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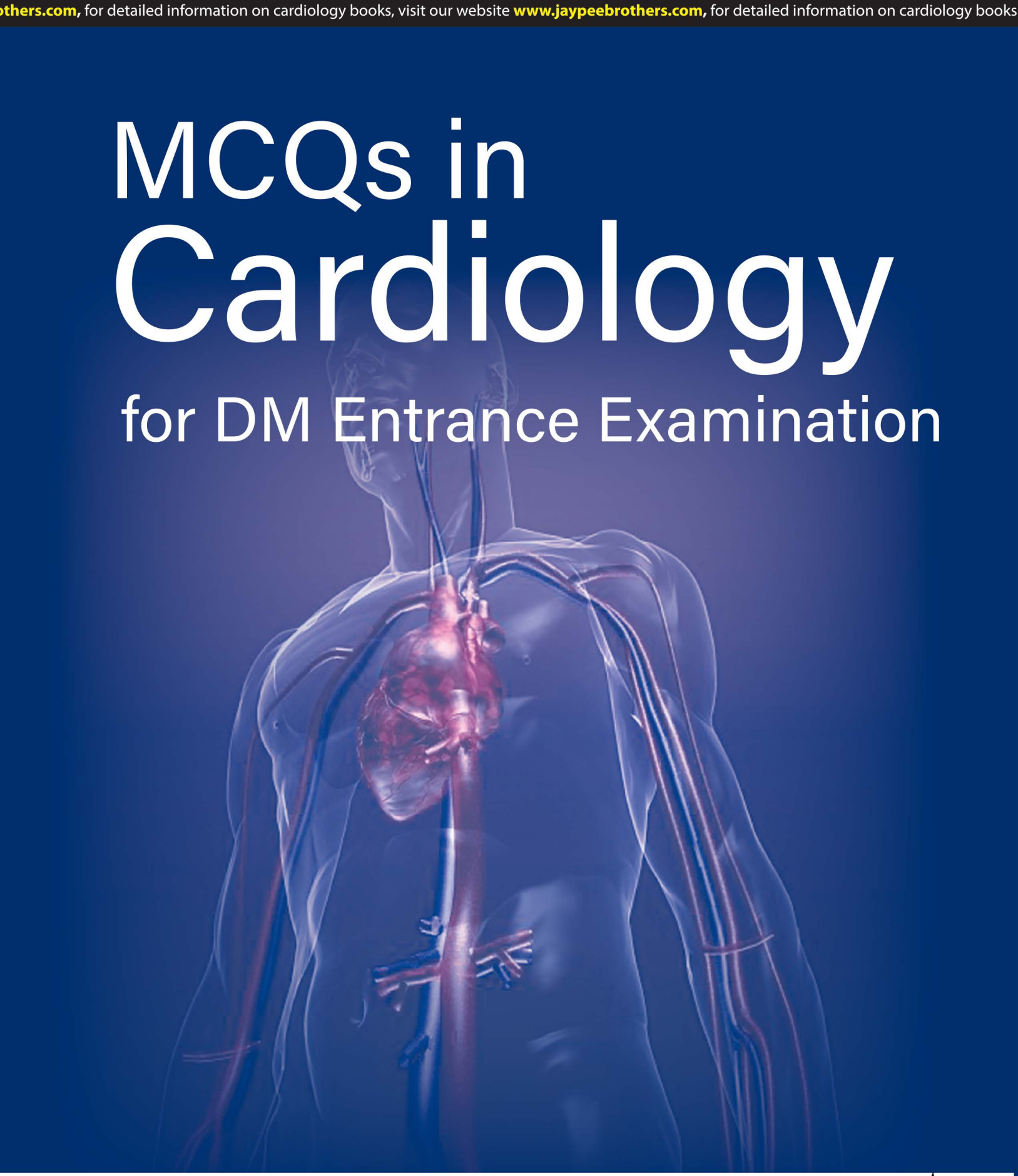
MCQs in Cardiology

for DM Entrance Examination



Vinod Sharma

2nd Edition

JAYPEE

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CHAPTER

2 Cardiac Physiology

1. Anrep effect includes all *except*:

a. A sudden increase in BP is compensated by an increase force of contraction
b. There is reflex decrease of peripheral resistance mediated by baroreceptors
c. It is seen in only experimental preparations
d. It is seen in both normal humans and experimental preparations

2. Which of the following parameter is taken as measure of afterload in clinical practice?

a. Venous pressure
b. Arterial blood pressure
c. Aortic impedance
d. Product of heart rate and blood pressure

3. Aortic impedance is derived by:

a. It is the aortic pressure divided by the aortic flow
b. It is the aortic flow divided by aortic pressure
c. It is the aortic pressure multiplied by aortic flow
d. None of the above

4. According to Laplace law:

a. Wall stress = $\frac{\text{Pressure} \times \text{Radius}}{2 \times \text{Wall thickness}}$
b. Wall stress = $\frac{\text{Pressure} \times \text{Radius}}{\text{Wall thickness}}$
c. Wall stress = $\frac{\text{Radius} \times \text{Wall thickness}}{\frac{1}{2}\ \text{Pressure}}$
d. Wall stress = $\frac{\text{Wall thickness}}{\sqrt{\text{Pressure} \times \text{Radius}}}$

5. In physiological preparation, an increased heart rate progressively increases the force of ventricular contraction, even in isolated papillary muscle preparation—It is called as:

a. Frank–Starling mechanism
b. Anrep effect
c. Treppe or Bowditch effect
d. Laplace effect

6. Which is *correct* regarding coronary blood flow?

a. Resting coronary blood flow is normally between 60–90 mL/min/100 g of myocardium
b. It can increase four to fivefold during exercise
c. Coronary artery perfusion pressure is an important determinant of coronary blood flow
d. All are correct

7. The increase in heart rate associated with atrial distention is known as:

a. Bezold–Jarisch reflex
b. Bainbridge reflex
c. Anrep effect
d. Frank–Starling effect

8. Ventricular distention producing depressor reflex is called as:

a. Bezold–Jarisch reflex
b. Bainbridge reflex
c. Anrep effect
d. Frank–Starling effect

9. Increasing left ventricular end diastolic volume increases stroke volume in ejecting beats is:

a. Frank–Starling effect
b. Anrep effect
c. Bowditch effect
d. Bainbridge reflex

10. Echocardiographically depressed regional wall motion, the hypocontractile segment that still have a sustain glucose extraction as shown by positron emission tomography (PET) is suggestive of:

a. Stunned myocardium
b. Ischemic preconditioning
c. Hibernating myocardium
d. Myocardial embalment

11. Bernheim effect (when a large left ventricle compresses the right ventricle, the volume on the left side being so great that right side is unable to fill properly) is as a:

a. Systolic ventricular interaction
b. Diastolic ventricular interaction
c. Systolic ventricular interference
d. Diastolic ventricular interference

12. Which statement is *not* correct regarding the relation of tension, pressure and radius?

a. Tension is directly related to thickness of wall
b. Tension is inversely related to thickness of wall
c. Tension is directly related to pressure
d. Tension is directly related to radius

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13. A nonproductive cough is a patient with heart failure is called as:
a. Dyspnea
b. Dyspnea equivalent
c. Orthopnea equivalent
d. Trepopnea

14. Orthopnea limited to one lateral decubitus position is named as:
a. Dyspnea equivalent
b. Orthopnea equivalent
c. Trepopnea
d. Paroxysmal nocturnal dyspnea

15. Which of following is *not* a mechanism for occurrence of paroxysmal nocturnal dyspnea?
a. Sudden expansion of thoracic blood volume due to resorption of interstitial fluid from the periphery and dependent portion
b. Sudden elevation of thoracic blood volume and of diaphragm on assuming recumbency
c. Enhanced adrenergic flow to support LV function during sleep
d. Normal nocturnal depression of the respiratory center

16. Paroxysmal nocturnal dyspnea reflects the presence of:
a. Pulmonary venous congestion
b. Interstitial edema
c. Alveolar edema
d. Reduction in vital capacity

17. Trepopnea means:
a. Breathlessness coming on brisk walking
b. Breathlessness that develops in the recumbent position and is relieved by elevation of the head with pillows
c. Breathlessness which develops on climbing stairs
d. Orthopnea limited to one lateral decubitus position

18. Enzyme neprilysin is responsible for breakdown of:
a. Natriuretic peptides
b. Bradykinin
c. Angiotensin II
d. All of the above

19. Which statement is *correct* regarding use of Sacubitril/Valsartan combinations?
a. At least 36 hours washout period should be allowed when switching from an ACEI before starting Sacubitril/Valsartan
b. Patient must be able to tolerate an ACEI or an ARB prior to being started on Sacubitril/Valsartan
c. Brain natriuretic peptide (BNP) will be elevated in patients taking this drug
d. All of the above

20. Which of the following should be utilized in patient on Sacubitril/Valsartan combination to assess heart failure exacerbation?
a. Brain natriuretic peptide (BNP) level
b. NT-proBNP
c. Serum creatinine level
d. Serum potassium level

21. Which of the following are chief contractile proteins in myocyte?
a. Actin and myosin
b. Troponin C
c. Titin
d. None of them

22. Which is the functional contractile unit of cardiac muscle?
a. Cardiac myocyte
b. Sarcomere
c. Actin and myosin
d. Myofibril

23. Which of the following is known to be a stretch sensor, that transmit the signals resulting into the myocyte growth pattern characteristics of volume overload condition?
a. Titin protein
b. LIM protein (MLP)
c. Adenosine gynaculate
d. ATP

24. Conditions in which contraction of cardiac muscle is strengthen independent of sarcomere length is labelled as:
a. Frank–Starling effect
b. Positive Inotropic states
c. Lusitropy
d. All

25. Enhanced conduction velocity through the conduction system is called as:
a. Positive chronotropy
b. Positive inotropy
c. Positive lusitropy
d. Positive dromotropy

26. All the following are the main determinants of ventricular mechanical performance *except*:
a. Preload and afterload
b. Contractility
c. Heart rate
d. Peripheral resistance

27. The cardiac cycle was assembled and conceived by:
a. Schmaroth
b. Braunwald
c. Forssman
d. Lewis and Wiggers

28. The phase of LV contraction after mitral valve closure and before aortic opening when the LV volume is fixed is referred as:
a. Isovolumic contraction
b. Isovolumic relaxation
c. Phase of reduced ejection
d. None of the above

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29. In cardiac cycle Diastasis occurs during:
a. End of left ventricular filling phase
b. In the mid part of left ventricular filling phase
c. The beginning of left ventricular ejection
d. At the end of left ventricular ejection

30. Atrial systole or left atrial kick is important for left ventricular filling during:
a. High heart rate
b. During exercise
c. In LV hypertrophy
d. All

31. The interval between the first heart sound (M1) to the closure of aortic valve (second heart sound A2) is called as:
a. Cardiologic systole
b. Physiologic systole
c. Physiologic diastole
d. Proto diastole

32. Physiologic systole lasts from:
a. Start of isovolumic contraction to the peak of the ejection phase
b. The start of isovolumic contraction to the end of ejection phase
c. Between the first heart sound (M1) and the second heart sound (A2)
d. None of the above

33. The degree of myocardial stretch or distention before contraction starts is called as:
a. Inotropic state
b. Preload
c. Afterload
d. Wall stress

34. The component of Frank–Starling effects are:
a. Only positive inotropic effect
b. Only increased lusitropic effect
c. Both positive inotropic effect and increased lusitropic effect
d. None of the above

35. The slow force response or adaptation of left ventricular inotropy in response to the elevation of aortic pressure is called as:
a. Frank–Starling effect
b. Anrep effect
c. Treppe phenomenon
d. Staircase phenomenon

36. Which state is *correct* regarding cardiac mechanics?
a. Preload is represented by the LV end diastolic volume (EDV).
b. Preload and afterload are interlinked
c. When preload increases the stroke volumes rises conversely when afterload increases stroke volume drops
d. All of the above

37. According to Laplace's Law left ventricular wall stress is:
a. Directly proportional to the product of pressure and radius and inversely proportional to the double of wall thickness
b. It is directly proportional to the double of wall thickness and inversely proportional to the product of pressure and radius.
c. It is directly proportional to the product of wall thickness and radius and inversely proportional to the double of pressure.
d. It is directly proportional to the double of wall thickness and inversely proportional to the double of radius.

38. Peak systolic wall stress reflects which components of afterload?
a. Peripheral resistance
b. Arterial compliance
c. Peak intraventricular pressure
d. All of the above

39. Bowditch staircase phenomenon is:
a. A larger heart volume increases the initial length of muscle fiber, to increase stroke volume at the cardiac output.
b. An increased heart rate progressively enhances the force of ventricular muscle contraction.
c. When the aortic pressure is elevated abruptly it limit ejection and tend to increase end diastolic volume, which acutely increases force and pressure at next beats.
d. None of the above

40. Which of the following is the best Index of cardiac work or O_2 uptake?
a. Double product (systolic blood pressure × heart rate)
b. External work (pressure × volume)
c. Minute work (SBP × SV × HR)
d. Pressure volume area

41. The wall stress in right ventricular function is lower due to which of the following factor?
a. Different chamber geometry of right ventricle.
b. Lower pressure in the right ventricle and pulmonary circulation
c. Lower right ventricular wall thickness
d. All the factors are contributory

42. Which chamber of the heart is called as "blood volume sensor"?
a. Left ventricle
b. Left atrium
c. Right ventricle
d. Right atrium

43. Which of the following cellular process plays important role in progressive cardiac dysfunction and LV remodelling in failing myocardium?
a. Necrosis
b. Apoptosis
c. Autophagy
d. All of the above

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ANSWERS WITH EXPLANATIONS

1. Ans. c. It is seen in only experimental preparations

Braunwald 11th Ed; Page 435–436

Anrep effect: During the systole of cardiac cycle, the left ventricle contract against the afterload (the load after the onset of contraction, against which LV contracts during LV ejection). It was studied in experimental animal that abruptly increasing the blood pressure of the heart lung preparation, the left ventricular performance increases to overcome the greater peripheral resistance. Both systolic and diastolic LV volumes rises indicating that left ventricle did not empty completely. The increased diastolic volume 'x' in a manner similar to an increase preload to prevent the stroke volume from falling as the afterload increased. Such an afterload dependent myocardium is probably the result of experimental condition in which the left ventricle is in a state of incipient failure. In experimental preparations and in normal humans, a sudden increase in the blood pressure is compensated for by an increase force of contraction, i.e., anrep effect and by a reflex decrease in the peripheral vascular resistance mediated by the baroreflexes.

When the aortic pressure is elevated abruptly a positive inotropic effect follows within 1–2 minutes. This is called as Homeometric autoregulation because it is apparently independent of muscle length and by definition it is a true inotropic effect. Stress develop in the ventricle when tension is applied to a cross sectional area and the units are force per unit area. According to Laplace law, wall stress equal to pressure into radius divided by 2 × wall thickness.

This equation emphasizes two points: (1) The bigger the left ventricle and greater its radius, the greater is the wall stress. (2) At any given radius (LV size) the greater the pressure developed by the left ventricle, the greater is the wall stress. An increase in the wall stress achieved by either of these two mechanism (LV size or intraventricular pressure) increases myocardial oxygen uptake. This is because a greater rate of ATP used is required, as the myofibrils develop greater tension.

2. Ans. b. Arterial blood pressure

Braunwald 11th Ed; Page 435–436

Wall stress, preload and afterload: Preload can be defined as the wall stress at the end of diastole and therefore at the maximal resting length of sarcomere. Measurement of wall stress in vivo is difficult because the radius of left ventricle neglects the confounding influence of the complex anatomy of left ventricle. Surrogate measurement of indices of preload include LV diastolic pressure or dimension. The afterload, being the load on the contractile myocardium is also the wall stress during left ventricle ejection. Increased afterload means that an increased intraventricular pressure has to be generated first to open the aortic valve and then during the ejection phase. These increases translate themselves into an increased myocardial wall stress which can be measured either as an average value or at a given phase of systole such as end systole. Systolic wall stress reflects the two major components of the afterload, namely the arterial blood pressure and the arterial compliance. Decreased arterial compliance and increase afterload can be anticipated when there is a aortic dilatation as in severe systemic hypertension or in elderly person. In clinical practice, it is a sufficient approximation to take the arterial blood pressure as a measure of afterload provided there is no significant aortic stenosis or change in arterial compliance.

The aortic impedance also term the arterial input impedance, gives another accurate measure of afterload. The aortic impedance is the aortic pressure divided by the aortic flow at that instance, so that this index of the afterload varies at each stage of contraction cycles. Factors reducing aortic flow such as high arterial blood pressure or aortic stenosis or loss of aortic compliance increases impedance and hence the afterload.

3. Ans. a. It is the aortic pressure divided by the aortic flow

Braunwald 11th Ed; Page 435–436

Refer to explanation for Q. No 1.

4. Ans. a. $$\textbf{Wall stress} = \frac{\textbf{Pressure} \times \textbf{Radius}}{\textbf{2} \times \textbf{Wall thickness}}$$

Braunwald 11th Ed; Page 435–436

Refer to explanation for Q. No 1.

5. Ans. c. Treppe or Bowditch effect

Braunwald 11th Ed; Page 437

Treppe or Bowditch effect: An increased heart rate progressively increases the force of ventricular contraction, even in an isolated papillary muscle preparation. This is also called as Treppe phenomenon or positive inotropic effect of activation or force frequency relation. Conversely, a decreased heart rate has a negative staircase effect. When stimulation becomes too rapid force decreases. It is proposed that during rapid stimulation more sodium and calcium ions enter the myocardial cells than can be handled by the sodium pump and the mechanism for calcium exit. Opposing the force frequency effect is the negative contractile influence of the decreased duration of ventricular filling at high heart rate. The longer the filling interval the better ventricular filling and the stronger subsequent contraction. This phenomenon is also seen

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in patient with mitral stenosis and atrial fibrillation with a variable filling interval.

6. Ans. d. All are correct

Hurst's 14th Ed; Page 120

Resting coronary blood flow is normally between 60 and 90 ml/min/100 gm of myocardium and can rapidly increase four to five folds during exercise or other conditions requiring augmented flow. The coronary flow rate is determined by the coronary artery perfusion pressure and by the resistance to flow exaggerated by forces generated within and outside the coronary vascular bed. The complexity of these forces is highlighted by the unexpected finding that the coronary diastolic pressure at the time of zero flow is greater than coronary sinus pressure. Control of coronary blood flow is metabolic, mechanical, autonomic and endothelial. The exact local feedback control mechanism that match coronary blood flow to myocardial oxygen consumption is still poorly understood.

7. Ans. b. Bainbridge reflex

Hurst's 14th Ed; Page 123

Mechanoreceptors are there in the heart, which possess both vagal and sympathetic efferent. Sensors on the atria and ventricle receive vagal efferent and sensors on pulmonary veins and coronary vessel receives sympathetic efferents. Atrial A and B receptors are located at the venoatrial junctions and have distinct function. Type 'A' receptors react primarily to heart rate but adapt to long-term changes in atrial volume. Type 'B' receptors increase their discharge during atrial distention. C-fibers arise from receptors is scattered throughout through the atria and these discharge with a low frequency and respond with increased discharge to increase in atrial pressure. The A and B receptors are thought to mediate the increase in heart rate associated with atrial distention (as it occurs with intravenous infusion) known as Bainbridge reflex. In contrast, activation of atrial C-fibers produces a vasodepressor effect (bradycardia and peripheral vasodilatation). Ventricular mechanoreceptor efferent discharge decreases periodically with inspiration. Ventricular C-fibers are primarily located in the epicardium and discharge more rapidly in response to increase in both systolic and diastolic blood pressure. Ventricular distention can produce a powerful depressor reflex called Bezold–Jarisch reflex. Vagal efferent of this cardiopulmonary reflex are also activated by chemical stimulation, e.g., cytokines, serotonin and classically veratrum alkaloids.

8. Ans. a. Bezold–Jarisch reflex

Hurst's 14th Ed, Page 123

Refer to explanation for Q. No 7.

9. Ans. a. Frank–Starling effect

Hurst's 14th Ed; Page 113

The influence of preload on measures of ventricular performance defines the LV function curve known as the Frank–Starling curve. Increasing LV end diastolic volume increases stroke volume in ejecting beats an increase peak LV pressure in isovolumic beats. The modulation of ventricular performance by changes in the preload is termed as heterometric regulation, operates on a beat by beat basis and is responsible for matching output of the right and left ventricle as with the changes in posture and breathing. The Frank–Starling curve also represents and important compensatory mechanism that maintains left ventricular stroke volume when left ventricular shortening is impaired, owing either to myocardial contractile dysfunction or due to excessive afterload. The atria also exhibit a Frank–Starling curve that becomes clinically important during exercise and when there is resistance to early diastolic left ventricular filling.

10. Ans. c. Hibernating myocardium

Hurst's 11th Ed; Page 710

The hibernating myocardium like the hibernating animal is temporarily asleep and can wake up to function normally when blood supply is fully restored. It is proposed that the fall of myocardial function to a lower level copes with the reduced myocardial oxygen supply and leads to self- preservation, the so called smart heart. Hibernation is a complex clinical situation without a good animal model. When ischemia in patient is delineated echocardiographically as depressed regional wall motion, the hypocontractile segment that still have a sustained glucose extraction has shown by positron emission tomography (PET), have a high chance of recovery after coronary artery bypass surgery. In contrast those segments with the decreased glucose extraction almost uniformly fail to recover. It has also been proposed that hibernation can occur even when the resting coronary blood flow is normal despite the presence of coronary artery disease. The proposed mechanism is that recurrent episode of ischemia leave behind stunned myocardium so that hibernation is the sum of repetitive and cumulative stunning.

11. Ans. b. Diastolic ventricular interaction

Hurst's 11th Ed; Page 710

The left ventricular function is intimately linked to that of right ventricle; both functionally and anatomically. The cardiac output of left ventricle must equal that of the right ventricle unless there is state of imbalance, as in condition of acute left ventricular failure when blood may accumulate in the lungs to cause pulmonary edema. In general, the right ventricle is working against a low resistance circuit and afterload is not a major problem in physiological condition. What the right ventricle received

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by means its filling pressure in the venous system, it empties in response to the Starling effect. The amount of pressure work generated by the right ventricle is relatively low and therefore the right ventricle is of thin wall. Anatomically, the two ventricles are interlinked. They share a common septum. That septum constitutes part of the load against which each ventricle must work. In the left ventricular hypertrophy, which includes the septum the right ventricle must work harder and tends to become hypertrophied. This is systolic ventricular interaction. One type of diastolic ventricular interaction is the 'Bernheim effect', whereby a large left ventricle compresses the right ventricle, the volume on the left side being so great that the right side is unable to fill properly. A converse ventricular interaction can occur in severe heart failure, when the dilated right ventricle may impinge on the left. When the right ventricle is unloaded by the venodilator agents (nitroglycerine) it can decrease in size and allow the left ventricular function to improve. When there is a physical impairment of mechanical function on the other as a result of volume overloading with blood the result is diastolic ventricular interference.

12. Ans. a. Tension is directly related to thickness of wall

Braunwald 11th Ed; Page 435–436

Refer to explanation for Q. Nos 1 to 4.

13. Ans. b. Dyspnea equivalent

Braunwald 11th Ed; Page 404–405

In patients with congestive heart failure, cough may be caused by pulmonary congestion, occurs under the same circumstances as dyspnea (i.e., during exertion or recumbency), and is relieved by treatment of heart failure. Thus a nonproductive cough in patients with heart failure is often a dyspnea equivalent, whereas a cough on recumbency may be considered an orthopnea equivalent. Patients with severe chronic obstructive lung disease sometime complain of orthopnea. Trepopnea is a rare form of orthopnea limited to one lateral decubitus position. It has been attributed to distortions of the great vessel in one position but not in the other.

14. Ans. c. Trepopnea

Braunwald 11th Ed; Page 404–405

Refer to explanation for Q. No 13.

15. Ans. c. Enhanced adrenergic flow to support LV function during sleep

Braunwald 11th Ed; Page 404–405

Paroxysmal nocturnal dyspnea: Attacks of paroxysmal dyspnea usually occur at night. The patient awakens, often quite suddenly and with a feeling of severe anxiety and suffocation, sits bolt upright and gasps for breath. Bronchospasm, which may be caused by congestion of the bronchial mucosa and by interstitial pulmonary edema compressing the small bronchi, increases ventilator difficulty and the work of breathing and is a common complicating factor of paroxysmal nocturnal dyspnea. In contrast to orthopnea, which may be relieved immediately by sitting upright at the side of the bed with the legs dependent, attacks of paroxysmal nocturnal dyspnea may require 30 minutes or longer in this position for relief. The reason for the common occurrence of these episodes at night is not clear, but it seems likely that the combination of: (1) The slow resorption of interstitial fluid from the dependent portion of the body and the resultant expansion of thoracic blood volume, (2) Sudden elevation of thoracic blood volume and of the diaphragm which occurs immediately on assuming recumbency (3) Reduced adrenergic support of left ventricular function during sleep, and (4) Normal nocturnal depression of the respiratory center. All the factors play major roles.

The mechanism of dyspnea: Increase awareness of respiration or difficulties in breathing is commonly associated with pulmonary capillary hypertension caused by an elevation of left atrial or left ventricular filling pressure. Patients with left ventricular failure typically exhibit restrictive ventilatory defect, characterized by a reduction of vital capacity as a consequence of replacement of the air in the lung with blood are interstitial fluid or both. Dyspnea during exertion and orthopnea are usually the clinical expression of pulmonary venous or capillary congestion. Paroxysmal nocturnal dyspnea reflect the presence of primarily interstitial edema, whereas pulmonary edema in which there is transudation and expectoration of blood tinged fluid is often a manifestation of alveolar edema.

16. Ans. b. Interstitial edema

Braunwald 11th Ed; Page 404–405

Refer to explanation for Q. No 15.

17. Ans. d. Orthopnea limited to one lateral decubitus position

Braunwald 11th Ed; Page 404–405

Refer to explanation for Q. No 13.

18. Ans. d. All of the above

Hurst's 14th Ed; Page 1709

The drug LCZ696 used in heart failure is composed of the ARB valsartan and the neprilysin inhibitor sacubitril. Valsartan blocks the AT1 receptor. Sacubitril is converted to the active neprilysin inhibitor, which inhibits neprilysin, an enzyme that breaks down atrial natriuretic peptide, BNP, and C-type natriuretic peptide, as well as other vasoactive substances such as bradykinin, angiotensin II, substance P.

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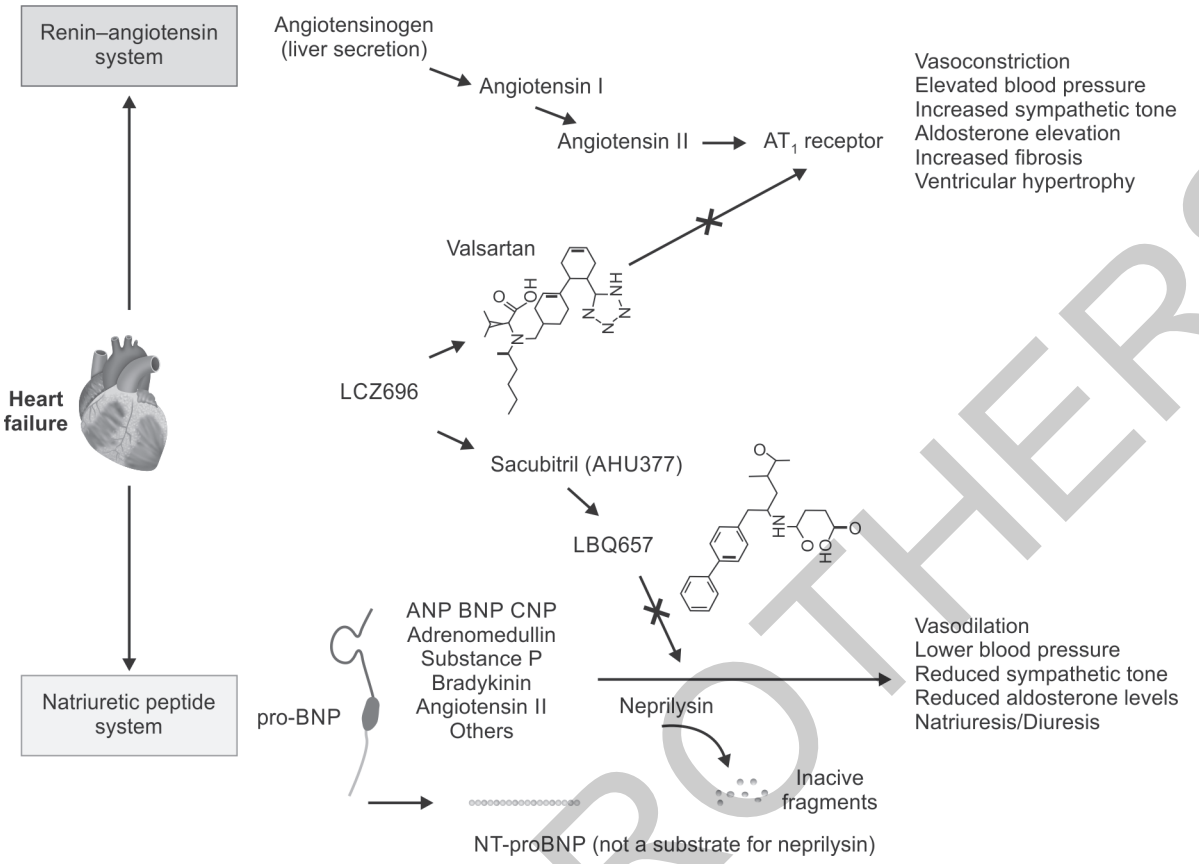

Schematic showing the mechanism of action of LCZ696

19. Ans. d. All of the above

ACC/AHA Guidelines for diagnosis and management of Heart Failure 2022

Oral neprilysin inhibitors, used in combination with angiotensin converting enzyme inhibitor, can lead to angioedema, and concomitant use is contraindicated and should be avoided. This adverse effect is thought to occur because ACE inhibitor and neprilysin break down bradykinin, which can directly or indirectly cause angioedema. An ARNI should not be administered within 36 hours of switching from or to an ACEI.

20. Ans. b. NT-proBNP

ACC/AHA Guidelines for diagnosis and management of Heart Failure 2022

B-type natriuretic peptide (BNP), atrial natriuretic peptide (ANP), and C-type natriuretic peptide (CNP) are the three main natriuretic peptides. They share a common structure and ultimately mediate their action by activating cyclic guanosine monophosphate (cGMP). ARNI therapy results in a decrease of N-terminal pro-BNP (NT-proBNP) and increase of BNP levels respectively and therefore, NT-proBNP should be utilized to assess heart failure exacerbation.

21. Ans. a. Actin and myosin

Braunwald, 12th Ed; Page 892–893 & 895

The two chief contractile proteins are the motor protein myosin on the thick filament and the actin on the thin filament. Calcium ions initiates the contraction cycle by binding to the thin filament regulatory protein Troponin-C to relieve the inhibition, otherwise exerted by this Troponin complex. The thin actin filaments are connected to Z-line at the either end of Sarcomere which is the functional contractile unit that is repeated through the filaments.

Titin is a large protein molecule. It is extraordinarily long, elastic, and slender. Titin extends from Z-line into thick filament approaching the M-line and connect the thick filament to Z-line. Titin has two distinct segments an inextensible anchoring segment and an extensible elastic segment that stretches as sarcomere length increases. Titin has multiple functions. First, tethers myosin and thick filaments to the Z-line therefore stabilizing sarcomeric structure. Second, as it stretches and relaxes, its elasticity contributes to the stress-strain relationship of cardiac muscles. Similar to spring, it helps in re-lengthening the sarcomere and aid early diastolic filling. Third, the increased diastolic stretch of the titin as the length of sarcomere in cardiac muscle is increased causes the unfolded part of titin molecules to straighten. This stretched molecular spring limits overstretching of

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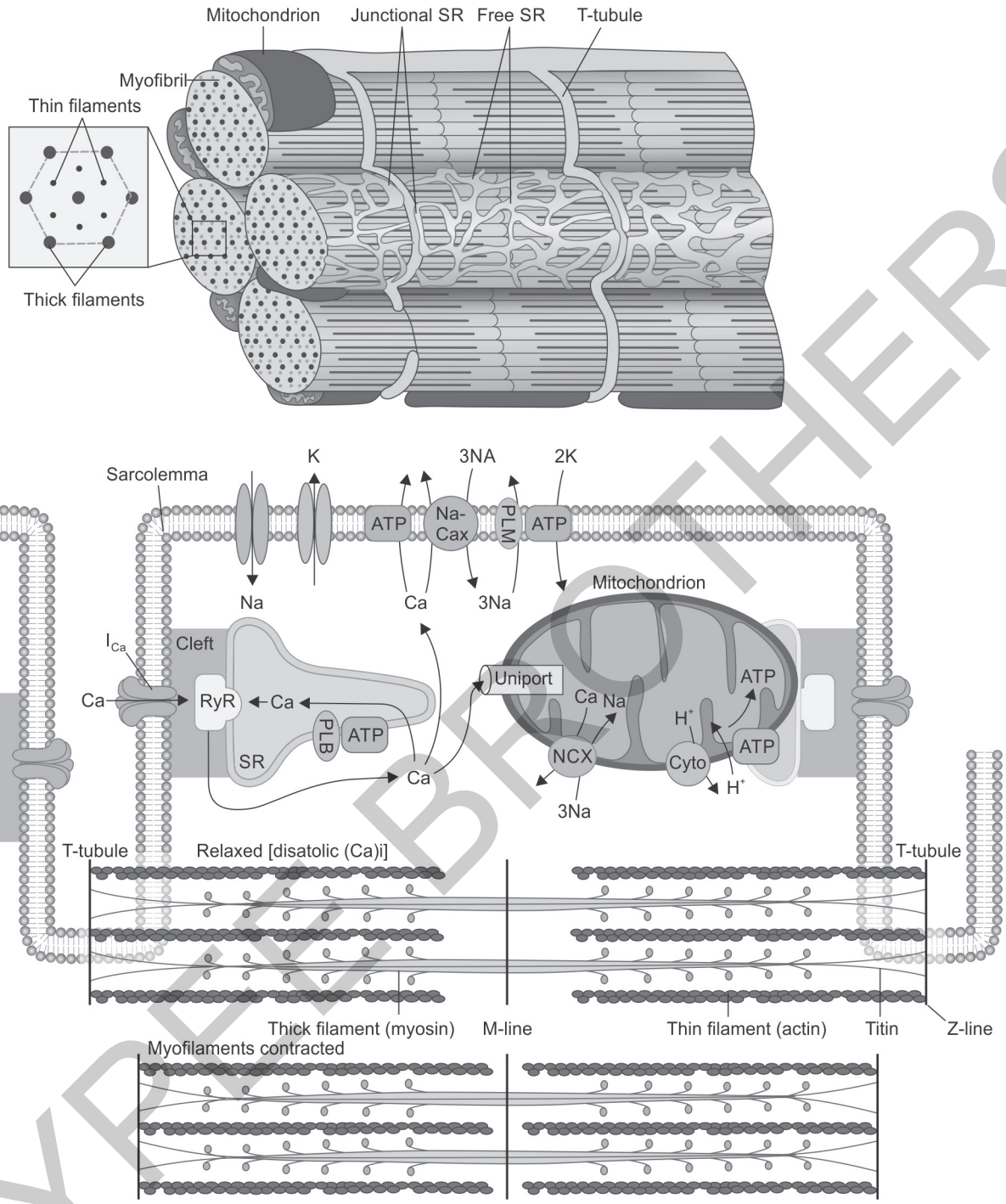

sarcomeres and end-diastolic volume. Fourth, titin may transduce mechanical stretch into the growth signals. Sustained diastolic stretch, as in volume overload condition can cause titin dependents signalling to muscle LIM protein (MLP) attached to the Z-line end of titin. MLP is known to be a stretch sensor that transmits the signal that results in the myocyte growth pattern characteristic of volume overload. This mechanism is defective in a subset of dilated cardiomyopathy patients.

When changes in diastolic length (or preload) are the cause of altered contractile-strength, it is called as Frank–Starling effect. Conditions in which contraction is strengthen independent of sarcomere length (typically by increased Calcium ion transient amplitude) are referred to as positive inotropic states or enhanced contractility.

22. Ans. b. Sarcomere

Braunwald, 12th Ed; Page 892–893 & 895

Refer to explanation for Q. No 23.

23. Ans. b. LIM protein (MLP)

Braunwald, 12th Ed; Page 892–893 & 895

Refer to explanation for Q. No 23.

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24. Ans. b. Positive Inotropic states

Braunwald, 12th Ed; Page 892–893 & 895

Refer to explanation for Q. No 23.

25. Ans. d. Positive Dromotropy

Braunwald 12th Ed; Page 899–900

During the classic adrenergic fight or flight response, cardiac myocyte beta-adrenergic receptors are activated which leads to increased cAMP production and PKA activation and consequent phosphorylation and altered function of numerous myocyte targets. This results iin an increased heart rate (positive chronotropy), increased contractility (positive inotropy), faster cardiac relaxation (positive lusitropy) and enhanced conduction velocity through the conduction system (positive dromotropy). These events enhance cardiac output by enhancing the heart rate, stroke volume, and diastolic filling. The adrenergic response is a key physiologic mechanism for increasing cardiac output in response to increased metabolic and hemodynamic demands.

26. Ans. d. Peripheral resistance

Braunwald 12th Ed; Page 904

There are five main determinants of ventricular mechanical performance, they are: (a) preload (Frank–Starling mechanism), (b) afterload; (c) contractility; (d) Lusitropy (Diastolic function) and € the heart rate.

27. Ans. d. Lewis and Wiggers

Braunwald 12th; Page 904

The cardiac cycle was fully assembled by Lewis but was first conceived by Wiggers. It describes the temporal sequence of events during cardiac cycle. The three basic events with respect to the mechanical event in the left ventricle are, LV contraction, LV relaxation and LV filling. Similar mechanical event occurs in right ventricle also.

28. Ans. a. Isovolumic contraction

Braunwald 12th Ed; Page 904

Left ventricular contraction begins with the LV pressure increased as calcium ion arrives at contractile protein after cellular depolarization triggers actin myosin interaction. This occurs soon after the upstroke of ventricular action potential indicated by QRS complex of electrocardiogram. When LV pressure exceeds that in left atrium (normally 8 to 15 mm Hg), the mitral valve closes, causing the mitral components of first heart sound. Right ventricular pressure changes are usually slightly delayed because of the electrical conduction such that tricuspid valve closure (T1) follows M1. The phase of LV contraction after mitral valve closure and before aortic opening when the LV volume is fixed is referred to as Isovolumic Contraction. As more myofibers become activated, LV pressure proceed to increase until it exceeds aortic pressure, causing the aortic valve to open. Opening of the aortic valve is followed by the phase of rapid ejection. The rate of ejection is determined by the pressure gradient across aortic valve, as well as elastic properties of aorta and the arterial tree, which undergoes systolic expansion. LV pressure rises to peak and to start fall. As myocytes Ca^{2+} starts to decline because of sarcoplasmic calcium uptake. Calcium ion dissociates from troponin C and thereby preventing further cross-bridge formation. As this state of relaxation progresses the rate of LV ejection of blood into aorta falls, that is called as "phase of reduced ejection".

29. Ans. b. In the mid part of left ventricular filling phase

Braunwald 12th Ed; Page 433–434

Following mitral valve opening the phase of rapid or early filling occurs and accounts for most of the increase in LV volume during diastole. Under normal circumstances, this is caused by a negative pressure gradient from atrium to LV apex, creating a suction effect, especially during exercise, when LV filling rate must be augmented to increase cardiac output. Such rapid filling may cause physiologic third heart sound (S3), when there is hyperkinetic circulation or a pathologic S3 when left atrial and left ventricular diastolic pressure are elevated in congestive heart failure. As pressure in the atrium and ventricle are equalize, LV filling virtually stops (diastasis, separation). Renewed filling requires that atrial pressure exceeds LV pressure, and this is achieved by atrial systole (or the LA kick), which is especially important at high heart rates, during exercise or when the left ventricle fails to relax normally such as in patients with left ventricular hypertrophy or increased chamber stiffness.

30. Ans. d. All

Braunwald 12th Ed; Page 433–434

Refer to explanation for Q. No 29.

31. Ans. a. Cardiologic systole

Braunwald 12th Ed; Page 905

The start of ventricular systole can be regarded as the beginning of isovolumic contraction, when left ventricular pressure exceeds the atrial pressure so as to bring mitral valve closure. Physiologic systole lasts from the start of isovolumic contraction to the peak of ejection phase. Physiologic diastole commences as calcium ion is taken back into sarcoplasmic reticulum, so that myocyte relaxation dominates over contraction, and as the LV pressure start to fall. Cardiologic systole is longer than physiologic systole and is demarcated by the interval between the first heart sound to the closure of aortic valve (A2). The remainder of the cardiac cycle automatically becomes cardiologic diastole. Proto diastole is the early phase of rapid filling, the time when S3 can be heard.

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32. Ans. a. Start of isovolumic contraction to the peak of the ejection phase

Braunwald 12th Ed; Page 905

Refer to explanation for Q. No 33.

33. Ans. b. Preload

Braunwald 12th Ed; Page 906

Contractility or the inotropic state is the inherent capacity of the myocardium to contact independently of changes in preload or afterload. At the molecular level, an increased inotropic state is usually explained by either enhanced calcium ion transients or enhanced myofilament calcium sensitivity and typically means a greater rate of contraction to reach a greater peak force. The preload describes the degree of myocardial stretch or distention before contract has started and is best represented at the chamber level by LV end-diastolic volume (EDV). Because, volume is difficult to measure accurately and precisely in practice, preload is often estimated by LV end-diastolic pressure. Afterload refers to the forces opposing LV ejection. Afterload is usually equal to aortic blood pressure but more accurately described as aortic impedance or elastance, which incorporates steady and oscillatory components of cardiac load. LV afterload can also be expressed by the wall stress that exists during systole.

Wall stress developed when the tension is applied to a cross sectional area and the unit are force per unit area.

34. Ans. c. Both positive inotropic effect and increased lusitropic effect

Braunwald 12th Ed; Page 906

If a larger heart volume increases the initial length of muscle fiber, to increase stroke volume and thus cardiac output, diastolic stretch of left ventricle (and increased sarcomere length) increases the force of contraction. Frank initially reported that the greater the initial LV volume, the more rapid the rate of rise, the greater the peak pressure reached, and the faster rate of relaxation. He described both positive inotropic effect and an increased lusitropic effect. These complimentary findings of Frank and Starling are often combined into Frank–Starling Law. Thus an increase in the strength of contraction can generally be categorized as either a Frank–Starling effect (increased sarcomere length) or an inotropic effect (altered calcium transient or myofilament calcium sensitivity), although both effects can occur simultaneously as during physical exercise.

35. Ans. b. Anrep effect

Braunwald 12th Ed; Page 906

When the aortic pressure is elevated abruptly, it limits ejection and tends to increase end diastolic volume, which acutely increases force and pressure at the next beat by the Frank–Starling effect. However, in a slower adaptation that takes seconds to minutes, the inotropic state of the heart increases (and calcium transients are larger). This slow force response or adaptation is referred to as the Anrep effect.

36. Ans. d. All of the above

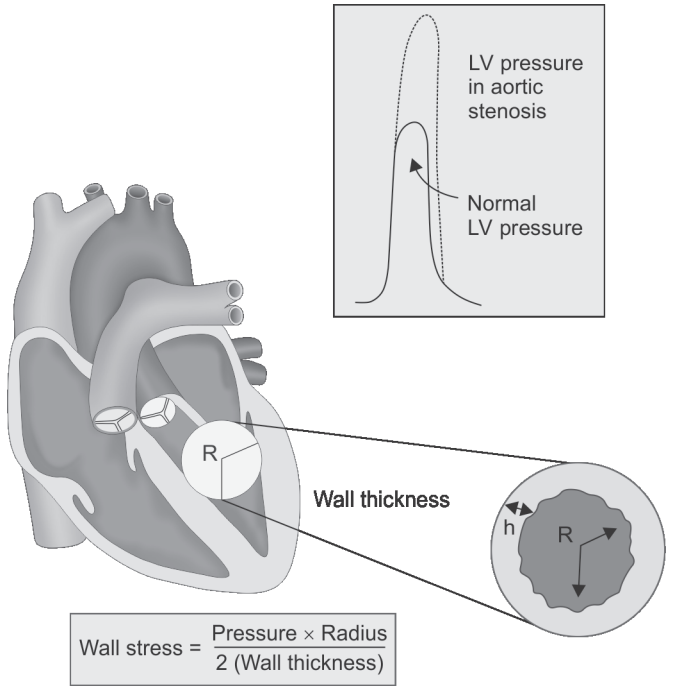
Braunwald 12th Ed; Page 906

The preload describes the degree of myocardial stretch or distention before contraction has started and is represented at the chamber level by LV end diastolic volume. Afterload refers to the forces opposing LV ejection. Afterload is more accurately described as aortic impedance or elastance. When preload increases stroke volume rises according to Starling's law, if all other factors are held constant. Conversely, when afterload increases, stroke volume drops. The preload and afterload influence each other. By the Frank–Starling Law, an increased LV volume leads to increase contractile function which in turn will increase systolic aortic pressure and thus the afterload in the subsequent contraction cycle. During LV ejection, sarcomere length progressively declines, decreasing both myofilament calcium and sensitivity and maximal force, which along with progressive calcium decline, reduces the contractile force. Afterload also dynamically changes during ejection and decline as ejection wanes.

37. Ans. a. Directly proportional to the product of pressure and radius and inversely proportional to the double of wall thickness

Braunwald 12th Ed; Page 907

Wall stress develops when the tension is applied to a cross-sectional area, and the units are force per unit area. According to Laplace's law, wall stress = (pressure × radius)/(2 × wall thickness). According to this equation,



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the larger the LV size and radius, the higher is the wall stress. Secondly, at any given radius (LV size), greater the pressure developed by the left ventricle, greater is the wall stress. An increase in wall stress is achieved either by increasing the LV size or increasing intraventricular pressure. Both of this will increase myocardial oxygen uptake because a greater rate of ATP use is required for the myofibrils to develop more tension. In cardiac hypertrophy, Laplace's law explains the effect of changes in wall thickness on wall stress. The increased wall thickness from hypertrophy balances the increased pressure, and wall stress remains unchanged during the phase of compensatory hypertrophy. Similarly, in congestive heart failure, the heart dilates so that increased radius will elevates wall stress. Moreover, in patient with congestive heart failure, the quantum of ejected blood is inadequate, the radius stays too large throughout the contractile cycle, and both end-diastolic and end-systolic wall stress is higher. This decreases LV efficiency, increases myocardial oxygen demand, and augments release of natriuretic peptide levels. The overall reduction in heart size decreases wall stress and improves LV function.

38. Ans. d. All of the above

Braunwald 12th Ed; Page 907

Preload is defined as the wall stretch at the end of diastole and therefore at the maximal resting length of sarcomere. The surrogate marker of preload is LVEDP or dimensions (the dimensions being the major or minor axis of the heart in 2-D Echocardiographic view). Afterload being the load on the contractile myocardium is the wall stress during LV ejection. Increased afterload means that increased intraventricular pressure is to be generated to open the aortic valve and then during the ejection phase. This increase is translate into increased myocardial wall stress, which can be measured either as an average value or at end-systole. Peak systolic wall stress reflects the three major components of afterload, peripheral resistance, arterial compliance and peak intraventricular pressure. Decreased arterial compliance and increased afterload can be anticipated with aortic remodeling and dilation, as seen in severe systemic hypertension or in elderly patients.

39. Ans. b. An increased heart rate progressively enhances the force of ventricular muscle contraction.

Braunwald 12th Ed; Page 908

An increased heart rate progressively enhances the force of ventricular muscle contraction, even in isolated papillary muscle preparations and isolated myocytes. This is called as "Bowditch phenomenon" or positive inotropic effect of activation or force frequency relationship. A decreased heart rate has a negative staircase effect.

40. Ans. c. Minute work (SBP × SV × HR)

Braunwald 12th Ed; Page 909

There are three determinants of myocardial oxygen uptake, they are: Preload, afterload and heart rate. External work (pressure × volume) is done by the heart, with stroke volume (cardiac output) being the volume moved against the arterial blood pressure. Volume work (associated with increased stroke volume) requires less oxygen than pressure work. Minute work can be defined as the product of systolic blood pressure, stroke volume and heart rate (SBP × SV × HR). This pressure work index takes into accounts both the double product (SBP × HR) and HR × SV (cardiac output). The pressure volume areas is another useful index of cardiac work or O_2 uptake but it requires invasive monitoring for accurate measurements.

41. Ans. b. Lower pressure in the right ventricle and pulmonary circulation.

Braunwald 12th Ed; Page 911

Right ventricular myocytes are fundamentally the same as those in left ventricle, with some minor quantitative differences in their ion channel, calcium ion handling, electrophysiology, and myofilament properties. The most important functional differences are in the chamber geometry related to Laplace's law and normal levels of pressure developed. The pressure in the right ventricle and pulmonary circulation is much lower than the left sided circulation. The right ventricle has a larger radius of curvature which would tend to increase wall tension, but it normally develops much lower pressure, which greatly reduces wall tension. Right ventricular wall thickness is also lower such that the normal characteristics of right ventricular shape and size are functionally matched to the different prevailing conditions on right ventricle. Once there is a pathophysiological condition like pulmonary hypertension where the afterload dependent is heightened, the right ventricle being poorly suited to eject against high pressure, it leads to right heart failure.

42. Ans. b. Left atrium.

Braunwald 12th Ed; Page 911

The left atrium has several important functions.

1. The left atrium functions as a blood receiving reservoir chamber;
2. It is a contractile chamber that by presystolic contraction helps to complete left ventricular filling with an atrial kick;
3. The left atrium function as a conduit that empties its contents into the left ventricle down a pressure gradient after the opening of mitral valve;

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4. It is called as "blood volume sensor" of the heart and releases atrial natriuretic peptide (ANP) in response to stretch so that ANP induced diuresis can help restore blood volume to normal;
5. Left atrium contains receptors for a afferent arms of various reflexes including mechanoreceptors that increase the sinus discharge rate, thereby contributing to the tachycardia of exercise as venous return increases (Bainbridge reflex).

43. Ans. b. All of the above.

Braunwald 12th Ed; Page 926

In failing myocardium, there are several changes occurs both in volume of cardiac myocytes and also in the volume and composition of the extra cellular matrix. Progressive myocytes loss occurs through process of necrosis, apoptosis or autophagic cell death.

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