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Semester I

Textbook of APPLIED PHYSIOLOGY for NURSING Students

As per the Revised INC Syllabus for BSc Nursing

N Geetha

3rd Edition

JAYPEE

Contents

Chapter

23 Cardiac Output

LEARNING OBJECTIVES

- Define cardiac output and stroke volume
- Describe the regulation of cardiac output
- Heterometric and homometric regulation of stroke volume
- Explain sinoaortic reflex
- Explain end-diastolic volume and the factors affecting it
- Cardiovascular homeostasis in exercise
- Application in nursing

STROKE VOLUME AND CARDIAC OUTPUT

The amount of blood pumped by each ventricle per beat is called **stroke volume**. During a single contraction, each ventricle pumps out 70 mL of blood into the aorta or pulmonary artery. Stroke volume is the difference between end-diastolic ventricular volume and end-systolic ventricular volume.

- End-diastolic volume (EDV) = 140 mL
- End-systolic volume (ESV) = 70 mL
- Stroke volume (SV) = EDV – ESV = 70 mL

The amount of blood pumped by each ventricle per minute is **cardiac output** (CO). Normal cardiac output is 5–6 L/min. It is the product of stroke volume and heart rate (HR).

Cardiac output = SV × HR = 70 mL × 72/min = 5 L/min.

Cardiac index is CO/min/square meter body surface area and is approximately 3.4 L/min/m^2 (2.8–4.2 L).

Cardiac reserve refers to the maximum increase in cardiac output above normal and is expressed in percentage. In a healthy adult, it is 300–400% and in athletes, it is 500–600%.

Distribution of Cardiac Output

About 75% of cardiac output is distributed to the vital organs and the rest 25% occurs to skeletal muscles, other organs, and skin **(Table 23.1)**.

Table 23.1: Distribution of cardiac output at rest.

Organ	*Distribution (mL/min)*
Liver	1,500
Kidneys	1,250
Brain	750
Skeletal muscle	750
Skin	450
Heart	250
Other regions	550

MEASUREMENT OF CARDIAC OUTPUT

Fick Method

The cardiac output can be estimated by using the **Fick's principle**. It states that the amount of substance taken up by the body in unit time is equal to the arteriovenous concentration difference (A–V difference) of the substance times the blood flow. From this law, the blood flow can be calculated by the formula.

Amount of substance taken up by the body in unit time = (A–V concentration difference) × (blood flow)

So, blood flow = amount of substance taken up/A–V concentration difference.

The above principle can be applied to determine cardiac output by measuring the amount of O_2 consumed by the body in a given period and dividing this value by the A–V concentration difference of O_2 across the lungs.

$$\text{Cardiac output} = \frac{O_2 \text{ consumption in mL/min}}{\text{Arterial } O_2 - \text{Venous } O_2 \text{ (mL/L)}}$$

$$= \frac{250 \text{ mL/min}}{(190 \text{ mL/L}) - (140 \text{ mL/L})} = 5 \text{ L}$$

VARIATIONS IN CARDIAC OUTPUT

Physiological Variation

1. Age—cardiac output increases from infancy to adulthood.
2. Sex—value is less in females than in males.
3. Exercise—in severe exercise, CO can increase from 5 L/min to 30–35 L/min.
4. CO increases by about 30% during digestion, i.e. after meals.
5. Posture—CO is maximal in the lying down posture and minimum in the standing posture.

Pathological Variation

1. **Pathological increase** in cardiac output is seen in hyperthyroidism, fever, etc.
2. **Pathological decrease** is seen in hypothyroidism, fibrillation, hemorrhage, circulatory shock, cardiac failure, myocardial infarction, etc.

REGULATION OF CARDIAC OUTPUT

Factors Affecting Cardiac Output

1. **Stroke volume**
2. **Heart rate**

Regulation of cardiac output includes all factors that regulate stroke volume and heart rate, since cardiac output is the product of heart rate and stroke volume.

REGULATION OF STROKE VOLUME

Stroke volume is regulated mainly by two mechanisms:

1. **Heterometric regulation** includes EDV and aortic impedance. **EDV** is the preload and **aortic impedance** is the afterload on heart. The preload is the degree to which the cardiac muscle is stretched before it contracts. Afterload is the resistance against which the ventricles pump the blood. **Heterometric regulation is independent of cardiac innervation**.
2. **Homometric regulation** includes neural and chemical factors.

Heterometric Regulation of Stroke Volume

End-diastolic Volume

The amount of blood in each ventricle at the end of diastole is EDV. EDV is the preload, i.e. the load that operates in the heart before the beginning of contraction. **Frank–Starling law** states that the force of contraction of the heart is directly proportional to the EDV within **physiological limits**. When the EDV increases, the stroke volume also increases.

Factors Affecting End-diastolic Volume

- **Venous return:**
  - Skeletal muscle pump
  - Venous tone
  - Respiratory pump
  - Posture and gravity
  - Blood volume
- Ventricular filling:
  - Atrial contraction
  - Intrapericardial pressure
  - Blood volume.

Factors Affecting Venous Return

Venous return is the amount of blood flowing from the veins into each atrium. The great veins like inferior vena cava, superior vena cava and cardiac vein empties into the right atrium and the 4 pulmonary veins empties blood into the right atrium. At rest, venous return to each atrium per minute is equal to cardiac output, i.e., 5 L/min.

The factors are:

1. **Skeletal muscle pump:** When there is movement, there is more contraction of skeletal muscles leading to the compression of blood vessels especially veins leading to increase in venous return. The back flow of blood in the veins is prevented by the presence of one-way valves along the course of veins. Thus, in exercise, there is increase in venous return.
2. **Venous tone:** The walls of veins contain smooth muscles which play an important role in propelling blood back to the heart. Large veins act as blood reservoirs by accommodating

large volumes of blood when blood volume is increased. In times of need like hypovolemia, they contract, increasing venous return and cardiac output. Sympathetic stimulation causes venoconstriction and increase in venous tone.

3. **Respiratory pump**: Negative intrathoracic pressure especially during inspiration exerts a sucking effect on the great veins leading to increased venous return. This is referred to as respiratory pump.
4. **Posture and gravity:** Venous return is decreased in standing posture due to pooling of blood in the lower extremities and is maximal in the lying down posture.
5. **Blood volume:** Decrease in the blood volume decreases venous return and thus decreases cardiac output. In pregnancy, there is increase in blood volume and increase in venous return and cardiac output.

Factors Affecting Ventricular Filling

- **Atrial contraction:** 70% of ventricular filling occurs passively. The rest is contributed by atrial contraction.
- **Intrapericardial pressure:** When there is increase in pericardial pressure as in pericardial effusion, pericarditis, etc. There is a limit to the extent of distension of the chambers of heart. This leads to reduced filling and decrease in EDV.
- **Blood volume:** When blood volume is decreased, ventricular filling will be reduced due to decrease in venous return. This leads to decrease in stroke volume.

Aortic Impedance

The aortic resistance offered to the outflow of blood when the ventricle contract is aortic impedance. It is the hydrostatic pressure of the blood in the aorta. It acts as a load against ventricular shortening. This is the **afterload**, i.e. the load acting on the heart after it begins to contract. It is the resistance against which ventricles pump the blood. If the resistance offered is high, as in systemic hypertension, the force of cardiac contraction should be increased to overcome this afterload. If the ventricular pressure is not adequate, then there will be a reduction in stroke volume and cardiac output.

Homometric Regulation of Stroke Volume

Those factors operating from outside on the heart are included under homometric regulation of cardiac output. The factors involved in homometric regulation are:

1. **Neural factors:**
   - Sympathetic stimulation
   - Parasympathetic stimulation
   - Influence from higher centers like spinal cord, medulla oblongata, etc.
2. **Chemical factors:**
   - Blood chemistry
   - Hormones
   - Drugs.

Neural Factors

- **Sympathetic stimulation** increases force of cardiac contraction due to the effect of norepinephrine liberated at the sympathetic nerve endings. Inhibition of sympathetic activity decreases the force of cardiac contraction. Sympathetic stimulation not only increases the force of contraction but also increases heart rate. Since cardiac output is the product of stroke volume and heart rate, sympathetic stimulation leads to a marked increase in cardiac output.
- **Parasympathetic stimulation** decreases the force of contraction.

Chemical Factors

- **Hypercapnia**, **hypoxia**, and **acidosis** produce negative inotropic effect and decrease the force of contraction and decrease cardiac output.
- **Hormones like catecholamines** produced from adrenal medulla exert positive inotropic effect and increases myocardial contractility.
- **Xanthines** like caffeine and theophylline increase CO.
- Drugs like **digitalis** increases cardiac contractility.

REGULATION OF HEART RATE

Regulation of heart rate includes the following factors:
1. Neural factors
2. Chemical factors
3. Physical factors.

Neural Factors

Autonomic Control

Heart is supplied by sympathetic and parasympathetic divisions of the nervous system.

Sympathetic fibers supply the whole of the heart, i.e. the nodal tissues, atrial- and ventricular musculature. **Norepinephrine** is the neurotransmitter. Sympathetic stimulation produces a positive chronotropic effect (increase in heart rate). In the heart, norepinephrine produces its effect via α-**receptors**.

Parasympathetic preganglionic fibers reach the heart through the right and the left vagus nerves. The ganglion is close to the heart. Postganglionic parasympathetic fibers supply the sinoatrial (SA) node, atrioventricular (AV) node, and atrial musculature. **Ventricle is devoid of parasympathetic supply**.

Parasympathetic stimulation has an inhibitory effect on heart, i.e. they depress the SA node and decrease heart rate. Neurotransmitter at the postganglionic ending is **acetylcholine**. Heart rate is kept constant by vagal tone. Sympathetics come into action only in times of emergencies.

Central Influences on Heart Rate

Control of Heart Rate at Spinal Cord Level

The lateral horn cells in the thoracic segments (T1–T5) of the spinal cord form the **spinal cardio-acceleratory center**.

Control of Heart Rate by Medullary Centers

In the reticular formation of medulla, many vital centers are present that control cardiac function called cardiac centers. These centers control the lateral horn cells of spinal cord. The medullary centers exert their effect almost entirely through the autonomic nervous system (ANS). The medullary centers are:

- Cardioinhibitory center (CIC or parasympathetic center).
- Vasomotor center (VMC or sympathetic center) now referred to as rostral ventrolateral medulla (RVLM).

Cardioinhibitory center is a group of neurons constituted by **dorsal motor nucleus of vagus**, **nucleus tractus solitarius (NTS)** and **nucleus ambiguus**. There is a constant discharge of impulses from the CIC through the vagus which influences the activity of the heart. CIC when stimulated leads

to a reduction in heart rate since the vagus is inhibitory to the heart. Continuous stream of impulses from the CIC to the heart is responsible for **vagal tone** which is very prominent in humans.

Vasomotor center consists of groups of neurons whose cell bodies are located in the **RVLM**. The axons of the VMC go to the lateral horn cells of spinal cord and influence the discharge through the sympathetic fibers. The excitatory neurotransmitter at the axonal endings of VMC neurons is **glutamate**. When the VMC is stimulated due to sympathetic stimulation, there is increase in heart rate.

The cardiac centers receive afferents from baroreceptors, from chemoreceptors and respiratory system, and from receptors within the heart and great blood vessels.

Impulses from Baroreceptors

Baroreceptors are stretch receptors located in the carotid sinus and aortic arch. The nerve fibers arising from the carotid sinus- and aortic arch baroreceptors are also called **sinoaortic nerves**. These fibers relay in the NTS in the medulla. The NTS sends excitatory impulses directly to the CIC and inhibitory impulses through inhibitory interneurons to the VMC. The inhibitory neurotransmitter is gamma-aminobutyric acid (GABA). Thus, when NTS is stimulated, the VMC is inhibited via inhibitory interneurons and the CIC is stimulated **(Fig. 23.1)**. The net effect is decrease in sympathetic tone and decrease in heart rate. This is **sinoaortic reflex or baroreceptor reflex**.

Marey's law states that heart rate is inversely proportional to blood pressure (BP). Whenever BP is increased, there will be reflex bradycardia, and whenever BP falls, there will be reflex tachycardia. Exception to this law is Cushing's reflex associated with raised intracranial tension (*refer* Cushing's reflex).

Respiratory Afferents

In forced respiration, changes in heart rate are observed which is called **sinus arrhythmia**. Forced inspiration produces increase in heart rate, and forced expiration produces decrease in heart rate.

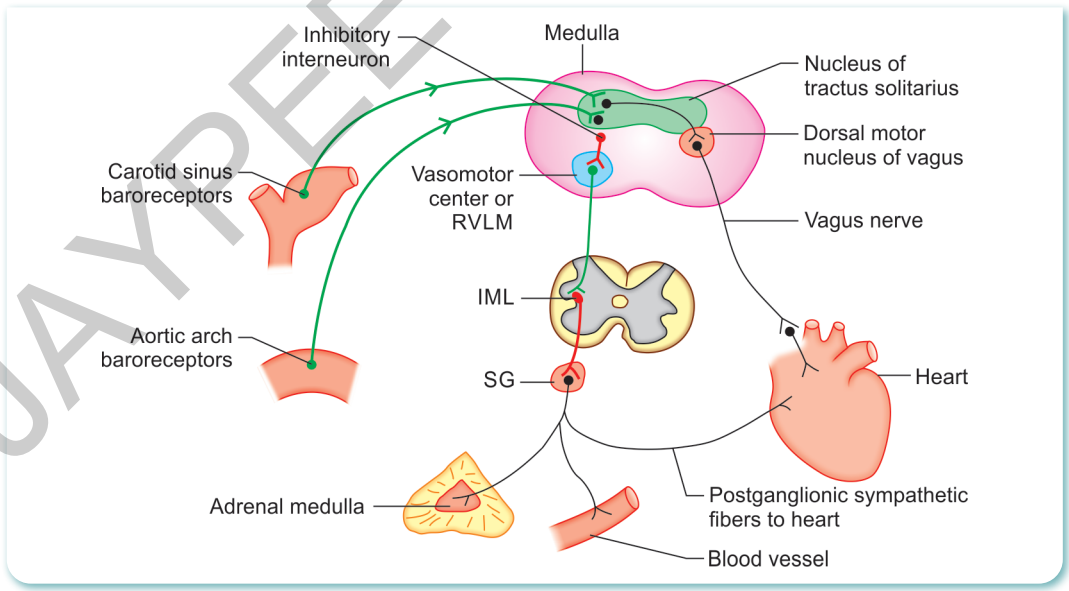

Fig. 23.1: Pathway for sinoaortic reflex or baroreceptor reflex.
(IML: Intermediolateral horn cells; SG: Sympathetic ganglion; RVLM: Rostral ventrolateral medulla)

Afferents from Chemoreceptors

Chemoreceptors present in carotid and aortic bodies send impulses through the afferent fibers passing through glossopharyngeal- and vagus nerves. Chemoreceptors are mainly stimulated by hypoxia, i.e., decrease in PO_2. Impulses from chemoreceptors reach the respiratory center and cardiac centers. The chemoreceptor afferents stimulate the inspiratory center and causes hyperpnea. The increased activity in the inspiratory center suppresses activity in the cardiac vagal centers, i.e., NTS and nucleus ambiguus and causes tachycardia.

Afferents from Receptors in Heart and Great Blood Vessels

Impulses from Right Atrium or Bainbridge Reflex

The atrial wall contain atrial baroreceptors. When there is stretch to the atrial wall, these baroreceptors get stimulated. The effect is inhibition of the CIC leading to tachycardia and inhibition of VMC leading to hypotension.

Bezold–Jarisch Reflex or Coronary Chemoreflex

In myocardial infarction, there will be reflex apnea followed by rapid breathing, hypotension, and bradycardia. Receptors are mainly present in the left ventricular wall and they are stimulated by certain substances released from the ischemic myocardium. The afferent fibers go through vagus nerve to the cardiac centers. This reflex is called Bezold–Jarisch reflex.

Chemical Factors

The chemical factors that affect heart rate are:

1. Catecholamines
2. Thyroxine
3. PCO_2
4. Hypoxia
5. Acidosis
6. Drugs.

Catecholamines increase rhythmicity and contractility of the heart. Norepinephrine is a net vasoconstrictor which causes increase in the mean arterial pressure. This produces reflex bradycardia via the sinoaortic mechanism. The net effect of epinephrine on blood vessels is vasodilatation. Hence, the mean arterial pressure is not raised and reflex bradycardia is not seen with epinephrine.

Increased level of **thyroid hormones** increases heart rate. In hypothyroidism, there is decrease in heart rate.

Hypercapnia (increase in PCO_2) decreases heart rate and hypocapnia increases heart rate.

Hypoxia in the initial stages increases heart rate. When the degree of hypoxia becomes severe, heart rate becomes irregular and arrhythmias occur.

Acidosis decreases heart rate.

Parasympatholytic **drugs** increase heart rate. Parasympathomimetic drugs decrease heart rate. Nicotine increases heart rate.

Physical Factors Regulating Heart Rate

When there is an increase in body **temperature** as in increased metabolic activity, heart rate is increased. **Muscular exercise** increases heart rate.

Regulation of Cardiac Output in Transplanted Heart

Normally during exercise, there is increased sympathetic discharge so that myocardial contractility and heart rate are increased and there is increase in cardiac output. But transplanted heart is devoid of cardiac innervation.

However, patients with transplanted heart are able to increase their cardiac output during exercise through the operation of **Frank-Starling mechanism**. During exercise there is increase in venous return and ventricular end-diastolic volume increases and the cardiac muscle contracts more forcefully. Increase in venous return is due to the action of **skeletal muscle pump and respiratory pump**. In addition, there is vasodilatation in the muscle leading to decrease in peripheral resistance and thus the afterload **(aortic impedance) is decreased**. Thus there is marked increase in cardiac output in transplanted heart.

CARDIOVASCULAR ADJUSTMENTS IN EXERCISE

The most important factor in exercise is to deliver the required O_2 and nutrients to the exercising muscles. Due to large mass of skeletal muscles in the body, during exercise it requires large amounts of blood flow. For this, the muscle blood flow should increase very much. During exercise in between each contraction, the blood flow increases as much as 30-fold. In **intermittent contraction**, the muscle blood flow decreases during each contraction because the contracting muscle compresses the intramuscular blood vessels. But in **tonic contraction**, there will be rapid muscle fatigue due to lack of O_2 and nutrients because of sustained contraction.

At rest, the skeletal muscle blood flow is 2–4 mL/100 g/min. But during maximal exercise the flow increases to 90 mL/100 g/min. This occurs by the following mechanisms:

- Stimulation of sympathetic nervous system
- Increase in arterial pressure
- Increase in cardiac output
- Local factors.

Increase in Sympathetic Discharge

Blood flow to the muscle increases at or even before the start of exercise and this is a neurally mediated response. Impulses coming through the sympathetic vasodilator system may be involved. At the onset of exercise, signals are transmitted from brain to muscles as well as to the vasomotor center to initiate mass sympathetic discharge. Once exercise has started, increase in flow is due to local mechanisms. The effects of increased sympathetic discharge are increase in heart rate and force of contraction of heart. The maximum heart rate that can be attained is 210/min. Increase in heart rate is seen even before the start of exercise. This is because of increased impulses coming from the hypothalamus and limbic system to the sympathetic neurons even at the thought of exercise.

Sympathetic stimulation also causes contraction of veins which diverts blood to the arterial side so that arterial blood volume is increased. There is constriction of the vascular beds of skin and splanchnic area so that more blood is made available to perfuse the exercising muscles.

Increase in Arterial Pressure

Arterial pressure is increased due to increase in heart rate and stroke volume. Systolic blood pressure is increased in all types of exercise. Diastolic pressure depends on peripheral resistance. Usually vasodilatation in the skeletal muscles balances the vasoconstriction in other tissues so that diastolic BP is not changed much. Pulse pressure and mean blood pressure are increased.

Increase in Cardiac Output

During exercise the cardiac output increases from 5.5 L/ min to 20–30 L/min depending on the severity of exercise. In an untrained person, the cardiac output increases by about 4-fold whereas in a well-trained athlete, the increase is about 6-fold. Increase in cardiac output is mainly due to increase in heart rate. Additional factors are increase in stroke volume and increase in venous return. Venous return is facilitated by the contraction of the exercising muscles.

Local Mechanisms

In exercise, depending on the severity, muscle blood flow is increased 10 to 100-fold. Local mechanisms that increase muscle blood flow during exercise include the following factors that produce vasodilatation:

- Decrease in tissue PO_2 and increase in tissue PCO_2.
- Accumulation of K^+, lactic acid, adenosine, H^+ and other vasodilator metabolites.
- Increase in tissue temperature due to muscular activity further dilates the vessels.

Effect of Training

- Cardiac hypertrophy by about 40%.
- Increase in the force of contraction of heart.
- Decrease in the heart rate at rest due to increased vagal tone.
- Increase in stroke volume at rest due to increase in the force of contraction.
- Since heart rate is decreased and stroke volume increased, there is no change in the cardiac output at rest.
- During exercise, the stroke volume increases by 50% and the heart rate increases by 270%. So, increase in cardiac output is mainly due to increase in heart rate.
- Increase in **VO_2 max**, which is the maximum amount of O_2 that can be consumed by a person while doing severe exercise. VO_2 max in an adult is 3 L/min and in a trained athlete, it may be as high as 5 L/min. Increase in VO_2 max depends on the degree to which cardiac output can increase and not by the ventilatory capacity or oxygen diffusion capacity of the lungs.

MULTIPLE CHOICE QUESTIONS

1. Increase in cardiac output during exercise is due to:
   a. Increased heart rate
   b. Decreased heart rate
   c. Increased total peripheral resistance
   d. Increased blood pressure
2. Cardiac index ratio is determined by:
   a. Cardiac output and surface area
   b. Stroke volume and surface area
   c. Surface area only
   d. Peripheral resistance
3. Cardiac output can be determined by all *except:*
   a. Fick's principle
   b. V/Q ratio
   c. Echocardiography
   d. Thermodilution method
4. Volume determining preload is:
   a. End-diastolic volume of ventricles
   b. End-systolic volume
   c Volume of blood in aorta
   d Ventricular ejection volume
5. A man consumes 1.5 L of O_2/min. His arterial PO_2 is 190 mL/L and O_2 content of mixed venous blood is 140 mL/L. Find out his cardiac output:
   a. 3 L/min
   b. 15 L/min
   c. 30 L/min
   d. 60 L/min

6. In a person with cardiac output 5 L/min and body surface area 1.7 m^2, what will be the cardiac index?
   a. 3 L/min/m^2 b. 4 L/min/m^2
   c. 5 L/min/m^2 d. 2.5 L/min/m^2
7. Cardiac output in liter per minute divided by heart rate equals:
   a. Cardiac efficiency
   b. Cardiac index
   c. Stroke volume
   d. Mean arterial pressure
8. A man consumes 1.5 L of O_2/min. His arterial PO_2 is 190 ml/L and O_2 content of mixed venous blood is 140 ml/L. Find out his cardiac output:
   a. 3 L/min b. 15 L/min
   c. 30 L/min d. 60 L/min
9. In a person with cardiac output 5 L/min and body surface area 1.7 m^2, what will be the cardiac index?
   a. 3 L/min/m^2 b. L/min/m^2
   c. L/min/m^2 d. 2.5 L/min/m^2
10. Cardiac index is defined as:
    a. Stroke volume per square meter body surface area
    b. Cardiac output per unit body surface area
    c. Systolic pressure per square meter body surface area
    d. End-diastolic volume
11. Cardiac index in a normal person is:
    a. 2.1 L/min/m^2 body surface area
    b. 3.2 L/min/m^2 body surface area
    c. 4.6 L/min/m^2 body surface area
    d. 5.9 L/min/m^2 body surface area
12. Basal cardiac output in an adult is approximately:
    a. 7.5 L b. 5 L
    c. 12 L d. 10 L
13. The work performed by the left ventricle is greater than that performed by the right ventricle because:
    a. Contraction of left ventricle is slower
    b. Left ventricular wall is thicker
    c. Stroke volume is greater
    d. After load is greater on the left ventricle
14. Starling's law of heart:
    a. Explains the increase in cardiac output that occur when venous return is increased
    b. Explains the increase in cardiac output when sympathetic nerves to heart are stimulated
    c. Explains the increase in heart rate in exercise
    d. Does not operate during exercise
15. Capacitance vessels are:
    a. Large arteries b. Small arteries
    c. Arterioles d. Veins
16. Cardiac output:
    a. Increases in arrhythmia
    b. Decreases during sympathetic stimulation
    c. Is equal to the stroke volume times the heart rate
    d. Remains the same during exercise
17. Cardiac output in a normal newborn baby in mL/kg/min is:
    a. 350 b. 150
    c. 40 d. 80
18. Preload of the heart is determined by:
    a. End-diastolic volume
    b. Ejection systolic volume
    c. End-systolic volume
    d. Systolic vascular resistance
19. Direct Fick method of measuring cardiac output requires estimation of:
    a. O_2 content of arterial blood
    b. O_2 consumption per unit time
    c. O_2 content of blood from right ventricle
    d. All of the above
20. All the following factors increase cardiac output *except:*
    a. Late pregnancy
    b. Adrenaline
    c. Food intake
    d. Sitting from lying posture
21. Ejection fraction of the ventricle refers to the ratio of:
    a. Amount of blood received/amount of blood ejected
    b. Stroke volume/end diastolic volume
    c. End-systolic/end-diastolic volume
    d. Stroke/end-systolic volume
22. Ejection fraction increases with:
    a. End-systolic volume
    b. End-diastolic volume
    c. Peripheral vascular resistance
    d. Venodilation
23. All the following factors normally increase the length of the ventricular cardiac muscle fibers *except*:
    a. Increased venous tone
    b. Increased total blood volume
    c. Increased negative intrathoracic pressure
    d. Lying to standing change in posture

24. Increase in cardiac output in exercise is due to:
    a. Decrease in peripheral resistance
    b. Increase in blood pressure
    c. Increase in heart rate
    d. Increase in peripheral resistance
25. The following may be the cause for right ventricular hypertrophy:
    a. Aortic stenosis
    b. Pulmonary stenosis
    c. Athletic training
    d. Mitral stenosis
26. All are increased during exercise *except*:
    a. Cardiac out put
    b. Venous return
    c. Coronary blood flow
    d. Peripheral vascular resistance
27. In a patient with transplanted heart, which of these are the reasons for the increased cardiac output during exercise?
    a. Reinnervation of transplanted heart
    b. Intrinsic mechanism
    c. Epinephrine from medulla
    d. Frank-Starling mechanism
28. Maximum heart rate that can occur with exercise:
    a. 120 b. 140
    c. 160 d. 200
29. Blood supply during exercise is increased in:
    a. Cutaneous circulation
    b. Hepatosplanchnic circulation
    c. Renal circulation
    d. Coronary circulation

Answers

1. a	2. a	3. b	4. a	5. c	6. a
7. c	8. c	9. a	10. b	11. b	12. b
13. d	14. a	15. d	16. c	17. a	18. a
19. d	20. d	21. b	22. b	23. d	24. c
25. b	26. d	27. c	28. d	29. d	

Textbook of Applied PHYSIOLOGY for NURSING Students

Salient Features

- Contains hand-drawn figures that can be easily and effectively reproduced.
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- Highlights important terminologies and nomenclatures in bold.
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- MCQs given at the end of each chapter.

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