Introduction and Principle:

The total volume of air in the lungs at full (maximal) inspiration is conventionally divided into four volumes. Each of these, except residual volume, can be measured directly with a spirometer. These volumes are the tidal volume (TV), inspiratory reserve volume (IRV), expiratory reserve volume (ERV), and residual volume (RV).

Respiratory physiology is quantitative in nature, in clinical practice certain combinations of volumes called "capacities" are more convenient to measure and interpret. The ones most used are.

Inspiratory capacity (IC = TV+IRV): The maximal volume that can be inspired following a normal expiration.
**Vital capacity** (VC=ERV+TV+IRV): The maximal volume of air that can be expired following a maximal inspiration (i.e. the largest possible breath you can make). It is a useful single test of the ability to inflate and deflate the lungs.

**Functional residual capacity** (FRC=RV+ERV): The volume of air remains in the lungs at the end of normal expiration.

**Total lung capacity** (TLC=RV+ERV+TV+IRV = FRC+IC): The volume in the lungs at the end of maximal inspiration.

**Tidal volume:** the volume of air that enters and go out of the lungs per breath at rest.

**Note:** Because FRC and TLC include RV, they cannot be measured directly, and the RV, must be estimated by a dilution method.

**Forced Vital Capacity** (FVC):

The maximum volume of air that can be forcefully expired following maximum inspiration.

In this test, the subject breaths-out his/her vital capacity as fast and forceful as possible. The entire expiration is recorded and the speed, duration and overall shape of the breath curve are examined.

**Objectives:**

Upon completion of this session the attendees will acquire:

1. The knowledge how instruct the patient to do lung function test.
2. Indicate changes in lung functions.
3. Interpret these changes.
4. Diagnose obstructive and restrictive lung diseases.
5. Asses the physical fitness of the subject.
**Instruments and Subjects:**

Spirometer.

A normal subject.

**Procedure:**

1. Fit the nose clip while the subject is in the standing position.
2. Instruct the subject to inspire as deeply as possible, then to expire as rapidly and forcefully as possible into the spirometer through the mouth piece until the end of expiration.

**Results:**

1. Measurement of the FVC: This value may directly be read-off from the top of the curve at the point where the curve finally levels-off on the Y-axis.

   **Normal values** 4-6L in young adult male.
   
   4.2 L in young adult female.

2. Measurement of \( FEV_1 \): This can be done by drawing a vertical line from the one second graduation to the point where it intersects the curve, then drawing a horizontal line between the point of intersection and the volume graduation.

3. Measurement of the \( FEV_1 \)% = \( FEV_1/FVC \times 100 \)

   Normally \( FEV_1 \)% should be greater than 75%.
spirometer.
Lab.7:

**Mouth-to-Mouth Respiration**

**Introduction and Principle:**

Artificial Respiration (Pulmonary Resuscitation)

Artificial respiration (AR; assisted ventilation) may be given by manual methods, mouth-to-mouth method (sometimes called “kiss of life” or “rescue breath”), or by mechanical methods.

**Manual Methods**

The old prone or supine position methods are no longer employed since they rely on compressing the thorax to cause expiration, and then allowing lungs to expand passively. The Holger-Nielson method described below is used when mouth-to-mouth respiration is not possible.
A. **Holger-Nielson Method (Back-pressure arm-lift, BPAL method)**

1. Place the victim, face downwards, on a hard surface, with the arms bent, and the head turned to one side and resting on the hands.
2. Kneel down on one knee at the victim’s head, with the opposite foot placed near the elbow.
3. Place your hands, with fingers widespread, on the victim’s back just below the scapulae. Now rock forward, with the arms held straight at the elbows, until your arms are vertical and pressing down on the back. This compresses the chest and produces expiration.
4. Slide your hands sideways and outwards on to the victim’s arms just above the elbows. Now rock backwards, lifting the victim’s elbows until some resistance is felt at her/his shoulders. This movement expands the thorax, decreasing the intrathoracic pressure and causing inspiration.
5. Repeat this cycle of compression and expansion (that lasts for about 3 seconds each) for about 12 times a minute.

![Fig(1): Holger-Nielson Method (Back-pressure arm-lift, BPAL method)](image)
B. **Mouth-to-Mouth Respiration (Rescue breath)**

Mouth-to-Mouth respiration has proved to be superior to all the manual methods in all age groups.

**Advantages and Disadvantages of mouth-to-mouth Respiration.**

The method is simple, safe, and easy to perform, even by a layman with minimum instruction. Above all, it does not require any apparatus. The only disadvantage is that the victim’s flaccid (toneless) tongue tends to fall back into the pharynx and thus obstruct the airway.

**PROCEDURES:**

1. Place the victim on his/her back on firm ground, and loosen the clothing around the neck, chest and waist.
2. Remove any mucus, food, saliva, or any foreign material from the mouth and nose with your fingers.
3. Open the airway by tilting the head back. Kneel by the right side of the victim. Place your right hand under the neck and lift it, while keeping a pressure on the forehead with the heel of the other hand. Using your right thumb and fingers, lift the chin and angle of the jaw upward and forward. This simple procedure keeps the airway open.
4. Clamp the nostrils with your left thumb and fingers, take a deep breath, apply your mouth firmly on the victim’s mouth, and blow a liter of air into the victim’s lungs, watching the expansion of the chest at the same time.
5. Remove your mouth, turn your head to one side and take another deep breath as the elastic recoil of the chest causes expiration. You may feel and hear the expiratory airflow from the victim’s mouth and nose.
6. Repeat the cycle of blowing out—turning the head—breathing in—about 14–16 times a minute.
7. Important. Feel the carotid pulse. If, after 6–8 lung inflations, there is no improvement in the color of the victim, suspect cardiac arrest, and start external cardiac massage as well.

**Fig(2): Mouth-to-Mouth Respiration**

**Mechanical Respirators**

Mechanical ventilation is employed when AR has to be given for long periods, e.g. during chronic respiratory failure.

1. Drinker’s Tank Respirator (also called the “iron lung”). It is an iron chamber in which the subject is placed, with the head kept outside, an air-tight collar sealing the body inside.
2. **Sahlin’s jacket model**, Brag Paul Pulsator, and their modifications employ inelastic chest jackets in which pressure can be increased and decreased at intervals.

![Photo of a patient using a Sahlin's jacket model.]

2. **Eve’s rocking method.** The victim is laid on a stretcher or a plank and the shoulders and ankles are fastened to it. A rhythmic rocking up and down like a see-saw causes the abdominal viscera to push up against the diaphragm (expiration) or pull it down (inspiration).

![Photo of a patient being岩石ed.]

*Good Luck*
Al-Noor University College  
Dept. of Anesthesiology  
Subject: Practical Applied Physiology  
Second Year  
Lecturer: Asmaa J. Al-lella  

Lab.8: Muscle and nerve  

**Introduction:-**

The **muscular system** is an organ system consisting of skeletal, smooth and cardiac muscles. It permits movement of the body, maintains posture and circulates blood throughout the body. The muscular systems in vertebrates are controlled through the nervous system. Together with the skeletal system, it forms the musculoskeletal system, which is responsible for movement of the human body.

It is convenient to consider the physiology of muscle and nerve together. Nerve fibers control the activity of skeletal muscle fibers. The functional division of skeletal muscle is the motor unit-a grouping of up to more than a hundred muscle fibers innervated by a single nerve fiber. In the
intact animal, skeletal muscle is excited via its nerve supply by pro-partition of the nerve impulse along the motor neuron to the specialized ending of the axon at the myoneural junction.

Here, acetylcholine is released, leading to a depolarization of the cell membrane. In the experimental situation, a muscle can be stimulated directly or indirectly. Such stimuli can be electrical, mechanical or chemical. Electrical stimulation will be used in the experiments because it can be conveniently applied and easily controlled.

The gastrocnemius muscle and the sartorius muscle of the frog have been favorite experimental objects in both research and teaching laboratories when the properties of muscle to be studied, especially the mechanical properties. Frog tissue has the practical advantage that it will function at room temperature without a blood supply; its oxygen requirements are met by diffusion from the air into the solution bathing the preparation.

Much of the physiological information about skeletal muscle activity has been obtained from experiments using a muscle nerve preparation connected to a recording instrument and a source of electrical stimulation.

**How does the neuromuscular system work?**

Nerves have cells called neurons. Neurons carry messages from the brain via the spinal cord. The neurons that carry these messages to the muscles are called motor neurons.

Each motor neuron ending sits very close to a muscle fibre. Where they sit together is called a neuromuscular junction. The motor neurons can release a chemical, which is picked up by the muscle fibre. This tells the muscle fibre to contract, which makes the muscles move.
Function of Muscle Tissue

1- The main function of the muscular system is movement.
2- maintenance of posture and body position.
3- Another function related to movement is the movement of substances inside the body. The cardiac and visceral muscles are primarily responsible for transporting substances like blood or food from one part of the body to another.
4- The final function of muscle tissue is the generation of body heat. As a result of the high metabolic rate of contracting muscle, our muscular system produces a great deal of waste heat.
Introduction and Principle:

Muscle contraction includes many chemical reactions that are affected by changing the temperature of the surrounding. High temperatures accelerate the reactions while low temperatures decelerate the reactions. This means that increasing the temperature around the muscle as well as the nerve fiber will no doubt increase the contractility of the muscle and conduction velocity of the nerve, whereas the reactions will go slower when reducing the temperature so muscle contraction and nerve conduction will be slower.

Objectives:

Upon completion of this session the attendees will:

1. Acquire the knowledge and skills of how muscle twitch is affected by different temperatures.
2. Make use of this knowledge in clinical practice.
Methods and materials:

The same as in experiment no. one.

Speed of the drum (speed five).

Stimulus intensity (threshold stimulus or greater).

Questions and Discussion:

1. What is the significance of this experiment?

   The experiment demonstrates the following facts.

   i- When a muscle is stimulated at a temperature higher than room temperature, this results in shortening of the latent period, contraction and relaxation phases. The contraction phase of the curve goes higher and becomes steeper.

   ii- When the muscle is stimulated at a temperature lower than room temperature, the effects will be exactly the opposite i.e. the latent period, contraction, and relaxation periods are all increased. The amplitude of the curve will be lower and less steep.

2. What are the causes of the above results?

   Increasing the temperature will enhance the metabolic reactions in the muscle and nerve fiber that in turn lead to:

   i- Acceleration of the conduction velocity of the impulses in the nerve fiber.

   ii- Activation of the chemical reactions in the muscle that make the contraction faster and steeper.

   iii- Decrease the resistance to sliding myofilaments (actin and myosin), probably due to a decrease in the viscosity of the muscle.

   All of the above mentioned effects are responsible for the shortened latent period, contraction, and relaxation periods.
In cool temperatures exactly the opposite effects take place that will increase the latent period, contraction and relaxation phases.

**Clinical Application:**

For a better performance of muscle contraction, this can be achieved with mild to moderate increase in temperature of the muscle surrounding, and fatigue will go more rapidly with higher temperatures.

1- The effects of cold are used to minimize energy expenditure by the stored tissues before transplantation. This experiment is now extended to whole human body, to keep it frozen for hundreds of years.

2- The hyperdynamic circulation as in fever (increased efficiency of cardiac muscle, circulation and blood pressure) is a good example of the effects of increased temperature.

![Effects of temperature on simple muscle twitch.](image-url)
Lab.9: Effect of Temperature on Simple Muscle Twitch

Introduction and Principle:

The motor control system is a complex system including many disperse areas at different levels of the central nervous system, brain, cerebellum, and spinal cord. Functions integrated at the level of the cerebrum are complex patterns of movement, those integrated at the level of the brain stem are less complex, and those at the level of the spinal cord are the simplest usually described as reflexes.

All voluntary motor functions begin as an idea, this idea is converted into a program the program is relayed to the primary motor cortex that in turn pass the commands to the executers (muscles). These movements can be of different intensities, in different directions, rapid, or slow…etc. All of these functions are controlled and integrated by certain areas in the brain. Therefore, examining the motor system in the form of testing for the power,
tone of the muscle, coordination of movements, as well as the reflexes is used to check for the integrity of these areas.

A reflex is an involuntary simple, jerky, and purposeless movement. Each reflex has its reflex arcs. This consist of a receptor, afferent (sensory) fiber, integrating center (spinal cord or higher in the brain). Efferent (motor or secretory) fiber, and an effector organ (muscle or a gland). Reflexes are always initiated by stimuli.

**Types of reflexes:**

- anatomically they can be classified as:
  1. superficial (cutaneous) reflexes
  2. deep (jerk, myotatic, stretch) reflexes, and eye reflexes.

  Physiologically they can be classified as:
  1. monosynaptic (stretch reflexes)
  2. polysynaptic (withdrawal reflexes and cross extensor reflexes).

**Examination of motor system**

- Inspection
- Palpation

**Objectives:**

Upon completion of this session the attendees will:

1. Acquire the knowledge and skills of how to examine the motor system.
2. Detect any abnormality in muscle power, tone, or coordination of motor activities as well as abnormal reflexes.
3. Indicate the significance of having abnormal motor activity.

**Material and Instruments:**

- Neurological hammer.
**Procedure:**

1. Examination of muscle power:

   This is done by asking the subject or patient to flex or extend the arm or leg or muscles of the shoulders or neck muscles against resistance offered by the examiner to indicate whether the power is normal, less than normal as in flaccid paralysis of poliomyelitis.

2. Examination of muscle tone:

   This is done by feeling the muscle between fingers to see whether it feels soft (normal) or very soft (flaccid) as in flaccid paralysis or tense (increased tone) as in Parkinson's disease.

3. Examination for coordination of movement:

   This is done by asking the patient to do some task as to point to the index finger of the examiner with his index finger then to put it back to his nose, or to walk on a straight line steadily.

4. Examining muscle stretch reflexes (knee jerk, ankle jerk Fig (1), biceps jerk, triceps jerk). These reflexes can be elicited by taping briskly with the neurological hammer on the tendon of the muscle in question or the muscle belly, the muscle will go into sudden jerky contraction. The muscle to be tested must be fully relaxed. Always compare with the other side on condition that the same stimulus intensity must be applied on both sides. The response will be in the form of contraction of the same muscle and movement of the attached limb.
Superficial reflexes (plantar reflex) figure (2) the plantar reflex (Babinski sign) represents an important test in assessing the role of the higher control centers on the neuronal pools of the spinal cord. The outer edge of the sole of the foot is scratched firmly by a key or a stick from the heel toward the little toe then go medially across the metatarsal bones.

a. Flexor plantar response (Babinski sign negative) the response is in the form of plantar flexion and adduction of the toes particularly of the big toe. Figure (2). This is the usual response in normal healthy, adults.

b. Extensor plantar reflex (Babinski sign positive). An abnormal plantar response first described by Babinski. There will be dorsiflexion (extension) of the big toe and fanning of the other toes. This is the normal response in infants bellow the age of one and a half year due to the incomplete development of the corticospinal tract. But abnormally found in adults when increased excitatory signals coming from the higher control centers to the neurons of the spinal cord.
Figure (2): Superficial reflex (Babinski's sign).
Lab. 5: Electrocardiography (ECG)

Introduction and Principle:

Immediately before contraction (systole) and relaxation (diastole) of the heart (cardiac cycle), there are electrical events (depolarization and repolarization) taking place in the cardiac muscle. The electrical potential generated by the cardiac muscle spreads through the body tissues and fluids which are considered as good electrical conductors to the skin, making it easy to record this activity from the skin by using surface electrodes. This process is called as electrocardiography (ECG). The machine used for this purpose is electrocardiograph and the print-out is electrocardiogram.
Electrocardiography is one of the noninvasive, easy, and reproducible investigations that can be done for patients with heart diseases, because it reflects the condition of the cardiac muscle as well as the conducting system of the heart. Certainly this will help the physician in making an accurate diagnosis.

**Objectives:**

Upon completion of this session the attendees will:

1. Acquire the skills of recording ECG.
2. Acquire the knowledge of preliminary reading of the ECG.
3. Detect abnormal rate and rhythm of the heart.

**Materials and Instruments:**

1. Electrocardiograph with its accessories.
2. Electrode gel.
3. Subject.
Figure (1): Voltage and time calibration of the electrocardiogram.

The horizontal lines are the voltage calibration lines. Each 10 divisions represent 1 mV. The vertical lines are the time calibration lines. Each large square represents 0.2 seconds (five large squares = one second). Each large square is divided into five small divisions 0.04 second each when the speed of the paper is at 25 mm/second (standard).

**Procedure:**

The subject lies supine with the chest, forearms, and ankle joints exposed. The skin is moistened with electrolyte gel to ensure good conductance between the skin and electrodes. The metal
piece of the electrode should preferably be put against a bone, the radius in the forearm and the lower end of the tibia in the leg.

In each lead the electrical potential difference is recorded between two points. In standard limb lead I the electrical potential difference is recorded between the right arm and left arm, in standard limb lead II between the right arm and left leg, and in standard limb lead III between the left arm and left leg. In the augmented limb leads, the potential difference is recorded between one point on an arm (the active electrode) and another point that is derived from the other two electrodes of Einthoven's triangle, in aVR the active electrode is on the right arm, in aVL on the left arm, and in aVF on the left foot.

The third type of leads is the chest or precordial leads. They are six denoted as (V) V1-6, in these leads the electrical potential difference is recorded between a point on the chest (the position of the searching electrode) and another point derived from connecting the three electrodes of the Einthoven's triangle to a single resistance.
The places on the chest wall where the searching electrode is put are:

V 1 in the 4 th intercostal space to the right of the sternal margin.

V 2 in the 4 th intercostal space to the left of the sternal margin.

V 3 between V 2 and V 4 .

V 4 at the point of intersection between mid clavicular line and the 5 th intercostal space.

V 5 at the point of intersection between anterior axillary line and the 5 th intercostal space.

V 6 at the point of intersection between midaxillary line and the 5 th intercostal space.

Now you are ready to record the ECG by switching the machine on and use the knobs of the indicated leads.

Figure (2) Diagram to shown the placing of unipolar precordial (chest) leads for recording ECG
Lab.5: Electrocardiography (ECG)

The result:

1-Rhythm:

   a- Regular:

Occur If the distance between R-wave and R-wave is uniform.
b- Irregular:

Occur if the distance between R-wave and R-wave is variable.

2-Heart rate:

The heart rate can be easily calculated from the ECG strip:

a) When the rhythm is regular, the heart rate is 300 divided by the number of large squares between the QRS complexes.

For example, if there are 4 large squares between regular QRS complexes, the heart rate is 75 (300/4=75).

b) The second method can be used with an irregular rhythm to estimate the rate. Count the number of R waves in a 6 second strip and multiply by 10.

For example, if there are 7 R waves in a 6 second strip, the heart rate is 70 (7x10=70).
فسلجة عملي

محافظة 1

Ramadan
Introduction:

Body temperature represents a balance between metabolic heat production and heat losses to the environment. This control is achieved by the hypothalamus. The thermoregulatory center in the hypothalamus and functions to modify heat production and heat losses as a means of regulating body temperature. Thermoregulatory center in the hypothalamus regulates the core body temperature, not the surface temperature.

Body temperature depends upon the balance between heat generation and loss, and the ambient environment. Body heat is generated in the tissues of the body, transferred to the skin surface by the blood, and then released into the environment surrounding the body.

Temperature measurement is an important part of the general examination of the patient. It is measured by centigrade (Celsius) scale but many countries (USA) are using Fahrenheit scale. The normal range of the body temperature is 36.6-37.2 °C.

A high temperature is a fever.

Metabolic rate depends on:

1- Sex
2- Age
3- Recent food ingestion
4- weight and surface area
5- Growth
6- Emotional state
Heat is produced by:

1- Metabolic process.
2- Food intake (dynamic action).
3- Exercise (contraction of skeletal muscles).
4- Hot environment such as sun, heater.

Heat is lost through:

1- Skin 97 % (radiation, conduction, convection and evaporation)
2- Expired air 2%.
3- Urine and feces 1%.

Objectives:

At the end of this session the students will be able to:

1- Identify places of measuring body temperature.
2- Indicate the significance of this test.

Subjects and instruments:

1 Medical or clinical thermometer (figure 1-2).
2- Antiseptic substance.
3- Cotton.
4- Container.

Routs of Temperature Measurement:

1- Mouth route, the most commonly used route and used in a conscious adult and in children older than 5 years.
2- Axilla route is used in unconscious adult and children
3- Groin route is used in infant younger than 1 year.
4- Rectal route is now rarely taken.
Procedure:

1- Before inserting thermometer in the mouth, wash it by antiseptic solution.

2- Hold the thermometer by fingers and watch the level of mercury, if it is higher than 35°C, shake it down to bring mercury below this level.

3- Put it in the mouth under the tongue for (3-5) minutes.

4- Mouth should be firmly closed, breathing is taken through nose.

5- Prior to reading, no hot or cold substance is placed in mouth and no gum chewing.

Physiological variation:

1- In women, the temperature may be one Celsius degree higher during ovulation.

2- The temperature of people who live in hot area 0.5°C more than those live in cold area

3- The temperature at evening 0.5°C more than morning due to increase metabolism.

4- The temperature in groin and axilla is low 0.5°C than that in the mouth while in the rectum its 0.5°C higher.

Figure: (1-2) shows different types of thermometers:

A- Mercurial.
B- Electronic

C- Tympanic
فسلجة عملي
محاضرة 2
Ramadan
Lab.2: Arrhythmia and Arterial Pulse

Introduction and principle:

One of the manifestations of cardiovascular system functions is the pulse. This can be detected by palpating any superficial artery, for instance the radial artery the most commonly used, the temporal artery, the dorsalis pedis artery and others.

Estimating the pulse rate is very informative, sometimes indicative of certain pathology of the heart and circulatory system at other times even diagnostic as in the case of ectopic beats and atrial fibrillation. In ectopic beat there is two successive beats followed by a compensatory pause. In hypovolemic shock the pulse is weak (low volume) or low pulse pressure and rapid.

Objectives:

Upon completion of this session the attendees will be able to:

1. Estimate the pulse rate, identify the rhythm, pulse pressure (pulse volume), and the character of the arterial wall whether it feels soft elastic or it has lost its elasticity (atherosclerotic) as in elderly people.

2. Identify any change in one or more the already mentioned variables.

3. Indicate the meanings of these changes.
The best places to find your pulse are the:

- wrists
- inside of elbow
- side of neck
- top of the foot

Factors effects on the pulse rate:

Nutrition and Heart Health

Smoking (Nicotine) and caffeine

Exercise

Body size.

Medications.

Methods:

1. Palpatory method

This is the most commonly used method for pulse estimation, more informative, easy to do and reproducible.

Procedure:

With the use of the middle three fingers of the right hand in right handed people, apply all three fingers on the course of the radial artery against the radius bone. Apply slight pressure with the ring finger on the artery distal ward so that the pulse can easily be detected by the index and middle fingers.

The examiner should be able to estimate first, the pulse rate whether normal (60-100 beat/minute) in young adults, or increased (tachycardia) as in fever, hyperthyroidism or physiological increase as in unusual exercise or in infants. Decreased heart (pulse) rate can be detected in hypothyroidism, A-V bundle block, or physiologically in professional
athletes. Second, pulse rhythm whether regular (normal) or irregular as in the case of multiple ectopic beats or in atrial fibrillation. Third, the pulse pressure (pulse volume), this is the force offered by the blood to push the examiner's fingers, or the amplitude the fingers move upward with each pulse. After performing the test for a number of times the student will acquire the ability to judge whether this pulse pressure is normal or less or greater than normal. If the pulse pressure is less than normal as in the case of hypovolemic conditions induced by fluid or blood loss, or in failing heart, the amplitude that the fingers move upward will be only slight, sometimes the pressure is so much reduced that no pulse can be detected. In hyperdynamic circulation as in the case of fever or hyperthyroidism the pulse volume will be greater than normal. Fourth, the arterial wall characteristics whether normal soft and elastic or it feels rigid non elastic as in atherosclerosis in elderly people.

2. Auscultatory method:

Using the stethoscope the pulse rate and rhythm can easily be detected directly from the heart across the chest wall but not the pulse pressure and arterial wall characteristic.
Lab.3: Arterial Blood

Introduction and principle:

Blood pressure is the force applied by the blood on a surface area (cm$^2$) of blood vessel wall expressed as mmHg. It represents one of the important parameters of cardiovascular physiology. Therefore, it is essential to learn how to measure blood pressure both in clinical practice and in research work.

The principle of this experiment depends on applying external pressure on the brachial artery greater than the systolic pressure so that the brachial artery is closed. Then the external pressure is reduced by gradually deflating the cuff, the artery is slightly opened so that blood starts flowing through the little orifice in the artery producing tapping sounds (Korotkov's sounds). At this moment the pressure in the cuff is equal to the systolic blood pressure. As you continue deflating the cuff the sound will increase in intensity become tapping then decrease and become muffled lastly it disappears, now the pressure in the cuff is equal to the diastolic pressure.
Objectives:

Upon completion of this session the attendees will:

1. Acquire the skills of measuring blood pressure.
2. Indicate the conditions that must be fulfilled before measuring blood pressure.
3. Identify the best position of the patient for measuring blood pressure.
4. Detect changes of blood pressure and indicate the reasons behind these changes.

Methods:

1. **Indirect method.**
   
   This method is used for measuring blood pressure in every day practice of medicine. It is noninvasive, accurate, easy, and reproducible.

Instruments and subjects:

1. Sphygmomanometer.
2. Stethoscope.
3. A subject.
Procedure:

The subject should preferably lie supine or sit comfortably, both physically and mentally rested. The upper arm is exposed so that the antecubital fossa is fully exposed.

Figure: Blood pressure measurement.

Tight or thick clothing must be removed, not merely rolled up, since it may cause inadvertent compression.

Place the air bag of the cuff flat against the inner aspect of the upper arm. Wind the cuff around the arm, taking care that each turn lies flat. The cuff should not be too tight or too loose. The lower edge of the cuff should be 3cm above the elbow.

Locate (by palpation) and mark the position of the brachial pulse, which should be clear off the cuff. Place the manometer so that the scale
should be out of the subject's view, and the mercury tank at the level of the heart. Place the chest piece of the stethoscope on the brachial artery. Close the screw and pump up the pressure to above the systolic blood pressure of the subject.

**Palpatory method:**

Feel the radial pulse by palpation. Place the three middle fingers on the course of the radial artery against the radius bone with mild pressure applied with the ring finger on the artery distal ward, so that you can palpate the pulse by the index and middle fingers. Raise the pressure in the cuff until no more pulse can be detected from the radial artery. This is taken as the systolic pressure. The diastolic pressure cannot be measured by this method.

**Auscultatory method:**

Raise the pressure in the cuff to above the systolic pressure as detected by the palpatory method. Open the screw slightly to release the pressure in the cuff gradually. Carefully listen to the appearance of the Korotkov's sounds. The moment the sounds appear, this is taken as the systolic pressure. Continue releasing the screw and deflating the cuff. The sound become louder, tapping, then muffled, and eventually fades away, this is taken as the diastolic pressure. In some cases the sounds continue until complete deflation of the cuff. In such a case the diastolic blood pressure is taken at the moment the sound changes in character from tapping to muffled.
2. **Direct method:**

   This is a more accurate method for measuring arterial pressure, but it is much more difficult, invasive, and irreproducible method. It is usually used in experimental research work.
فسلجة عملي
محاضرة 4
Ramadan
Lab.4: Effect of exercise on blood pressure and Pulse rate

Introduction and principles:

Exercise is bodily activity that enhances or maintains physical fitness and overall health or wellness.

The effect of muscular exercise on blood pressure depends on the following:
1. The type of muscular exercise, whether aerobic or anaerobic, and isotonic or isometric.
2. The severity and degree of exercise. Depending on the increase in heart rate.
3. The duration of exercise.
4. It also depends on whether the subject is a trained athlete or an untrained individual.

Blood pressure = Cardiac Output x peripheral resistance.
Cardiac Output: is The amount of blood pumped by the heart per minute
Cardiac Output = Heart Rate x Stroke Volume.

Objectives:

Upon completion of this session the attendees will:
1. Acquire the knowledge and skills about the effects of exercise on blood pressure.
2. Indicate any abnormality in the response obtained.
3. Detect changes of blood pressure and heart rate.
**Instruments and subjects:**

1. Sphygmomanometer.
2. Stethoscope
3. A subject
4. Couch or chair
5. Treadmill.
6. Timer

**Procedures:**

Various types of exercises, of varying degrees, and duration may be devised and their effects may be compared.

1. Records the blood pressure by the palpatory, auscultatory methods and notes the heart rate at the resting.

2. Make the subject comfortable and explain the procedures to be followed in this experiment.

3. You may employ any of these exercises:

   a) “Running in place” (spot running) with the thighs brought up to the horizontal alternately, for 3–5 minutes (if a metronome is available, the speed of running can be varied);
   b) Hopping on each foot for 3 minutes, raising the feet 12–15 inches off the ground; climbing up and down the stairs; jogging.

4. Records the blood pressure by the palpatory, auscultatory methods and notes the heart rate after exercise, at 2 minutes, at 4 minutes and at 6 minutes sequentially.
Results and Interpretation:
Depending upon the obtained results, compare the recorded results with table 2.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Rest</th>
<th>Strenuous Dynamic Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial blood pressure</td>
<td>120/80mmHg</td>
<td>160-220/7.5-8mmHg</td>
</tr>
<tr>
<td>Pulse rate</td>
<td>60 to 100 beats/min</td>
<td>For adults, 220 beats per minute (bpm) minus your age</td>
</tr>
</tbody>
</table>

Table (2): shows the changes that occur to arterial blood pressure and heartbeat as a result of exercise.

Cardiorespiratory adaptations to acute aerobic exercise:

1) Increased heart rate (HR)
2) Increased stroke volume (SV)
   a. The amount of blood pumped from each ventricle each time the heart beats, b. Measured in mL per beat
3) Increased cardiac output
4) Increased systolic blood pressure
5) No change (or a slight decrease) in diastolic blood pressure

Things that can increase risk of getting high blood pressure

- overweight
- eat too much salt
- drink too much alcohol or coffee
- smoke
- do not get much sleep or have disturbed sleep
The purpose of the cardio-respiratory system is to extract oxygen from the atmosphere and deliver it to the mitochondria of cells.

**OXYGEN CASCADE :**

The oxygen cascade describes the process of declining oxygen tension from atmosphere to mitochondria. When air down through the body to the cell, oxygen is diluted down, extracted or otherwise lost, so that at cellular level the PO2 may only be 3 or 4 mmHg.

With every breath, the inspired gas mixture is humidified at 37°C in the upper airway. The inspired tension of O2 (PiO2) is therefore reduced by the added water vapor. Water vapor pressure is dependent only upon temperature and is 47 mmHg at 37°C. In humidified air, the normal partial pressure of O2 at sea level is 149 mm Hg:

\[
(760 - 47) \times 0.21 = 149 \text{ mmHg(approx.).}
\]
The general equation is \( \text{PiO}_2 = (\text{Pb} - \text{PH}_2\text{O}) \times \text{FiO}_2 \)

Air consists of oxygen and nitrogen, but as gas moves into the alveoli, a third gas, carbon dioxide, is present. The alveolar carbon dioxide level. The alveolar partial pressure of oxygen PAO2 can be calculated from the following equation: \( \text{PAO}_2 = \text{PiO}_2 - \frac{\text{PaCO}_2}{R} \).

\( R \) is the respiratory quotient, which represents the amount of carbon dioxide excreted for the amount of oxygen utilized, and this in turn depends on the carbon content of food (carbohydrates high, fat low).

For now let us assume that the respiratory quotient is 0.8, the PAO2 will then be \( 149 - (40/0.8) = 100\text{mmHg} \) (approx).

The next step is the movement of oxygen from alveolus to pulmonary capillary (where the partial pressure of oxygen in mixed venous blood, PVO2, is approx (47mmHg), and as you would expect, there is a significant gradient, usually 5-10 mmHg, explained by small ventilation perfusion abnormalities and the physiologic shunt. Oxygen is progressively extracted from the capillary network, may only be 3 or 4mmHg.

The amount of oxygen in the bloodstream is determined by the:

1. Oxygen binding capacity of Hb.
2. The serum hemoglobin level.
3. The percentage of this hemoglobin saturated with oxygen.
4. The cardiac output
5. amount of oxygen dissolved.
Oxygen Transport:

Oxygen is carried in the blood in two forms:

1. dissolved in plasma
2. bound to hemoglobin.

Dissolved oxygen

obeys Henry’s law – the amount of oxygen dissolved is proportional to the partial pressure. For each mmHg of PO2 there is 0.003 ml O2/dl. The solubility coefficient of oxygen in plasma is 0.003. Therefore, with a Pao2 of 100 mmHg, only 0.3 mL of O2 is transported dissolved per deciliter of plasma.

Bound oxygen

97-98% Bound to Hb. Hemoglobin is the main carrier of oxygen. Each gram of hemoglobin can carry 1.34ml of oxygen. This means that with a hemoglobin concentration of 15g/dl, the O2 content is approximately 20ml/100ml. With a normal cardiac output of 5.6 L/min in men and 4.9 L/min in women, the delivery of oxygen to the tissues at rest is approximately 1000 ml/min: a huge physiologic reserve. Hemoglobin has 4 binding sites for oxygen, and if all of these in each hemoglobin molecule were to be occupied, then the oxygen capacity would be filled or saturated. The amount of oxygen in the blood is thus related to the oxygen saturation of hemoglobin.
**Oxygen Saturation:**

**Oxygen Saturation**: is the Ratio of oxygen bound to Hb compared to total amount that can be bound with Oxygen. Up to four oxygen molecules can bind to one hemoglobin (Hb).

**O2 Content in blood**: The sum of O2 carried on Hb and dissolved in plasma • 97-98% Carried in Combination With Hb • 2% Dissolved in Plasma We can calculate the oxygen content of blood where the PO2 is 100mmHg, when the hemoglobin concentration is 15g/L : O2 content in 100 ml blood (if normal adult with Hb 15 gm/dl) ~ 20 ml/dl (20 ml as OxyHb + 0.3 ml in plasma)
Hemoglobin:

Hemoglobin is a complex molecule consisting of: • four heme. Heme is an iron–porphyrin compound that is have four O2 -binding sites • four protein subunits. Each gram of hemoglobin can theoretically carry up to 1.34 mL of O2 .

Hemoglobin Dissociation Curve :-

Represent the relationship between the partial pressure of oxygen and the saturation of oxygen.
The complex interaction between the hemoglobin subunits results in nonlinear (an elongated S Sigmoid shape) binding with O2.

**Factors Influencing the Hemoglobin Dissociation Curve:**

- Important factors altering O2 binding include
  
  1. hydrogen ion concentration.
  2. CO2 tension.
  3. temperature.

Each factor shifts the dissociation curve either to the right or to the left. A rightward shift in the oxygen–hemoglobin dissociation curve lowers O2 affinity, displaces O2 from hemoglobin, and makes more O2 available to tissues; (At cellular site) leftward shift increases hemoglobin’s affinity for O2, reducing its availability to tissues. (the lower CO2 content in pulmonary capillaries increases hemoglobin’s affinity for O2 again, facilitating O2 uptake from alveoli.)
Abnormal Ligands & Abnormal Forms of Hemoglobin’s

Carbon monoxide, cyanide, nitric acid, and ammonia can combine with hemoglobin at O2–binding sites. They can displace O2 and shift the saturation curve to the left. Carbon monoxide is particularly potent, having 200–300 times the affinity of O2 for hemoglobin, combining with it to form carboxyhemoglobin. Carbon monoxide decreases hemoglobin’s O2-carrying capacity and impairs the release of O2 to tissues.

Arterial Oxygen Content:

The total O2 content of blood is the sum of that in solution plus that carried by hemoglobin. **Total O2 content is expressed by the following equation:**

\[
\text{Arterial Oxygen Content } \text{CaO2 (ml/dL)} = (\text{SaO2} \times \text{Hb} \times 1.34) + (\text{PO2} \times 0.003)
\]

where Hb is hemoglobin concentration in g/dL blood, and SaO2 is hemoglobin saturation at the given PO2. Using the above formula and a hemoglobin of 15 g/dL, the normal O2 content for both arterial and mixed venous blood and the arteriovenous difference can be calculated as follows:

\[
\text{CaO2} = (0.003 \times 100) + (0.97 \times 15 \times 1.34) = 20 \text{ mL/dL blood}
\]

\[
\text{Cvo2} = (0.003 \times 40) + (0.75 \times 15 \times 1.34) = 15 \text{ mL/dL blood}
\]

Oxygen Stores:

The concept of O2 stores is important in anesthesia. When the normal flux of O2 is interrupted by apnea existing O2 stores are consumed by cellular metabolism; if stores are depleted, hypoxia and eventual cell death follow. This amount includes:

1- The O2 remaining in the lungs
2- That bound to hemoglobin and myoglobin
3- That dissolved in body fluids.
The O2 contained within the lungs at FRC (initial lung volume during apnea), therefore, becomes the most important source of O2. Of that volume, however, probably only 80% is usable. Apnea in a patient previously breathing room air leaves approximately 480 mL of O2 in the lungs. (If fraction of inspired oxygen (FiO2) = 0.21 and FRC = 2300 mL, O2 content = FiO2 × FRC.) The metabolic activity of tissues rapidly depletes this reservoir severe hypoxemia usually occurs within 90 sec. The onset of hypoxemia can be delayed by increasing the FiO2 prior to the apnea. Following ventilation of O2; this delays hypoxemia following apnea for 4–5 min. This concept is the basis for preoxygenation prior to induction of anesthesia

**Carbon Dioxide**

Carbon dioxide is transported in blood in three forms:

1. dissolved in plasma 7%
2. as bicarbonate 70%
3. combine with proteins in the form of carbamino compounds 23%.

The sum of all three forms is the total CO2 content of blood

**Dissolved Carbon Dioxide:**

Carbon dioxide is more soluble in blood than O2,

**Bicarbonate:** In aqueous solutions, CO2 slowly combines with water to form carbonic acid and bicarbonate, according to the following reaction:

\[ H_2O + CO_2 \leftrightarrow H_2CO_3 \leftrightarrow H^+ + HCO_3^- \]

In plasma, although less than 1% of the dissolved CO2 undergoes this reaction, the presence of the enzyme carbonic anhydrase within erythrocytes and endothelium greatly accelerates the reaction. As a result, bicarbonate represents the largest fraction of the CO2 in blood.
Hypoxia: is defined as lack of oxygen at tissue level. Or supply O2 is insufficient for normal life function.

Anoxia: is defined as complete absence of oxygen in the tissues.

Types of hypoxia

A. Hypoxic hypoxia
B. Anaemic hypoxia
C. Stagnant(ischaemic) hypoxia
D. Histotoxic hypoxia

A. Hypoxic hypoxia

☐ It is characterized by low arterial pO2 when oxygen carrying capacity of blood and rate of blood flow to tissues are normal or elevated
Causes:

1) Low pO2 of inspired air
2) Decreased pulmonary ventilation
3) Defect in exchange of gases

B. Anemic hypoxia

In anemic hypoxia arterial pO2 is normal but the amount of hemoglobin available to carry oxygen is reduced.

Causes:

i. Anemia
ii. Hemorrhage
iii. Conversion of haemoglobin to some abnormal form

C. Stagnant(ischemic) Hypoxia

Blood flow to the tissue is so low that adequate oxygen is not delivered to them. despite normal arterial pO2 and hemoglobin concentration

Causes:

i. Circulatory failure
ii. Hemorrhage via baroreceptors leading to reflex vasoconstriction

D. Histotoxic hypoxia

Amount of oxygen delivered to the tissues is adequate but because of the action of toxic agents the tissues cannot make use of the oxygen supplied to them.

Cause:

Cyanide poisoning causing damage to enzyme cytochrome oxidase.
Stages of hypoxia:

1- Indifferent
At beginning, depression of eye function.

2- Compensatory:
Rise in respiration and heart rates, blood pressure.

3- Disturbance:
Obvious symptoms begin (numbness, tingling)

4- Critical:
Loss of consciousness.

Adaptation to low oxygen

1- A great increase in pulmonary ventilation.
2- Increase number of red blood cells
3- Increase efficiency of cellular metabolism.

Treatment of hypoxia

1. Treatment of the underlying cause - depending upon the type of hypoxia

2. Oxygen therapy
   i. Inhalation of 100% pure oxygen
   ii. Hyperbaric oxygen therapy
Low oxygen pressure at high altitude

The carotid body, a cluster of specialized cells in the carotid artery, detects low oxygen levels in the blood and alerts the brain.

In response, the brain sends signals to the rest of the body to:

- Increase breathing rate and constrict vessels in the lung
- Increase heart rate
- Dilate peripheral blood vessels in arms, legs, hands, and feet

Good Luck
Obstructive and restrictive Pulmonary Disease:

Obstructive and restrictive breathing are the two most common abnormal patterns, as determined by PFTs.

**Obstructive lung diseases** are the most common form of pulmonary dysfunction. They include:

- asthma, emphysema, chronic bronchitis, cystic fibrosis, and bronchiolitis.

The primary characteristic of these disorders is:

1- resistance to airflow.

2- forced expiratory volume <70% .

3- Elevated airway resistance and air trapping lead to:
1. Increase the work of breathing

2. Respiratory gas exchange is impaired because of ventilation/perfusion (V·/Q·) imbalance.

3. Wheezing is a common finding and represents turbulent airflow.

**ASTHMA**

Asthma is a common disorder, affecting 5% to 7% of the population. Its primary characteristic is

- airway (bronchiolar) inflammation
- hyper reactivity in response to a variety of stimuli.
- Airway obstruction, bronchial smooth muscle constriction.
- edema.
- increased secretions.

**causes**

- a variety of airborne substances, including pollens, animal dander, dusts, and various chemicals.

- Some patients also develop bronchospasm following ingestion of aspirin, nonsteroidal anti-inflammatory agents such as sulfites.

- Exercise, emotional excitement, and viral infections

Clinically, asthma is manifested by episodic attacks of dyspnea, cough, and wheezing

**Asthma is classified as acute or chronic.**
**Chronic asthma** is further classified as intermittent mild, moderate, and severe persistent disease.

**Diagnosis:**

expiratory airflow measurements such as FEV₁, FEV₁/FVC, and peak expiratory flow rate FEF help in assessing the severity of airway obstruction

**Intraoperative Management:**

- Regional anesthesia will circumvent this problem, but some clinicians believe that high spinal or epidural anesthesia may aggravate bronchoconstriction by blocking sympathetic tone to the lower airways (T₁–T₄) and allowing unopposed parasympathetic activity

- Drugs often associated with histamine release (e.g., morphine) should be avoided or given very slowly when used.

**COPD**

defined as a disease state characterized by airflow limitation that is not fully reversible Most patients with COPD are asymptomatic or only mildly symptomatic, but show expiratory airflow obstruction upon PFTs. With advancing disease, mal distribution of both ventilation and pulmonary blood flow results in areas of low (V·/Q·) ratios (intrapulmonary shunt).

**Intraoperative Management:**

- Regional anesthesia is often considered preferable to general anesthesia, high spinal or epidural anesthesia can decrease lung volumes, restrict the use of accessory respiratory muscles, and produce an ineffective cough, leading to dyspnea and retention of secretions

- Pre oxygenation prior to induction of general anesthesia prevents the rapid oxygen desaturation often seen in these patients and retention of secretions
Restrictive Pulmonary Disease: are characterized by

- decreased lung compliance.
- Lung volumes are typically reduced

Restrictive pulmonary diseases include many acute and chronic intrinsic pulmonary disorders, as well as extrinsic (extra pulmonary) disorders involving the pleura, chest wall, diaphragm, or neuromuscular function.

Reduced lung compliance lead to

- increases the work of breathing characteristic rapid, but shallow, breathing pattern.

- Respiratory gas exchange is usually maintained until the disease process is advanced
Restrictive lung diseases

are often divided into two groups, depending on their cause is intrinsic or extrinsic.

**Intrinsic restrictive lung disorders** cause an internal abnormality, usually leading to the stiffening, inflammation, and scarring of the lung tissues. Types of diseases and conditions involved in intrinsic restrictive lung disease can include: pneumonia, tuberculosis, sarcoidosis, idiopathic pulmonary fibrosis, interstitial lung disease, lung cancer, fibrosis caused by radiation, rheumatoid arthritis, infant and acute respiratory distress syndrome, inflammatory bowel disease (IBD) and systemic lupus.

**Extrinsic restrictive lung disease** is caused by complications with tissues or structures outside of the lungs are often associated with weakened muscles, damaged nerves, stiffening of the chest wall tissues.
Types of diseases and conditions involved in extrinsic restrictive lung disease can include:

- pleural effusions, or the buildup of excessive fluid between tissue layers surrounding the lungs
- scoliosis, or twisting of the spine
- neuromuscular disease such as Lou Gehrig's disease
- muscular dystrophy intermittent muscle weakness
- obesity
- rib damage, especially fractures
- ascites, or abdominal swelling connected with liver scarring or cancer
- diaphragm paralysis
- kyphosis, or hunching of the upper back
- diaphragmatic hernia and heart failure.

**Symptoms**

☐ Most people with restrictive lung diseases have similar symptoms, including:

- shortness of breath, especially with exertion
- inability to catch their breath or get enough breath
- chronic or a long-term cough, usually dry, but sometimes accompanied by white sputum or mucus
- weight loss
- chest pain • wheezing or gasping breath • fatigue , depression and anxiety
**Preoperative Management**

Patients with acute pulmonary disease should be procedures

- oxygenation and ventilation should be optimized preoperatively to the greatest extent possible.
- Fluid overload should be treated with diuretics.
- heart failure may also require vasodilators.

**Commonly used tests for restrictive lung disease include:**

1- Forced vital capacity (FVC) test
2- Chest X-ray, which creates images of the entire chest and lung area for evaluation
3- Computed tomography (CT) scans, which create more detailed images of the chest and lung area compared to chest
4- X-rays.
5- Bronchoscopy, where a flexible tube with a camera is inserted through the nose or mouth into the airways of the lung for examination.