Glycogen, a stored form of energy in muscle and liver is polysaccharide of glucose, while lactic acid is formed through anaerobic metabolism of

glucose. The muscle's stored glycogen is metabolised to produce energy, while that in liver is used in gluconeogenesis to produce glucose which is then metabolised to form ATP. The process involves splitting of glucose into two pyruvic acid molecules which in turn produce large number of ATPs in the presence of oxygen, through oxidative phosphorylation in mitochondria. However, the pyruvic acid in the absence of sufficient oxygen is converted into lactic acid in the muscles and diffuses out to interstitium and blood. The glycogen-lactate system produces ATP 2.5 times faster than oxidative phosphorylation in mitochondria. Therefore, the glycogen is converted into lactic acid to generate and supply energy where oxidative metabolism is unable to meet the energy demand of body. Under optimal conditions glycogen-lactic acid system can supply energy for 1.3-1.6 minutes of intense muscular activity.

### 2.3.3 The Aerobic Metabolism

Aerobic system consists of oxidation of food products in the mitochondria to provide energy. End products of food metabolism like glucose, fatty acids and amino acids, undergo oxidative phosphorylation to release tremendous amounts of energy which is used to form ATP inside the mitochondria via electron transport chain.

#### 2.3.3.1 Replenishing Muscle's Energy Stores after Exercise

As discussed previously when phosphocreatine system takes over, ATP is reconstituted. Glycogen-lactate system regenerates both phosphocreatine and ATP whereas aerobic system is used for reconstitution of all other systems (Figure 2).

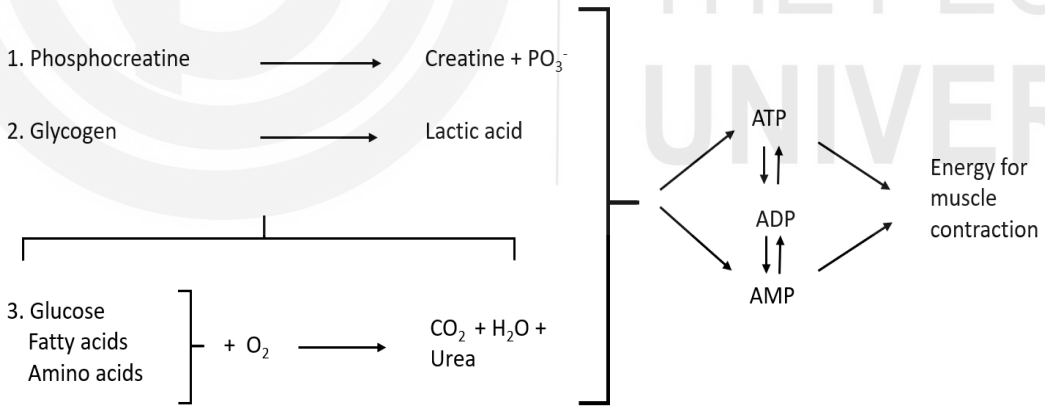



**Figure 2 Muscle's metabolic systems**

***Source***: Adapted from: Hall, J.E. 2015.

In lactate system, recovery refers to the removal of excess lactate accumulated in body fluid which is responsible for muscle soreness and fatigue.

Lactate removal is achieved by converting it:

- back to pyruvic acid which can be metabolised oxidatively.
- into glucose by gluconeogenesis in liver which can either be metabolised or be converted to glycogen in the muscles.

### 2.3.3.2 Replenishing Aerobic System

As intense exercise requires enormous amount of energy, one's aerobic energy capability is exhausted even during early stages of exercise. This exhaustion is caused by:

- Oxygen debt,
- Exhaustion of the muscle glycogen stores.

#### 2.3.3.2.1 Oxygen Debt

Your body stores about two litres $O_2$, which maintains aerobic metabolism in the events of transient anoxia. These stores comprise of:

- Half a litre in air filling the lungs
- 1/4 litre dissolved in plasma and interstitial fluid
- 0.3 litre attached to myoglobin in muscles

All of this stored oxygen is utilised in less than a minute of intense exercise for energy production, aerobically. So, to replenish it body continues to breathe extra amount of oxygen over and above the normal requirements even after the cessation of exercise (Figure 3).

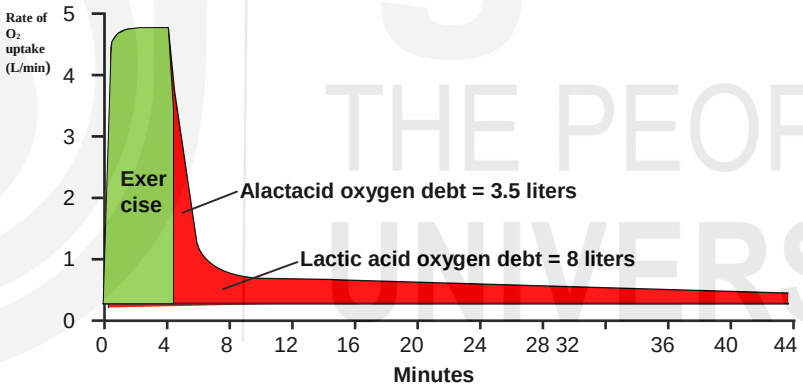



**Figure 3: Demonstrating the principle of oxygen debt - Oxygen uptake per minute during maximal exercise for 4 minutes and during rest for 40 minutes, following exercise.**

***Source***: Adapted from: Hall, J.E. 2015.

In addition to this about 9 litres of oxygen is further required to reconstitute the ATP, creatine phosphate and glycogen-lactate stores. All this extra amount of oxygen (~11.5 litres) that needs to be repaid after the exercise is known as oxygen debt (Hall, 2015).

#### 2.3.3.2.2 Recovery of Muscle Glycogen

Replenishing depleted glycogen stores is complex process and takes many days for complete recovery. It highly depends on the type of diet taken during recovery period. Athlete who takes high carbohydrate diet recovers glycogen stores in about 2 days while it may take 5-6 days in those who consume high

fat and high protein diet. The two important take home messages from the above discussion are:

- athletes should refrain from exhaustive exercise during 48 hours preceding the event and
- should take high carbohydrate diet before an endurance event.

**Check Your Progress 1**

1) Define fatigue and state its physiological significance.

……

……

……

……

……

## 2.4 NUTRIENT OF CHOICE DURING EXERCISE

The main source of energy for muscles especially during early stage of exercise is carbohydrates, followed by fat as fatty acids and sometimes proteins as amino acids. All endurance athletic events which last more than 4-5 hours, exhaust muscle glycogen stores even with best efficiency. To further continue the activity, energy is derived chiefly from fatty acids and from mobilising liver glycogen via gluconeogenesis. Figure 4 shows the relative usage of carbohydrates and fatty acids during endurance activities under different dietary conditions.

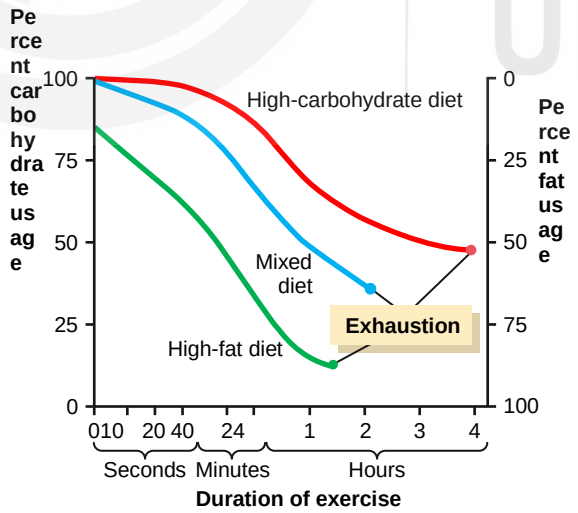



**Figure 4: Relative usage of carbohydrates and fatty acids during endurance activities under different dietary conditions.**

***Source:*** Adapted from: Hall. 2015

During first few seconds or minutes most of the energy is derived from carbohydrates whereas, by the time of exhaustion i.e. after 3 to 4 hours of

event 60% - 85% of energy is derived from fats. It can be noted from the earlier discussion that glycogen and blood glucose are the energy nutrients of choice when available (Hall, 2015).

## 2.4 ATHLETIC TRAINING AND MUSCULAR PLASTICITY

**Resistance Training**

Muscles hardly increase in strength even after prolonged muscular activity if performed under no or minimal load. While, working at more than half of maximal force of contraction, strength develops rapidly even if the contractions were performed only few times a day. Based on this principle it has been shown experimentally that performing three sets of six maximal muscle contractions for three days per week gives best results for increasing muscle strength. Muscle strength can be increased by about 30% in initial six to eight week with such a training program but reaches plateaus after that time. This increase in muscle strength is mainly attributed to increase in diameter of muscle fibres, process known as muscle hypertrophy. To lesser extent the increase in muscle mass is also contributed by increase in number of muscle fibres called muscle hyperplasia.

Following changes occur in hypertrophied muscle:

- Increase in the number of myofibrils i.e. diameter of the muscle fibres,
- Increased oxidative enzymes in mitochondria,
- Increased creatine phosphate and ATP,
- More than 50% rise in store glycogen, and
- 75% to 100% rise in stored triglycerides/fat.

These adaptations make both aerobic and anaerobic metabolism systems more capable and increase the maximal oxidation capability and efficiency of oxidative metabolism system.

## 2.6 TYPES OF MUSCLE FIBERS

Our muscles are made up of different proportions of two types of fibres, namely fast-twitch and slow-twitch muscle fibers. The basic differences between the two types of fibers are that the fast twitch fiber:

- are twice as large in diameter compared with slow-twitch fibers;
- have 2-3 times more active enzymes which expedite energy release from the phosphagen and glycogen–lactate systems, increasing maximal achievable power in similar proportion;
- contain lesser amounts of mitochondria, enzymes of aerobic metabolism and myoglobin; and
- have less capillary density.

The relative proportion of fast twitch versus slow twitch fibres in skeletal muscles of different individuals is also genetically determined. These

hereditary differences chiefly determine an individual's inherent propensity to endurance versus resistance activities. Athletic training programmes have not shown any significant change in the relative proportions of fast-twitch and slow twitch fibres in skeletal muscles. It implies that people with relatively higher proportion of slow twitch fibres have inherent advantage to perform better in endurance sports. More than 80% of muscle fibres in marathoner's muscles are slow twitch type while sprinter's muscle predominantly has fast twitch fibres (65%) (Hall, 2015).

**Check Your Progress 2**

2) Differentiate between fast twitch and slow twitch muscle fibers.

……

……

……

……

……

## 2.7 RESPIRATORY SYSTEM IN EXERCISE

Respiratory system is usually not a limiting factor in most athletic events but it becomes critical for maximal performance in endurance athletics. For better understanding we shall discuss structure and function of human respiratory system.

### 2.7.1 Alveolar Ventilation and Oxygen Utilisation during Exercise

An adult man at rest consumes an average of 250 ml/min. of oxygen. However, for men, during maximal exercise conditions, this increases to approximately 3600 ml/min, 4000 ml/min, 5000 ml/min in untrained adult, trained athlete and marathon runners respectively (Hall, 2015).

At different levels of exercise, there exists a linear relationship between oxygen utilisation and total alveolar ventilation. In trained athletes both the values increase by about 20-times from resting state to peak exercise capacity.

### 2.7.2 Limits of Pulmonary Ventilation

How severely can you stress your respiratory systems during exercise?

In an adult man at peak exercise, the pulmonary ventilation is about 100-110 L/min while the maximal breathing capacity is about 150-170 L/min. It means peak breathing capability is about 50% greater than the actual ventilation of lungs during peak exercise. This reserve acts as safety for extra ventilation which is required in conditions like:

- exercise at high altitudes,
- exercise under hot and humid conditions,
- abnormalities in the respiratory system.

Thus, it is not for the ability of respiratory system to deliver oxygen to muscles during exercise but the capacity of heart to pump the blood into circulation which is the main limiting factor for maximal exercise capacity.

### 2.7.3 Effect of Training on $V\dot{O}_2$max

$V\dot{O}_2$ max is the abbreviation for maximal oxygen uptake per minute during intense exercise. Short term exercise training increases by about 10% while long term endurance training can increase $V\dot{O}_2$ max considerably more than 10%. $V\dot{O}_2$ max of a marathon runner is about twice that of an average person. The main determinants of $V\dot{O}_2$ max of athletes are genetically determined anthropometric parameters (Hall, 2015).

Thus, persons with larger chest relative to body size, stronger muscles of respiration, higher $V\dot{O}_2$ max and greater proportion of slow twitch muscle fibres would prefer to become marathoners due to relative ease in performing endurance activities compared to others.

### 2.7.4 $O_2$ Diffusing Capacity in Athletes

The $O_2$ diffusing capacity denotes the rate of oxygen diffusion from the alveoli into the blood per millimetre of mercury difference between partial pressures of oxygen in alveoli and deoxygenated blood (ml $O_2$/min/mmHg). The diffusion capacity increases several fold from rest to peak exercise. This results mainly due to increased perfusion area by recruitment of previously collapsed capillaries and increased cardiac output. Diffusion capacity can be adversely affected by smoking and diseases that reduced alveolar surface area like emphysema.

### 2.7.5 Blood Gases during Exercise

During heavy exercise, oxygen consumption increases to meet the high energy demand of the body and carbon dioxide is also produced in similar proportion. Despite highly increased demand and production of $O_2$ and $CO_2$ respectively, partial pressure of oxygen ($PO_2$) and carbon dioxide ($PCO_2$) remain nearly normal in blood, demonstrating the extreme ability of the respiratory system to provide adequate aeration during stress. The nervous signals that are transmitted from the brain to the skeletal muscles to cause the exercise also stimulate medullary centres to increase heart rate and respiration. Sensory signals from the contracting muscles and active joints are also transmitted to the respiratory centre to stimulate respiration.

### 2.7.6 Effect of Smoking on Pulmonary Ventilation in Exercise

Smoking, as we know, is injurious to health for many reasons:

- nicotine in smoke causes direct destruction of terminal bronchioles;
- smoke irritates airway passage and causes inflammation and increases fluid secretion from epithelial lining; and
- nicotine suppresses ciliary clearance of airways.

Chronic smoking destroys the alveolar septa and decreases surface area of respiratory membrane limiting $V\dot{O}_2$ max.

## 2.8 CARDIOVASCULAR SYSTEM IN EXERCISE

The basic requirement of oxygen and other nutrients to the exercising muscles increases drastically during exercise. To meet these requirements blood flow to exercising muscles increases about 25 times, by:

- Increasing cardiac output, which is equal to stroke volume times heart rate. It increases blood flow & pressure,
- Decreasing vascular resistance due to increased luminal diameter,
- Vasodilation in exercising muscles,
- Vasoconstriction in visceral organs and non-exercising muscles shunting blood to general circulation.

### 2.8.1 Effect of Training on Cardiac Output

Cardiac output in a well-trained athlete can increase six to eight-fold while in a normal untrained person can increase a little over four-fold. Both the heart rate and stroke volume increase to about 95 percent of their maximal levels during maximal exercise increasing cardiac output to about 90 percent of the maximum achievable values, leaving hardly any reserve for further stress. As oxygen consumption by the body cannot exceed the rate at which the cardiovascular system can transport it, thus cardiovascular system becomes more limiting on $V\dot{O}_2$ max than respiratory system.

**Check Your Progress 3**

3) Define cardiac output and discuss the effect of exercise on cardiac output.

……

……

……

……

……

## 2.9 BODY HEAT IN EXERCISE

Energy released during exercise by metabolism of nutrients is finally dissipated as heat. Under optimal conditions also only 20%-25 % of nutrient energy is converted into muscle work, the rest is released as heat. Amount of heat liberated during exercise is directly proportional to the oxygen consumption which as we know can increase up to 20-fold in the well-trained athlete during intense exercise. This tremendous amount of heat liberated into the internal body tissues needs to be dissipated to surroundings in order to maintain core body temperature for normal body functioning. Hot and humid

surroundings prevent the sweating mechanism from eliminating this heat which may lead to heatstroke in the athlete.

**Heatstroke**

Exertion heat stroke occurs when metabolic heat production exceeds the body's heat-dissipating mechanisms and body temperature rises to dangerously high levels. Risk is highest during endurance athletics in hot and humid environment. The body temperature may rise to 106°F to 108°F which is destructive to tissue cells, especially the brain cells. Its symptom ranges from tiredness, extreme weakness, and headache to feeling dizzy, nauseating with profuse sweating which may cause confusion and even collapse and unconsciousness. In heatstroke, body's mechanism of temperature regulation is overwhelmed. The raised body temperature almost doubles the rates of cellular reactions, further liberating heat and compounding the situation. Reducing the body temperature as soon and as rapidly as possible is the main aim of treatment which comprises of removal of clothing, sponging the body with cold water and blowing air over the body, hospitalisation may be considered in severe cases.

## 2.10 WATER REQUIREMENT DURING EXERCISE

Apart from providing structure and form to our body, water is a medium for body's transport mechanisms, exchange of gases and metabolic waste. It helps in the maintenance of homeostasis through body's fluid, electrolyte balance and heat exchange mechanisms. Average daily intake of water in normal environment is about 2.5 L which can increase to between 5 and 10 L for an active person in a warm environment. Sources of water input to body are: liquids, foods and metabolic processes. Water is lost from body in four ways namely urine, through the skin, as water vapor in expired air and in faeces. Water loss through sweating is affected by severity of physical activity, environmental temperature and humidity. The major physiologic defence against overheating comes from evaporation of sweat from the skin's surface. The evaporation of 1 L of sweat releases about 600 kCal of heat energy from the body to the surroundings. Thus, appropriate hydration along with electrolytes is very crucial for optimum exercise output and preventing heat stroke (Plowman and Smith, 2013).

**Check Your Progress 4**

Write short notes on:

4) Oxygen debt

…….

…….

…….

…….

5) $V\dot{O}_2$ max

…….

…….

…….

…….

…….

6) Heat stroke

…….

…….

…….

…….

…….

## 2.11 SUMMARY

Exercise physiology is the study of body's response and adaptations to physical stress. It is applied by trainers and coaches to enhance physical performance and health of their subjects. Gender specific physiological differences in body built and composition are attributed to hormone testosterone. An athlete's strength and power are attributed mainly to muscle size while endurance to nutritive support and physical training.

ATP (adenosine triphosphate) produced as the end product of metabolism, is the currency of muscle energetics. Carbohydrate rich diet and rest helps body to replenish its depleted energy stores faster, following strenuous physical activity. Muscle's plasticity enables its fibres to increase in size and number as per body's demand. Cardio-respiratory system adapts remarkably to increasing body demand to physical training. In face of heightened oxygen demand pulmonary ventilation can rise up to 20 folds while cardiac output rises 6 to 8 folds, making it the limiting factor to maximal oxygen transport required during heavy exercise. 75% to 80% of energy produced during exercise is released as heat because only 20%-25% is utilised for work. Thus, heat dissipation and efficient cooling is very crucial to prevent heat stroke, seen commonly in conditions which decrease gradient for heat loss e.g. in hot and humid environment. Maintaining adequate hydration is equally important during physical training as water is body's medium to transport heat, gases, nutrients, electrolytes and products of metabolism. Dehydration could lead to heat stress, fatigue, electrolyte imbalance which could be fatal, if severe.

## 2.12 REFERENCES

Hall, J.E. (2015). *Guyton and Hall textbook of medical physiology (*13th ed.). Elsevier Health Sciences.

McArdle, W.D., Katch, F.I., & Katch, V.L. (2006). Essentials of exercise physiology (4th ed.). Lippincott Williams & Wilkins.

Plowman, S.A., & Smith, D.L. (2013). *Exercise physiology for health fitness and performance* (3rd ed.). Lippincott Williams & Wilkins.

## 2.13 HINTS/ANSWERS TO CHECK YOUR PROGRESS

1) Fatigue is temporary and transient decrease in strength of muscle contraction due to prolonged or repetitive muscular contractions. It is generally perceived as sense of tiredness, lack of energy and feeling of exhaustion. It helps prevent muscle damage due to lack of nutrients and provides body some reserve for emergencies like fight/flight situation. Lactate accumulation, increased $H^+$ ions in muscles stimulate pain fibres which set in the sense of fatigue.

2) As compared to slow twitch muscle fibres, fast twitch muscle fibers are twice as large in diameter, have 2-3 times more active enzymes for quick energy release from the phosphagen and glycogen–lactate systems which increases maximal power generation. They contain lesser number of mitochondria, enzymes of aerobic metabolism and myoglobin and have less capillary density. Thus, fast twitch muscle fibers are suited for short bursts of heavy exercise while slow twitch fibres for prolonged endurance activities.

3) Cardiac output is the amount of blood pumped by each ventricle per minute. It is calculated as stroke volume x heart rate. Exercise is a stress on cardiovascular system due to increased oxygen demand of contracting muscles. To facilitate this, both heart rate and stroke volume rises due to increased sympathetic stimulation to heart, increasing cardiac output 6 to 8 times of normal. Contracting muscles also helps increase venous return to heart, increasing preload while vasodilatation during exercise decreases afterload this helps increase cardiac output and decrease workload on heart, respectively.

4) At rest our body stores about two litres oxygen distributed in air filled in the lungs (0.5 litre), dissolved in plasma and interstitial fluid (0.25 litre), attached to myoglobin in muscles (0.3 litre). All of this stored oxygen is utilised in less than a minute of intense exercise for energy production, aerobically. So, to replenish this body continues to breathe extra amount of oxygen over and above the normal requirements even after the cessation of exercise. In addition to this about 9 litres of oxygen is further required to reconstitute the ATP, creatine phosphate and glycogen-lactate stores. All this extra amount of oxygen (~11.5 litres) that needs to be repaid after the exercise is known as oxygen debt.

5) $V\dot{O}_2$ max refers to maximal oxygen uptake per minute during intense exercise. Exercise training can increase $V\dot{O}_2$ max considerably. $V\dot{O}_2$ max of marathon runners is about twice that of an average person.

Anthropometric parameters like height, chest/body size and lean body mass affect $V\dot{O}_2$ max.

6) Heat stroke is a condition when body's mechanism of temperature-regulation is overwhelmed. It is usually caused by prolonged exposure to or physical exertion in high temperatures when body's heat production exceeds body's heat-dissipating mechanisms and body temperature rises to dangerously high levels (106°F to 108°F). Such high temperatures are detrimental to tissue cells, especially the brain cells.

# UNIT 3 HAEMODYNAMICS*

**Contents**



**Learning Objectives**

After reading this unit, you will be able to understand:

- the phases of a cardiac cycle;
- factors affecting cardiac output;
- variables determining the blood flow in vessels; and
- transportation and exchange of oxygen in the body.


*Contributor: Dr Deepali Verma, Research Consultant, Freelance.

# 3.0 INTRODUCTION

All living organisms have evolved to survive and reproduce. To do so, they share a common mechanism for distributing oxygen and nutrients throughout the body and excreting metabolic wastes, which is, the circulatory or cardiovascular system. This system also helps to maintain the body homeostasis by regulating body temperature and body fluids. It transports immune cells which help body fight against infections or other diseases, as well as hormones which control body functions (Monahan-Earley, 2013; Secomb, 2016).

Human cardiovascular system consists of the heart and an extensive network of blood vessels - arteries, veins and capillaries through which blood flows. It has three circulatory paths:

- pulmonary, which carries deoxygenated blood from the heart to the lungs and returns oxygenated blood;
- systemic, which carries oxygenated blood from heart throughout the body and returns deoxygenated blood; and
- coronary, which carries oxygenated blood intrinsic to heart muscle and returns deoxygenated blood (Malanga, 2007) (Figure 1).

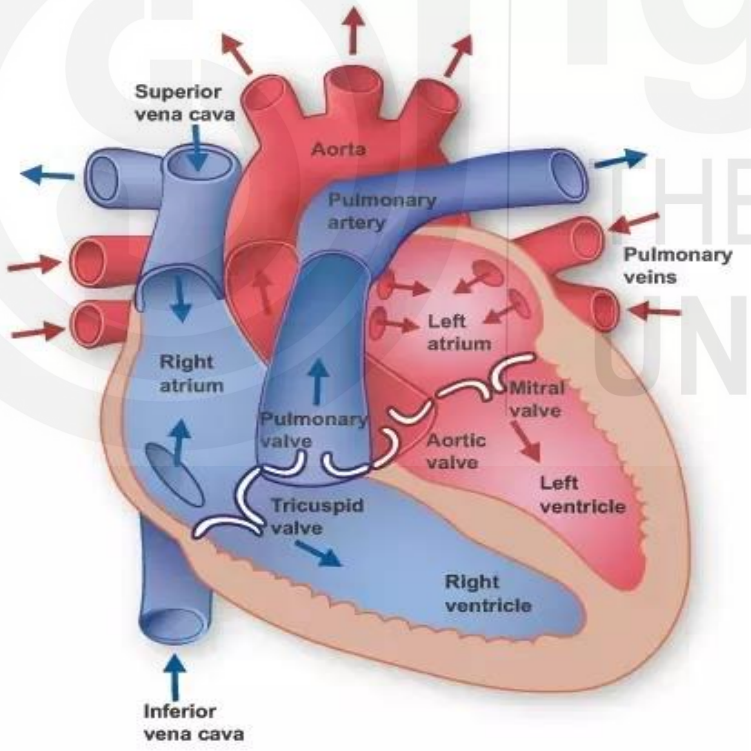



**Figure 1: The heart structure and blood flow**

***Source:*** https://www.quora.com/How-does-blood-flow-through-the-heart

The contraction and relaxation of heart creates a pumping action forcing blood to flow through the body while structural design of valves allows flow in only one direction. They open or close in response to the pressure differences across them (Laizzo, 2005).

*Haemodynamic refers to the study of physical factors which govern blood flow throughout the body or cardiovascular system (Klabunde, 2011; Secomb, 2016). Broadly, blood flow is determined by the pressure difference across the cardiovascular system and the resistance of the vessel. It flows from higher pressure to low pressure region and is impeded by the resistance of blood vessels.*

This relationship can be expressed by Ohm's law, in relation to fluid as: (Klabunde, 2011):

**Blood flow (Q) = Pressure gradient (ΔP)/ Resistance (R)**

Where, $\Delta P = P_2 - P_1$ (difference in pressure).

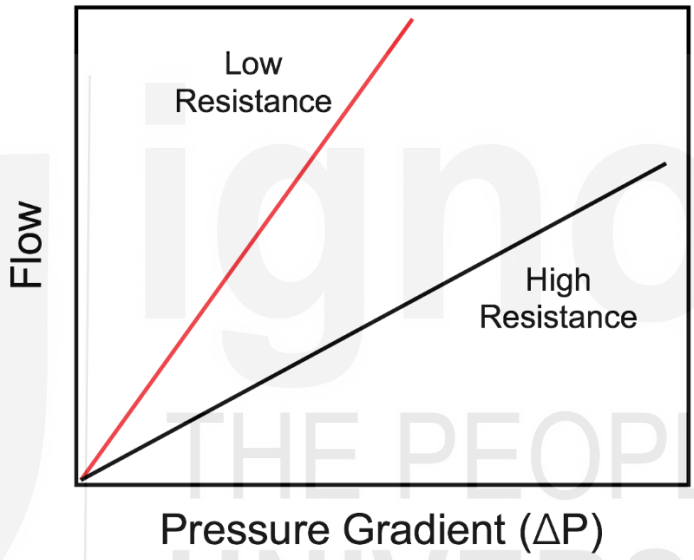



**Figure 2**: Blood flow at given resistance and pressure gradient

***Source:*** https://www.cvphysiology.com/Hemodynamics/H010

To understand this further, let's divide it into two parts:

- Blood flow in the heart
- Blood flow in the vessels

## 3.1 BLOOD FLOW IN THE HEART

### 3.1.1 Cardiac Cycle

A cardiac cycle is the sequence of contractions which pump blood through the body followed by relaxation where heart fills with blood. These two phases are called the systole (contraction) and diastole (relaxation), respectively (Figure 3).

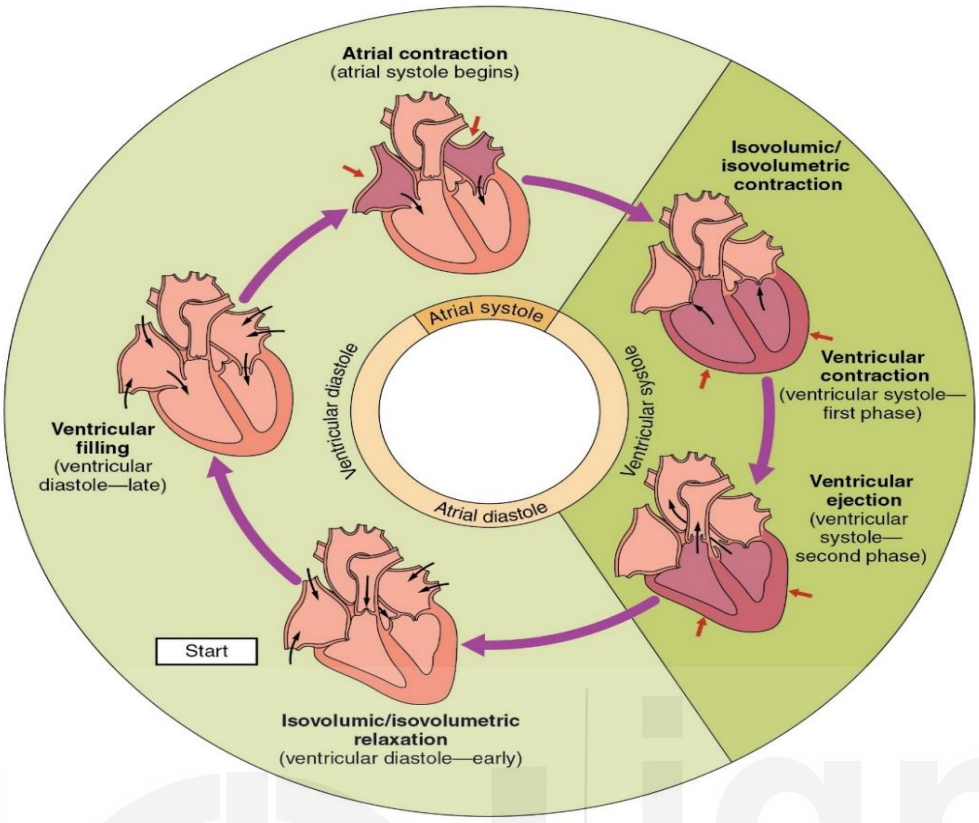



**Figure 3: Cardiac cycle**

***Source:*** https://www.sciencetechstudy.com/2019/03/cardiac-cycle.html

### 3.1.1.1 Atrial Systole

At the beginning of the cardiac cycle, both the atria and ventricles are relaxed (diastole). The right atrium receives deoxygenated blood from the

- superior vena cava, which drains blood from veins that come from the brain and arms;
- inferior vena cava which drains blood from the veins that come from the lower organs and the legs; and
- coronary sinus which drains blood from the heart itself.

At the same time, left atrium receives oxygenated blood from pulmonary veins. When the pressure of atriums exceeds the pressure of ventricles, the two atrio-ventricular valves - the tricuspid at right side of heart and mitral valves at left side of heart open allowing 70-80 percent of blood in the chambers to flow passively into the ventricles (Klabunde, 2011; Rubenstein et al., 2015; Voorhees and Hai-Chao Han, 2015; Ostadfar, 2016; OpenStax, 2019a).

Rest 20-30 percent of the ventricular filling occurs by atrial contraction stimulated by the impulse generated at Sino-atrial (SA) node (Figure 4). It is a set of specialised neurons located in the upper wall of right atrium in proximity to the superior vena cava. The impulse travels to the left atrium through the Bachmann's bundle causing both atria to contract in unison and emptying into the ventricles (Hall and Guyton, 2011; Baura, 2012).

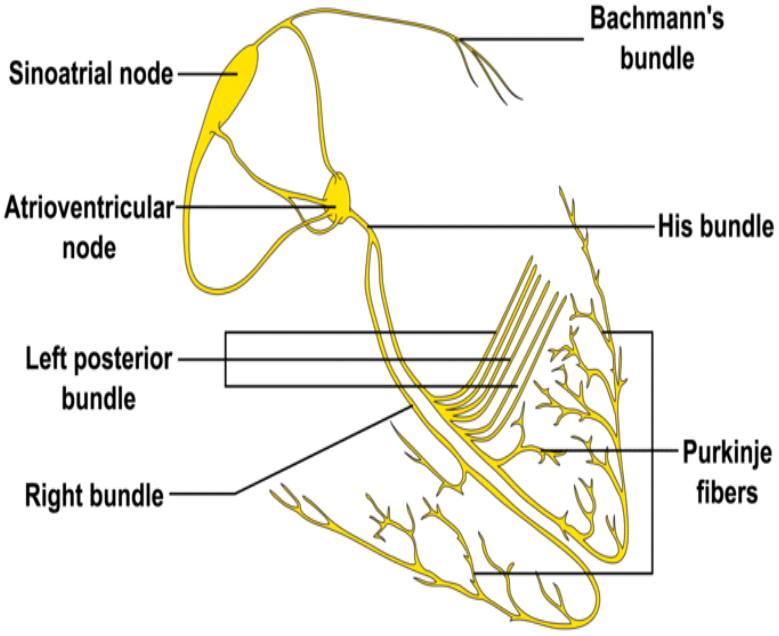



**Figure 4: The Cardiac Conduction System**

***Source:*** https://courses.lumenlearning.com/boundless-ap/chapter/physiology-of-the-heart/

### 3.1.1.2 Ventricular Systole

The impulse from SA node slowly reaches the atrio-ventricular (AV) node situated between the right atrium and right ventricle. It is delayed for about a tenth of a second which allows atria to empty completely into the ventricles (Guyton and Hall 2011, Baura 2012). The impulse then travels through the bundle of His and Purkinje fibres leading to ventricular contraction. As the ventricles contract, the pressures within them rapidly exceed that of atria and forces atrio-ventricular valves (tricuspid and mitral) to close. Closing of these valves produces the first heart sound 'lub' (Figure 5).

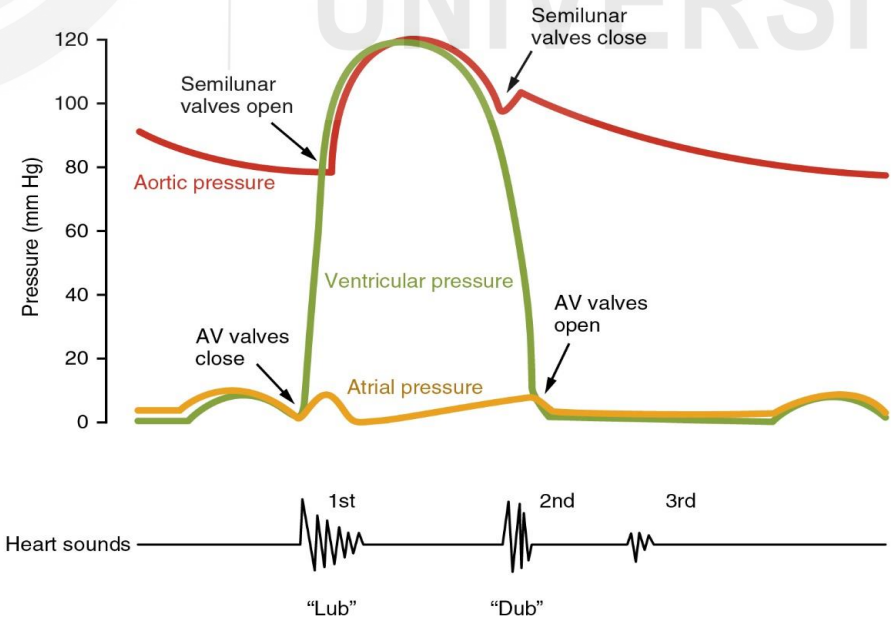



**Figure 5: Pressure difference and heart sound during a cardiac cycle**

***Source:*** https://teachmephysiology.com/cardiovascular-system/cardiac-cycle-2/cardiac-cycle/

However, the ventricular pressure is not high enough to open the semilunar valves - pulmonary and aortic valves. Since there is no ejection, the volume of blood in ventricles remains constant and so this initial phase of ventricular contraction (systole) is known as isovolumetric contraction (Rubenstein et al., 2015; Voorhees and Hai-Chao Han, 2015; Ostadfar, 2016; Ahmad, 2018; OpenStax, 2019a).

Continued contraction of ventricle causes ventricular pressure to exceed that of pulmonary artery and aorta. As a result, the semilunar valves are forced open and blood is ejected into pulmonary artery and aorta. Pressure generated in the left ventricle is slightly higher (8mmHg) than the pressure generated by the right ventricle as the existing aorta pressure is so much high. However, the amount of blood pumped from both ventricles remains same. The amount of blood ejected into the body is called stroke volume (Klabunde, 2011, Mohrman et al., 2018; OpenStax, 2019a).

### 3.1.1.3 Ventricular Diastole

As blood is pushed into pulmonary artery and aorta, the pressure within the ventricles begins to fall. When pressure drops below the pressure in both, the pulmonary trunk and aorta, the semilunar valves close to prevent the blood backflow into the heart. Closing of semilunar valves produces second heart sound ‘dup’. At this point, the atrio-ventricular valves remain close and there is no change in the volume of blood in ventricle. So, this early phase of ventricular diastole is called the isovolumetric ventricular relaxation phase (Klabunde, 2011; OpenStax, 2019a).

As the ventricular muscle continues to relax (late phase), pressure on the blood within the ventricles drops even further. Eventually, it drops below the pressure in the atria. The atrio-ventricular valves (tricuspid and mitral) open and the cardiac cycle begins once again with filling of blood into ventricles from atria (OpenStax, 2019a).

**Check Your Progress 1**

1) Explain the contraction and relaxation phases of a cardiac cycle.

……

……

……

……

……

## 3.2 BLOOD FLOW IN THE VESSELS

### 3.2.1 Resistance

In blood vessels, the resistance to blood flow is a function of viscosity, vessel radius, and vessel length. It can be represented by Poiseuille Law of Resistance as:

**Resistance (R) = 8 x viscosity (η) x vessel length (L) /π x radius ($r^4$)**

Where, 8 and π are constant.

The equation reflects that vascular resistance is directly proportional to the length (L) of blood vessel and the viscosity (η) of the blood while inversely proportional to the fourth power of radius ($r^4$). That is, increase in vessel length or blood viscosity will increase the resistance while increase in vessel radius will reduce the resistance to blood flow. The greater the resistance, the lower the flow. As change in radius alters resistance inversely to the fourth power, two times increase in radius will decrease the resistance by sixteen times (Klabunde, 2011; Faber and Stouffer, 2017).

Ohm's law and Poiseuille's law for resistance have been combined together to get an overall equation for the blood flow and is called Poiseuille's law for flow. The formula is:

$$\textbf{Blood flow (Q)} = \Delta P \pi r^4 / 8\eta L$$

**Check Your Progress 2**

2) Explore the relation of resistance to the blood flow in vessel.

……

……

……

……

……

## 3.2.2 Vessel Length and Diameter

Blood vessels increases in length as individual grows from a child to an adult but not later. In contrast, the diameter of blood vessels changes throughout the body. The aorta branches into arteries, which eventually branch into smaller arterioles and then capillaries. Blood from capillaries enters into venules. Venules progressively merge into larger veins and veins into vena cava. The diameter increases from capillaries to aorta and from venules to vena cava. Greater the diameter, lesser the resistance and increased blood flow (Klabunde, 2011, OpenStax, 2019a).

## 3.2.3 Blood Viscosity

Viscosity is the internal resistance of fluid to flow. It relates to the friction between fluid layers, that is, when a layer slides over adjacent layer during flow and is commonly expressed as thickness of the fluid. The more the thickness, the more viscous is the fluid and so greater the resistance to flow (Klabunde, 2011). The blood is composed of erythrocytes (red blood cells), leukocytes (white blood cells) and platelets suspended in the plasma. The properties of these components such as haematocrit levels, plasma viscosity, and the deformability and aggregation of red blood cells (RBCs) determine

the blood viscosity (Kwaan, 2010, Byrnes and Wolberg, 2017; Connes et al., 2019).

#### 3.2.3.1 Haematocrit

The haematocrit measures the volume of red blood cells compared to the total blood volume. It is expressed as percentage and is normally 40%-54% for men and 36%-44% for women (Billett, 1990). Increased haematocrit (policy thaemia) increases the blood viscosity and consequently the resistance to blood flow. Anaemic people have low haematocrits, therefore reduced blood viscosity (Klabunde, 2011).

#### 3.2.3.2 Plasma Viscosity

Plasma viscosity can alter only by changes in its electrolytes and plasma proteins (albumin, immunoglobulins and clotting factors). Among these, the clotting factor i.e., fibrinogen exert the major effect. In response to injury, it converts into thread-like protein fibrin and form a mesh where platelets and blood cells get trapped and form the clot (Smith et al., 2015). Since the plas ma is converting from fluid state to solid state, viscosity increases and blood flow reduces (Kwaan, 2010; Luyendyk et al., 2018). Also, during inflame matory condition, fibrinogen concentration may rise manifold leading to increased blood viscosity (Simmonds et al., 2013).

#### 3.2.3.3 RBC Deformability

RBCs have flexible membrane and can change from its biconcave discoid shape to ellipsoidal during flow. Such alterations lead to decreased blood viscosity and facilitate the microcirculation, that is, blood flow in the smaller blood vessels (such as capillaries, venules) where the resistance to flow is greater because of its smaller diameter. Any change in the membrane's deformability may restrict the blood flow to the tissues causing organ damage (Diez-Silva et al., 2010; Simmonds et al., 2013; Alizadeh and Dadvand, 2018).

#### 3.2.3.4 RBC Aggregation

RBCs in their biconcave discoid shape have a tendency to aggregate into a structure resembling stack of coins known as rouleaux. They tend to increase the frictional resistance and thus the blood viscosity (Simmonds et al., 2013; Lee et al., 2016). In normal condition, they disperse into individual RBC eventually after formation. However, the cell aggregation can be modified by the change in membrane properties of RBCs, increased concentration of plasma protein fibrinogen and high haematocrit level (Baskurt and Meiselman, 2003; Mehri et al., 2018).

### 3.2.4 Blood Pressure and Compliance

As blood travels through the blood vessel, it exerts a force on the walls of vessels which is termed as blood pressure. It is expressed as the ratio of systolic blood pressure (SBP) over diastolic blood pressure (DBP), where systolic pressure reflects the arterial pressure during ventricular contraction (systole) and diastolic pressure as the arterial pressure during ventricular

relaxation (diastole). The systolic pressure is higher than the diastolic pressure and is typically 120/80 mmHg in a normal adult. Pressures higher than this may indicate hypertension, while lower pressures may indicate hypotension. It is usually measured over brachial artery using sphygmo manometer and a stethoscope (OpenStax, 2019a). The blood pressure basically reflects the interaction between the force generated by the pumping heart and the resistance of the blood vessel walls. It increases with increase in cardiac output, peripheral resistance or both (Mayet and Hughes, 2003).

The average pressure of blood in the arteries can be calculated as:

$$\textbf{Mean Arterial Pressure (MAP)} = \text{diastolic BP} + \frac{(\text{systolic BP} - \text{diastolic BP})}{3}$$

where, the difference between the systolic pressure and the diastolic pressure is known as pulse pressure. MAP normally range from 70–110 mmHg. If the value falls below this for an extended time, the pressure will not be high enough to ensure circulation. This will lead to ischemia (insufficient blood flow) accompanied by hypoxemia (insufficient supply of oxygen to tissues) (Klabunde 2017; OpenStax 2019a).

Mean blood pressure decreases as blood moves away from the heart through arteries, capillaries, and veins (Figure 6). Although individual capillary has narrower diameter than that of arteriole, they have low blood pressure. This drop is because of the increased cross-sectional area of capillary bed, that is, there are more capillaries in the body than any other types of blood vessel (Hu et al., 2013; OpenStax, 2019a). While in veins, the low blood pressure is due to the flow of against the gravity (SEER, 2019). Concurrently, the rate of blood flow decreases dramatically from arteries to capillaries. Slow blood flow in capillaries allows more time for exchange of oxygen and nutrients from blood into tissue and waste products from tissue into blood.

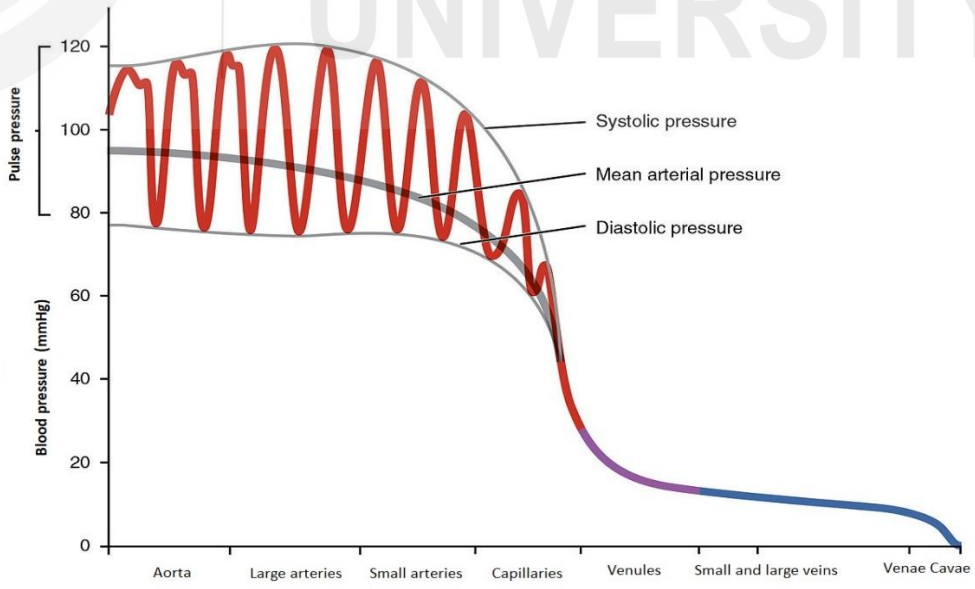



**Figure 6: Blood pressure in blood vessels**

***Source:*** https://opentextbc.ca/anatomyandphysiology/chapter/20-2-blood-flow-blood-pressure-and-resistance/

When blood passes through the capillary beds to the venules, veins, and main venae cavae (inferior and superior), the flow increases but is still much slower than that in aorta. The one-way valves in veins prevent blood to fall backward due to gravity (OpenStax, 2019a).

This blood flow is regulated by vasoconstriction and vasodilation of the blood vessels; particularly arteries, arterioles and veins. These modulate resistance and blood pressure within the vessels leading to increased or decreased blood flow (Figure 7).

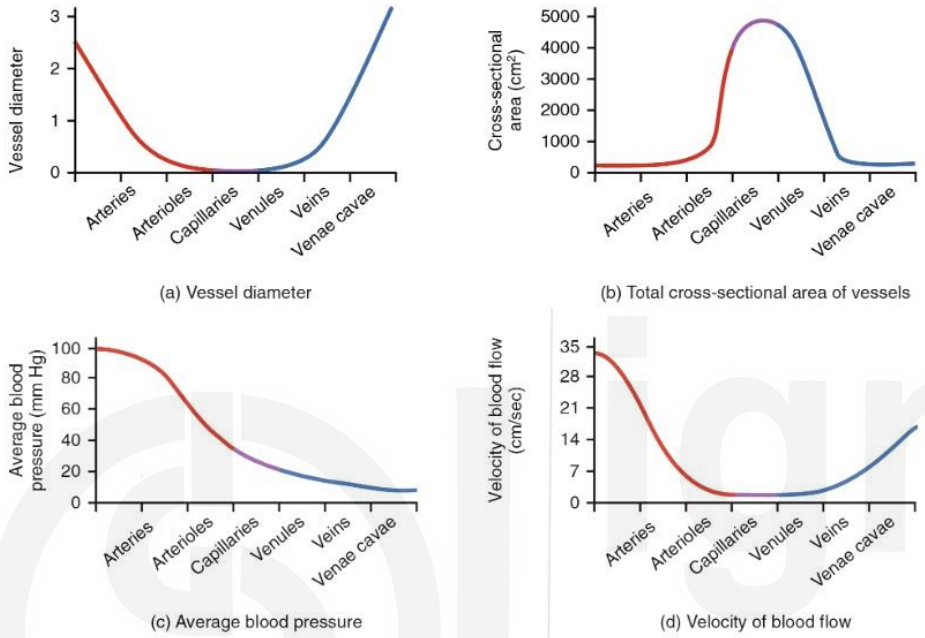



**Figure 7: A comparative relation of vessel diameter, total cross-sectional area, average blood pressure and velocity of blood flow in the blood vessels**

***Source:*** https://opentextbc.ca/anatomyandphysiology/chapter/20-2-blood-flow-blood-pressure-and-resistance/

Vasoconstriction is the narrowing of blood vessels caused by contraction of smooth muscle cells within the vessel wall. When blood vessel constricts, vascular resistance increases leading to increased blood pressure and decreased blood flow. While vasodilation is widening of blood vessels caused by relaxation of smooth muscle cells, dilation of blood vessels leads to decreased vascular resistance, decreased blood pressure and thus increased blood flow. This rhythmic contraction and relaxation generate pressure gradient and allow blood to flow in one direction (high to low pressure). Vasodilation occurs during cardiac systole while vasoconstriction during cardiac diastole (Siddiqui, 2011; OpenStax, 2019a; MedicineNet, 2019). In veins, the blood flow is maintained not only by rhythmic contraction and relaxation of vessels but also by the action of skeletal muscles as the body moves. That's why it is important to move frequently after long periods of sitting so that blood doesn't pool in the extremities (OpenStax, 2019a).

The ability of blood vessels to expand in response to increased pressure and accommodate blood pumped from the heart is called compliance. The arteries are low compliance vessels as their wall is made of thick layer of smooth muscle and it recoils back after expansion. While veins are high compliance

vessels as their wall has thin layer of smooth muscle, therefore, at a given pressure, arteries accommodate less volume of blood than the veins. In arteriosclerosis, compliance is reduced, and pressure and resistance within the vessel increase (Tozzi et al., 2000, Kopot, 2019).

**Check Your Progress 3**

3) Describe the role of vasoconstriction and vasorelaxation in blood flow.

…….

…….

…….

…….

…….

### 3.2.5 Pulse rate

The pressure wave generated by contraction of ventricle i.e., systole travels through the artery and is maintained through its expansion and recoiling. This pressure wave is known as pulse and can be palpated manually by placing and lightly pressing the tips of the fingers over an artery close to body surface, such as at the neck (carotid artery), wrist (radial artery), behind the knee (popliteal artery), the groin (femoral artery), near the ankle joint (posterior tibial artery), and on foot (dorsalispedis artery) (OpenStax, 2019a).

Clinically, both pulse rate (number of pulsations per minute) and pulse strength are important. Elevated (tachycardia) or educed pulse rate (bradycardia) may be due to temporary physiological condition or stress but also indicate a heart condition. The pulse strength indicates the strength of ventricular contraction and cardiac output. A strong pulse reflects high systolic pressure while a weak pulse means low systolic pressure. In such situation, a person might need medical intervention (OpenStax, 2019a, Physiopedia.com).

Measure of pulse rate is considered equivalent to heart rate but it can be a crude and inaccurate measurement when cardiac output is low. Low cardiac output causes minor change in the pressure and so in the pulse. Heart rate can be considerably higher than pulse rate (OpenStax, 2019a).

## 3.3 CARDIAC OUTPUT AND ITS DETERMINANTS

Cardiac output (CO) refers to the amount of blood pumped by the heart per minute. It is determined by multiplication of heart rate (HR), which is the number of times heart beats per minute and stroke volume (SV), which is the amount of blood pumped per beat (Malanga, 2007; Mohrman and Heller, 2018).

**Cardiac output (CO) = Heart rate (HR) x Stroke volume (SV)**

In an adult standing in resting position, the ventricles contain approximately 130ml blood by the end of atrial systole (i.e., after closure of the AV valves and before ventricular contraction) and is called end diastolic volume (EDV). On contraction, the ventricles eject 70ml blood into circulation which means 60 ml blood still remains in the ventricles (Klabunde 2011; OpenStax 2019a). This volume is known as end systolic volume (ESV). So, the volume of blood pumped with each heart beat i.e., stroke volume will be:

**Stroke Volume (SV) = End diastolic volume (EDV)-End systolic volume (ESV)**

These relationships implicate that the changes in HR and SV influence cardiac output.

### 3.3.1 Stroke Volume

Stroke volume varies and depends on how much cardiac muscle fibre will be able to shorten while working against arterial pressure. It is determined by the interplay of the following factors that affect the resting length and contractile property of these cardiac muscle fibers:

#### 3.3.1.1 Preload

According to Otto Frank (1895), the strength of ventricular contraction depends on the length or stretch of cardiac muscles prior to the contraction. Greater the stretch, greater is the force of contraction. The degree of stretch at the end of diastole (prior to contraction) is called preload. This observation was further investigated by Ernest Starling and co-workers, who found that the increased ventricular filling i.e., end diastolic volume, increases the pressure on ventricle muscles and so the preload, causing increased force of contraction. Increased force of contraction expels more blood into circulation which reflects in an increase in stroke volume and thus, cardiac output. Conversely, stroke volume will be reduced if ventricular filling falls. These concepts have been combined into the Frank–Starling law which indicates that the increased volume of blood in the ventricle (the end diastolic volume) results in increased stroke volume (Malanga, 2007; Vincent, 2008; Klabunde, 2011; Feher, 2012; Widmaier et al., 2016).

#### 3.3.1.2 Contractility

Contractility is the inherent ability of cardiac muscles to increase force of contraction (Ranek et al., 2017). The stretching of the cardiac muscle during diastole stimulates the release of calcium as well as sensitivity of troponin for binding calcium in the muscle fibres. It results into greater number of actin-myosin cross-bridges within the muscle fibres and so increased force of contraction (Klabunde, 2005; Widmaier et al., 2016; Martini, 2018) ( Figure 8). However, if muscle fibres are stretched beyond an optimal point, there will be insufficient cross-bridges and the muscle fibres will no longer be able to efficiently generate force (Martini et al., 2018; Boron and Boulpeap, 2009).

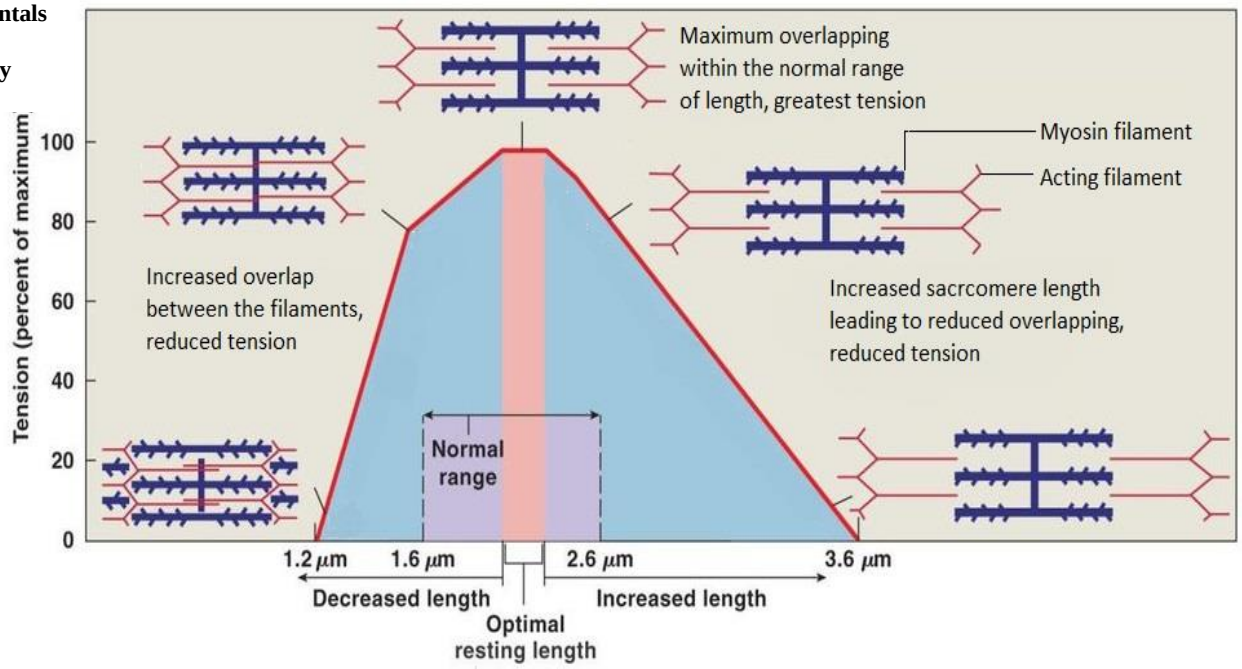



**Figure 8: Length-tension relation of muscle**

***Source:*** http://faculty.pasadena.edu/dkwon/chapt_11/images/image78.png

In addition to the intrinsic Frank–Starling mechanism, cardiac contractility is influenced by activation of sympathetic nervous system ("fight-or-flight" response). It releases noradrenaline which increases the heart rate (HR) and the force of contraction independent of the change in muscle fibre length or ventricular filling (EDV). The change in cardiac contractility independent of changes in muscle fibre length has been referred as inotropy. It results in a lower ESV at the end of contraction and consequently, an enhanced stroke volume at the same preload (Malanga, 2007; Kumar et al., 2019). Cardiac muscle contractility is usually expressed as ejection fraction which is, the ratio of amount of blood pumped out (SV) to amount of blood in ventricles at the end of diastole (EDV). An increase in cardiac contractility at a given EDV increases SV and so the ejection fraction (Klabunde, 2011; Kumar et al., 2019).

**Ejection fraction = Stroke (SV)/ End diastolic volume (EDV)**

### 3.3.1.3 Afterload

Afterload is the amount of tension or force ventricles must develop against the resistance in the systemic or pulmonary vasculature to pump blood into the circulation during contraction (Klabunde, 2011; OpenStax, 2019a).

As afterload increases, heart takes longer period to develop pressure enough to force open the semilunar valves and correspondingly shorter period to eject blood. Consequently, less volume of blood is pumped out of the ventricles with each cardiac cycle, and EDV increases. The increased EDV leads to greater preload to enable the ventricles to contract with greater force (Frank-Starling mechanism) and there is increase in stroke volume and cardiac output (Klabunde, 2011; Malanga, 2007; O'Keefe and Singh, 2019).

Therefore, in a normal heart, increased afterload may not have major effect on stroke volume but, in patients with heart failure, increased afterload causes fall in stroke volume (Sidebotham, 2007; O'Keefe and Singh, 2019).

### 3.3.2 Heart Rate

Initially an increase in heart rate leads to an increase in SV. However, as it continues to rise, there is less time for diastole i.e., ventricular filling. Low ventricular filling means decreased SV. CO will initially remain stable as the increasing HR compensates for the decreasing SV. But at very high rates, CO eventually decreases as increased heart rates are no longer able to compensate for the decreasing SV (Vincent, 2008; King and Lowrey, 2019, OpenStax, 2019a).

**Check Your Progress 4**

4) Specify the factors affecting amount of blood pumped by the heart per minute.

……

……

……

……

……

## 3.4 OXYGEN TRANSPORTATION

### 3.4.1 Exchange of Gases

The deoxygenated blood from the right ventricle is pumped into lungs through pulmonary artery. In lungs, it branches and eventually forms capillary network around the alveoli where exchange of gases ($O_2$ and $CO_2$) takes place. The oxygenated blood then returns to heart through pulmonary vein and is pumped into body. Thus, gas exchange occurs at two sites: in the lungs, where oxygen is picked up and carbon dioxide is released, and at the tissues, where oxygen is released and carbon dioxide is picked up. It is mediated by the difference in the partial pressure of gases across alveoli, capillaries and tissues (West, 2008, Mader, 2018).

In lungs, the partial pressure of oxygen ($Po_2$) is high in the alveoli (105 mmHg) and low (40 mmHg) in the blood of the pulmonary capillaries. In contrast, the partial pressure of carbon dioxide ($Pco_2$) is high in the blood of pulmonary capillaries (45 mmHg) and low (40 mmHg) in alveoli. This pressure gradient causes diffusion of $O_2$ from the alveoli into the blood and $CO_2$ from blood into alveoli. Similarly, the pressure gradient at tissue level, provide oxygen to the tissues ($Po_2$) and eliminate carbon dioxide. The partial pressure gradient around tissue is opposite to that in lungs – $Po_2$ is high in blood (100 mmHg) and low in tissues (40 mmHg) while $Pco_2$ is high in tissue (45 mmHg) and low in blood (40 mmHg) (Pittman, 2011; OpenStax, 2019b)

## 3.4.2 Oxygen-Haemoglobin Dissociation Curve

Oxygen is majorly transported to the tissues by haemoglobin protein found in the erythrocytes (RBCs). It is made of four subunits, each composed of an iron-containing *heme* group and a globin polypeptide chain. Each *heme* group bounds to an oxygen molecule, allowing a haemoglobin molecule to carry up to four molecules of oxygen. Together they form the oxy-haemoglobin ($Hb–O_2$)– a bright red coloured molecules which gives bright red colour to oxygenated blood (Marengo-Rowe, 2006; Collins et al., 2015; Rhodes and Varacallo, 2019).

Binding of the first oxygen molecule causes a conformational change in haemoglobin which facilitates binding of other molecules readily. Similarly, when first oxygen molecule dissociates (in tissues), the other molecules dissociate readily. Haemoglobin is said to be saturated when all four *heme* sites are bound with oxygen, and partially saturated if only one to three *heme* sites are bound. The percent of the heme units bound to oxygen at a given time is called haemoglobin saturation and generally range from 95 to 99 percent in an individual with normal haemoglobin level (Figure 9) (Collins et al., 2015; Rhodes and Varacallo, 2019; OpenStax 2019b).

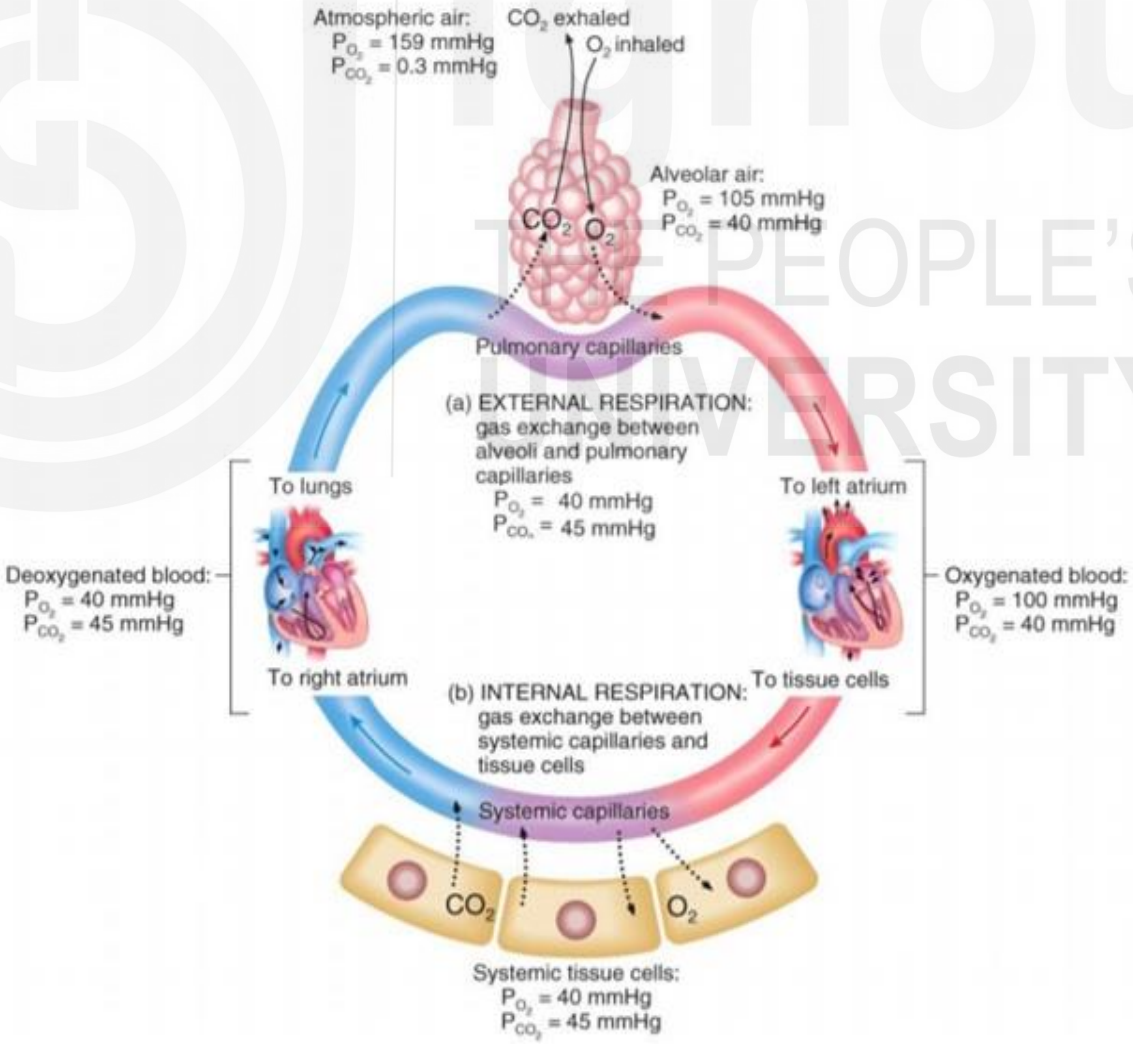



**Figure 9: Exchange of gases in lungs and tissues**

***Source:*** http://www.mc3cb.com/pdf_ap_lectures/C23_2_gas_exchange_transport.pdf

The association and dissociation of oxygen from haemoglobin depends on the $Po_2$. In pulmonary capillaries, the $Po_2$ is high and so oxygen binds with the haemoglobin. While in tissue capillaries, the $Po_2$ is low and oxygen is released from the haemoglobin. This relationship is represented by a sigmoid shaped oxygen-haemoglobin dissociation curve where percentage of haemoglobin saturation (percentage of haemoglobin bound with oxygen) is plotted against varying levels of $Po_2$ (Figure 10).

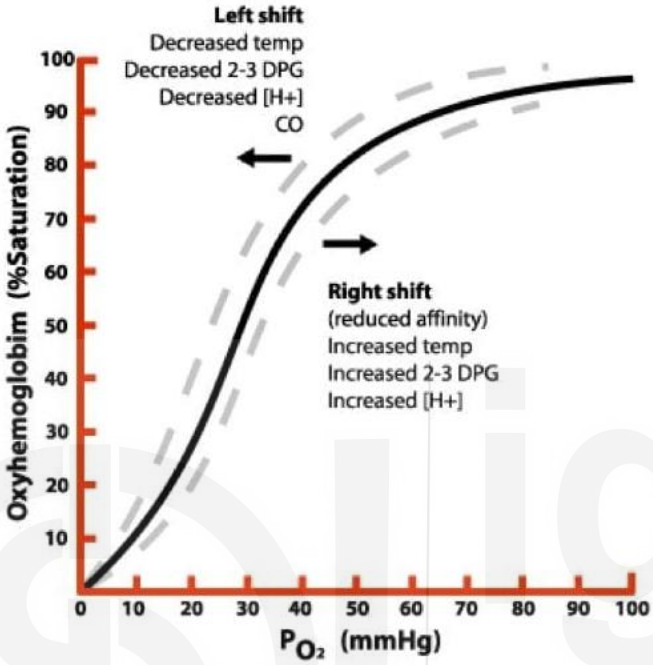



**Figure 10: Oxygen–haemoglobin dissociation curve**

***Source***: https://litfl.com/oxygen-haemoglobin-dissociation-curve/

It illustrates progressive increase in haemoglobin saturation with the increase in $Po_2$. The steep lower portion of the dissociation curve shows that the peripheral tissues can withdraw large amounts of oxygen at low $Po_2$. While the flat upper portion represent plateau in oxygen saturation in spite of an increasing partial pressure of oxygen in the blood. It signifies that even if alveolar gas $Po_2$ falls to some degree, oxygen saturation will be less affected (Collins et al., 2015, OpenStax, 2019b).

The oxygen–haemoglobin dissociation curve can shift either to the left or right. It reflects the change in affinity of haemoglobin for oxygen. A rightward shift illustrates decreased affinity of haemoglobin for oxygen and may results from an increase in body temperature, increase in $CO_2$ concentration, increase in 2,3-bisphosphoglycerate and decrease in pH (Kaufman and Dhamoon, 2019; Rhodes and Varacallo, 2019). Also, diseases like sickle cell anaemia and thalassemia affect oxygen-carrying or delivering capacity of erythrocytes. In sickle cell anaemia, the red blood cell become stiff crescent shaped and is unable to pass through the capillaries reducing its ability to deliver oxygen, while thalassemia is an inherited genetic disease characterised by low haemoglobin level due to defect in its structure. This condition affects the oxygen carrying capacity of erythrocytes (Rhodes and Varacallo, 2019).

**Check Your Progress 5**

5) Define oxygen-haemoglobin dissociation curve.

……

……

……

……

……

6) Ventricular contraction when no blood is ejected into circulation is termed as …….. .

7) The force heart must overcome to pump blood into pulmonary artery and aorta is

   a) Cardiac output

   b) Stroke volume

   c) Afterload

   d) Preload

8) Which of the following statement(s) is true?

   a) The wider the blood vessel, the higher the resistance and slower the blood flow.

   b) Closing of atrioventricular valves produce second heart sound ‘dub’.

   c) Haemoglobin affinity to bound with oxygen increase when $Po_2$ is high.

   d) Thick layer of smooth muscles around arteries makes them more compliant

## 3.5 SUMMARY

A cardiac cycle begins with the passive flow of blood into atria and then into ventricles through arterio-ventricular valves. The impulse from sinoatrial node causes contraction of atria and completes emptying of atria into ventricles. In a fraction of time, the impulse initiates contraction of ventricles and the pressure within ventricles begins to rise. When the pressure reaches above that of atria, atrioventricular valves close and produce first heart sound ‘lub’. When the ventricular pressure rises above the pressure in pulmonary artery and aorta, semilunar valves force open and blood is pumped out for circulation in the body. Eventually ventricular pressure begins to fall and semilunar valves close to prevent back flow of blood and produce second heart sound ‘dub’. Once again, the cardiac cycle begins with filling of blood into ventricles from atria.

In blood vessels, the blood flow is determined by the vessel diameter, blood viscosity and vessel length. An increase in vessel diameter reduces the resistance and increases the blood flow. While the more viscous the blood, the higher the resistance and lower the blood flow. RBC characteristics such as deformability and aggregation also influence the blood flow in the vessels. Vasoconstriction and vasodilation modulated the resistance and blood pressure within the vessels leading to increased or decreased blood flow.

The cardiac output is determined by several factors including preload, contractility of cardiac muscle, afterload and heart rate. Higher the preload, greater will be the force of contraction. While at a given preload, high contractility results in a lower ESV at the end of contraction and consequently, an enhanced stroke volume. An increase in afterload causes heart to pump less volume of blood from ventricles during each cardiac cycle and increases EDV. Consequently, it might not have major effect on stroke volume in a normal heart but stroke volume falls in an individual with cardiac problem.

Oxygen is transported bound to haemoglobin protein found in erythrocytes. Its structure allows attaching four molecules of oxygen. The association and dissociation of oxygen from haemoglobin depends on the partial pressure of oxygen and the relation can be illustrated by oxyhaemoglobin dissociation curve. The curve may shift to right or left depending on the body temperature, partial pressure (concentration) of $CO_2$, 2,3-bisphosphoglycerate and pH.

## 3.6 REFERENCES


Ahmad, M. (2018). *Cardiac cycle, phases of cardiac cycle, cardiac cycle & ECG*. Medicosite.com. Retrieved from: https://www.medicosite.com/cardiac-cycle/

Alizadeh, A., & Dadvand, A.R. (2018). Effects of deformability of RBCs on their dynamics and blood flow passing through a stenosed microvessel: An immersed boundary-lattice Boltzmann approach. *Theoretical and Computational Fluid Dynamics, Vol. 32(*1), pp. 91-107

Baskurt, O.K., Meiselman, H.J. (2003 Oct.) Blood rheology and hemodynamics. *Semin Thromb Hemos*t. *29(5),* 435-50

Baura, G.D. (2012). Electrocardiograph. *Medical device technologies - A systems based overview using engineering standards*. Academic Press, pp. 39-57. Retrieved from https://doi.org/10.1016/B978-0-12-374976-5.00002-5

Billett, H.H. (1990). Hemoglobin and hematocrit. *Clinical methods: The history, physical, and laboratory examinations*. (3rd ed.). Boston: Butterworths.

Boron, W.F., & Bouleap, E.l. (2009). The cardiovascular system. *Medical physiology – A cellular and molecular approach*. (2nd ed., pp. 427-593). Saunders Elsevier.

Byrnes, J.R., & Wolberg, A.S. (2017). Red blood cells in thrombosis. *Blood, Vol.130* (16), pp. 1795-1799

Collins, J.A., Rudenski, A., Gibson, J., Howard, L., & O'Driscoll, R. (2015). Relating oxygen partial pressure, saturation and content: the haemoglobin–Oxygen dissociation curve. *Breathe, Vol. 11(3),* pp. 194-201

Connes, P., Dufour, S., Pichon, A., & Favret, F. (2019). Blood rheology, blood flow, and human health. *Nutrition and enhanced sports performance-Muscle building, endurance, and strength.* (2nd ed., pp. 359-369). Academic Press.

Diez-Silva, M., Dao, M., Han, J., Chwee-Teck, L., & Subra S. (2010). Shape and biomechanical characteristics of human red blood cells in health and disease. *MRS Bulletin, Vol. 35(*5), pp. 382-388

Faber, J.E., & Stouffer, G.A. (2017). Introduction to basic hemodynamic principles. *Cardiovascular hemodynamics for the clinician.* (2nd ed., pp. 3-9). Wiley Online Library.

Feher, J. (2012). The cardiac function curve. *Quantitative human physiology-An introduction*. (2nd ed., pp. 486-564). Academic Press.

Frank, O. (1895). Zur Dynamik des herzmuskels Zeitschrift für Biologie. 33(3), 370-447. Translated version: Chapman, C.B and Wasserman, E (1959). On the dynamics of cardiac muscle. *American Heart Journal, 58*(2), 282-317.

Hall, J.E., & Guyton, A.C. (2011). *Textbook of medical physiology*. (12th ed., pp 101-118). Philadelphia: Saunders Elsevier.

Hu, Y., Song, S., & Hu, Yanxia. (2013). Flow resistance of vessels with an enlarged total cross-sectional area in the midsection. *Open Circulation & Vascular Journal, Vol. 6*, pp. 9-12

Kaufman, D.P., & Dhamoon, A.S. (2019). *Physiology, oxyhemoglobin dissociation curve.* StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing. Retrieved from https://www.ncbi.nlm.nih.gov/books/NBK499818/

King, J., & Lowery, D.R. (2019). *Physiology, cardiac output.* StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing. https://www.ncbi.nlm.nih.gov/books/NBK470455/

Klabunde, R.E., (2011). *Cardiovascular physiology concepts*. (2nd ed., pp. 60-123). Lippincott Williams & Wilkins.

Kopot, A. (2019 May 2). Compliance of blood vessels. *AK lectures*. Retrieved from http://www.aklectures.com/lecture/compliance-of-blood-vessels

Kumar, K.R., Kirsch, R.E., & Hornik, C.P. (2019). Cardiovascular physiology for intensivists. *Critical heart disease in infants and children*. (3rd ed., pp. 111-133.e5). Elsevier.

Kwaan, H.C. (2010). Role of plasma proteins in whole blood viscosity: A brief clinical review. *Clinical Hemorheology and Microcirculation, Vol. 44*(3), pp. 167-176

Laizzo, P.A. (2005). General features of the cardiovascular system. *The handbook of cardiac anatomy, physiology and devices* (pp. 3-12). New Jersey: Humana Press.

Lee et al. (2016). Optical tweezers study of red blood cell aggregation and disaggregation in plasma and protein solutions. *Journal of Biomedical Optics, Vol. 21*(3), pp. 35001

Luyendyk, J.P., Schoenecker, J.G., & Flick, M.J. (2018). The multifaceted role of fibrinogen in tissue injury and inflammation. *Blood, Vol. 134*(4), pp. 11-39

Mader, S.S. (2018). *Human Biology*. (15th ed., pp. 197-217). McGraw Hill.

Malanga, C.J. (2007). Structure and function of the heart. *xPharm: The com prehensive pharmacology reference*. Elsevier, pp 1-5. Retrieved from https:// doi.org/10.1016/B978-008055232-3.60302-2

Marengo-Rowe, A.J. (2006). Structure-function relations of human hemog lobins. *Proceedings* (Baylor University. Medical Center), *Vol. 19(3)*, pp. 239-45

Martini, F.H., Nath, J.L., & Bartholomew, E.F. (2018). Muscle tissue. *Fundamentals of anatomy and physiology* (11th ed., pp. 337-378). Pearson.

Mayet, J., & Hughes, A. (2003). Cardiac and vascular pathophysiology in hypertension. *Heart, Vol. 89*(9), pp. 1104–1109

MedicineNet.com. *Definition of vasodilation*. (2019 May 2). Retrieved from https://www.medicinenet.com/script/main/art.asp?articlekey=5965

Mehri, R., Mavriplis, C., & Fenech, M. (2018). Red blood cell aggregates and their effect on non-Newtonian blood viscosity at low hematocrit in a two-fluid low shear rate microfluidic system. *PLoS ONE, Vol. 13*(7), pp. e0199911

Mohrman, D.E., & Heller, L.J. (2018). *Cardiovascular physiology*. McGraw-Hill Medical.

Monahan-Earley, R., Dvorak, A.M., & Aird, W.C. (2013). Evolutionary origins of the blood vascular system and endothelium. *Journal of Thrombosis and Haemostasis, Vol. 11* (1), pp. 46–66

O'Keefe, E., & Singh, P. (2019). *Physiology, cardiac preload*. StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing. Retrieved from https://www.ncbi.nlm.nih.gov/books/NBK541109/

OpenStax textbook. (2019a). The cardiovascular system. *Anatomy and physiology*. BC campus, pp. 124-136. Retrieved from https://opentextbc.ca/ anatomyandphysiology/front-matter/preface-2/

(2019b). The respiratory system: Gas exchange. *Anatomy and physiology*. BC campus, pp. 149. Retrieved from https:/ /opentextbc.ca/ anatomy and physiology / chapter/22-4-gas-exchange/

Ostadfar, A. (2016). Biofluid dynamics in human organs. *Biofluid mechanics - principles and applications*. Academic Press, pp. 111-204. Retrieved from https://doi.org/10.1016/B978-0-12-802408-9.00004-1

Physiopedia. (2019 May 14). *Pulse rate*. Retrieved from https://www.physiopedia.com/index.php?title=Pulse_rate&oldid=207012

Pittman, R.N. (2011). *Regulation of tissue oxygenation*. San Rafael (CA): Morgan & Claypool Life Sciences. Retrieved from https://www.ncbi.nlm.nih.gov books/NBK54103/

Ranek, M.J., Vu, A., Willis, M., & Lymperopoulos, A. (2017). Neuronal hormones and the sympathetic/ parasympathetic regulation of the heart. *Endocrinology of the heart in health and disease - Integrated, cellular, and molecular endocrinology of the heart* (pp. 207-227). Academic Press.

Rhodes, C.E., & Varacallo, M. (2019). *Physiology, oxygen transport. StatPearls [Internet]*. Treasure Island (FL): StatPearls Publishing. Retrieved from https://www.ncbi.nlm.nih.gov/books/NBK538336/

Rubenstein, D.A., Yin, W., & Frame, M.D. (2015). The heart. *Biofluid mechanics - An introduction to fluid mechanics, macrocirculation, and microcirculation*. (2nd ed.). Academic press, pp. 123-160. Retrieved from https://doi.org/10.1016/B978-0-12-800944-4.00004-4

Secomb, T.W. (2016). Hemodynamics. *Comprehensive Physiology, Vol. 6*(2), pp. 975–1003

SEER Training Modules. (2019 March 9). Cardiovascular system: Physiology of circulation. *Module Anatomy & Physiology*. U. S. National Institutes of Health, National Cancer Institute. Retrieved from: https:// training.seer.cancer.gov/anatomy/cardiovascular/heart/physiology.html

Siddiqui, A. (2011). Effects of vasodilation and arterial resistance on cardiac output. *Journal of Clinical and Experimental Cardiology*, *Vol. 2*, pp. 170. doi: 10.4172/2155-9880.1000170

Sidebotham, D., & Le Grice, I.J. (2007). Physiology and pathophysiology. *Cardiothoracic critical care* (pp. 3-27). Butterworth Heineman.

Simmonds, M.J., Meiselman, H.J. & Baskurt, O.K. (2013). Blood rheology and aging. *Journal of Geriatric Cardiology, Vol. 10*(3), pp. 291-301

Smith, S.A., Travers, R.J., & Morrissey, J.H. (2015). How it all starts: Initiation of the clotting cascade. *Critical Reviews in Biochemistry and Molecular Biology, Vol. 50*(4), pp. 326–336

Tozzi, P., Corno, A., & Hayoz, D. (2000). Definition of arterial compliance. *American Journal of Physiology, Vol. 278(*4), pp H1407

Vincent, J.L. (2008). Understanding cardiac output. *Critical Care, Vol. 12*(4), pp. 174

Voorhees, A.P., & Han, H.C. (2015). Biomechanics of cardiac function. *Comprehensive Physiology, Vol. 5*(4), pp. 1623–1644

West, J.B. (2008). *Respiratory physiology- The essentials.* (8th ed., pp. 12-24). Lippincott Williams & Wilkins.

Widmaier, E.P., Raff, H., & Strang, K.T. (2016). *Vander's human physiology: The mechanisms of body function.* (14th ed.). New York, NY: McGraw-Hill Education.

## 3.7 HINTS/ANSWERS TO CHECK YOUR PROGRESS

1) The atria and ventricles of the heart undergo a sequence of alternating contraction (systole) and relaxation (diastole) to circulate blood throughout the body. Atrial contraction marks the beginning of a cardiac cycle and it ends with ventricular relaxation.

2) Resistance to the blood flow is determined by three factors: blood viscosity, vessel radius, and vessel length. The greater the resistance, the lower the flow.

3) Vasoconstriction i.e., narrowing of the blood vessels increases the vascular resistance and decreases the blood flow. While vasodilation i.e., widening of the blood vessels decreases the vascular resistance and increases the blood flow. The rhythmic vasoconstriction and vasodilation maintain the blood flow in the blood vessels.

4) The changes in cardiac output (amount of blood pumped by the heart per minute) are majorly determined by the stroke volume and heart rate. Though their relationship is direct, it is affected by many other factors - venous return (preload), cardiac contractibility, arterial blood pressure (afterload) and ventricular filling.

5) The oxygen-haemoglobin dissociation curve is a graphic expression of the relationship between haemoglobin saturation (percentage of haemoglobin bound with oxygen) and partial pressure of oxygen.

6) iso-volumetric contraction

7) c) Afterload

8) c) Haemoglobin affinity to bound with oxygen increases when $Po_2$ is high.

ignou
THE PEOPLE'S
UNIVERSITY