بسم الله الرحمن الرحيم

Physiology Team 436
Cardiovascular Block

Questions File

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References:

This file contains all the questions related to the CVS block from the following textbooks:

1. Guyton and Hall Physiology Review.
2. Linda: Physiology Cases and Problems.

The aim of this file is to spare you the hassle of searching for questions in multiple references; but if you would like a copy of the books just email us at:
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5. In which phase of the ventricular muscle action potential is the potassium permeability the highest?
A) 0
B) 1
C) 2
D) 3
E) 4

Answer=**D)** During phase 3 of the ventricular muscle action potential, the potassium permeability of ventricular muscle greatly increases, which causes a more negative membrane potential.

8. Which of the following statements about cardiac muscle is most accurate?
A) The T-tubules of cardiac muscle can store much less calcium than T-tubules in skeletal muscle
B) The strength and contraction of cardiac muscle depends on the amount of calcium surrounding cardiac myocytes
C) In cardiac muscle the initiation of the action potential causes an immediate opening of slow calcium channels
D) Cardiac muscle repolarization is caused by opening of sodium channels
E) Mucopolysaccharides inside the T-tubules bind chloride ions

answer=**B)** The cardiac muscle stores much more calcium in its tubular system than skeletal muscle and is much more dependent on extracellular calcium than the skeletal muscle. An abundance of calcium is bound by the mucopolysaccharides inside the T-tubule. This calcium is necessary for contraction of cardiac muscle, and its strength of contraction depends on the calcium concentration surrounding the cardiac myocytes. At the initiation of the action potential, the fast sodium channels open first, followed later by the opening of the slow calcium channels.

17. Sympathetic stimulation of the heart
A) Releases acetylcholine at the sympathetic endings
B) Decreases sinus nodal discharge rate
C) Decreases excitability of the heart
D) Releases norepinephrine at the sympathetic endings
E) Decreases cardiac contractility

Answer=**D)** Increased sympathetic stimulation of the heart increases heart rate, atrial contractility, and ventricular contractility and also increases norepinephrine release at the ventricular sympathetic nerve endings. It does not release acetylcholine. It does cause an increased sodium permeability of the A-V node, which increases the rate of upward drift of the membrane potential to the threshold level for self-excitation, thus increasing heart rate.

29. What is the resting membrane potential of the sinus nodal fibers?
A) 2100 mV
B) 290 mV
C) 280 mV

D) 255 mV
E) 220 mV

Answer=**D)** The resting membrane potential of the sinus nodal fibers is 255 mV, and this is in contrast with the 285 to 290 mV membrane potential of cardiac muscle. Other major differences between the sinus nodal fibers and ventricular muscle fibers are that the sinus fibers exhibit self-excitation from inward leaking of sodium ions.

31. Sympathetic stimulation of the heart normally causes
which of the following conditions?
A) Acetylcholine release at the sympathetic endings
B) Decreased heart rate
C) Decreased rate of conduction of the cardiac
impulse
D) Decreased force of contraction of the atria
E) Increased force of contraction of the ventricles

Answer=**E)** Sympathetic stimulation of the heart normally causes an increased heart rate, increased rate of conduction of the cardiac impulse and increased force of contraction in the atria and ventricles. However, it does not cause acetylcholine release at the sympathetic endings because they contain norepinephrine. Parasympathetic stimulation causes acetylcholine release. The sympathetic nervous system firing increases the permeability of the cardiac muscle fibers, the S-A node, and the A-V node to sodium and calcium.

24. Which of the following are caused by acetylcholine?
A) Hyperpolarization of the S-A node
B) Depolarization of the A-V node
C) Decreased permeability of the S-A node to potassium
ions
D) Increased heart rate
E) Increased permeability of the cardiac muscle to
calcium ions

answer=**A)** Acetylcholine does not depolarize the A-V node or increase permeability of the cardiac muscle to calcium ions but causes hyperpolarization of the S-A node and the A-V node by increasing permeability to potassium ions. This results in a decreased heart rate.

4. Currents caused by opening of which of the following channels
contribute to the repolarization phase of the action potential of
ventricular muscle fibers?
A) Na+ channels
B) Cl– channels
C) Ca2+ channels
D) K+ channels
E) HCO3– channels
Answer=D

19. Which of the following best explains how sympathetic

stimulation affects the heart?
A) Permeability of the S-A node to sodium decreases
B) Permeability of the A-V node to sodium decreases
C) Permeability of the S-A node to potassium increases
D) There is an increased rate of upward drift of the resting membrane potential of the S-A node
E) Permeability of the cardiac muscle to calcium Decreases

Answer=**D)** During sympathetic stimulation, the permeabilities of the S-A node and the A-V node increase. Also, the permeability of cardiac muscle to calcium increases resulting in an increased contractile strength. In addition, there is an upward drift of the resting membrane potential of the S-A node. Increased permeability of the S-A node to potassium does not occur during sympathetic stimulation.

29. What is the resting membrane potential of the sinus

14. A 25-year-old, well-conditioned athlete weighs 80 kg (176 lb). During maximal sympathetic stimulation, what is the plateau level of his cardiac output function curve?
A) 3 L/min
B) 5 L/min
C) 10 L/min
D) 13 L/min
E) 25 L/min

E) The normal plateau level of the cardiac output function curve is 13 L/min. This level decreases in any kind of cardiac failure and increases markedly during sympathetic stimulation.

20. Which of the following structures will have the slowest rate of conduction of the cardiac action potential?
A) Atrial muscle
B) Anterior internodal pathway
C) A-V bundle fibers
D) Purkinje fibers
E) Ventricular muscle

C) The atrial and ventricular muscles have a relatively rapid rate of conduction of the cardiac action potential, and the anterior internodal pathway also has fairly rapid conduction of the impulse. However, A-V bundle myofibrils have a slow rate of conduction because their sizes are considerably smaller than the sizes of the normal atrial and ventricular muscle. Also, their slow conduction is partly caused by diminished numbers of gap junctions between successive muscle cells in the conducting pathway, causing a great resistance to conduction of the excitatory ions from one cell to the next.

21. If the S-A node discharges at 0.00 seconds, when will the action potential normally arrive at the epicardial

surface at the base of the left ventricle?
A) 0.22 sec
B) 0.18 sec
C) 0.16 sec
D) 0.12 sec
E) 0.09 sec

A) After the S-A node discharges, the action potential travels through the atria, through the A-V bundle system and finally to the ventricular septum and throughout the ventricle. The last place that the impulse arrives is at the epicardial surface at the base of the left ventricle, which requires a transit time of 0.22 sec.

22. If the S-A node discharges at 0.00 seconds, when will the action potential normally arrive at the A-V bundle (bundle of His)?
A) 0.22 sec
B) 0.18 sec
C) 0.16 sec
D) 0.12 sec
E) 0.09 sec

D) The action potential arrives at the A-V bundle at 0.12 sec. It arrives at the A-V node at 0.03 sec and is delayed.09 sec in the A-V node, which results in an arrival time at the bundle of His of 0.12 sec.

23. Which of the following conditions at the S-A node will cause heart rate to decrease?
A) Increased norepinephrine levels
B) Increased sodium permeability
C) Increased calcium permeability
D) Increased potassium permeability
E) Decreased acetylcholine levels

D) Increases in sodium and calcium permeability at the S-A node result in an increased heart rate. An increased potassium permeability causes a hyperpolarization of the S-A node, which causes the heart rate to decrease.

25. What is the membrane potential (threshold level) at which the S-A node discharges?
A) 240 mV
B) 255 mV
C) 265 mV
D) 285 mV

E) 2105 mV

A) The normal resting membrane potential of the S-A node is 255 mV. As the sodium leaks into the membrane an upward drift of the membrane potential occurs until it reaches 240 mV. This is the threshold level that initiates the action potential at the S-A node.

30. If the Purkinje fibers, situated distal to the A-V junction, become the pacemaker of the heart, what is the expected heart rate?
A) 30/min
B) 50/min
C) 60/min
D) 70/min
E) 80/min

A) If the Purkinje fibers are the pacemaker of the heart, the heart rate ranges between 15 and 40 beats/min. In contrast, the rate of firing of the A-V nodal fibers are 40 to 60 times a minute, and the sinus node fires at 70 to 80 times per minute. If the sinus node is blocked for some reason, the A-V node will take over as the pacemaker; and if the A-V node is blocked, the Purkinje fibers will take over as the pacemaker of the heart.

26. Which of the following conditions at the A-V node will cause a decrease in heart rate?
A) Increased sodium permeability
B) Decreased acetylcholine levels
C) Increased norepinephrine levels
D) Increased potassium permeability
E) Increased calcium permeability

D) An increase in potassium permeability causes a decrease in the membrane potential of the A-V node. Thus, it will be extremely hyperpolarized, making it much more difficult for the membrane potential to reach its threshold level for conduction. This results in a decrease in heart rate. Increases in sodium and calcium permeability and norepinephrine levels increase the membrane potential, causing a tendency to increase the heart rate.

27. If the ventricular Purkinje fibers become the pacemaker of the heart, what is the expected heart rate?
A) 30/min
B) 50/min
C) 65/min
D) 75/min
E) 85/min

A) If there is a failure in conduction of the S-A nodal impulse to the A-V node or if the S-A node stops firing, the A-V node will take over as the pacemaker of the heart. The intrinsic rhythmical

rate of the A-V node is 40 to 60 times per minute. If the Purkinje fibers take over as pacemakers, the heart rate will be between 15 and 40 beats/min.

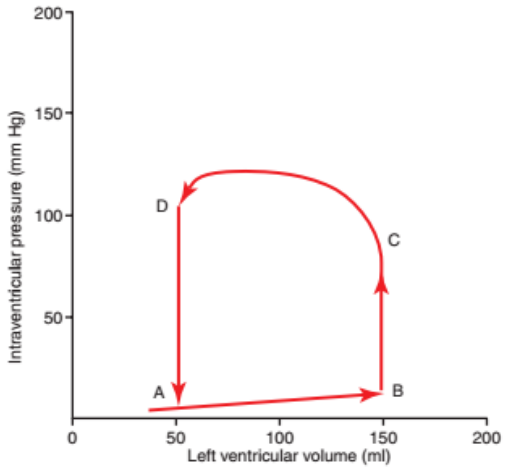
28. What is the normal total delay of the cardiac impulse in the A-V node and the A-V bundle system?
A) 0.03 sec
B) 0.06 sec
C) 0.09 sec
D) 0.13 sec
E) 0.17 sec

D) The impulse coming from the S-A node to the A-V node arrives at 0.03 sec. Then there is a total delay of 0.13 sec in the A-V node and bundle system allowing the impulse to arrive at the ventricular septum at 0.16 sec.

2. Which of the following normally has a slowly depolarizing “prepotential”?
A) sinoatrial node
B) atrial muscle cells
C) bundle of His
D) Purkinje fibers
E) ventricular muscle cells

Answer =A

A 60-year-old woman has a resting heart rate of 70 beats/min, arterial pressure is 130/85 mm Hg, and body temperature is normal. Her pressure-volume diagram of the left ventricle is shown above.



When does the second heart sound occur in the ven-tricular pressure–volume relationship?

A) At point D

B) Between point A and point B

C) Between point B and point C

D) Between point C and point D

E) Between point D and point A

A) During the ejection phase, the aortic and pulmo-nary valves open and blood flows into the aorta and pulmonary artery. The ejection phase is between C and D, so the aortic and pulmonary valves open at C and then close at D. The closing of these valves causesthe second heart sound.

When does the third heart sound occur in the ventri-cular pressure–volume relationship?

A) At point D

B) Between point A and point B

C) Between point B and point C

D) Between point C and point D

E) Between point D and point A

B) Between points A and B is the period of ventricu-lar filling. The vibration of the ventricular walls makesthis sound after sufficient blood has entered the ven-tricular chambers.

4. What is her ventricular ejection fraction?

A) 33%

B) 50%

C) 60%

D) 67%

E) 80%

D) The ejection fraction is the stroke volume/end diastolic volume. Stroke volume is 100 ml, and the end systolic volume at point D is 150 ml. This gives you an ejection fraction of 0.667 or in terms of percentage 66.7%.

In a resting adult, the typical ventricular ejection frac-tion has what value?

A) 20%

B) 30%

C) 40%

D) 60%

E) 80%

D) The typical ejection fraction is 60%, and lower values are indicative of a weakened heart.

A 30-year-old man has an ejection fraction of 0.25 and an end systolic volume of 150 ml. What is his enddiastolic volume?

A) 50 ml

B) 100 ml

C) 125 ml

D) 200 ml

E) 250 ml

D) The end diastolic volume is always greater than theend systolic volume. Multiplication of the ejectionfraction by the end diastolic volume gives you thestroke volume, which is 50 ml in this problem. There-fore, the end diastolic volume is 50 ml greater than the end systolic volume and has a value of 200 ml.

Which of the following events occurs at the end of the period of ventricular ejection?

A) A-V valves close

B) Aortic valve opens

C) Aortic valve remains open

D) A-V valves open

E) Pulmonary valve closes

E) At the end of ventricular ejection, both the aorticvalves and the pulmonary valves close. This is followedby the period of isovolumic relaxation.

Which of the following phases of the cardiac cycle fol-lows immediately after the beginning of the QRS wave?

A) Isovolumic relaxation

B) Ventricular ejection

C) Atrial systole

D) Diastasis

E) Isovolumic contraction

E) Immediately after the QRS wave, the ventriclesbegin to contract and the first phase that occurs is isovolumic contraction. This occurs before the ejec-tion phase and increases the ventricular pressure enough to mechanically open the aortic and pulmo-nary valves.

Which of the following events is associated with thefirst heart sound?

A) Closing of the aortic valve

B) Inrushing of blood into the ventricles during diastole

C) Beginning of diastole

D) Opening of the A-V valves

E) Closing of the A-V valves

E) As seen in Chapter 9, the first heart sound bydefinition occurs just after the ventricular pressure exceeds the atrial pressure. This causes the A-V valves to mechanically close. The second heart sound occurs when the aortic and pulmonary valves close.

Case 1

This case is designed to take you through important basic calculations involving the cardiovascular system. Use the information provided in Table 2–1 to answer the questions. Part of the challenge in answering these questions will be in deciding which information you need in order to perform each calculation. Good luck!

table 2–1 Cardiovascular Values for Case 10

Parameter	Value
Systolic pressure (aorta)	124 mm Hg
Diastolic pressure (aorta)	82 mm Hg
R-R interval	800 msec
Left ventricular end-diastolic volume	140 mL
Left ventricular end-systolic volume	70 mL
Mean pulmonary artery pressure	15 mm Hg
Right atrial pressure	2 mm Hg
Left atrial pressure	5 mm Hg
O_2 consumption (whole body)	250 mL/min
O_2 content of systemic arterial blood	0.20 mL O_2/mL blood
O_2 content of pulmonary arterial blood	0.152 mL O_2/mL blood

R-R interval, time between R-waves on the ECG.

Questions

1. Mean arterial pressure is not the simple average of systolic and diastolic pressures. Why not? How is mean arterial pressure estimated? From the information given in Table 2–1, calculate the mean arterial pressure in this case.

2. Calculate the stroke volume, cardiac output, and ejection fraction of the left ventricle.

3. Calculate cardiac output using the Fick principle.

4. What is the definition of total peripheral resistance (TPR)? Which equation describes the relationship between TPR, arterial pressure, and cardiac output? What is the value of TPR in this case?

5. How is pulmonary vascular resistance calculated? What is the value of pulmonary vascular resis-tance in this case? Compare the calculated values for pulmonary vascular resistance and TPR and explain any difference in the two values.

6. What is the total blood flow (in mL/min) through all of the pulmonary capillaries?

7. What is the total blood flow (in mL/min) through all of the systemic arteries?

8. What information, in addition to that provided in Table 2–1, is needed to calculate the resistance of the renal vasculature?

9. If the diameter of the aorta is 20 mm, what is the velocity of aortic blood flow? Would you expect the velocity of blood flow in systemic capillaries to be higher than, lower than, or the same as the veloc-ity of blood flow in the aorta?

ANSWERS

1. Systemic arterial pressure is not a single value because arterial pressure varies over the course of each cardiac cycle. Its highest value is systolic pressure, which is measured just after the blood is ejected from the left ventricle into the aorta (i.e., systole). Its lowest value is diastolic pressure, which is measured as the blood flows from the arteries into the veins and back to the heart (i.e., diastole).Mean arterial pressure cannot be calculated as the simple average of systolic and diastolic pressures because averaging does not take into account the fact that a greater fraction of each cardiaccycle is spent in diastole (approximately two-thirds) than in systole (approximately one-third).Thus, mean arterial pressure is closer to diastolic pressure than to systolic pressure. Figure 2–1shows an arterial pressure tracing over a single cardiac cycle. The difference between systolic pressure and diastolic pressure is called pulse pressure.

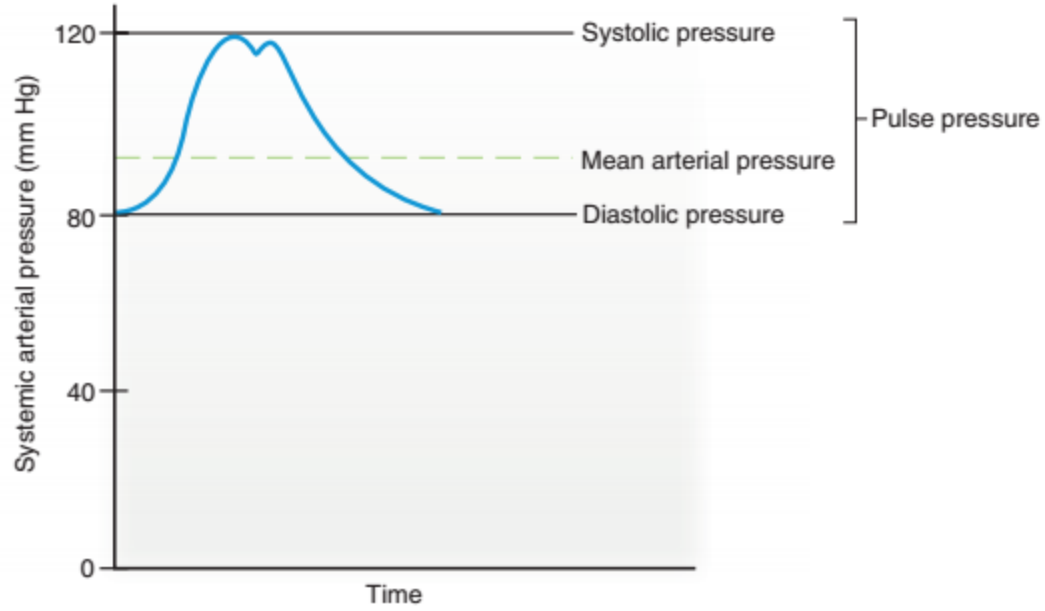


FIGURE 2–1. Systemic arterial pressure during the cardiac cycle.

Although this approach is impractical, the mean arterial pressure can be determined by measuring the area under the arterial pressure curve. Alternatively, the mean arterial pressure can be estimated as follows:

Mean arterial pressure = diastolic pressure + 1/3 pulse pressure

= diastolic pressure + 1/3 (systolic pressure – diastolic pressure)

Where Diastolic pressure = lowest value for arterial pressure in a cardiac cycle

Systolic pressure = highest value for arterial pressure in a cardiac cycle

Pulse pressure = systolic pressure – diastolic pressure

Therefore, in this case:

Mean arterial pressure = 82 mm Hg + 1/3 (124 – 82 mm Hg)

= 82 mm Hg + 1/3 (42 mm Hg)

= 82 + 14 mm Hg

= 96 mm Hg

2- These calculations concern the cardiac output of the left ventricle. The basic relationships are as follows:

Stroke volume = end-diastolic volume - end-systolic volume

Where Stroke volume = volume ejected by the ventricle during systole (mL)

End-diastolic volume = volume in the ventricle before ejection (mL)

End-systolic volume = volume in the ventricle after ejection (mL)

Cardiac output = stroke volume ¥ heart rate

Where Cardiac output = volume ejected by the ventricle per minute (mL/min)

Stroke volume = volume ejected by the ventricle (mL)

Heart rate = beats/min

Ejection fraction = stroke volume/end-diastolic volume

Where Ejection fraction = fraction of the end-diastolic volume ejected in one stroke

Now we can use these basic equations to calculate the stroke volume, cardiac output, and ejection fraction in this case.

Stroke volume = left ventricular end-diastolic volume – left ventricular end-systolic volume

= 140 mL – 70 mL

= 70 mL

Cardiac output is the volume ejected by the left ventricle per minute. It is calculated as the product of stroke volume (determined to be 70 mL) and heart rate. Heart rate is not given in Table 2–1, but it can be calculated from the R-R interval. “R” is the R-wave on the electrocardiogram and represents electrical activation of the ventricles. The R-R interval is the time elapsed from one R-wave to the next(Fig. 2–2). It is also called cycle length (i.e., time elapsed in one cardiac cycle).

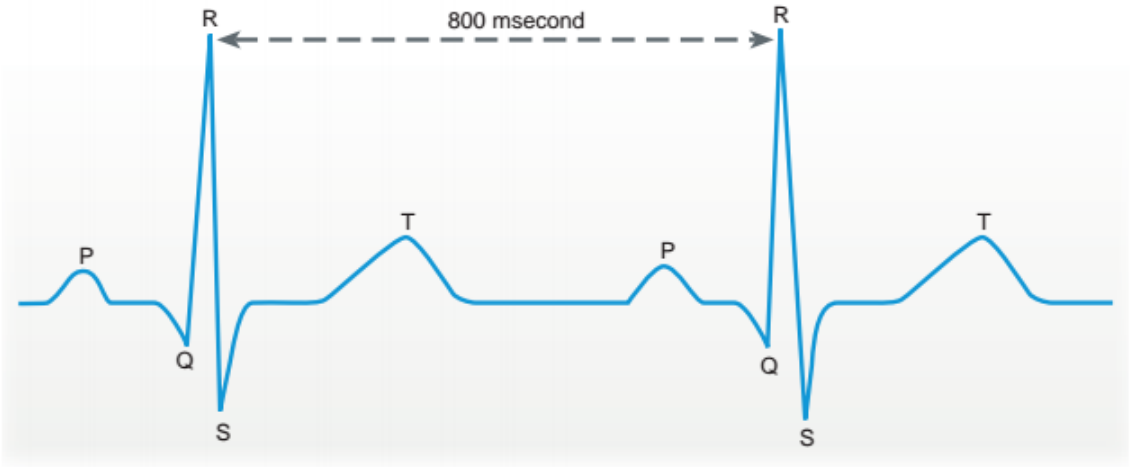


FIGURE 2–2. ECG measured from lead II. The interval between R-waves is the cycle length.

Cycle length can be used to calculate the heart rate as follows:

Heart rate = 1/cycle length

= 1/800 msec

= 1/0.8 sec

= 1.25 beats/sec

= 75 beats/min

Cardiac output = stroke volume × heart rate

= 70 mL × 75 beats/min

= 5,250 mL/min

Ejection fraction = stroke volume/end-diastolic volume

= 70 mL/140 mL

= 0.5, or 50%

3. As shown in Question 2, we calculate cardiac output as the product of stroke volume and heart rate.However, we measure cardiac output by the Fick principle of conservation of mass. The Fick principle for measuring cardiac output employs two basic assumptions: (i) pulmonary blood flow (the cardiac output of the right ventricle) equals systemic blood flow (the cardiac output of the left ventricle) in the steady state, and (ii) the rate of O2 utilization by the body is equal to the difference between the amount of O2 leaving the lungs in pulmonary venous blood and the amount of O2 returning to the lungs in pulmonary arterial blood. This relationship can be stated mathematically as follows:

O2 consumption = cardiac output × [O2]pulmonary vein – cardiac output × [O2]pulmonary artery

$$\text{Cardiac output} = \frac{\text{O}_2 \text{ consumption}}{[\text{O}_2]_{\text{pulmonary vein}} - [\text{O}_2]_{\text{pulmonary artery}}}$$

Rearranging to solve for cardiac output:

Where

Cardiac output = cardiac output (mL/min)

O2 consumption = O2 consumption by the body (mL O2/min)

[O2]pulmonary vein = O2 content of pulmonary venous blood (mL O2/mL blood)

[O2]pulmonary artery = O2 content of pulmonary arterial blood (mL O2/mL blood)

In this case, cardiac output can be calculated by substituting values from Table 2–1. To find the appropriate values in the table, recall that systemic arterial blood is equivalent to pulmonary venous blood.

$$\begin{aligned}\text{Cardiac output} &= \frac{250 \text{ mL/min}}{0.20 \text{ mL O}_2/\text{mL blood} - 0.152 \text{ mL O}_2/\text{mL blood}} \\ &= \frac{250 \text{ mL/ min}}{0.048 \text{ mL O}_2/\text{mL blood}} \\ &= 5,208 \text{ mL/min}\end{aligned}$$

Thus, the value for cardiac output measured by the Fick principle (5,208 mL/min) is very close to the value of 5,250 mL/min calculated as the product of stroke volume and heart rate in Question 2.

4. TPR is the collective resistance to blood flow that is provided by all of the blood vessels on the systemic side of the circulation. These blood vessels include the aorta, large and small arteries, arterioles, capillaries, venules, veins, and vena cava. Most of this resistance resides in the arterioles.The fundamental equation of the cardiovascular system relates blood flow, blood pressure, and resistance. The relationship is analogous to the one that relates current (I), voltage (V), and resistance (R) in electrical circuits as expressed by the Ohm's law ($I = \Delta V/R$). Blood flow is analogous to current flow, blood pressure is analogous to voltage, and hemodynamic resistance is analogous to electrical resistance. Thus, the equation for blood flow is:

$Q = DP/R$

or, rearranging and solving for R,

$R = DP/Q$

where

Q = blood flow (mL/min)

ΔP = pressure difference (mm Hg)

R = resistance (mm Hg/mL per min)

Therefore, to calculate TPR, it is necessary to know the total blood flow through the systemic circulation (i.e., cardiac output of the left ventricle) and the pressure difference across the entire systemic circulation. In solving this problem, it may be helpful to visualize the organization and circuitry of the cardiovascular system (Fig. 2–3).

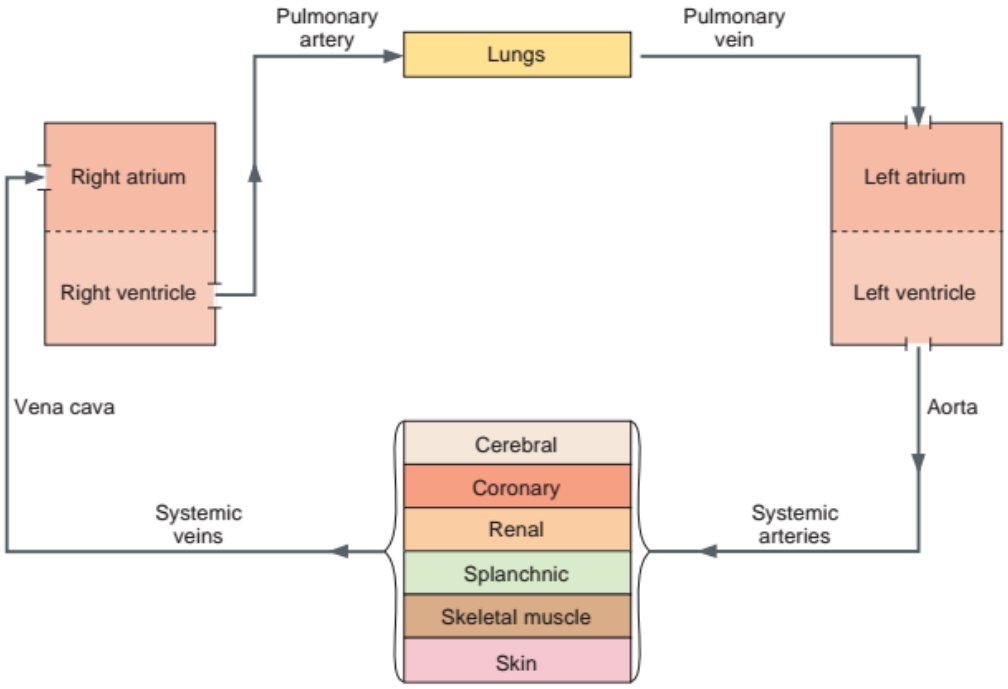


FIGURE 2–3. Circuitry of the cardiovascular system. (Reprinted, with permission, from Costanzo LS. *BRS Physiology*. 5th ed. Baltimore: Lippincott Williams & Wilkins; 2011:65.)

Cardiac output was calculated by different methods in Questions 2 and 3 as 5,250 mL/min and 5,208 mL/min, respectively. These values are similar, and we can (arbitrarily) take the average value (5,229 mL/min) to represent cardiac output. The pressure difference across the systemic circulation (ΔP) is the difference in pressure at the inflow and outflow points. Inflow pressure to the systemic circulation is aortic pressure, and outflow pressure from the systemic circulation is right atrial pressure. In Question 1, the mean aortic pressure was calculated as 96 mm Hg, which is also approximately the value of mean arterial pressure. The right atrial pressure is given in Table 2–1 as 2 mm Hg. Thus, ΔP across the systemic circulation is 96 mm Hg – 2 mm Hg, or 94mmHg.Resistance (R), which represents TPR, is:

R = DP/Q

or

TPR = (mean arterial pressure – right atrial pressure)/cardiac output

= (96 mm Hg – 2 mm Hg)/5,229 mL/min

= 94 mm Hg/5,229 mL/min

= 0.018 mm Hg/mL per min

5. Pulmonary vascular resistance (PVR) is calculated in the same way that TPR was calculated in Question

4. We need to know the values for pulmonary blood flow (cardiac output of the right ventricle) and the pressure difference across the pulmonary circulation. To determine the pulmonary blood flow,

it is necessary to understand that the left and right sides of the heart operate in series (i.e., blood flows sequentially from the left heart to the right heart and back to the left heart). Thus, in the steady state, the cardiac output of the right ventricle (pulmonary blood flow) equals the cardiac output of the left ventricle, or 5,229 mL/min. The pressure difference across the pulmonary circulation is inflow pressure minus outflow pressure. The inflow pressure is mean pulmonary artery pressure (15 mm Hg),and the outflow pressure is left atrial pressure (5 mm Hg). Thus, pulmonary vascular resistance is:

PVR = ΔP/Q

= (mean pulmonary artery pressure – left atrial pressure)/cardiac output

= (15 mm Hg – 5 mm Hg)/5,229 mL/min

= 10 mm Hg/5,229 mL/min

= 0.0019 mm Hg/mL per min

Although pulmonary blood flow is equal to systemic blood flow, pulmonary vascular resistance is only one-tenth the value of systemic vascular resistances. How is this possible? Since pulmonary resistance is lower than systemic resistance, shouldn't pulmonary blood flow be higher than systemic blood flow? No, because pulmonary pressures are also much lower than systemic pressures.Thus, pulmonary blood flow can be exactly equal to systemic blood flow because pulmonary vascular resistance and pressures are proportionately lower than systemic vascular resistance and pressures.

6. Because of the serial arrangement of blood vessels within the lungs (i.e., blood flows from the pulmonary artery to smaller arteries to arterioles to capillaries to veins), the total blood flow at any level of the pulmonary vasculature (e.g., at the level of all of the pulmonary capillaries) is the same. Thus,the total blood flow through all of the pulmonary capillaries equals the total blood flow through the pulmonary artery, which is the cardiac output of the right ventricle, or 5,229 mL/min.

7. This question addresses the same issue as Question 6, but as applied to the systemic circulation.Because of the serial arrangement of blood vessels in the systemic circulation (i.e., blood flows from the aorta to smaller arteries to arterioles, and so forth), the total blood flow at any level of the systemic vasculature (e.g., at the level of all of the arteries) is the same. Thus, the total blood flow through all of the systemic arteries equals the cardiac output of the left ventricle, or 5,229 mL/min.

8. The principles that were used to determine TPR (or to determine pulmonary vascular resistance) can also be used to calculate the vascular resistance of individual organs (e.g., kidney). Recall how the pressure, flow, and resistance relationship was rearranged to solve for resistance: R = ΔP/Q. R can also represent the resistance of the blood vessels in an individual organ (e.g., kidney), ΔP can represent the pressure difference across the organ's vasculature (e.g., for the kidney, the pressure in the renal artery minus the pressure in the renal vein), and Q can represent the organ's blood flow (e.g.,renal blood flow).Actually, none of the exact information needed to calculate renal vascular resistance is available in Table 2–1 or from the previous calculations. Renal arterial pressure is close, but not exactly equal, to the mean arterial pressure that was calculated for the aorta in Question 1. The mean pressure in large "downstream" arteries is slightly lower than the pressure in the aorta. (It must be lower in order for blood to flow in the right direction, that is, from the aorta to the distal arteries.) Like the pressure in any large vein,

renal venous pressure must be slightly higher than the right atrial pressure. Because of the parallel arrangement of arteries off the aorta, renal blood flow is only a fraction of the total systemic blood flow.

9. The velocity of blood flow is the rate of linear displacement of blood per unit time:

$v = Q/A$

where

v = linear velocity of blood (cm/min)

Q = blood flow (mL/min)

A = cross-sectional area of a blood vessel (cm2)

In words, velocity is proportional to blood flow and is inversely proportional to the crosssectional area of the blood vessel. Blood flow through the aorta is the total systemic blood flow, or cardiac output, which is 5,229 mL/min. The cross-sectional area can be calculated from the diameter of the aorta, which is 20 mm (radius, 10 mm).

$$
\begin{aligned}
V &= \frac{Q}{\pi r^2} \\
&= \frac{5{,}229\ \text{ml/min}}{3.14 \times (10\ \text{mm})^2} \\
&= \frac{5{,}229\ \text{ml/min}}{3.14 \times 1\ \text{cm}^2} \\
&= \frac{5{,}229\ \text{cm}^3/\text{min}}{3.14\ \text{cm}^2} \\
&= 1{,}665\ \text{cm/min}
\end{aligned}
$$

Based on the inverse relationship between velocity and radius of blood vessels, the velocity of blood flow should be lower in all of the capillaries than in the aorta. (Of course, a single capillary has a smaller radius than the aorta, but all of the capillaries have a larger collective radius and crosssectional area than the aorta.)

Case2

Ventricular Pressure–Volume Loops

Figure 2–4 shows a pressure–volume loop for the left ventricle. This loop shows the relationship between left ventricular pressure (in mm Hg) and left ventricular volume (in mL) over a single cardiac cycle. Use Figure 2–4 to answer the following questions.

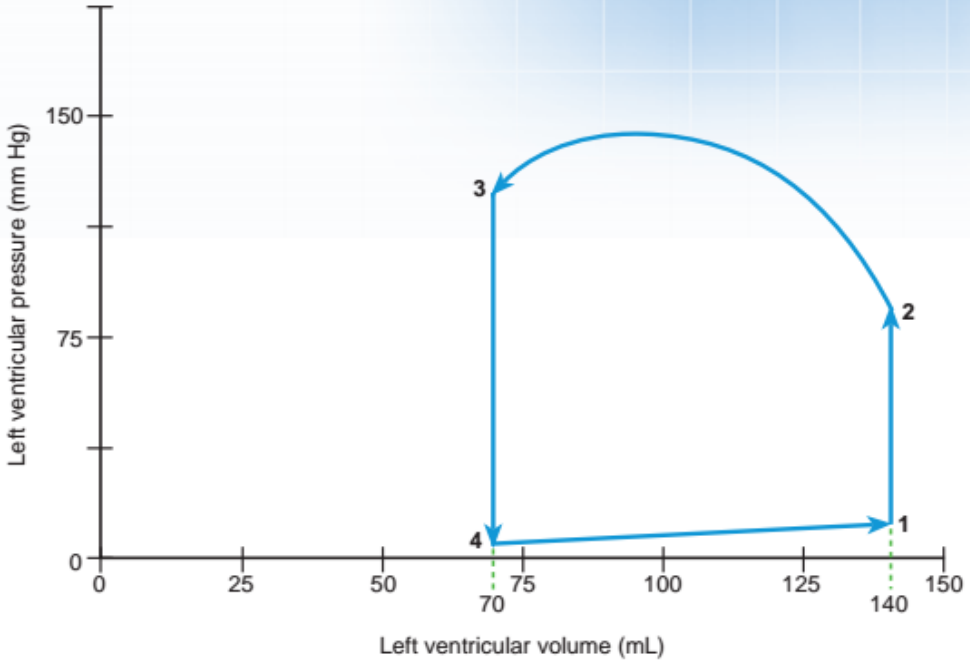


FIGURE 2–4. Left ventricular pressure–volume loop. (Adapted, with permission, from Costanzo LS. *BRS Physiology*. 5th ed. Baltimore: Lippincott Williams & Wilkins; 2011:77.)

Questions

1. Describe the events that occur in the four segments between numbered points on the pressure–volume loop (e.g., 1 → 2, 2 → 3). Correlate each segment with events in the cardiac cycle.

2. According to Figure 2–4, what is the value for left ventricular end-diastolic volume? What is the value for end-systolic volume?

3. What is the approximate value for stroke volume? What is the approximate value for ejection fraction?

4. Which portion, or portions, of the pressure–volume loop correspond to diastole? To systole?

5. Which portions of the pressure–volume loop are isovolumetric?

6. At which numbered point does the aortic valve open? At which numbered point does the aorticvalve close? At which numbered point does the mitral valve open?

7. At which numbered point, or during which segment, would the first heart sound be heard?

8. At which numbered point, or during which segment, would the second heart sound be heard?

9. Superimpose a new pressure–volume loop to illustrate the effect of an increase in left ventricular end-diastolic volume (i.e., increased preload). What is the effect on stroke volume?

10. Superimpose a new pressure–volume loop to illustrate the effect of an increase in contractility.What is the effect on end-systolic volume? What is the effect on ejection fraction?

11. Superimpose a new pressure–volume loop to illustrate the effect of an increase in aortic pressure(i.e., increased afterload). What is the effect on end-systolic volume? What is the effect on ejection fraction?

ANSWERS

1. Figure 2–4 shows a single left ventricular cycle of contraction, ejection of blood, relaxation, and filling (to begin another cycle). This figure can be used to describe the events as follows. 1 → 2 is isovolumetric contraction. During this phase, the ventricle (which was previously filled from the atrium) is contracting. Contraction causes a steep increase in ventricular pressure. However,because the aortic valve is closed, no blood is ejected and left ventricular volume remains constant(i.e., is isovolumetric). 2 → 3 is ventricular ejection. The ventricle is still contracting, causing ventricular pressure to increase further. The aortic valve is now open, and blood is ejected from the left ventricle, which causes ventricular volume to decrease. 3 → 4 is isovolumetric relaxation. The left ventricle relaxes, and ventricular pressure decreases. Both the aortic and the mitral valves are closed, and ventricular volume remains constant. 4 → 1 is ventricular filling. The left ventricle is still relaxed, but now the mitral valve is open and the ventricle is filling with blood from the atrium.Because the ventricle is relaxed, the increase in ventricular volume causes only a small increase in ventricular pressure.

2. End-diastolic volume is the volume present in the ventricle after filling is complete, but before any blood is ejected into the aorta. Therefore, end-diastolic volume is present at points 1 and2 (approximately 140 mL). End-systolic volume is the volume that remains in the left ventricle after ejection is complete, but before the ventricle fills again (i.e., the volume at points 3 and 4, which is approximately 70 mL).

3. Stroke volume is the volume ejected during systole (ventricular ejection). Thus, stroke volume is represented by the width of the pressure–volume loop, or approximately 70 mL (140 mL – 70 mL).Ejection fraction is stroke volume expressed as a fraction of end-diastolic volume (i.e., stroke volume/end-diastolic volume), or 70 mL/140 mL, or 0.5 (50%).

4. Diastole is the portion of the cardiac cycle when the ventricle is relaxed (i.e., is not contracting).Diastole corresponds to segments 3 → 4 (isovolumetric relaxation) and 4 → 1 (ventricular filling).Systole is the portion of the cardiac cycle when the ventricle is contracting. Thus, systole corresponds to segments 1 → 2 (isovolumetric contraction) and 2 → 3 (ventricular ejection).

5. By definition, isovolumetric portions of the ventricular cycle are those in which ventricular volume is constant (i.e., the ventricle is neither filling with blood nor ejecting blood). Isovolumetric segments are 1 → 2 and 3 → 4.

6. The aortic valve opens at point 2, when ventricular pressure exceeds aortic pressure. Opening of the aortic valve is followed immediately by ejection of blood and a decrease in ventricular volume. The aortic valve closes at point 3, and ejection of blood ceases. The mitral valve (the atrioventricular valve of the left heart) opens at point 4, and ventricular filling begins.

7. The first heart sound corresponds to closure of the atrioventricular valves. This closure occurs at the end of ventricular filling, at the beginning of isovolumetric contraction. Thus, the first heart sound occurs at point 1.

8. The second heart sound corresponds to closure of the aortic valve, at point 3.

9. End-diastolic volume (preload) is the volume of blood contained in the ventricle just before contraction. Therefore, an increase in ventricular end-diastolic volume (e.g., produced by an infusion of saline) means the ventricle has filled to a greater volume during diastole. In Figure 2–5, point 1 shifts to the right to represent the increased end-diastolic volume. The Frank–Starling relationship for the ventricle states that the greater the end-diastolic volume, the greater the stroke volume. Therefore, without any change in contractility, an increase in end-diastolic volume causes an increase in stroke volume, as evidenced by increased width of the pressure–volume loop.

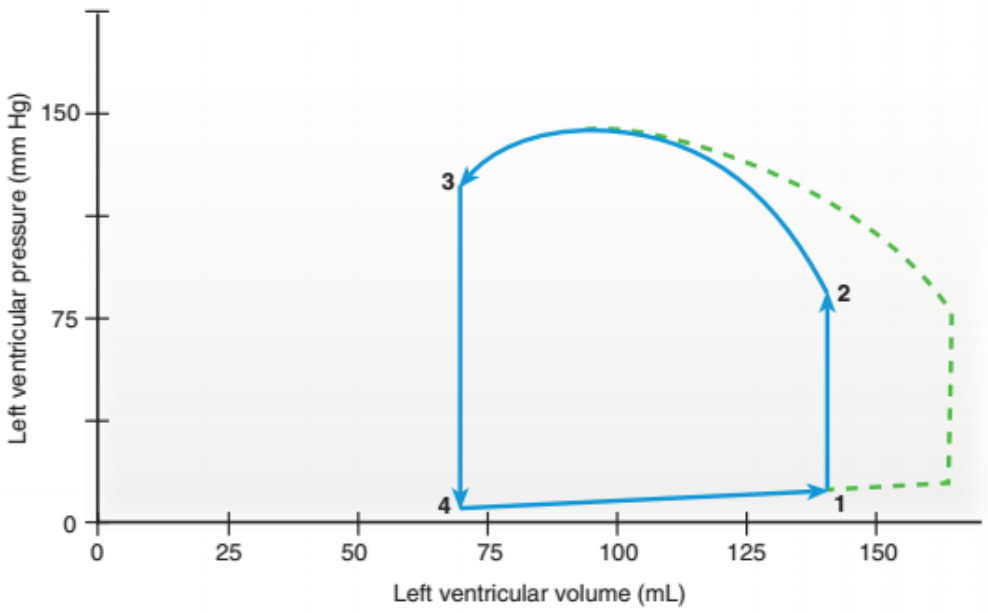


FIGURE 2–5. Effect of an increase in preload on the left ventricular pressure–volume loop. (Adapted, with permission, from Costanzo LS. *BRS Physiology*. 5th ed. Baltimore: Lippincott Williams & Wilkins; 2011:78.)

10. Contractility (inotropy) is the intrinsic ability of myocardial fibers to develop tension at a given muscle length (i.e., at a given end-diastolic volume). Contractility is directly correlated with the intracellular Ca2+ concentration, which dictates how many cross-bridges cycle and, therefore, how much tension is generated. When contractility is increased (e.g., by positive inotropic agents, such as norepinephrine or digitalis), the ventricle can develop greater tension and pressure during systole. As a result, stroke volume increases (Fig. 2–6), less blood remains in the ventricle after ejection, and,therefore, end-systolic volume decreases. Because ejection fraction is stroke volume expressed as a fraction of end-diastolic volume, if stroke volume increases and end-diastolic volume is unchanged, ejection fraction must have increased.

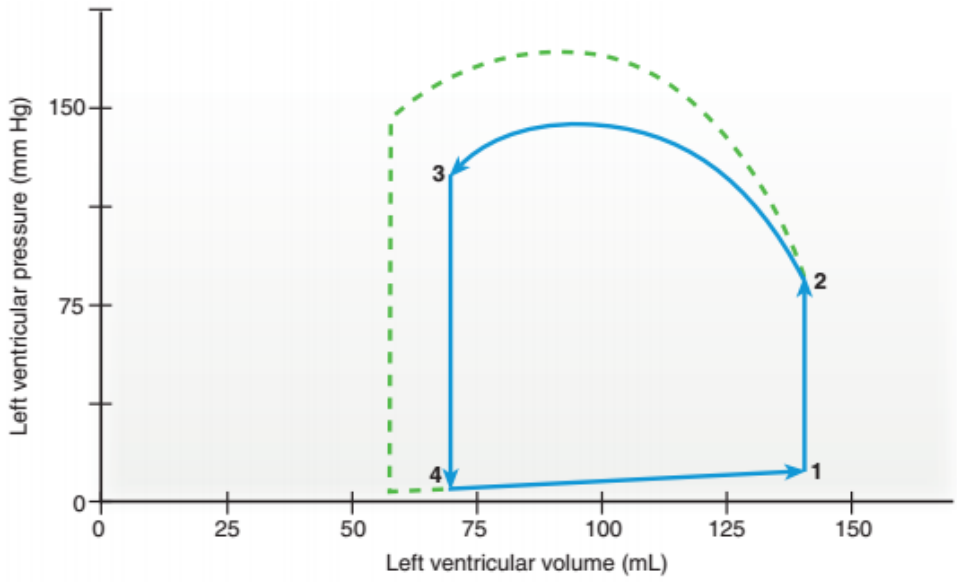


FIGURE 2–6. Effect of an increase in contractility on the left ventricular pressure–volume loop. (Adapted, with permission, from Costanzo LS. *BRS Physiology*. 5th ed. Baltimore: Lippincott Williams & Wilkins; 2011:78.)

11. Afterload is the pressure against which the ventricles must eject blood. Afterload of the left ventricle is aortic pressure. To open the aortic valve and eject blood, left ventricular pressure must increase to a level greater than aortic pressure. Thus, if afterload increases, the left ventricle must work harder than usual to overcome this higher pressure. Figure 2–7 shows the consequences of an increase in afterload. During isovolumetric contraction (1 → 2) and ventricular ejection (2 → 3), ventricular pressure increases to a higher level than normal. Because of the increased afterload, stroke volume is compromised, more blood remains in the left ventricle after ejection, and end systolic volume is increased. Because stroke volume decreases and end-diastolic volume is unchanged, ejection fraction must have decreased.

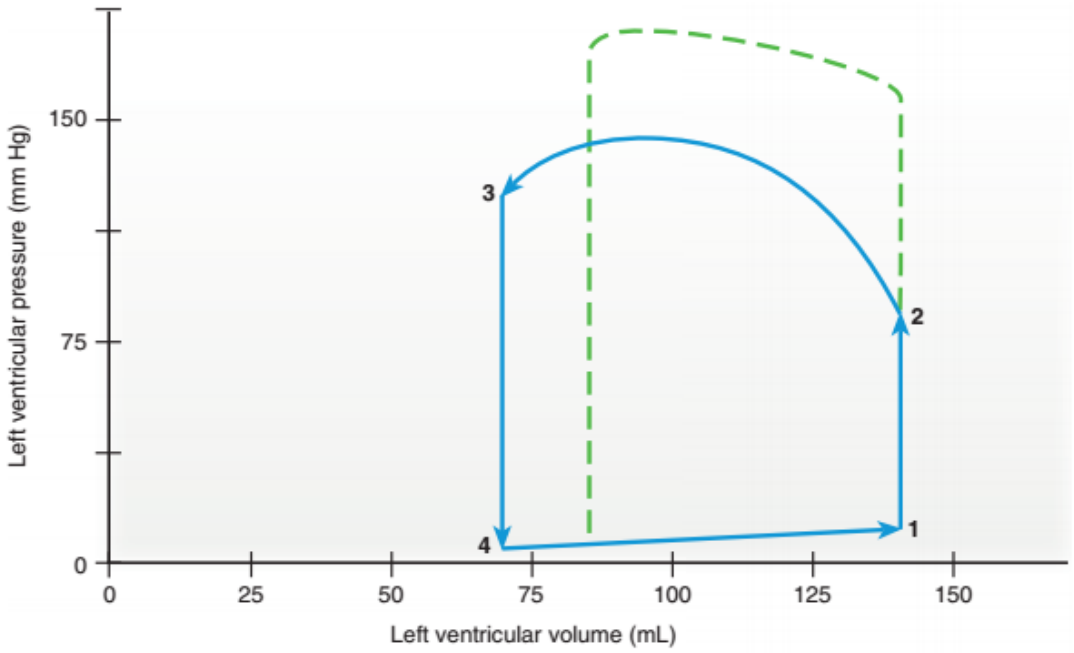


FIGURE 2–7. Effect of an increase in afterload on the left ventricular pressure–volume loop. (Adapted, with permission, from Costanzo LS. *BRS Physiology*. 5th ed. Baltimore: Lippincott Williams & Wilkins; 2011:78.)

CASE 3

Responses to Changes in Posture

Joslin Chambers is a 27-year-old assistant manager at a discount department store. One morning, she awakened from a deep sleep and realized that she was more than an hour late for work. She panicked,momentarily regretting her late-night socializing, and then jumped out of bed. Briefly, she felt light-headed and thought she might faint. She had the sensation that her heart was "racing." Had she not been so late for work, she would have returned to bed. As she walked toward the bathroom, she noticed that her light-headedness dissipated. The rest of her day was uneventful.

Questions

1. When Joslin moved rapidly from a supine (lying) position to a standing position, there was a brief,initial decrease in arterial pressure that caused her light-headedness. Describe the sequence of events that produced this transient fall in arterial pressure.

2. Why did the decrease in arterial pressure cause Joslin to feel light-headed?

3. Joslin's light-headedness was only transient because a reflex was initiated that rapidly restored arterial pressure to normal. Describe the specific effects of this reflex on heart rate, myocardial contractility, TPR, and capacitance of the veins. What receptors are involved in each of these responses?

4. How does each component of the reflex (e.g., the effect on heart rate) help to restore arterial pressure? (Hint: It may help to write the equation that relates arterial pressure, cardiac output, and TPR.)

5. In addition to the reflex correction of blood pressure, the fact that Joslin walked to the bathroom helped return her arterial pressure to normal. How did walking help?

ANSWERS

1. Orthostatic hypotension is the phenomenon whereby arterial pressure decreases when one stands up.

When a person suddenly moves from a supine (lying) position to a standing position, blood pools in the veins of the legs. (Because the capacitance, or compliance, of the veins is high, they can hold large volumes of blood.) This pooling decreases venous return to the heart, which decreases cardiac output by the Frank–Starling mechanism. (The Frank–Starling mechanism describes the relationship between venous return and cardiac output. Increases in venous return lead to increases in end diastolic volume. Up to a point, increases in end-diastolic volume lead to increases in cardiac output. Conversely, decreases in venous return lead to decreases in cardiac output.) Because arterial pressure is affected by the volume of blood in the arteries, a decrease in cardiac output (i.e., less blood is pumped into the arterial system) causes a decrease in arterial pressure.

2. When Joslin stood up quickly, she felt light-headed because a brief period of cerebral ischemia occurred as a result of the decrease in arterial pressure. The autoregulatory range for cerebral blood flow is 60 to 140 mm Hg. In other words, cerebral blood flow is maintained constant as long as arterial pressure is greater than 60 mm Hg and less than 140 mm Hg. When Joslin stood up, her arterial pressure briefly decreased below this critical autoregulatory range. As a result, cerebral blood flow decreased, and she felt light-headed.

3. Baroreceptors located in the carotid sinus and the aortic arch sensed the decrease in arterial pressure.The baroreceptor reflex then orchestrated a series of compensatory responses, including increased sympathetic outflow to the heart and blood vessels. There are four consequences of this increased

sympathetic outflow:

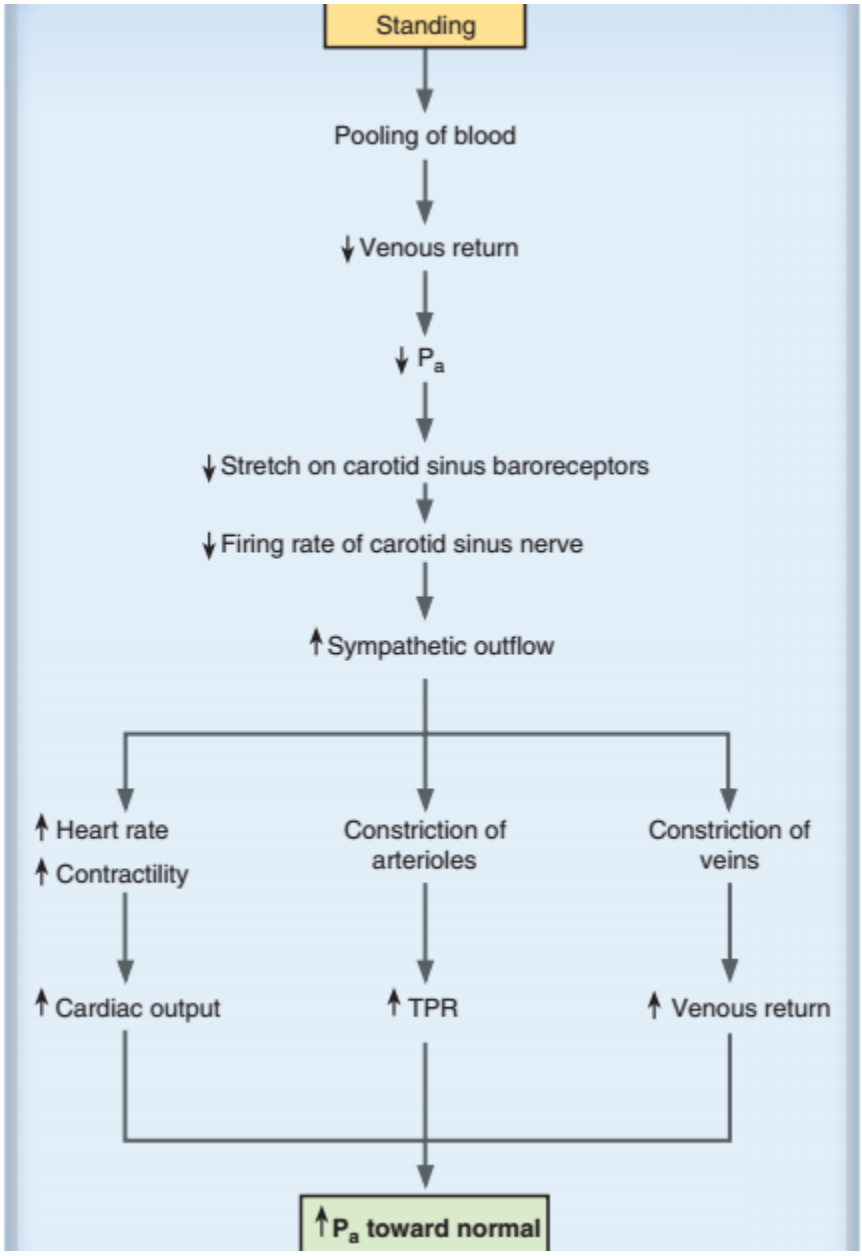
• Increased heart rate (the sensation of a racing heart), a positive chronotropic effect mediated by b1-adrenergic receptors in the sinoatrial node.

• Increased contractility of the ventricles, a positive inotropic effect mediated by b1-adrenergic receptors in the ventricular muscle.

• Increased arteriolar constriction, mediated by a1-adrenergic receptors on vascular smooth muscle of the arterioles.

• Increased venoconstriction, mediated by a1-adrenergic receptors on vascular smooth muscle of the veins.

4. All of the components of the baroreceptor reflex contributed to the restoration of Joslin's arterial pressure (Fig. 2–8).



These contributions can be appreciated by reviewing the relationship between arterial pressure,cardiac output, and TPR:

Pa = cardiac output ¥ TPR

where

Pa = mean arterial pressure

Cardiac output = volume of blood ejected from the left ventricle/min

TPR = total peripheral resistance

In words, arterial pressure depends on the volume of blood pumped into the arteries from the left ventricle and the resistance of the arterioles. (It may be helpful to think of arteriolar resistance as“holding” blood on the arterial side of the circulation.)Now, using the equation, consider how each portion of the baroreceptor reflex helped to restore Joslin’s arterial pressure back to normal. The increased heart rate and contractility combined to produce an increase in cardiac output. The increased cardiac output caused an increase in arterial pressure. The increased arteriolar constriction produced an increase in TPR, which also increased the arterial pressure. Finally, venoconstriction led to decreased capacitance of the veins, which increased venous return to the heart and increased the cardiac output (by the Frank–Starling mechanism).

5. As Joslin walked toward the bathroom, the muscular activity compressed the veins in her legs and decreased venous capacitance (i.e., the volume of blood the veins can hold). This compression,combined with sympathetic venoconstriction, increased venous return to the heart and cardiac output.

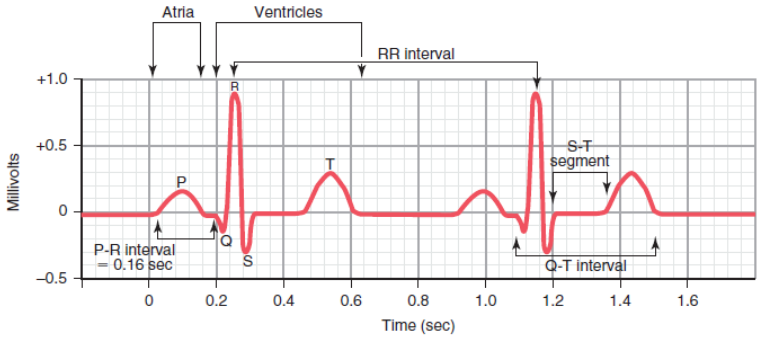
9. A 60-year-old man’s EKG shows that he has an R-R interval of 0.55 sec. Which of the following best explains his condition?
A) He has fever
B) He has a normal heart rate
C) He has excess parasympathetic stimulation of the S-A node
D) He is a trained athlete at rest
E) He has hyperpolarization of the S-A node

A) Heart rate is determined by the formula 60/R-R interval, and the heart rate for this patient is 109 beats/min. This is a fast heart rate, which would occur during fever. A trained athlete has a low heart rate. Excess parasympathetic stimulation and hyperpolarization of the S-A node both decrease heart rate.

2. Which of the following phases of the cardiac cycle follows immediately after the beginning of the QRS wave?
A) Isovolumic relaxation
B) Ventricular ejection
C) Atrial systole
D) Diastasis
E) Isovolumic contraction

E) Immediately after the QRS wave, the ventricles begin to contract and the first phase that occurs is isovolumic contraction. This occurs before the ejection phase and increases the ventricular pressure enough to mechanically open the aortic and pulmonary valves.

3. When recording lead I on an EKG, the right arm is the negative electrode, and the positive electrode is the
A) left arm
B) left leg
C) right leg
D) left arm 1 left leg
E) right arm 1 left leg



A) By convention, the left arm is the positive electrode for lead I of an EKG.

A) 64
B) 70
C) 88
D) 94
E) 104

B) The heart rate can be calculated by 60 divided by the R-R interval, which is 0.86 sec. This results in a heart rate of 70 beats/min.

33. When recording lead aVL on an EKG, the positive electrode is the
A) left arm

B) left leg
C) right leg
D) left arm 1 left leg
E) right arm 1 left leg

A) By convention, the left arm is the positive electrode for lead aVL of an EKG.

34. A 70-year-old man was had the following EKG during his annual physical exam. What is his Q-T interval?
A) 0.12 sec
B) 0.16 sec
C) 0.22 sec
D) 0.30 sec
E) 0.40 sec

E) The contraction of the ventricles lasts almost from the beginning of the Q wave and continues to the end of the T wave. This interval is called the Q-T interval and ordinarily lasts about 0.35 sec. In this particular example the Q-T interval is a little bit longer than average and equals 0.40 sec.

36. What is the normal QT interval?
A) 0.03 seconds
B) 0.13 seconds
C) 0.16 seconds
D) 0.20 seconds
E) 0.35 seconds

E) The contraction of the ventricles lasts almost from the beginning of the Q wave and continues to the end of the T wave. This interval is called the Q-T interval and ordinarily lasts about 0.35 sec.

37. When recording lead II on an EKG, the positive electrode is the
A) left arm
B) left leg
C) right leg

D) left arm 1 left leg
E) right arm 1 left leg

B) By convention, the left leg is the positive electrode for lead II of an EKG.

38. When recording lead III on an EKG, the negative electrode is the
A) left arm
B) left leg
C) right leg
D) left arm 1 left leg
E) right arm 1 left leg

A) By convention, the left arm is the negative electrode for lead III of an EKG.

39. A 65-year-old man had an EKG recorded at a local emergency room following a biking accident. His weight was 80 kg and his aortic blood pressure was 160/90 mm Hg. The QRS voltage was 0.5 mV in lead I and 1.5 mV in lead III. What is the QRS voltage in lead II?
A) 0.5 mV
B) 1.0 mV
C) 1.5 mV
D) 2.0 mV
E) 2.5 mV

D) Einthoven's law states that the voltage in lead I plus the voltage in lead III is equal to the voltage in lead II, which in this case is 2.0 mV.

40. A ventricular depolarization wave when traveling 290° in the frontal plane will cause a large negative deflection in which lead?
A) aVR
B) aVL
C) Lead II
D) Lead III
E) aVF

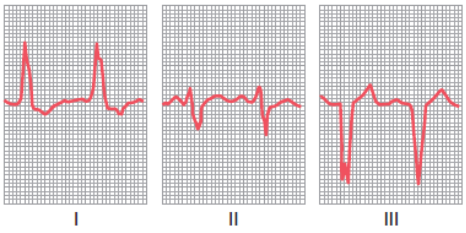
E) As can be seen in Figure 12-3 (TMP12), the positive portion of lead aVF has an axis of 90° and the negative part of this lead has an axis of 290°. Note the difference between the positive and the negative ends of this vector is 180°.

A 60-year-old woman had the following EKG recorded at a local emergency room following an automobile accident.Her weight was 70 kg and her aortic blood pressure was 140/80 mm Hg.

41. What is the mean electrical axis calculated from standard leads I, II, and III shown in her EKG?
A) 290°
B) 250°
C) 212°
D) 1100°
E) 1170°

B) The mean electrical axis can be determined plotting the resultant voltage of the QRS for leads I, II, and III. The result is as is shown above and has a value of 250°.

42. What is the heart rate using lead I for the calculation?
A) 70
B) 88
C) 100
D) 112
E) 148



A) The heart rate can be calculated by 60 divided by the R-R interval, which is 0.68 sec. This results in a heart rate of 88 beats/min.

43. What is her likely diagnosis?
A) Mitral valve stenosis
B) Left bundle branch block
C) Pulmonary valve stenosis
D) Right bundle branch block
E) Left ventricular hypertrophy

B) Note in Figure 12-14 (TMP12), which is shown above, that there is a QRS width greater than 0.12 sec.

This indicates a bundle branch block. There is also a left axis deviation, which is consistent with a left bundle branch block.

44. Which of the following conditions will usually result in right axis deviation in an EKG?
A) Systemic hypertension
B) Aortic valve stenosis
C) Aortic valve regurgitation
D) Excess abdominal fat
E) Pulmonary hypertension

E) Systemic hypertension results in a left axis deviation because of the enlargement of the left ventricle. Aortic valve stenosis and aortic valve regurgitation also result in a large left ventricle and left axis deviation. Excessive abdominal fat, because of the mechanical pressure of the fat, causes a rotation of the heart to the left resulting in a leftward shift of the mean electrical axis. Pulmonary hypertension causes enlargement of the right heart and thus causes right axis deviation.

45. A ventricular depolarization wave when traveling 60° in the frontal plane will cause a large positive deflection in which of the following leads?
A) aVR
B) aVL
C) Lead I
D) Lead II
E) aVF

D) Lead II has a positive vector at the 60° angle. The positive end of lead II is at 2120°.

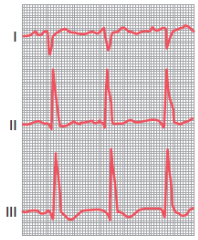
A male long-term smoker who is 62 years old weighs 250 lb. He had the following EKG recorded at his local hospital.

46. Which of the following is the mean electrical axis calculated from standard leads I, II, and III shown in his EKG?
A) 2110°
B) 220°
C) 190°
D) 1105°
E) 1180°

D) Note that lead III has the strongest vector, therefore the mean electrical axis will be closer to this lead than to leads I or II. The angle of lead III is 120°, and the resultant vector (mean electrical axis) is close to that lead and has a value of 1105°.

47. Which of the following is the likely diagnosis?
A) Left ventricular hypertrophy
B) Left bundle branch block
C) Tricuspid stenosis
D) Right bundle branch block
E) Right ventricular hypertrophy

D) The diagnosis is right bundle branch block. This can be determined by a rightward shift in mean electrical axis as well as the greatly prolonged QRS complex. In right ventricular hypertrophy, the QRS complex is only moderately prolonged.



48. A 60-year-old woman has lost some ability to perform normal household tasks and is not feeling well. An EKG shows a QRS complex with a width of 0.20 sec, the T wave is inverted in lead I, and the R wave has a large negative deflection in lead III. Which of the following is the likely diagnosis?
A) Right ventricular hypertrophy
B) Left bundle branch block
C) Pulmonary valve stenosis
D) Right bundle branch block
E) Left ventricular hypertrophy

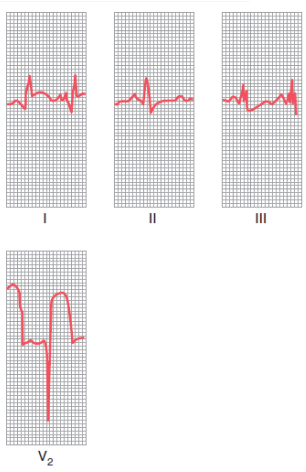
D) The patient has a left axis deviation because of the large negative deflection of the R wave in lead III. Also, her T wave was inverted in lead I, which means that it is in the opposite direction of the QRS complex. This is characteristic of bundle branch block. Also, the QRS complex had a width of 0.20 sec, a very prolonged QRS complex. A QRS complex that has a width greater than

0.12 sec is normally caused by a conduction block. All these factors indicate that this patient has a left bundle branch block.

49. A 70-year-old woman sought assistance at a hospital emergency department because she is experiencing chest pain. Based on the following EKG tracing, which of the following is the likely diagnosis?

A) Acute anterior infarction in the left ventricle of the heart
B) Acute anterior infarction in the right ventricle of the heart
C) Acute posterior infarction in the left ventricle of the heart
D) Acute posterior infarction in the right ventricle of the heart
E) Right ventricular hypertrophy

A) This patient has an acute anterior infarction in the left ventricle of the heart. This can be determined by plotting the currents of injury from the different leads. The limb leads are used to determine whether the infarction is coming from the left or right side of the heart and from the base or apex of the heart. The chest leads are used to determine whether it is an anterior or posterior infarct. When we analyze the currents of injury, a negative potential, caused by the current of injury, occurs in lead I and a positive potential, caused by the current of injury, occurs in lead III. This is determined by subtracting the J point from the TP segment. The negative end of the resultant vector originates in the ischemic area, which is therefore the left side of the heart. In lead V2, the chest lead, the electrode is in a field of very negative potential, which occurs in patients with an anterior lesion.



50. A 30-year-old man had his EKG measured at his physician's office, but his records were lost. The EKG technician remembered that the QRS deflection was large and positive in lead aVF and 0 in lead I. What is the mean electrical axis in the frontal plane?
A) 90°
B) 60°
C) 0°

D) 260°
E) 290°

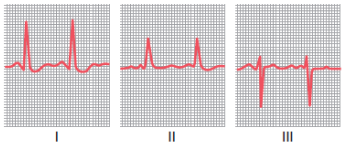
A) Since the deflection in this EKG is 0 in lead I, the axis has to be 90° away from this lead. Therefore, the mean electrical axis has to be 190° or 290°. Since the aVF lead has a positive deflection, the mean electrical axis must be at 190°.

51. Which of the following is most likely at the “J point” in an EKG of a patient with a damaged cardiac muscle?
A) Entire heart is depolarized
B) All the heart is depolarized except for the damaged cardiac muscle
C) About half the heart is depolarized
D) All of the heart is repolarized
E) All of the heart is repolarized except for the damaged cardiac muscle

A) At the J point the entire heart is depolarized in a patient with a damaged cardiac muscle or a patient with a normal cardiac muscle. The area of the heart that is damaged will not repolarize, but remains depolarized at all times.

52. A 50-year-old man is a new employee at ABC Software. The EKG shown here was recorded during a routine physical examination. His likely diagnosis is which of the following?

A) Chronic systemic hypertension
B) Chronic pulmonary hypertension
C) Second-degree heart block
D) Paroxysmal tachycardia
E) Tricuspid valve stenosis



A) Note that the QRS complex has a positive deflection in lead I and a negative in lead III, which indicates that there is a leftward axis deviation. This occurs during chronic systemic hypertension. Pulmonary hypertension increases the ventricular mass on the right side of the heart, which gives a right axis deviation.

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53. A 55-year-old man had his EKG measured at an annual physical, and his net deflection (R wave minus Q or S wave) in standard limb lead I is 21.2 mV. Standard

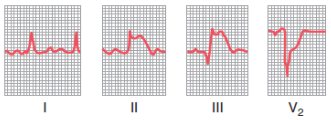
limb lead II has a net deflection of 11.2 mV. What is the mean electrical axis of his QRS?
A) 230°
B) 130°
C) 160°
D) 1120°
E) 2120°

D) The QRS wave plotted on lead I was 21.2 mV and lead II was 11.2 mV so the absolute value of the deflections were the same. Therefore, the mean electrical axis has to be exactly halfway in between these two leads, which is halfway between the lead II axis of 60° and the lead I negative axis of 180°, resulting in a value of 120°.

55. A 60-year-old woman tires easily. Her EKG shows a QRS complex that is positive in the aVF lead and negative in standard limb lead I. A likely cause of this condition is which of the following?
A) Chronic systemic hypertension
B) Pulmonary hypertension
C) Aortic valve stenosis
D) Aortic valve regurgitation

B) The EKG from this patient has a positive deflection in aVF and a negative deflection in standard limb lead I. Therefore, the mean electrical axis is between 90° and 180°, which is a rightward shift in the EKG mean electrical axis. Systemic hypertension, aortic valve stenosis, and aortic valve regurgitation cause hypertrophy of the left ventricle and thus a leftward shift in the mean electrical axis. Pulmonary hypertension causes a rightward shift in the axis, and is therefore characterized by this EKG.

56. A 60-year-old woman came to the hospital emergency department and complained of chest pain. Based on the EKG tracing shown here, which of the following is the most likely diagnosis?



A) Acute anterior infarction in the base of the heart
B) Acute anterior infarction in the apex of the heart
C) Acute posterior infarction in the base of the heart
D) Acute posterior infarction in the apex of the heart
E) Right ventricular hypertrophy

D) Note in the following figure that the current of injury is plotted on the graph at the bottom. This is not a plot of the QRS voltages but the current of injury voltages. They are plotted for leads II and III, which are both negative, and the resultant vector is nearly vertical. The negative end of the vector points to where the current of injury originated, which is in the apex of the heart. The elevation of the TP segment above the J point indicates a posterior lesion. Therefore, the EKG is consistent with acute posterior infarction in the apex of the heart.

57. A 50-year-old man has a blood pressure of 140/85 and weighs 200 lb. He reports that he is not feeling well, his EKG has no P-waves, he has a heart rate of 46, and the QRS complexes occur regularly. What is his likely condition?
A) First-degree heart block
B) Second-degree heart block
C) Third-degree heart block
D) Sinoatrial heart block
E) Sinus bradycardia

D) When a patient has no P waves and a low heart rate, it is likely that the impulse leaving the sinus node is totally blocked before entering the atrial muscle. This is called sinoatrial block. The ventricles pick up the new rhythm usually initiated in the A-V node at this point, which results in a heart rate of 40 to 60 beats/min. In contrast, during sinus bradycardia you still have P waves associated with each QRS complex. In first-, second-, and third-degree heart block, you have P waves in each of these instances, although some are not associated with QRS complex.

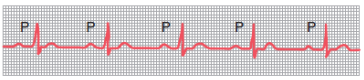
58. An 80-year-old man had an EKG taken at his local doctor's office, and the diagnosis was atrial fibrillation. Which of the following statements are likely conditions in someone with atrial fibrillation?
A) Ventricular fibrillation normally accompanies atrial fibrillation
B) P waves of the EKG are strong
C) Rate of ventricular contraction is irregular and fast
D) Atrial "a" wave is normal
E) Atria have a smaller volume than normal

C) Atrial fibrillation has a rapid irregular heart rate. The P waves are missing or are very weak. The atria

exhibit circus movements, and atrial volume is often increased, causing the atrial fibrillation.

59. Circus movements in the ventricle can lead to ventricular fibrillation. Which of the following conditions in the ventricular muscle will increase the tendency for circus movements?
A) Decreased refractory period
B) Low extracellular potassium concentration
C) Increased refractory period
D) Shorter conduction pathway (decreased ventricular volume)
E) Increase in parasympathetic impulses to the heart

A) Circus movements occur in ventricular muscle particularly if you have a dilated heart or decreases in conduction velocity. High extracellular potassium and sympathetic stimulation, not parasympathetic stimulation, increase the tendency for circus movements. A longer refractory period tends to prevent circus movements of the heart, because when the impulses travel around the heart and contact the area of ventricular muscle that has a longer refractory period, the action potential stops at this point.



54. A 65-year-old patient with a heart murmur has a mean QRS axis of 120°, and the QRS complex lasts 0.18 sec. Which of the following is the likely diagnosis?
A) Aortic valve stenosis
B) Aortic valve regurgitation
C) Pulmonary valve stenosis
D) Right bundle branch block
E) Left bundle branch block

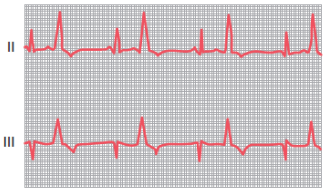
D) A QRS axis of 120° indicates a rightward shift. Since the QRS complex is 0.18 sec, this indicates a conduction block. Therefore, this EKG, which fits with these characteristics, is a right bundle branch block.

60. A 75-year-old man goes to the hospital emergency department and faints. Five minutes later he is alert. An EKG shows 75 P waves per minute and 35 QRS waves per minute with a normal QRS width. Which of the following is the likely diagnosis?
A) First-degree A-V block
B) Stokes-Adams syndrome
C) Atrial paroxysmal tachycardia

D) Electrical alternans
E) Atrial premature contractions

B) A sudden onset of A-V block that comes and goes is called the Stokes-Adams syndrome. The patient depicted here has about 75 P-waves/min, which means that the atria are contracting normally. But the A-V block that occurs allows only 35 QRS waves to occur each minute.

61. A 60-year-old man weighing 220 lb had the following EKG, which shows the standard lead II. What is his diagnosis?



A) A-V nodal rhythm
B) First-degree A-V heart block
C) Second-degree A-V heart block
D) Third-degree A-V heart block
E) Atrial flutter

D) By definition, first-degree A-V heart block occurs when the P-R interval exceeds a value of 0.20 sec, but without any dropped QRS waves. In the following figure, the P-R interval is about 0.30 sec, which is considerably prolonged. However, there are no dropped QRS waves. During second-degree A-V block or thirddegree A-V block, QRS waves are dropped.

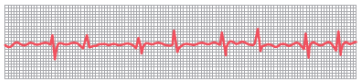
62. A 35-year-old woman had unusual sensations in her chest after she smoked a cigarette. Her EKG is shown here. Which of the following is the likely diagnosis?

A) Premature contraction originating in the atrium
B) Premature contraction originating high in the A-V node
C) Premature contraction originating low in the A-V node
D) Premature contraction originating in the apex of the ventricle
E) Premature contraction originating in the base of the ventricle

E) Note that the premature ventricular contractions (PVCs) have wide and tall QRS waves in the EKG. The

mean electrical axis of the premature contraction can be determined by plotting these large QRS complexes on the standard limb leads. The PVC originates at the negative end of the resultant mean electrical axis, which is at the base of the ventricle. Note that the QRS of the PVC is wider and much taller than the normal QRS waves in this EKG.

A 55-year-old man had the following EKG recorded at his doctor's office during a routine physical examination.



63. What is his diagnosis?
A) Normal EKG
B) Atrial flutter
C) High A-V junctional pacemaker
D) Middle A-V junctional pacemaker
E) Low A-V junctional pacemaker

B) This patient has atrial flutter characterized by several P waves for each QRS complex. In this EKG, you see some areas that have two P waves for every QRS and other areas that have three P waves for each QRS. Note the rapid heart rate, which is characteristic of atrial flutter, and the irregular R-R intervals.

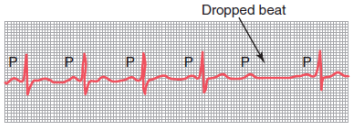
64. What is his ventricular heart rate in beats per minute?
A) 37.5
B) 60
C) 75
D) 100
E) 120

E) The average ventricular rate is 120 beats/min in this EKG, which is typical of atrial flutter. Once again, note that the heart rate is irregular due to the inability of the impulses to quickly pass through the A-V node because of its refractory period.

65. A 60-year-old woman has been diagnosed with atrial fibrillation. Which of the following statements best describe this condition?
A) Ventricular rate of contraction is 140 beats/min
B) P waves of the EKG are pronounced
C) Ventricular contractions occur at regular intervals

D) QRS waves are more pronounced than normal
E) Atria are smaller than normal

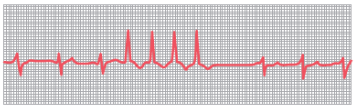
A) Atrial fibrillation has a rapid irregular heart rate. The P waves are missing or are very weak. The atria exhibit circus movements, and often are very enlarged, causing the atrial fibrillation.



66. Which of the following is most characteristic of atrial fibrillation?
A) Occurs less frequently in patients with atrial enlargement
B) Ventricular heart rate is about 40 beats per min
C) Efficiency of ventricular pumping is decreased 20 to 30 percent
D) Ventricular beat is regular
E) Atrial P wave is easily seen

C) Atrial fibrillation occurs often with patients with an atrial enlargement. This causes an increased tendency for circus movements to occur. The ventricular beat is irregular because impulses are rapidly arriving at the A-V node; however, many times the A-V node is in a refractory period. Therefore the A-V node will not pass a second impulse until about 0.35 sec elapses after the previous one. There is also a variable interval between when the atrial impulses reach the A-V node. This results in a very irregular heartbeat but one that is very rapid with a rate of 125 to 150 beats/min.

67. A 65-year-old woman who had a myocardial infarction 10 days ago returns to her family physician's office and reports that her pulse rate may be rapid. Based on the above EKG, which of the following is the likely diagnosis?



A) Stokes-Adams syndrome
B) Atrial fibrillation
C) A-V nodal tachycardia
D) Atrial paroxysmal tachycardia
E) Ventricular paroxysmal tachycardia

E) The term paroxysmal means that the heart rate becomes rapid in paroxysms, with the paroxysm beginning suddenly and lasting for a few seconds, a few minutes, a few hours, or much longer. Then the paroxysm usually ends as suddenly as it began and the pacemaker shifts back to the S-A node. The mechanism by which this is believed to occur is by a re-entrant circus movement feedback pathway that sets up an area of local repeated

self-re-excitation. The EKG shown is ventricular paroxysmal tachycardia. That the origin is in the ventricles can be determined because of the changes in the QRS complex that have high voltages and look much different than the preceding normal QRS complexes. This is very characteristic of a ventricular irritable locus.

68. A 65-year-old man had the EKG shown here recorded at his annual physical examination. Which of the following is the likely diagnosis?

A) Atrial paroxysmal tachycardia
B) First-degree A-V block
C) Second-degree A-V block
D) Third-degree A-V block
E) Atrial flutter

C) Note in this EKG that a P wave precedes each of the first four QRS complexes. After that we see a P wave but a dropped QRS wave. This is characteristic of second-degree A-V block.

69. Which of the following decreases the risk of ventricular fibrillation?
A) Dilated heart
B) Increased ventricular refractory period
C) Decreased electrical conduction velocity
D) Exposure of the heart to 60-cycle alternating current
E) Epinephrine administration

B) A dilated heart increases the risk of occurrence of ventricular fibrillation because of an increase in likelihood of circus movements. Also, if the conduction velocity decreases, it will take longer for the impulse to travel around the heart, which decreases the risk of ventricular fibrillation. Exposure of the heart to 60-cycle alternating current or epinephrine administration increases the irritability of the heart. If the refractory period is long, the likelihood of the re-entrant type of pathways decreases, because when the impulse travels around the heart, the ventricles remain in a refractory period.

71. Which of the following statements best describes a patient with premature atrial contraction?

A) Pulse taken from the radial artery immediately following the premature contraction will be weak
B) Stroke volume immediately following the premature contraction will be increased
C) P wave is never seen
D) Probability of these premature contractions occurring is decreased in people with a large caffeine intake
E) Causes the QRS interval to be lengthened

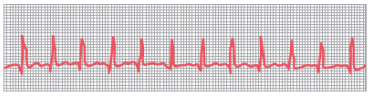
A) The heartbeat immediately following a premature atrial contraction weakens because the diastolic period is very short in this condition. Therefore, the ventricular filling time is very short, and thus the stroke volume decreases. The P wave is usually visible in this arrhythmia unless it coincides with the QRS complex. The probability of these premature contractions increases in people with toxic irritation of the heart and local ischemic areas.

A male patient had a myocardial infarction at age 55. He is now 63 years old. Standard limb lead I is shown here.

72. What is his heart rate?
A) 40 beats/min
B) 50 beats/min
C) 75 beats/min
D) 100 beats/min
E) 150 beats/min
E) The heart rate can be determined by 60 divided by the R-R interval, which gives you a value of 150 beats/min. This is tachycardia, defined as a heart rate greater than 100 beats/min.

73. What is his current diagnosis?
A) Sinus tachycardia
B) First-degree heart block
C) Second-degree heart block
D) ST segment depression
E) Third-degree heart block

A) The relationship between the P waves and the QRS complexes appears to be normal and there are no missing beats. Therefore, this patient has a sinus rhythm, and there is no heart block. There is also no ST segment depression in this patient. Since we have normal P and QRS and T waves, this condition is sinus tachycardia.

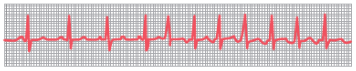


74. A 55-year-old man has been diagnosed with Stokes-Adams syndrome. Two minutes after the syndrome starts to cause active blockade of the cardiac impulse, which of the following is the pacemaker of the heart?
A) Sinus node
B) A-V node
C) Purkinje fibers
D) Cardiac septum
E) Left atrium

B) During a Stokes-Adams syndrome attack total A-V block suddenly begins, and the duration of the block may be a few seconds or even several weeks. The new pacemaker of the heart is distal to the point of blockade but is usually the A-V node or the A-V bundle.

75. If the origin of the stimulus that causes atrial paroxysmal tachycardia is near the A-V node, which of the following statements about the P-wave in standard limb lead I is most accurate?
A) P wave will originate in the sinus node
B) It will be upright
C) It will be inverted
D) P wave will be missing

C) During atrial paroxysmal tachycardia the impulse is initiated by an ectopic focus somewhere in the atria. If the point of initiation is near the A-V node the P wave travels backward toward the S-A node and then forward into the ventricles at the same time. Therefore, the P wave will be inverted.



76. A 45-year-old man had the EKG below recorded at his annual physical. Which of the following is the likely diagnosis?

A) Atrial paroxysmal tachycardia
B) First-degree A-V block
C) Second-degree A-V block
D) Ventricular paroxysmal tachycardia
E) Atrial flutter

A) This EKG has characteristics of atrial paroxysmal tachycardia. This means the tachycardia may come and go at random times. The basic shape of the QRS complex and its magnitude are virtually unchanged from the normal QRS complexes, which eliminates the possibility of ventricular paroxysmal tachycardia. This

EKG is not characteristic of atrial flutter since there is only one P wave for each QRS complex.

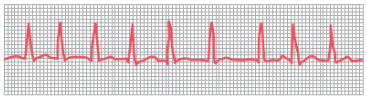
77. A 60-year-old woman sees her physician for her annual physical examination. The physician orders an EKG, which is shown below. Which of the following is the likely diagnosis?

A) First-degree A-V block
B) Second-degree A-V block
C) Third-degree A-V block
D) Atrial paroxysmal tachycardia
E) Atrial fibrillation

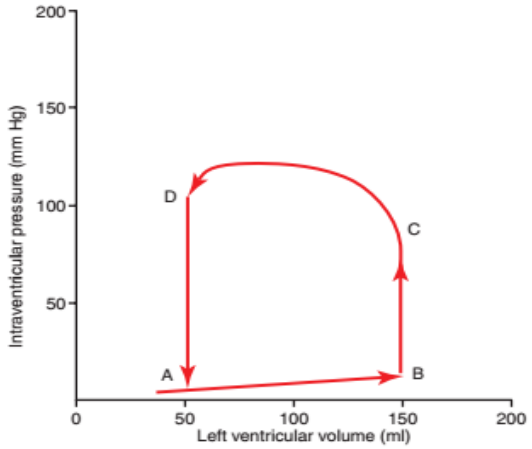
E) First-, second-, and third-degree heart blocks as well as atrial paroxysmal tachycardia all have P waves in the EKG. However, there are usually no evident P waves during atrial fibrillation, and the heart rate is irregular. Therefore, this EKG is characteristic of atrial fibrillation.

HS

Use this figure to answer the next 3 questions



A 60-year-old woman has a resting heart rate of 70 beats/min, arterial pressure is 130/85 mm Hg, and body temperature is normal. Her pressure-volume diagram of the left ventricle is shown above.



When does the second heart sound occur in the ven
tricular pressure–volume relationship?

A) At point D
B) Between point A and point B
C) Between point B and point C
D) Between point C and point D
E) Between point D and point A

When does the third heart sound occur in the ventri
cular pressure–volume relationship?

A) At point D
B) Between point A and point B
C) Between point B and point C
D) Between point C and point D
E) Between point D and point A

Which of the following events is associated with the first heart sound?
A) Closing of the aortic valve
B) Inrushing of blood into the ventricles during diastole
C) Beginning of diastole
D) Opening of the A-V valves
E) Closing of the A-V valves

Which of the following is associated with the second heart sound?
A) In-rushing of blood into the ventricles due to atrial contraction

B) Closing of the A-V valves
C) Closing of the pulmonary valve
D) Opening of the A-V valves
E) In-rushing of blood into the ventricles in the early to middle part of diastole

-year-old woman has been diagnosed with a heart murmur A 40A
"blowing" murmur of relatively high pitch is heard maximally over the left ventricle. The chest x-ray shows an enlarged heart. Arterial pressure in the aorta is 140/40mmHg .
What is the diagnosis?
A) Aortic valve stenosis
B) Aortic valve regurgitation
C) Pulmonary valve stenosis
D) Mitral valve stenosis
E) Tricuspid valve regurgitation

Which of the following heart murmurs is heard during systole?
A) Aortic valve regurgitation
B) Pulmonary valve regurgitation
C) Tricuspid valve stenosis
D) Mitral valve stenosis
E) Patent ductus arteriosus

An increase in left atrial pressure is most likely to occur in which of the following heart murmurs?
A) Tricuspid stenosis
B) Pulmonary valve regurgitation
C) Mitral regurgitation
D) Tricuspid regurgitation

A – 50 year-old female patient at UMC has been diagnosed with a heart murmur. murmur of relatively low pitch is heard maximally over the second intercostal space to the right of the sternum. The chest x-ray shows an enlarged heart. The mean QRS axis of the EKG is 245.
The diagnosis is :
A) Mitral valve stenosis
B) Aortic valve stenosis
C) Pulmonary valve stenosis
D) Tricuspid valve stenosis
E) Tricuspid valve regurgitation

Which of the following heart murmurs are only heard during systole?
A) Patent ductus arteriosus
B) Mitral stenosis

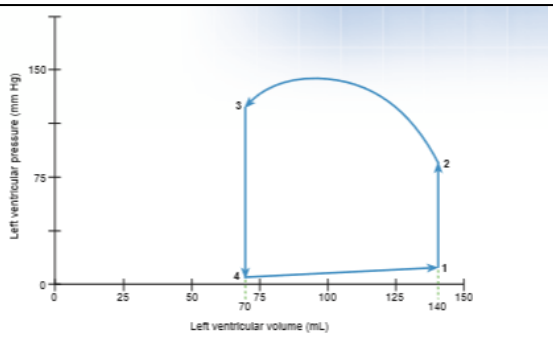
C) Tricuspid valve stenosis
D) Interventricular septal defect
E) Aortic regurgitation

Which of the following is associated with the first heart sound?
A) In-rushing of blood into the ventricles due to atrial contraction
B) Closing of the A-V valves
C) Closing of the pulmonary valve
D) Opening of the A-V valves
E) In-rushing of blood into the ventricles in the early to middle part of diastole

Which of the following heart murmurs is only heard during diastole?
A) Patent ductus arteriosus
B) Mitral regurgitation
C) Tricuspid valve stenosis
D) Interventricular septal defect
E) Aortic stenosis

Which of the following is associated with the third heart sound?
A) In-rushing of blood into the ventricles due to atrial contraction
B) Closing of the A-V valves
C) Closing of the pulmonary valve
D) Opening of the A-V valves
E) In-rushing of blood into the ventricles in the early to middle part of diastole

Use this figure to answer the next two questions



At which numbered point, or during which segment, would the first heart sound be heard?

the first heart sound corresponds to closure of the atrioventricular valves. This closure occurs at the end of ventricular filling, at the beginning of isovolumetric contraction. Thus, the first heart sound occurs at point 1.

At which number or during which segment, would the second heart sound be heard?

The second heart sound corresponds to closure of the aortic valve, at point 3

The second heart sound is caused by :

A) closure of the aortic and pulmonary valves.

B) vibrations in the ventricular wall during systole.

C) ventricular filling.

D) closure of the mitral and tricuspid valves.

E) retrograde flow in the vena cava.

The fourth heart sound is caused by:

A) closure of the aortic and pulmonary valves.

B) vibrations in the ventricular wall during systole.

C) ventricular filling.

D) closure of the mitral and tricuspid valves.

E) retrograde flow in the vena cava

Which of the following is most likely to cause the heart to go into spastic contraction?
A) Increased body temperature B) Increased sympathetic activity C) Decreased extracellular fluid potassium ions D) Excess extracellular fluid potassium ions E) Excess extracellular fluid calcium ions

Which of the following conditions will result in a dilated, flaccid heart?

A) Excess calcium ions in the blood B) Excess potassium ions in the blood C) Excess sodium ions in the blood D) Increased sympathetic stimulation E) Increased norepinephrine concentration in the blood

Which of the following conditions at the A-V node will cause a decrease in heart rate?
A) Increased sodium permeability B) Decreased acetylcholine levels C) Increased norepinephrine levels D) Increased potassium permeability E) Increased calcium permeability

Sympathetic stimulation of the heart :
A) Releases acetylcholine at the sympathetic endings B) Decreases sinus nodal discharge rate C) Decreases excitability of the heart D) Releases norepinephrine at the sympathetic endings E) Decreases cardiac contractility

Which of the following best explains how sympathetic stimulation affects the heart?
A) Permeability of the S-A node to sodium decreases B) Permeability of the A-V node to sodium decreases C) Permeability of the S-A node to potassium increases D) There is an increased rate of upward drift of the resting membrane potential of the S-A node E) Permeability of the cardiac muscle to calcium decreases

Which of the following conditions at the S-A node will cause heart rate to decrease?

A) Increased norepinephrine levels B) Increased sodium permeability C) Increased calcium permeability D) Increased potassium permeability E) Decreased acetylcholine levels

Which of the following conditions at the A-V node will cause a decrease in heart rate?
A) Increased sodium permeability B) Decreased acetylcholine levels C) Increased norepinephrine levels D) Increased potassium permeability E) Increased calcium permeability

Sympathetic stimulation of the heart normally causes which of the following conditions?
A) Acetylcholine release at the sympathetic endings B) Decreased heart rate C) Decreased rate of conduction of the cardiac impulse D) Decreased force of contraction of the atria E) Increased force of contraction of the ventricles

Circus movements in the ventricle can lead to ventricular fibrillation. Which of the following conditions in the ventricular muscle will increase the tendency for circus movements?
A) Decreased refractory period B) Low extracellular potassium concentration C) Increased refractory period D) Shorter conduction pathway (decreased ventricular volume) E) Increase in parasympathetic impulses to the heart

A balloon catheter is advanced from the superior vena cava into the heart and inflated
To increase the atrial pressure by 5mmhg.

An increase in which of the following would be expected to occur in response to the elevated atrial pressure ?
A) Atrial natriuretic peptide B) Angiotensin II C) Aldosterone D) Renal sympathetic nerve activity

Blood flow to tissues remains relatively constant despite a reduction in arterial pressure (autoregulation). Which of the following would be expected to occur in response to the reduction in arterial pressure ?
A) Decreased conductance B) Decreased tissue carbon dioxide concentration C) Increased tissue oxygen concentration D) Decreased vascular resistance E) Decreased arteriolar diameter

Autoregulation of tissue blood flow in response to an increase in arteria pressure occur as a result of which of the following?
A) Decrease in vascular resistance B) Initial decrease in vascular wall tension C) Excess delivery of nutrients such as oxygen to the tissues D) Decrease in tissue metabolism

. Excess production of which of the following would most likely result in chronic hypertension? A) Atrial natriuretic peptide B) Prostacyclin C) Angiotensin II D) Nitric oxide

A decrease in which of the following would be expected to occur in response to an increase in sodium intake?
A) Angiotensin II B) Nitric oxide C) Sodium excretion D) Atrial natriuretic peptide

An increase in atrial pressure results in which of the following?
A) Decrease in plasma atrial natriuretic peptide B) Increase in plasma angiotensin II concentration C) Increase in plasma aldosterone concentration D) Increase in heart rate

1. A healthy 28-year-old woman stands up from a supine position. Which of the following sets of cardiovascular changes is most likely to occur?

	Heart rate	Renal blood flow	Total peripheral resistance
A)	↑	↑	↑
B)	↑	↓	↑
C)	↑	↓	↓
D)	↑	↑	↓
E)	↓	↓	↓
F)	↓	↑	↓
G)	↓	↑	↑
H)	↓	↓	↑

2. A healthy 25-year-old male medical student has an exercise stress test at a local health club. Which of the following sets of physiological changes is most likely to occur in this man's skeletal muscles during exercise?

	Arteriolar resistance	Adenosine concentration	Vascular conductance
A)	↑	↑	↑
B)	↑	↓	↑
C)	↑	↓	↓
D)	↑	↑	↓
E)	↓	↓	↓
F)	↓	↑	↓
G)	↓	↑	↑
H)	↓	↓	↑

3. A 60-year-old woman has experienced dizziness for the past 6 months when getting out of bed in the morning and when standing up. Her mean arterial pressure is 130/90 mm Hg lying down and 95/60 sitting. Which of the following sets of physiological changes would be expected in response to moving from a supine to an upright position?

	Parasympathetic nerve activity	Plasma renin activity	Sympathetic activity
A)	↑	↑	↑
B)	↑	↓	↑
C)	↑	↓	↓
D)	↑	↑	↓
E)	↓	↓	↓
F)	↓	↑	↓
G)	↓	↑	↑
H)	↓	↓	↑

4. Which of the following sets of physiological changes would be expected to occur in response to an increase in atrial natriuretic peptide?

	Angiotensin II	Aldosterone	Sodium excretion
A)	↑	↑	↑
B)	↑	↓	↑
C)	↑	↓	↓
D)	↑	↑	↓
E)	↓	↓	↓
F)	↓	↑	↓
G)	↓	↑	↑
H)	↓	↓	↑

9. A 65-year-old man with a 5-year history of congestive heart failure is being treated with an angiotensin-converting enzyme (ACE) inhibitor. Which of the following sets of changes would be expected to occur in response to the ACE inhibitor drug therapy?

	Arterial pressure	Angiotensin II	Total peripheral resistance
A)	↑	↑	↑
B)	↑	↓	↑
C)	↑	↓	↓
D)	↑	↑	↓
E)	↓	↓	↓
F)	↓	↑	↓
G)	↓	↑	↑
H)	↓	↓	↑

11. A 55-year-old man with a history of normal health visits his physician for a checkup. The physical examination reveals that his blood pressure is 170/98 mm Hg. Further tests indicate that he has renovascular hypertension as a result of stenosis in the left kidney. Which of the following sets of findings would be expected in this man with renovascular hypertension?

	Total peripheral resistance	Plasma renin activity	Plasma aldosterone concentration
A)	↑	↑	↑
B)	↑	↓	↑
C)	↑	↓	↓
D)	↑	↑	↓
E)	↓	↓	↓
F)	↓	↑	↓
G)	↓	↑	↑
H)	↓	↓	↑

28. A 22-year-old man enters the hospital emergency room after severing a major artery in a motorcycle accident. It is estimated that he has lost approximately 700 ml of blood. His blood pressure is 90/55 mm Hg. Which of the following sets of changes would be expected in response to hemorrhage in this man?

	Heart rate flow	Sympathetic nerve activity	Total peripheral resistance
A)	↑	↑	↑
B)	↑	↓	↑
C)	↑	↓	↓
D)	↑	↑	↓
E)	↓	↓	↓
F)	↓	↑	↓
G)	↓	↑	↑
H)	↓	↓	↑

ganong

The inverse stretch reflex :

A) has a lower threshold than the stretch reflex. B) is a monosynaptic reflex. C) is a disynaptic reflex with a single interneuron inserted between the afferent and efferent limbs. D) is a polysynaptic reflex with many interneurons inserted between the afferent and efferent limbs. E) requires the discharge of central neurons that release acetylcholine.

Thermoreceptors:

A) are activated only by severe cold or severe heat. B) are located on superficial layers of the skin. C) are a subtype of nociceptors. D) are on dendritic endings of Aδ fibers and C fibers. E) all of the above

In questions 3–8, select A if the item is associated with (a) below, B if the item is associated with (b) below, C if the item is associated with both (a) and (b), and D if the item is associated with neither (a) nor (b).

(a) V1A vasopressin receptors (b) V2 vasopressin receptors

3. Activation of Gs **B**

4. Vasoconstriction **A**

5. Increase in intracellular inositol triphosphate **A**

6. Movement of aquaporin **B**

7. Proteinuria **D**

In its action in cells, aldosterone:

A) increases transport of ENaCs from the cytoplasm to the cell membrane. B) does not act on the cell membrane. C) binds to a receptor in the nucleus. D) may activate a heat shock protein. E) also binds to glucocorticoid receptors.

A young man presents with a blood pressure of 175/110 mm Hg. He is found to have a high circulating aldosterone but a low circulating cortisol. Glucocorticoid treatment lowers his circulating aldosterone and lowers his blood pressure to 140/85 mm Hg. He probably has an abnormal:

A) 17α-hydroxylase. B) 21β-hydroxylase. C) 3β-hydroxysteroid dehydrogenase. D) aldosterone synthase. E) cholesterol desmolase.

When a pheochromocytoma (tumor of the adrenal medulla) suddenly discharges a large amount of epinephrine into the circulation, the patient's heart rate would be expected to:

A) increase, because the increase in blood pressure stimulates the carotid and aortic baroreceptors. B) increase, because epinephrine has a direct chronotropic effect on the heart. C) increase, because of increased tonic parasympathetic discharge to the heart. D) decrease, because the increase in blood pressure stimulates the carotid and aortic chemoreceptors. E) decrease, because of increased tonic parasympathetic discharge to the heart

Activation of the baroreceptor reflex:

A) is primarily involved in short-term regulation of systemic blood pressure. B) leads to an increase in heart rate because of inhibition of the vagal cardiac motor neurons. C) inhibits neurons in the CVLM. D) excites neurons in the RVLM. E) all of the above

Sympathetic nerve activity would be expected to increase:

A) if glutamate receptors were blocked in the NTS. B) if GABA receptors were blocked in the RVLM. C) if there was a compression of the RVLM. D) during hypoxia. E) for all of the above

Which of the following does not dilate arterioles in the skin?

A) increased body temperature B) epinephrine C) bradykinin D) substance P E) vasopressin

Dehydration increases the plasma concentration of all the following hormones except:

A) vasopressin. B) angiotensin II. C) aldosterone. D) norepinephrine. E) atrial natriuretic peptide

Renin is secreted by:

A) cells in the macula densa. B) cells in the proximal tubules. C) cells in the distal tubules. D) juxtaglomerular cells. E) cells in the peritubular capillary bed.

Erythropoietin is secreted by:

A) cells in the macula densa. B) cells in the proximal tubules. C) cells in the distal tubules. D) juxtaglomerular cells. E) cells in the peritubular capillary bed

When a woman who has been on a low-sodium diet for 8 d is given an intravenous injection of captopril, a drug that inhibits angiotensin-converting enzyme, one would expect:

A) her blood pressure to rise because her cardiac output would fall. B) her blood pressure to rise because her peripheral resistance would fall. C) her blood pressure to fall because her cardiac output would fall. D) her blood pressure to fall because her peripheral resistance would fall. E) her plasma renin activity to fall because her circulating angiotensin I level would rise.

Which of the following is the principal buffer in interstitial fluid?

A) hemoglobin B) other proteins C) carbonic acid D) H_2PO_4 E) compounds containing histidine

Increasing alveolar ventilation increases the blood pH because:

A) it activates neural mechanisms that remove acid from the blood. B) it makes hemoglobin a stronger acid. C) it increases the PO_2 of the blood. D) it decreases the PCO_2 in the alveoli. E) the increased muscle work of increased breathing generates more CO_2.

Linda

CASE: (for Qs from 1-5)

Joslin Chambers is a 27-year-old assistant manager at a discount department store. One morning, she awakened from a deep sleep and realized that she was more than an hour late for work. She panicked, momentarily regretting her late-night socializing, and then jumped out of bed. Briefly, she felt lightheaded and thought she might faint. She had the sensation that her heart was “racing.” Had she not been so late for work, she would have returned to bed. As she walked

toward the bathroom, she noticed that her light-headedness dissipated. The rest of her day was uneventful.

When Joslin moved rapidly from a supine (lying) position to a standing position, there was a brief, initial decrease in arterial pressure that caused her light-headedness. Describe the sequence of events that produced this transient fall in arterial pressure

Orthostatic hypotension is the phenomenon whereby arterial pressure decreases when one stands up. When a person suddenly moves from a supine (lying) position to a standing position, blood pools in the veins of the legs. (Because the capacitance, or compliance, of the veins is high, they can hold large volumes of blood.) This pooling decreases venous return to the heart, which decreases cardiac output by the Frank–Starling mechanism. (The Frank–Starling mechanism describes the relationship between venous return and cardiac output. Increases in venous return lead to increases in enddiastolic volume. Up to a point, increases in end-diastolic volume lead to increases in cardiac output. Conversely, decreases in venous return lead to decreases in cardiac output.) Because arterial pressure is affected by the volume of blood in the arteries, a decrease in cardiac output (i.e., less blood is pumped into the arterial system) causes a decrease in arterial pressure.

Why did the decrease in arterial pressure cause Joslin to feel light-headed?

When Joslin stood up quickly, she felt light-headed because a brief period of cerebral ischemia occurred as a result of the decrease in arterial pressure. The autoregulatory range for cerebral blood flow is 60 to 140 mm Hg. In other words, cerebral blood flow is maintained constant as long as arterial pressure is greater than 60 mm Hg and less than 140 mm Hg. When Joslin stood up, her arterial pressure briefly decreased below this critical autoregulatory range. As a result, cerebral blood flow decreased, and she felt light-headed.

Joslin's light-headedness was only transient because a reflex was initiated that rapidly restored arterial pressure to normal. Describe the specific effects of this reflex on heart rate, myocardial contractility, TPR, and capacitance of the veins. What receptors are involved in each of these responses?

Baroreceptors located in the carotid sinus and aortic arch sensed the decrease in arterial pressure. The baroreceptor reflex then orchestrated a series of compensatory responses, including increased sympathetic outflow to the heart and blood vessels. There are four consequences of this increased sympathetic outflow:

- Increased heart rate (the sensation of a racing heart), a positive chronotropic effect mediated by b 1-adrenergic receptors in the sinoatrial node

- Increased contractility of the ventricles, a positive inotropic effect mediated by b 1-adrenergic receptors in the ventricular muscle

- Increased arteriolar constriction, mediated by a 1-adrenergicreceptors on vascular smooth muscle of the arterioles

- Increased venoconstriction, mediated by a 1-adrenergic receptors on vascular smooth muscle of the veins

How does each component of the reflex (e.g., the effect on heart rate) help to restore arterial pressure? (Hint: It may help to write the equation that relates arterial pressure, cardiac output, and TPR.)

4. All of the components of the baroreceptor reflex contributed to the restoration of Joslin's arterial pressure (Fig. 2-4).

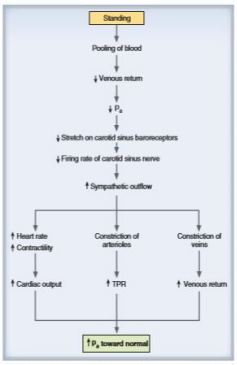


FIGURE 2-4. Cardiovascular responses in a person moving suddenly from a supine to a standing position. P_a, arterial pressure; TPR, total peripheral resistance.

These contributions can be appreciated by reviewing the relationship between arterial pressure , cardiac output, and TPR:

Pa = CO x TPR

Where

Pa = mean arterial pressure

Cardiac output = volume of blood ejected from the left ventricle/min

TPR = total peripheral resistance

In words, arterial pressure depends on the volume of blood pumped into the arteries from the left ventricle and the resistance of the arterioles. (It may be helpful to think of arteriolar resistance as "holding" blood on the arterial side of the circulation.) Now, using the equation, consider how each portion of the baroreceptor reflex helped to restore Joslin's arterial pressure back to normal. The increased heart rate and contractility combined to produce an increase in cardiac output. The increased cardiac output caused an increase in arterial pressure. The increased arteriolar constriction produced an increase in TPR, which also increased the arterial pressure. Finally, venoconstriction led to decreased capacitance of the veins, which increased venous return to the heart and increased the cardiac output (by the Frank–Starling mechanism).

In addition to the reflex correction of blood pressure, the fact that Joslin walked to the bathroom helped return her arterial pressure to normal. How did walking help?

As Joslin walked toward the bathroom, the muscular activity compressed the veins in her legs and decreased venous capacitance (i.e., the volume of blood the veins can hold). This compression, combined with sympathetic venoconstriction, increased venous return to the heart and cardiac output.

Case :

Cassandra Farias is a 34-year-old dietician at an academic medical center. She believes in the importance of a healthy lifestyle and was intrigued when the division of cardiology recruited healthy female volunteers for a study on the cardiovascular responses to exercise. Cassandra met the study criteria (i.e., 25 to 40 years old, no medications, normal weight for height, normal blood pressure), and she was selected for participation. Control measurements were taken of Cassandra's blood pressure, heart rate, and arterial and venous Po2; her stroke volume was estimated. Cassandra then walked on the treadmill for 30 min at 3 miles per hour. Her blood pressure and heart rate were monitored continuously, and her arterial and venous Po2 were measured at the end of the exercise period (Table 2–2)

To set the stage for the following questions, describe the cardiovascular responses to moderate exercise, including the roles of the autonomic nervous system and local control of blood flow in skeletal muscle. What is the ultimate "purpose" of these cardiovascular responses?

table 2–2 Cassandra's Cardiovascular Responses to Exercise

Parameter	Control (Pre-exercise)	Exercise
Systolic blood pressure	110 mm Hg	145 mm Hg
Diastolic blood pressure	70 mm Hg	60 mm Hg
Heart rate	75 beats/min	130 beats/min
Stroke volume (estimated)	80 mL	110 mL
Arterial Po_2	100 mm Hg	100 mm Hg
Venous Po_2	40 mm Hg	25 mm Hg

The "goal" of the cardiovascular responses to exercise is to increaseO2 delivery to muscles that are working harder (skeletal and cardiac muscle). The major mechanism for providing this additional O2 is increased blood flow to the exercising skeletal muscle and the myocardium. In principle, blood flow in an organ can be increased in two ways: (i) total blood flow (cardiac output) can increase, which also increases blood flow to individual organs; or (ii) blood flow can be redistributed so that the percentage of total flow to some organs is increased at the expense of other organs. During exercise, both of these mechanisms are utilized: cardiac output increases significantly (through increases in heart rate and stroke volume), and blood flow is redistributed to the skeletal muscle and myocardium, so that these tissues receive a greater percentage of the (increased) cardiac output. Figure 2–9 summarizes these responses.

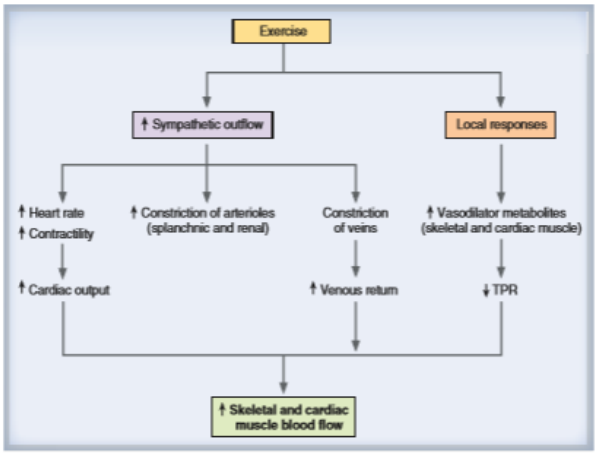


FIGURE 2–8. Cardiovascular responses to exercise.

At the initiation of exercise, muscle mechanoreceptors and chemoreceptors trigger reflexes that send afferent signals to the cerebral motor cortex. The cerebral cortex then directs responses that include increased sympathetic outflow to the heart and blood vessels. (i) In the heart, increased sympathetic activity, through activation of b 1 receptors, produces an increase in heart rate and an increase in contractility. The increase in contractility results in increased stroke volume. Together with increased heart rate, this increased stroke volume produces an increase in cardiac output. (Recall that cardiac output = stroke volume × heart rate.) (ii) In addition, increased sympathetic activity,

through a 1 receptors, produces arteriolar constriction in some vascular beds (e.g., splanchnic, renal) and venoconstriction. (iii) Venoconstriction (combined with compression of the veins by the squeezing action of skeletal muscle) increases venous return to the heart. Increased venous return is an essential component of the response to exercise; it provides the increased end-diastolic volume that is needed to produce the increase in cardiac output (Frank–Starling mechanism). In addition to these central responses that are orchestrated by the sympathetic nervous system, local responses occur in skeletal and cardiac muscle to increase their blood flow. In skeletal muscle, as the metabolic rate increases, metabolites such as lactate, K+, nitric oxide, and adenosine are generated. These metabolites produce vasodilation of skeletal muscle arterioles, thereby increasing the local blood flow. This local vasodilation in skeletal muscle is so prominent that it is responsible for an overall decrease in total peripheral resistance (TPr). (If these local responses in skeletal muscle did not occur, TPR would have increased as a result of sympathetic vasoconstriction.) Local responses also dominate in the myocardium, where they are primarily mediated by adenosine and decreased Po2 and cause vasodilation and increased coronary blood flow

Early in the exercise period, Cassandra's skin was cool to the touch. However, at the peak of exercise, her skin was flushed and very warm to the touch. What mechanisms were responsible for these changes in skin color and temperature as the exercise progressed?

Cutaneous blood flow exhibits a biphasic response to exercise. Early in exercise, vasoconstriction of cutaneous arterioles occurs as a result of the activation of sympathetic α1 receptors. Blood flow is shunted away from the skin, and the skin is cool. As exercise progresses, body temperature increases secondary to increased O2 consumption, and sympathetic centers controlling cutaneous blood flow in the anterior hypothalamus are inhibited. This selective inhibition of sympathetic activity produces vasodilation in cutaneous arterioles. As a result, warmed blood is shunted from the body core to venous plexus near the skin surface, as evidenced by redness and warmth of the skin.

Case:

Stewart Hanna is a 58-year-old partner in a real estate firm. Over the years, the pressures of the job have taken their toll. Mr. Hanna has smoked two packs of filtered cigarettes a day for 40 years. He tries to watch his diet, but "required" business lunches and cocktail hours have driven his weight up to 210 lb. (He is 5 feet, 9 inches tall.) He recently separated from his wife of 35 years and is dating a much younger woman. Suddenly realizing how out of shape he had become, he made an appointment for a physical examination. In his physician's office, Mr. Hanna's blood pressure was 180/125 (normal, 120/80). The physician heard a continuous abdominal bruit (sound). Because of Mr. Hanna's elevated blood pressure and the bruit, the physician drew a venous blood sample to determine plasma renin levels. After receiving the results, the physician ordered an additional test called a differential renal vein renin. Mr. Hanna's plasma renin activity was 10 ng/mL per hour (normal, 0.9 to 3.3 ng/mL per hour). His differential renal vein renin (left to right) was 1.6 (normal is 1.0). The test results were consistent with left renal artery stenosis. Mr. Hanna was scheduled for a renal arteriogram, which showed 80% occlusion of the left renal artery as a result of severe atherosclerotic disease. A balloon angioplasty was performed immediately to clear the occlusion. Mr. Hanna's blood pressure was expected to return to normal after the procedure. He was ordered to stop smoking, follow a low-fat diet, exercise regularly, and undergo periodic physical examinations.

How did occlusion of Mr. Hanna's left renal artery lead to an increase in plasma renin activity ?

Atherosclerotic disease caused occlusion (narrowing) of Mr. Hanna's left renal artery. This occlusion caused a decrease in renal perfusion pressure, which then stimulated renin secretion from the kidney's juxtaglomerular cells (Fig. 2–11). Increased quantities of renin, secreted by Mr. Hanna's left kidney, entered renal venous blood and then the systemic circulation (increased plasma renin activity).

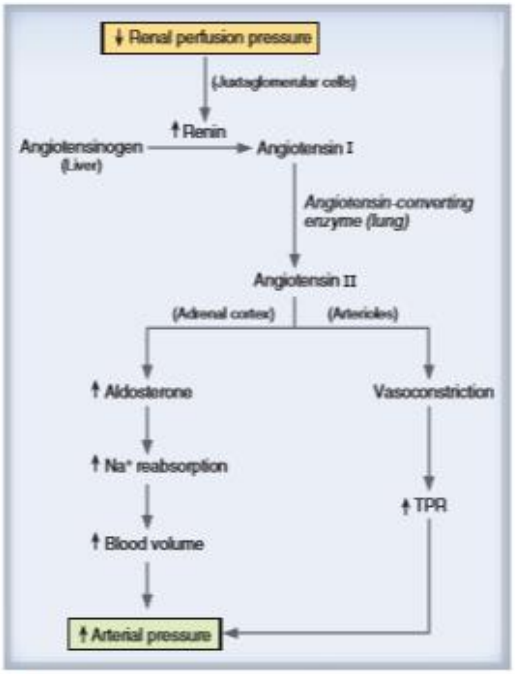


FIGURE 2–11. The renin–angiotensin II–aldosterone system. TPR, total peripheral resistance.

How did the increase in plasma renin activity cause an elevation in Mr. Hanna's arterial blood pressure (called renovascular hypertension) ?

Renin is an enzyme that catalyzes the conversion of angiotensinogen (renin substrate) to angiotensin I. Angiotensin I is then converted, primarily in the lungs, to angiotensin II, which has several biologic actions. The first action of angiotensin II is to stimulate the synthesis and secretion of aldosterone by the adrenal cortex; aldosterone increases renal Na+ reabsorption, extracellular fluid volume, and blood volume. The second action of angiotensin II is to cause vasoconstriction of arterioles; this vasoconstriction increases the TPR. In Mr. Hanna, the increase in blood volume (which increased venous return and cardiac output) combined with the increase in TPR to produce an increase in his arterial pressure. (Recall from Case 10 that Pa = cardiac output × TPR.) Mr. Hanna had renovascular hypertension, in which his left kidney incorrectly sensed low arterial pressure. Because his left renal artery was stenosed, there was a decrease in left renal perfusion pressure that activated the renin–angiotensin II–aldosterone system and produced an increase in arterial pressure above normal

The differential renal vein renin measurement involves determining the renin level in venous blood from each kidney. In healthy persons, the renal vein renin level from each kidney is approximately the same; therefore, the ratio of left to right renin is 1.0. In Mr. Hanna, this ratio was elevated to 1.6. Although it is not apparent, the elevation of the ratio actually had two components: (i) his left renal vein renin was increased and (ii) his right renal vein renin was decreased. Why was renin secretion increased in the left kidney and decreased in the right kidney ?

In the question, you were told that the ratio of left to right renin was elevated for two reasons: (i) increased renin secretion by the left kidney and (ii) decreased renin secretion by the right kidney. Based on the earlier discussion, it is relatively easy to state why left renal renin secretion was increased: narrowing of the left renal artery led to decreased left renal perfusion pressure and increased left renal renin secretion. But how can we explain decreased renin secretion by the right kidney? The answer lies in the response of the normal right kidney to the increased arterial pressure (that resulted from stenosis of the left renal artery). The right kidney sensed increased arterial pressure and responded appropriately by decreasing its renin secretion.

If the balloon angioplasty was not successful, Mr. Hanna would be treated with an angiotensin converting enzyme (ACE) inhibitor (e.g., captopril). What is the rationale for using ACE inhibitors to treat hypertension caused by renal artery stenosis ?

The reason why angiotensin-converting enzyme (ACE) inhibitors such as captopril successfully lower arterial pressure in renovascular hypertension should be evident from the pathogenesis of the elevated blood pressure. In Mr. Hanna's case, unilateral renal artery stenosis led to increased plasma renin activity, which led to increased levels of angiotensin II. Angiotensin II caused the increase in arterial pressure, both directly by vasoconstriction and indirectly through the actions of aldosterone. Blocking the production of angiotensin II by inhibiting ACE activity interrupts this sequence of events.

Case:

Mavis Byrne is a 78-year-old widow who was brought to the emergency room one evening by her sister. Early in the day, Mrs. Byrne had seen bright red blood in her stool, which she attributed to hemorrhoids. She continued with her daily activities: she cleaned her house in the morning, had lunch with friends, and volunteered in the afternoon as a "hugger" in the newborn intensive care unit. However, the bleeding continued all day, and by dinnertime, she could no longer ignore it. Mrs. Byrne does not smoke or drink alcoholic beverages. She takes aspirin, as needed, for arthritis, sometimes up to 10 tablets daily. In the emergency room, Mrs. Byrne was light-headed, pale, cold, and very anxious. Her hematocrit was 29% (normal for women, 36 to 46%). Table 2–3 shows Mrs. Byrne's blood pressure and heart rate in the lying (supine) and upright (standing) positions.

An infusion of normal saline was started, and a blood sample was drawn to be typed and

table 2–3 Mrs. Byrne's Blood Pressure and Heart Rate

Parameter	Lying Down (Supine)	Upright (Standing)
Blood pressure	90/60	75/45
Heart rate	105 beats/min	135 beats/min

crossmatched to prepare for a blood transfusion. A colonoscopy showed that the bleeding came from herniations in the colonic wall, called diverticula. (When arteries in the colon wall rupture,

bleeding can be quite vigorous.) By the time of the colonoscopy, the bleeding had stopped spontaneously. Because of the quantity of blood lost, Mrs. Byrne received two units of whole blood and was admitted for observation. The physicians were prepared to insert a bladder catheter to allow continuous monitoring of urine output. However, by the next morning, her normal color had returned, she was no longer light-headed, and her blood pressure, both lying and standing, had returned to normal. No additional treatment or monitoring was needed. Mrs. Byrne was discharged to the care of her sister and advised to “take it easy”.

What is the definition of circulatory shock? What are the major causes ?

Shock (or circulatoryshock) is a condition in which decreased blood flow causes decreased tissue perfusion and O2 delivery. Untreated, shock can lead to impaired tissue and cellular metabolism and, ultimately, death. In categorizing the causes of shock, it is helpful to consider the components of the cardiovascular system that determine blood flow to the tissues: the heart (the pump), the blood vessels, and the volume of blood in the system. Shock can be caused by a failure of, or deficit in, any of these components. Hypovolemic shock occurs when circulating blood volume is decreased because of loss of whole blood (hemorrhagic shock), loss of plasma volume (e.g., burn), or loss of fluid and electrolytes (e.g., vomiting, diarrhea). Cardiogenic shock is caused by myocardial impairment (e.g., myocardial infarction, congestive heart failure). Mechanical obstruction to blood flow can occur anywhere in the circulatory system and cause a local decrease in blood flow. Neurogenic shock (e.g., deep general anesthesia, spinal anesthesia, spinal cord injury) involves loss of vasomotor tone, which leads to venous pooling of blood. Septic or anaphylactic shock involves increased filtration across capillary walls, which leads to decreased circulating blood volume.

After the gastrointestinal blood loss, what sequence of events led to Mrs. Byrne’s decreased arterial pressure?

Mrs. Byrne had a gastrointestinal hemorrhage and lost a significant volume of whole blood. How did this blood loss lead to decreased arterial pressure? Although it is tempting to picture blood pouring out of the arteries as the direct cause of her decreased arterial pressure, this explanation is an oversimplification. A number of intervening steps are involved. Recall that because the capacitance of the veins is high, most of the blood volume is contained in the veins, not in the arteries. Therefore, when a hemorrhage occurs, most of the blood volume that is lost comes from the veins. A decrease in venous volume leads to a decrease in venous return to the heart and a decrease in end-diastolic volume (preload). A decrease in end-diastolic volume leads to a decrease in cardiac output by the Frank–Starling mechanism (the length–tension relationship for the ventricles). A decrease in cardiac output leads to a decrease in arterial pressure, as expressed by the familiar relationship: Arterial pressure = cardiac output × TPR (symbolically, Pa = cardiac output × TPR). Thus, after blood loss, the fundamental problem is decreased venous volume and venous return, leading to decreased cardiac output. In textbooks, you will see references to filling pressure, venous filling pressure, or cardiac filling pressure. All of these terms refer to the relationships between venous volume, venous return, cardiac output, and (ultimately) arterial pressure.

Why was Mrs. Byrne's arterial pressure lower in the upright position than in the lying (supine) position ?

Mrs. Byrne's arterial pressure was lower in the upright position than in the supine position (orthostatic hypotension) because when she was upright, blood pooled in the veins of her legs and her venous return was further compromised. As a result, end-diastolic volume was further reduced, which led to further reductions in cardiac output and arterial pressure.

Mrs. Byrne's heart rate was elevated (105 beats/min) when she was supine. Why? Why was her heart rate even more elevated (135 beats/min) when she was upright?

Asking why Mrs. Byrne's heart rate was elevated brings us to the larger issues of compensatory responses to hemorrhage. Essentially, decreased arterial pressure triggers several compensatory mechanisms, including an increase in heart rate, that attempt to restore blood pressure to normal (Fig. 2–12). Two major mechanisms are activated in response to decreased arterial pressure: (i) the baroreceptor reflex and (ii) the renin–angiotensin II–aldosterone system (discussed in Question 8). In the baroreceptor reflex, sympathetic outflow to the heart and blood vessels is increased. As a result, heart rate and contractility increase and cause an increase in cardiac output. There is arteriolar constriction, which increases TPR, and there is venoconstriction, which increases venous return. Looking once again at the equation for arterial pressure (Pa = cardiac output × TPR), you can appreciate how each of these changes works to restore arterial pressure toward normal.

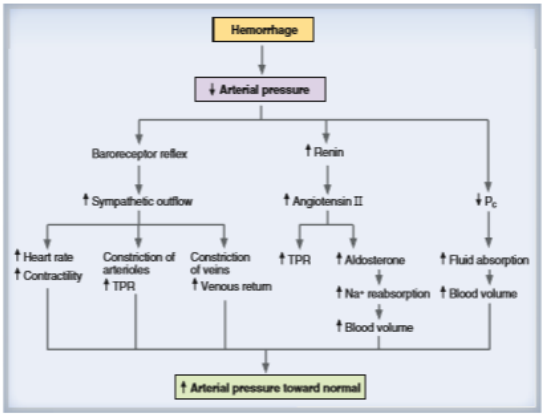


FIGURE 2–12. Cardiovascular responses to hemorrhage. P_c, capillary hydrostatic pressure; TPR, total peripheral resistance. (Reprinted, with permission, from Costanzo LS. *BRS Physiology*. 5th ed. Baltimore: Lippincott Williams & Wilkins; 2011:98.)

Mrs. Byrne's heart rate was more elevated in the upright position than in the supine position because her arterial blood pressure was lower when she was upright (venous pooling). Therefore, the baroreceptor mechanism was more strongly stimulated, and sympathetic stimulation of the heart and blood vessels (including the increase in heart rate) was exaggerated.

If central venous pressure and pulmonary capillary wedge pressure had been measured, would you expect their values to have been increased, decreased, or the same as in a healthy person ?

Central venous pressure is measured in the vena cava. Its value is related to the volume of blood in the veins and is approximately equal to the right atrial pressure. Pulmonary capillary wedge pressure is measured by advancing a catheter through the pulmonary artery until it "wedges" in the artery's smallest branch. At that point, the catheter senses pulmonary capillary pressure, which is nearly equal to the left atrial pressure. Thus, central venous pressure estimates the right atrial pressure, and pulmonary capillary wedge pressure estimates the left atrial pressure. The values reflect end-diastolic volume, or preload, of the right and left ventricles, respectively. Had they been measured, Mrs. Byrne's central venous pressure and pulmonary capillary wedge pressure both would have been decreased because of the loss of blood volume from the venous side of the circulation.

What is hematocrit? Why was Mrs. Byrne's hematocrit decreased, and why was this decrease potentially dangerous?

Hematocrit is the fraction (or percentage) of blood volume occupied by red blood cells; the remaining fraction of whole blood is plasma, which is mostly water. A decrease in hematocrit can be caused by any number of factors, including blood loss, decreased red blood cell production, increased red blood cell destruction, or an increase in plasma volume without an accompanying increase in red blood cell volume. In Mrs. Byrne's case, the decreased hematocrit was probably secondary to the hemorrhage of whole blood. But, wait a minute! You may be asking: If whole blood was lost from the gastrointestinal tract, why would hematocrit be changed (reasoning that red blood cells and plasma were lost

proportionately)? In the first hours after hemorrhage, it is true that hematocrit is unchanged. However, as plasma volume is restored (as a result of increased aldosterone levels [see the answer to Question 8], increased capillary absorption of fluid, and the infusion of saline), plasma volume increases, but red blood cell volume does not. (It takes about 7 days for stem cells to become mature red blood cells.) Therefore, Mrs. Byrne's hematocrit was decreased by dilution. A decrease in hematocrit is dangerous because red blood cells contain hemoglobin, the O2carrying protein of blood. Thus, after a hemorrhage, there are two potentially lethal consequences for O2 delivery to the tissues: the decrease in blood flow to the tissues (i.e., decreased cardiac output) and the decreased O2-carrying capacity of the blood (decreased hematocrit.(

Why was her skin pale and cold ?

Mrs. Byrne's pale, cold skin is typical of the response to hemorrhage, reflecting vasoconstriction of cutaneous arterioles. As the baroreceptor reflex was initiated in response to decreased arterial pressure (see Question 4), sympathetic vasoconstriction of arterioles occurred in many vascular beds, including the skin. Cutaneous vasoconstriction particularly makes sense as it allows the body to increase the arterial pressure and redirect blood flow to more vital organs (e.g., brain, heart). In contrast to hemorrhagic or hypovolemic shock, where the skin is cold and pale, in the

early stages of septic shock (e.g., secondary to a gram negative infection), the skin is warm and flushed due to increased circulating vasodilators.

If Mrs. Byrne's urinary Na+ excretion had been measured, would you expect it to be higher than, lower than, or the same as that of a healthy person? Why?

If urinary Na+ excretion had been measured, it likely would have been decreased. The reason for this decreased Na+ excretion is activation of the renin–angiotensin II–aldosterone system in response to decreased arterial pressure. Increased levels of aldosterone cause increased Na+ reabsorption in the late distal tubule and collecting duct of the kidney (i.e., decreased Na+ excretion). This mechanism is designed to increase the amount of Na+ in extracellular fluid, which increases extracellular fluid volume and blood volume. Increased blood volume leads to increased venous return, increased cardiac output, and ultimately, increased arterial pressure.

How was the saline infusion expected to help her condition?

In an attempt to restore venous return and cardiac output, Mrs. Byrne received an infusion of saline to increase her extracellular fluid volume and blood volume. The saline infusion accomplished a result similar to the body's endogenous aldosterone, only faster.

Why did the physicians consider monitoring her urine output? How do prostaglandins "protect" RBF after a hemorrhage? In this regard, why was it dangerous that Mrs. Byrne had been taking aspirin?

A critical element in the response to hemorrhage, and one that may determine the outcome for the patient, is the "balancing act" between vasoconstriction in some organs (e.g., kidney) and maintaining blood flow in those organs. Increased sympathetic activity and increased angiotensin II both produce vasoconstriction and an increase in TPR, which is important to the body's attempt to restore arterial pressure (recall that Pa = cardiac output × TPR). However, vasoconstriction, by increasing resistance, decreases the blood flow in the involved organs. Of particular note is the kidney, where both sympathetic activity and angiotensin II cause arteriolar vasoconstriction. If unopposed, this vasoconstriction can compromise renal blood flow and glomerular filtration rate (GFr), producing renal failure and even death. Thus, had Mrs. Byrne not recovered quickly, it would have been important to monitor her urine output as an indicator of renal perfusion and renal function. Notice the word "unopposed" in the previous paragraph. Perhaps this word led you to question whether there are endogenous "modulators" of the vasoconstricting effects of sympathetic activity and angiotensin II in the kidneys. Yes, there are! Prostaglandins serve this modulatory role. Both sympathetic activity and angiotensin II cause increased local production of prostaglandin E2 and prostaglandin I2, which are renal vasodilators. Thus, the vasoconstrictive effects of sympathetic activity and angiotensin II are offset by the vasodilatory effects of endogenous prostaglandins. Renal blood flow is thereby protected and maintained in high vasoconstrictor states, such as hemorrhage. The confounding and potentially harmful issue with Mrs. Byrne was her use of large amounts of aspirin for her arthritis. Aspirin, a nonsteroidal anti-inflammatory drug (NSAID), is a cyclooxygenase inhibitor that blocks prostaglandin synthesis.

Therefore, Mrs. Byrne was at risk for developing renal failure if her ingestion of aspirin prevented the protective, vasodilatory effects of prostaglandins.

Had her blood loss been more severe, Mrs. Byrne might have received a low dose of dopamine, which has selective actions in various vascular beds. In cerebral, cardiac, renal, and mesenteric vascular beds, dopamine is a vasodilator; in muscle and cutaneous vascular beds, dopamine is a vasoconstrictor. Why is low-dose dopamine helpful in the treatment of hypovolemic shock?

Mrs. Byrne's physicians were prepared to administer a low dose of dopamine if her blood pressure and blood flow (as reflected in the color returning to her skin) had not been corrected. Dopamine, a precursor of norepinephrine, has its own vasoactive properties, as explained in the question. Low doses of dopamine selectively dilate arterioles in critical organs (i.e., heart, brain, kidney) and selectively constrict arterioles in less critical organs (e.g., skeletal muscle, skin), thus redirecting blood flow where it is most needed. In particular, the kidneys, which might otherwise be vasoconstricted as a result of increased sympathetic activity and angiotensin II, may be spared by the vasodilatory actions of dopamine.

Case :

At the time of her death, Celia Lukas was a 38-year-old homemaker and mother of three children, 15, 14, and 12 years of age. She had an associate's degree in computer programming from a community college, but had not worked outside the home since the birth of her first child. Keeping house and driving the children to activities kept her very busy. To stay in shape, she took aerobics classes at the local community center. The first sign that Celia was ill was vague: she fatigued easily.

table 2–4 Results of Celia's Cardiac Catheterization

Pressure	Value
Mean pulmonary artery pressure	35 mm Hg (normal, 15 mm Hg)
Right ventricular pressure	Increased
Right atrial pressure	Increased
Pulmonary capillary wedge pressure	Normal

However, within 6 months, Celia was short of breath (dyspnea), both at rest and when she exercised, and she had swelling in her legs and feet. She made an appointment to see her physician. On physical examination, Celia's jugular veins were distended, her liver was enlarged (hepatomegaly), and she had ascites in her peritoneal cavity and edema in her legs. A fourth heart sound was audible over her right ventricle. The physician was very concerned and immediately scheduled Celia for a chest x-ray, an ECG, and a cardiac catheterization. The chest x-ray showed enlargement of the right ventricle and prominent pulmonary arteries. The ECG findings were consistent with right ventricular hypertrophy. The results of cardiac catheterization are shown in Table 2–4.

Consulting physicians in cardiology and pulmonology concluded that Celia had primary pulmonary hypertension, a rare type of pulmonary hypertension that is caused by diffuse pathologic changes in the pulmonary arteries. These abnormalities lead to increased pulmonary vascular resistance and pulmonary hypertension, which causes right ventricular failure (cor pulmonale). Celia was treated with vasodilator drugs, but they were not effective. Her name was added to a list of patients awaiting a heart–lung transplant. However, she died of right heart failure before a transplant could be performed.

Why did increased pulmonary vascular resistance cause an increase in pulmonary artery pressure (pulmonary hypertension)?

To explain why increased pulmonary vascular resistance (caused by intrinsic pathology of the small pulmonary arteries) led to increased pulmonary artery pressure, it is necessary to think about the relationship between pressure, flow, and resistance. Recall this relationship from Case 10: ΔP = blood flow × resistance. Mathematically, it is easy to see that if the blood flow (in this case, pulmonary blood flow) is constant and resistance of the blood vessels increases, then ΔP, the pressure difference between the pulmonary artery and the pulmonary vein, must increase. ΔP could increase because pressure in the pulmonary artery increases or because pressure in the pulmonary vein decreases. (Note, however, that a decrease in pulmonary vein pressure would have little impact on ΔP because its value is normally very low.) In Celia, ΔP increased because her pulmonary arterial pressure increased. As pulmonary vascular resistance increased, resistance to blood flow increased, and blood "backed up" proximal to the pulmonary microcirculation into the pulmonary arteries. Increased blood volume in the pulmonary arteries caused increased pressure.

Case:

Marvin Zimmerman is a 52-year-old construction manager who is significantly overweight. Despite his physician's repeated admonitions, Marvin ate a rich diet that included red meats and high-calorie desserts. Marvin also enjoyed unwinding with a few beers each evening. He joked with the guys, "I guess I'm a heart attack waiting to happen." He had occasional chest pains (angina) that were relieved by nitroglycerin. The evening of his myocardial infarction, Marvin went to bed early because he wasn't feeling well. He awakened at 2:00 a.m. with crushing pressure in his chest and pain radiating down his left arm that was not relieved by nitroglycerin. He was nauseated and sweating profusely. He also had difficulty breathing (dyspnea), especially when he was recumbent (orthopnea). His breathing was "noisy." Marvin's wife called 911, and paramedics arrived promptly and transported him to the nearest hospital. In the emergency room, Marvin's blood pressure was 105/80. Inspiratory rales were present, consistent with pulmonary edema, and his skin was cold and clammy. Sequential ECGs and serum levels of cardiac enzymes (creatine phosphokinase and lactate dehydrogenase) suggested a left ventricular wall myocardial infarction. Pulmonary capillary wedge pressure, obtained during cardiac catheterization, was 30 mm Hg (normal, 5 mm Hg). His ejection fraction, measured with two-dimensional echocardiography, was 0.35 (normal, 0.55). Marvin was transferred to the coronary intensive care unit. He was treated with a thrombolytic agent to prevent another myocardial infarction, digitalis (a positive inotropic agent), and furosemide (a loop diuretic). After 7 days in the hospital, he was sent home on a strict, low-fat, low-Na+ diet.

Why was Marvin sent home on a low-Na+ diet?

Extracellular fluid volume is determined by extracellular Na+ content. A low-Na+ diet was recommended to reduce the extracellular fluid volume and blood volume and to prevent subsequent episodes of pulmonary edema (similar to the idea of treating Marvin with a diuretic).

Calculate the stroke volume, cardiac output, and ejection fraction of the left ventricle.

2. These calculations concern the cardiac output of the left ventricle. The basic relationships are as follows:

Stroke volume = **end-diastolic volume** - **end-systolic volume**

where

Stroke volume □□volume ejected by the ventricle during systole (mL

End-diastolic volume □□) volume in the ventricle before ejection (mL)

End-systolic volume □□volume in the ventricle after ejection (mL)

Cardiac output = **stroke volume ¥ heart rate**

where

Cardiac output □□volume ejected by the ventricle per minute (mL/min)

Stroke volume □□volume ejected by the ventricle (mL)

Heart rate □□beats/min

Ejection fraction = **stroke volume/end-diastolic volume**

where

Ejection fraction □□fraction of the end-diastolic volume ejected in one stroke

Now we can use these basic equations to calculate the stroke volume, cardiac output, and **ejection fraction** in this case.

Stroke volume □□left ventricular end-diastolic volume – left ventricular end-systolic volume

□□□□□□□□□□□□□□□□□□□□□□□□□□□□□□□140 mL – 70 mL

□□□□□□□□□□□□□□□□□□□□□□□□□□□□□□□70 mL

Cardiac output is the volume ejected by the left ventricle per minute. It is calculated as the product of **stroke volume** (determined to be 70 mL) and **heart rate.** Heart rate is not given in Table 2–1, but it can be calculated from the **r-r interval.** "R" is the R-wave on the electrocardiogram and represents elec- trical activation of the ventricles. The R-R interval is the time elapsed from one R-wave to the next (Fig. 2–2). It is also called **cycle length** (i.e., time elapsed in one cardiac cycle).

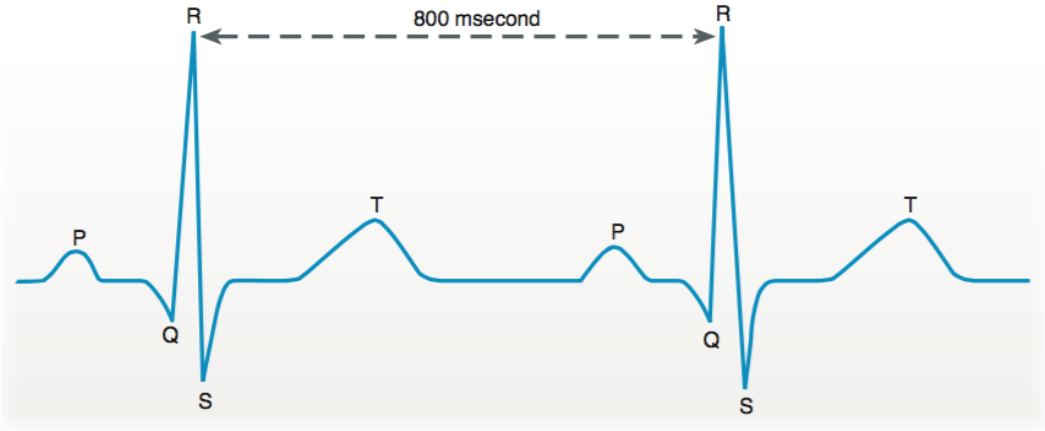


FIGURE 2–2. ECG measured from lead II. The interval between R-waves is the cycle length.

Cycle length can be used to calculate the heart rate as follows:

Heart rate □□1/cycle length

□□□□□□□□□□□□□□□□□□□□1/800 msec

□□□□□□□□□□□□□□□□□□□□1/0.8 sec

□□1.25 beats/sec

□□□□□□□□□□□□□□□□□□□□75 beats/min

Cardiac output □□stroke volume □□heart rate

□□70 mL □□75 beats/min

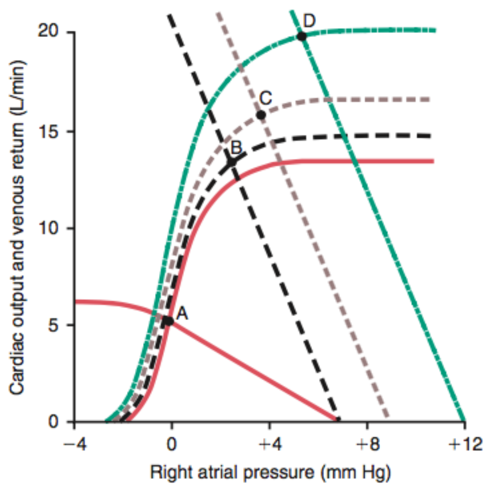
□□□□□□□□□□□□□□□□□□□□□□□□□□□□5,250 mL/min

Ejection fraction □□stroke volume/end-diastolic volume

□□□□□□□□□□□□□□□□□□□□□□□□□□□□70 mL/140 mL

□□0.5, or 50%

1)In the following graph, for the cardiac output and venous return curves defined by the solid red lines (with the equilibrium at A), which of the following is true?



A) Mean systemic filling pressure is 12 mm Hg

B) Right atrial pressure is 2 mm Hg

C) Resistance to venous return is 1.4 mm Hg/L/min

D) Pulmonary arterial flow is approximately 7 L/min

E) Resistance to venous return is 0.71 mm Hg/L/min

Explanation :

C) The formula for resistance to venous return is mean systemic filling pressure **2** right atrial pressure/ cardiac output. In this example the mean systemic filling pressure is 7 mm Hg and the right atrial pressure is 0 mm Hg. The cardiac output is 5 L/min. Using these values in the previous formula indicates that the resis- tance to venous return is 1.4 mm Hg/L/min. Note that this formula only applies to the linear portion of the venous return curve.

71)A30-year-old male is resting,and his sympathetic out- put increases to maximal values. Which of the follow- ing sets of changes would be expected in response to this increased sympathetic output?

	Resistance to venous return	Mean systemic filling pressure	Venous return
A)	↑	↑	↑
B)	↑	↓	↑
C)	↑	↓	↓
D)	↑	↑	↓
E)	↓	↓	↓
F)	↓	↑	↓
G)	↓	↑	↑
H)	↓	↓	↑

Explanation :

1. A) During increases in sympathetic output to maximal values, several changes occur. First, the mean systemic filling pressure increases markedly, but at the same time the resistance to venous return increases. Venous return is determined by the formula: mean systemic filling pressure – right atrial pressure/resistance to venous return. During maximal sympathetic output, the increase in systemic filling pressure is greater than the increase in resistance to venous return. Therefore, in this formula the numerator has a much greater increase than the denominator. This results in an increase in the venous return.

73)If the thorax of a normal person is surgically opened, what will happen to the cardiac output curve?

A) It shifts to the left 2 mm Hg

B) It shifts to the left 4 mm Hg

C) It shifts to the right 2 mm Hg

D) It shifts to the right 4 mm Hg

E) It does not shift

Explanation :

D) The normal intrapleural pressure is -4 mm Hg. When the thorax is surgically opened, all pressures inside the chest will immediately have a value of 0 mm Hg, which is the atmospheric pressure. This increased pressure in the chest tends to collapse the atria and decreases the transmural pressure across each of the atria. In particular, the right atrial transmural pressure

gradient decreases about 4 mm Hg. There- fore, the cardiac output curve will shift to the right by 4 mm H

74)Which of the following normally cause the cardiac output curve to shift to the left along the right atrial pressure axis?

A) Surgically opening the chest

B) Severe cardiac tamponade

C) Breathing against a negative pressure

D) Playing a trumpet

E) Positive pressure breathing

Explanation :

74. C) Several factors can cause the cardiac output to shift to the right or to the left. These factors include surgi- cally opening the chest, which makes the cardiac out- put curve shift 4 mm Hg to the right, and severe car- diac tamponade, which increases the pressure inside the pericardium, thus tending to collapse the heart, particularly the atria. Playing a trumpet or positive pressure breathing tremendously increases the inter- pleural pressure, thus collapsing the atria and shifting the cardiac output curve to the right. Breathing against a negative pressure will shift the cardiac output curve to the left.

75)Which of the following will elevate the plateau of the cardiac output curve?

A) Surgically opening the thoracic cage

B) Placing a patient on a mechanical ventilator

C) Cardiac tamponade

D) Increasing parasympathetic stimulation of the heart

E) Increasing sympathetic stimulation of the heart

Explanation:

E) The plateau level of thec ardiac output curve,which is one measure of cardiac contractility, decreases in several circumstances. Some of these include severe cardiac tamponade that

increases the pressure in the pericardial space and increasing parasympathetic stim- ulation of the heart. Increased sympathetic stimulation of the heart increases the level of the cardiac output curve by increasing heart rate and contractility.

76)Which of the following normally cause the cardiac output curve to shift to the right along the right atrial pressure axis?

A) Decreasing intrapleural pressure to -6 mm Hg

B) Increasing mean systemic filling pressure

C) Taking a patient off a mechanical ventilator and allowing normal respiration

D) Surgically opening the chest

E) Breathing against a negative pressure

Explanation:

D) Several factors can cause the cardiac output to shift to the right or to the left. These factors include surgically opening the chest, which makes the cardiac output curve shift 4 mm Hg to the right, and severe cardiac tamponade, which increases the pressure inside the pericardium, thus tending to collapse the heart, particularly the atria. Playing a trumpet or posi- tive pressure breathing including being on a mechani- cal ventilator tremendously increases the interpleural pressure, thus collapsing the atria and shifting the cardiac output curve to the right.

77)Which of the following conditions would be expected to decrease mean systemic filling pressure?

A) Norepinephrine administration

B) Increased blood volume

C) Increased sympathetic stimulation

D) Increased venous compliance

E) Skeletal muscle contraction

Explanation :

D) Mean systemic filling pressure is a measure of the tightness of fit of the blood in the circulation. Mean systemic filling pressure is increased by factors which increase blood volume and by factors that decrease the vascular compliance. Therefore, a decreased venous compliance, not an increased compliance, would cause an increase in mean systemic filling pressure. Norepi-

nephrine administration and sympathetic stimulation cause arteriolar vasoconstriction and decreased vascu- lar compliance resulting in an increase in mean systemic filling pressure. Increased blood volume and skeletal muscle contraction, which cause a contraction of the vasculature, also increase this filling pressure.

78)Which of the following is normally associated with an increased venous return of blood to the heart?

A) Decreased mean systemic filling pressure

B) Acute large vein dilation

C) Decreased sympathetic tone

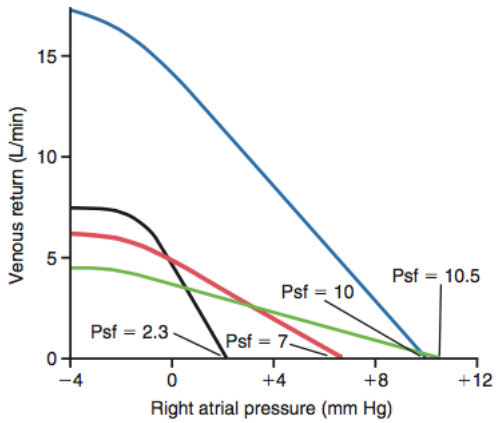
D) Increased venous compliance

E) Increased blood volume

Explanation :

E) Venous return of the heart is equal to the mean systemic filling pressure minus the right atrial pressure divided by the resistance to venous return. Therefore, decreased mean systemic filling pressure will decrease the venous return to the heart. Factors that will decrease the systemic filling pressure include large vein dilation, decreased sympathetic tone, increased venous compliance and increased blood volume.

79)Which of the curves in the following graph has the highest resistance to venous return?



A) Blue line with mean systemic pressure (Psf) 5 10

B) Green line with Psf 5 10.5

C) Black line with Psf 5 2.3

D) Red line with Psf 5 7

Explanation :

B) The resistance to venous return is the inverse of the slope of the linear portion of the venous return curve. Therefore, the curve with the lowest slope will have the highest resistance to venous return.

80)Which of the following is normally associated with an increased cardiac output?

A) Increased venous compliance

B) Cardiac tamponade

C) Surgically opening the chest

D) Moderate anemia

E) Severe aortic stenosis

Explanation :

D) Decreased cardiac output can result from a weak- ened heart or from a decrease in venous return. Increased venous compliance decreases the venous return of blood to the heart. Cardiac tamponade, sur- gically opening the chest and severe aortic stenosis will effectively weaken the heart and thus decrease cardiac output. Moderate anemia will cause an arteriolar vaso- dilation, which increases venous return of blood back to the heart thus increasing cardiac output.

81)In which of the following conditions would you nor- mally expect to find a decreased cardiac output?

A) Hyperthyroidism

B) Beriberi

C) A-V fistula

D) Anemia

E) Acute myocardial infarction

Explanation:

E) Cardiac output increases in several conditions because of increased venous return. Cardiac output increases in hyperthyroidism because of the increased oxygen use by the peripheral tissues resulting in arterio- lar vasodilation and thus increased venous return. Beri- beri causes increased cardiac output because a lack of the vitamin thiamine results in peripheral vasodilation. A-V fistulae also cause a decreased resistance to venous return thus increasing cardiac output. Anemia, because of the decreased oxygen delivery to the tissues, causes an increase in venous return to the heart and thus an increase in cardiac output. Cardiac output decreases in patients with myocardial infarction.

104)If a 21-year-old male patient has a cardiac reserve of 300% and a maximum cardiac output of 16 L/min, what is his resting cardiac output?

A) 3 L/min

B) 4 L/min

C) 5.33 L/min

D) 6 L/min

E) 8 L/min

Explanation:

B) This patient has a resting cardiac output of 4 L/min, and his cardiac reserve is 300% of this rest- ing cardiac output or 12 L/min. This gives a total maximum cardiac output of 16 L/min. Therefore, the cardiac reserve is the percentage increase that the cardiac output can be elevated over the resting cardiac output.

105)Which of the following occurs during heart failure and causes an increase in renal sodium excretion?

A) Increased aldosterone release

B) Increased atrial natriuretic factor release

C) Decreased glomerular filtration rate

D) Increased angiotensin II release

E) Decreased mean arterial pressure

Explanation:

B) Several factors cause sodium retention during heart failure including aldosterone release, decreased glomerular filtration rate, and an increased angioten- sin II release. A decrease in mean arterial pressure also results in decreases in glomerular hydrostatic pressure and causes a decrease in renal sodium ex- cretion. During heart failure blood volume increases, resulting in an increased cardiac stretch. In particu- lar, the atrial pressure increases causing a release of atrial natriuretic factor resulting in an increase in renal sodium excretion.

107)Which of the following conditions normally accom- panies acute unilateral right heart failure?

A) Increased right atrial pressure

B) Increased left atrial pressure

C) Increased urinary output

D) Increased cardiac output

E) Increased arterial pressure

Explanation :

A) In unilateral right heart failure, the right atrial pressure decreases and the overall cardiac output decreases. This results in a decrease in arterial pres- sure and urinary output. However, left atrial pressure does not increase but in fact decreases.

Capillary circulation

1. Listed below are the hydrostatic and oncotic pressures within a microcirculatory bed:
   Plasma colloid osmotic pressure = 25 mm Hg
   Capillary hydrostatic pressure = 25 mm Hg
   Venous hydrostatic pressure = 5 mm Hg
   Arterial pressure = 80 mm Hg
   Interstitial fluid hydrostatic pressure = -5 mm Hg
   Interstitial colloid osmotic pressure = 10 mm Hg
   Capillary filtration coefficient = 10 ml/min/mm Hg
   What is the rate of net fluid movement across the capillary wall?
   A) 25 ml/min
   B) 50 ml/min
   C) 100 ml/min
   D) 150 ml/min
   E) 200 ml/min

   Answer: D)

   The rate of net fluid movement across a capillary wall is calculated as capillary filtration coefficient **X** net filtration pressure. Net filtration pressure **=** capillary hydrostatic pressure **-** plasma colloid osmotic pressure **+** interstitial colloid osmotic pressure **-** interstitial hydrostatic pressure. Thus, the rate of net fluid movement across the capillary wall is 150 ml/min.

   Filtration rate **=** Capillary filtration coefficient (K_f) **X** Net filtration pressure. Filtration rate**=**K_f **X** [Pc **-**Πc **+** Πi **-** P_I]

   Filtration rate **=** 10 ml/min/mm Hg **X** [25 **-** 25 **+** 10 **-** (**-**5)]

   Filtration rate **=** 10 **X** 15 **=** 150 ml/min

2. Listed below are the hydrostatic and oncotic pressures and filtration rate across a muscle capillary wall:
   Capillary hydrostatic pressure (Pc) = 25 mm Hg
   Plasma colloid osmotic pressure (Πp) = 25 mm Hg
   Interstitial colloid osmotic pressure (Π_I) = 10 mm Hg
   Interstitial hydrostatic pressure (P_I) = -5 mm Hg
   Capillary filtration rate = 150 ml/min
   What is the capillary filtration coefficient?
   A) 0

B) 5
C) 10
D) 15
E) 20

Answer: C)

Filtration rate (FR) is the product of the filtration coefficient (Kf) and the net pressure (NP) across the capillary wall. Thus, the filtration coefficient is equal to filtration rate divided by the net pressure. The net pressure for fluid movement across a capillary wall = capillary hydrostatic pressure - plasma colloid osmotic pressure + interstitial colloid osmotic pressure - interstitial hydrostatic pressure.

The net pressure in this question calculates to be 15 mm Hg and the filtration is 150. Thus, the Kf is 150/15 or 10 ml/min/mm Hg.

NP = [Pc - ∏p + ∏I - PI]

NP = [25 – 25+ 10 - (-5)]

NP = 15

Kf = 150/15 = 10 ml/min/mm Hg

3. Histamine is infused into the brachial artery. Which of the following sets of microcirculatory changes would be expected in the infused arm?
A) Increased capillary water permeability, capillary hydrostatic pressure, capillary filtration rate.
B) Increased capillary water permeability, capillary hydrostatic pressure and decreased capillary filtration rate.
C) Increased capillary water permeability and decreased capillary hydrostatic pressure, capillary filtration rate.
D) Increased capillary water permeability, capillary filtration rate, and decreased capillary hydrostatic pressure.
E) Decreased capillary water permeability, capillary hydrostatic pressure, capillary filtration rate.
F) Decreased capillary water permeability, capillary hydrostatic pressure and increased capillary filtration rate.
G) Decreased capillary water permeability and increased capillary hydrostatic pressure, capillary filtration rate.
H) Decreased capillary water permeability, capillary filtration rate, and increased capillary hydrostatic pressure.

Answer: A)

Histamine is a vasodilator that is typically released by mast cells and basophils. Infusion of histamine into a brachial artery would decrease arteriolar resistance and increase water permeability of the capillary wall. The decrease in arteriolar resistance would also increase capillary hydrostatic pressure. The increase in capillary hydrostatic pressure and water permeability leads to an increase in capillary filtration rate.

4. Bradykinin is infused into the brachial artery of a 22-year-old man. Which of the following sets of microcirculatory changes would be expected in the infused arm?
A) Increased capillary water permeability, capillary hydrostatic pressure, lymph flow.
B) Increased capillary water permeability, capillary hydrostatic pressure and decreased lymph flow.
C) Increased capillary water permeability and decreased capillary hydrostatic pressure, lymph flow.
D) Increased capillary water permeability, lymph flow, and decreased capillary hydrostatic pressure.
E) Decreased capillary water permeability, capillary hydrostatic pressure, lymph flow.
F) Decreased capillary water permeability, capillary hydrostatic pressure and increased lymph flow.
G) Decreased capillary water permeability and increased capillary hydrostatic pressure, lymph flow.
H) Decreased capillary water permeability, lymph flow, and increased capillary hydrostatic pressure.

Answer: A)

Bradykinin is a vasodilator that is believed to play a role in regulating blood flow and capillary leakage in inflamed tissue. Infusion of bradykinin into the brachial artery would increase arteriolar diameter and decrease arteriolar resistance. The decrease in arteriolar resistance would also result in an increase in capillary hydrostatic pressure and filtration rate. The increase in filtration rate leads to an increase in interstitial hydrostatic pressure and lymph flow.

5. The diameter of a precapillary arteriole is increased in a muscle vascular bed. A decrease in which of the following would be expected?
A) Capillary filtration rate
B) Vascular conductance
C) Capillary blood flow
D) Capillary hydrostatic pressure
E) Arteriolar resistance

Answer: E)

An increase in the diameter of a precapillary arteriole would decrease arteriolar resistance. The decrease in arteriolar resistance would lead to an increase in vascular conductance and capillary blood flow, hydrostatic pressure, and filtration rate.

6. An increase in which of the following would tend to increase lymph flow?
   A) Hydraulic conductivity of the capillary wall
   B) Plasma colloid osmotic pressure
   C) Capillary hydrostatic pressure
   D) Arteriolar resistance
   E) A and C

Answer: E)

The two main factors that increase lymph flow are an increase in capillary filtration rate and an increase in lymphatic pump activity. An increase in plasma colloid osmotic pressure decreases capillary filtration rate, interstitial volume and hydrostatic pressure, and lymph flow. In contrast, an increase in hydraulic conductivity of the capillary wall and capillary hydrostatic pressure increase capillary filtration rate, interstitial volume and pressure, and lymph flow. An increase arteriole resistance would decrease capillary hydrostatic pressure, capillary filtration rate, interstitial volume and pressure, and lymph flow.

7. Which of the following substances in plasma is the major factor that contributes to plasma colloid osmotic pressure?
   A) Sodium chloride
   B) Glucose
   C) Albumin
   D) Cholesterol
   E) Potassium

Answer: C)

Those molecules or ions that fail to pass through the pores of the capillary wall exert osmotic pressure. The capillary wall is highly permeable to sodium chloride, glucose, cholesterol, and potassium but relatively impermeable to albumin. Thus, albumin in the plasma is the major contributor to plasma colloid osmotic pressure.

8. A twofold increase in which of the following would result in the greatest increase in the transport of oxygen across the capillary wall?
   A) Capillary hydrostatic pressure
   B) Intercellular clefts in the capillary wall
   C) Oxygen concentration gradient

D) Plasma colloid osmotic pressure
E) Capillary wall hydraulic permeability

Answer: C)

The transport of oxygen across a capillary wall is proportional to the capillary surface area, capillary wall permeability to oxygen, and oxygen gradient across the capillary wall. Thus, a twofold increase in the oxygen concentration gradient would result in the greatest increase in the transport of oxygen across the capillary wall. A twofold increase in intercellular clefts in the capillary wall would not significantly impact oxygen transport, because oxygen can permeate the endothelial cell wall.

9. Which of the following vessels has the greatest total cross-sectional area in the circulatory system?
   A) Aorta
   B) Small arteries
   C) Capillaries
   D) Venules
   E) Vena cava

Answer: C)

The capillaries have the largest total cross- sectional area of all vessels of the circulatory system. The venules also have a relatively large total cross- sectional area, but not as great as the capillaries, which explains the large storage of blood in the venous sys- tem compared with that in the arterial system.

10. An increase in which of the following tends to decrease capillary filtration rate?
    A) Capillary hydrostatic pressure.
    B) Plasma colloid osmotic pressure.
    C) Interstitial colloid osmotic pressure.
    D) Venous hydrostatic pressure.
    E) Arteriolar diameter.

Answer: B)

An increase in plasma colloid osmotic pressure would reduce net filtration pressure and capillary fil- tration rate. Increases in capillary hydrostatic pressure and interstitial colloid osmotic pressure would also favor capillary filtration. An increase in venous hydro- static pressure and arteriolar diameter would tend to increase capillary hydrostatic pressure and capillary filtration rate.

11. An increase in which of the following tends to increase capillary filtration rate? A) Capillary wall hydraulic conductivity

B) Arteriolar resistance

C) Plasma colloid osmotic pressure

D) Interstitial hydrostatic pressure

E) Plasma sodium concentration

Answer: A)

An increase in capillary wall permeability to water would increase capillary filtration rate, whereas increases in arteriolar resistance, plasma colloid osmotic pressure, and interstitial hydrostatic pressure would all decrease filtration rate. Plasma sodium concentration would have no effect on filtration.

12. A decrease in which of the following tends to increase lymph flow?

A) Capillary hydrostatic pressure

B) Interstitial hydrostatic pressure

C) Plasma colloid osmotic pressure

D) Lymphatic pump activity

E) Arteriolar diameter

Answer: C)

The rate of lymph flow increases in proportion to the interstitial hydrostatic pressure and the lymphatic pump activity. A decrease in plasma colloid osmotic pressure would increase filtration rate, interstitial volume, interstitial hydrostatic pressure, and lymph flow. A decrease in arteriolar diameter would decrease capillary hydro- static pressure, capillary filtration, and lymph flow.

13. Which of the following tends to increase the net movement of glucose across a capillary wall?

A) Increase in plasma sodium concentration

B) Increase in the concentration difference of glucose across the wall

C) Decrease in wall permeability to glucose

D) Decrease in wall surface area without an increase in the number of pores

E) Decrease in plasma potassium concentration

Answer: B)

The factors that determine the net movement of glucose across a capillary wall include the wall permeability to glucose, the glucose concentration gradient across the wall, and the capillary wall surface area. Thus, an increase in the concentration difference of

glucose across the wall would enhance the net movement of glucose.

14. A 65-year-old man is suffering from congestive heart failure. He has a cardiac output of 4 L/min, arterial pressure of 115/85 mm Hg, and a heart rate of 90 beats/min. Further tests by a cardiologist reveal that the patient has a right atrial pressure of 10 mm Hg. An increase in which of the following would be expected in this patient?

A) Plasma colloid osmotic pressure

B) Interstitial colloid osmotic pressure

C) Arterial pressure

D) Cardiac output

E) Vena cava hydrostatic pressure

Answer: E)

An increase in atrial pressure of 10 mm Hg would tend to decrease venous return to the heart and increase vena cava hydrostatic pressure. Plasma colloid osmotic pressure, interstitial colloid osmotic pressure, arterial pressure, and cardiac output would generally be low to normal in this patient.

15. Using the following data, calculate the filtration coefficient for the capillary bed:
Plasma colloid osmotic pressure = 30 mm Hg
Capillary hydrostatic pressure = 40 mm Hg
Interstitial hydrostatic pressure = 5 mm Hg
Interstitial colloid osmotic pressure = 5 mm Hg
Filtration rate = 150 ml/min
Venous hydrostatic pressure = 10 mm Hg
A) 10 ml/min/mm Hg
B) 15 ml/min/mm Hg
C) 20 ml/min/mm Hg
D) 25 ml/min/mm Hg
E) 30 ml/min/mm Hg

Answer: B)

Filtration coefficient (K_f) **=** filtration rate **/** net filtration pressure. Net filtration pressure **=** capillary hydrostatic pressure **-** plasma colloid osmotic pressure **+** interstitial colloid osmotic pressure **-** interstitial hydrostatic pressure. The net filtration pressure in this example is 10 mm Hg.

Thus, K_f **=** 150 ml/min **/** 10 mm Hg, or 15 ml/min/mm Hg.

16. Which of the following pressures is normally negative in a muscle capillary bed in the lower extremities?
    A) Plasma colloid osmotic pressure
    B) Capillary hydrostatic pressure
    C) Interstitial hydrostatic pressure
    D) Interstitial colloid osmotic pressure
    E) Venous hydrostatic pressure

Answer: C)

An increase in perfusion pressure to a tissue results in excessive delivery of nutrients such as oxy- gen to a tissue. The increase in tissue oxygen concen- tration constricts arterioles and returns blood flow and nutrient delivery toward normal levels.

17. Which of the following would decrease venous hydro- static pressure in the legs?
    A) Increase in right atrial pressure
    B) Pregnancy
    C) Movement of leg muscles
    D) Presence of ascitic fluid in the abdomen

Answer: C)

Interstitial hydrostatic pressure in a muscle capillary bed is normally negative (**-**3 mm Hg). Pumping by the lymphatic system is the basic cause of the negative pressure.

18. Which of the following has the fastest rate of movement across the capillary wall?
    A) Sodium
    B) Albumin
    C) Glucose
    D) Oxygen

Answer: D)

The primary mechanism whereby solutes move across a capillary wall is simple diffusion.

Coronary circulation

1. Which of the following vasoactive agents is usually the most important controller of coronary blood flow?
   A) Adenosine
   B) Bradykinin
   C) Prostaglandins
   D) Carbon dioxide
   E) Potassium ions

   Answer: A)

   Bradykinin, prostaglandins, carbon dioxide, and potassium ions serve as vasodilators for the coronary artery system. However, the major controller of coronary blood flow is adenosine. Adenosine is formed as ATP degrades to adenosine monophosphate. Then, small portions of the adenosine monophosphate are further degraded to release adenosine into the tissue fluids of the heart muscle, and this adenosine vasodilates the coronary arteries.

2. During mild exertion, a 70-year-old man experiences an ischemia-induced myocardial infarction and dies from ventricular fibrillation. In this patient, what factor was most likely to increase the tendency of the heart to fibrillate after the infarction?
   A) Increased parasympathetic stimulation of the heart
   B) A decrease in ventricular diameter
   C) Low potassium concentration in the heart extracellular fluid
   D) A more negative ventricular membrane potential
   E) Current of injury from the damaged area

   Answer: E)

   An acute loss of blood supply to a cardiac muscle causes depletion of potassium from the cardiac myo- cytes. This locally increases the extracellular potassium concentration. In turn, this increases the irritability of the cardiac musculature and therefore its likelihood for fibrillating. Therefore, a decreased potassium ion con- centration in the extracellular fluids of the heart does not lead to fibrillation. Powerful sympathetic and not parasympathetic reflexes also increase the irritability of the cardiac muscle and predispose it to fibrillation. A more negative membrane potential protects the heart from fibrillation, and a current of injury allows electrical current flow from an ischemic area of the heart to a normal area and can elicit fibrillation.

3. Which of the following statements about coronary blood flow is most accurate?
   A) Normal resting coronary blood flow is 500 ml/min
   B) The majority of flow occurs during systole

C) During systole the percentage decrease in subendocardial flow is greater than the percentage decrease in epicardial flow

D) Adenosine release will normally decrease coronary flow

Answer: C)

The normal resting coronary blood flow is approximately 225 ml/min. Infusion of adenosine or local release of adenosine normally increases the coronary blood flow. The concentration of the cardiac muscle around the vasculature, particularly in the subendocardial vessels, causes a decrease in blood flow. Therefore, during the systolic phase of the cardiac cycle the subendocardial flow clearly decreases while the decrease in epicardial flow is relatively minor.

4. Which of the following is the most frequent cause of decreased coronary blood flow in patients with ischemic heart disease?

A) Increased adenosine release

B) Atherosclerosis

C) Coronary artery spasm

D) Increased sympathetic tone of the coronary arteries

E) Occlusion of the coronary sinus

Answer: B)

Several factors contribute to decreased coronary flow in patients with ischemic heart disease. Some patients will have spasm of the coronary arteries which acutely decreases coronary flow. However, the major cause of decreased coronary flow is an atherosclerotic narrowing of the lumen of the coronary arteries.