Hemodynamics Concepts

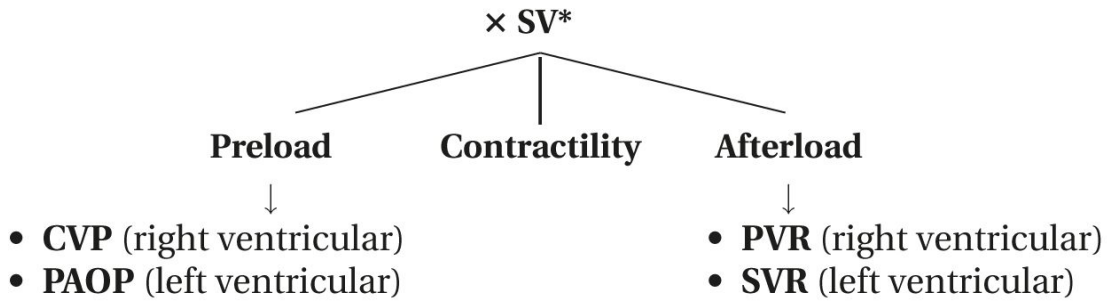
One of the most important keys to Success is having the discipline to do what you know you should do, even when you don't feel like doing it.

—Unknown

Hemodynamics is included in the Cardiovascular section of the Adult CCRN test blueprint. However, this topic has been placed after the Cardiovascular Concepts, Respiratory Concepts, and Multisystem Concepts chapters in this book because each of these content areas includes hemodynamic concepts.

Although most critically ill patients do not receive invasive hemodynamic monitoring, ALL patients have hemodynamics that reflect their specific problems. These, to a degree, determine the plan of care. Hemodynamics and invasive hemodynamic monitoring are included in numerous Adult CCRN exam questions.

☆ The equation in Figure 6-1 is the foundation of hemodynamics. The cardiac output (CO) is equal to the heart rate (HR) times the stroke volume (SV). The SV is dependent upon the preload, afterload, and contractility of the ventricles of the heart.



*Heart rate (HR); stroke volume (SV); central venous pressure (CVP); pulmonary artery occlusion pressure (PAOP); pulmonary vascular resistance (PVR); systemic vascular resistance (SVR)

Figure 6-1. Regulation of hemodynamics

Critical Points

- The normal cardiac output (CO) is 4 L to 8 L per minute. If it becomes critically low, the blood pressure will decrease.
  - Cardiac index (CI) takes into account the body surface area (BSA) and is **a more meaningful value than CO**.
  - The normal CI is 2.5 to 4.0 L/min/m^2.
- As **heart rate** (HR) increases, CO increases up to a point. The point is determined by the patient's age or the condition of his or her ventricles. Generally, 130–170 beats per minute is the maximal heart rate originating from the sinoatrial (SA) node that is attainable for most people.
  - Extreme bradycardia results in low CO and hypotension.
  - An increase in HR is the first sign of compensation for a low CO. This will occur before the BP drops. Patients on beta blocker drugs, or any drug that decreases heart rate, may not be able to compensate as well for a problem that is decreasing cardiac output.
  - Conversely, extreme tachycardia will also decrease the CO no matter the patient's age or the condition of his or her ventricles. Why? A loss of diastolic filling time occurs. If diastolic filling time is decreased, the ventricles do not have time to fill, ventricular preload drops, and ultimately the ventricle cannot put out what is not delivered.
- As **stroke volume** increases, CO increases. The stroke volume (SV) is how many mL per beat the left ventricle ejects. It is determined by the preload, afterload, and contractility. The normal SV is 50–100 mL per beat.

- **Preload**
  - Preload is the volume/pressure in the ventricle at the end of diastole after the AV valves close, just prior to ejection. The right atrial (RA) pressure or central venous pressure (CVP) reflects the right ventricular preload. The PAOP reflects the left ventricular preload.
  - As preload increases, the SV and CO increase up to a point.
  - Too high of a preload may lead to heart failure.
  - In general, preload will seldom be elevated if the heart is disease-free and if there are no metabolic abnormalities.
- **Afterload**
  - Afterload is the pressure (resistance) against which the ventricle must pump to open the valve (pulmonic or aortic).
  - Afterload is clinically measured by the pulmonary vascular resistance (PVR) for the right ventricle or the systemic vascular resistance (SVR) for the left ventricle.
  - As afterload increases, the SV and CO decrease.
- **Contractility**
  - Contractility is the contractile force of the myofibrils, independent of preload and afterload.
  - As contractility increases, the SV and CO increase.

☆ For the Adult CCRN exam, you need to know normal hemodynamic values and how various drugs and therapies affect preload, afterload, and contractility. Therefore, you need to know how these drugs and therapies affect stroke volume and, ultimately, cardiac output.

Table 6-1 summarizes normal hemodynamics, and Table 6-2 outlines how various clinical problems affect hemodynamics. Table 6-3 lists the hemodynamic effects of various cardiovascular agents. You **must** have

an understanding of this information, as it is the foundation for patient management.

☆ **Table 6-1. Normal Hemodynamic and Oxygenation Parameters**

Parameter	Normal	Formula
Heart rate (HR)	60–100 beats/minute	Direct measurement
Blood pressure (BP)	90/60–140/90 mmHg	Direct measurement
Mean arterial pressure (MAP)	70–110 mmHg	SBP + 2(DBP) ÷ 3
Cardiac output (CO)	4–8 L/min	Direct measurement
Cardiac index (CI)	2.5–4.0 L/min/m^2	CO ÷ BSA
Stroke volume (SV)	50–100 mL/beat	Direct measurement
Stroke index (SI)	25–45 mL/beat/m^2	SV + BSA
Right atrial pressure (RAP) [also known as central venous pressure (CVP)]	2–6 mmHg 3–8 cm H_2O	Direct measurement
Pulmonary artery pressure (PAP)	20/8–30/15 mmHg Mean: < 20 mmHg	Direct measurement
Pulmonary artery occlusion (wedge) pressure (PAOP)	8–12 mmHg (although it varies depending upon the LV function)	Direct measurement
Systemic vascular resistance (SVR)	800–1200 dynes/s/cm^{-5}	(MAP − CVP) ÷ CO × 80
Pulmonary vascular resistance (PVR)	50–250 dynes/s/cm^{-5}	(MPAP − PAOP) ÷ CO × 80
Coronary artery perfusion pressure (CAPP)	60–80 mmHg	DBP – PAOP
Mixed venous oxygen saturation (SvO_2)	60–75%	Direct measurement (pulmonary artery)
Central venous oxygen saturation ($ScvO_2$)	> 70%	Direct measurement (superior vena cava)
Arterial oxygen saturation (SaO_2)	95–99% on room air	Direct measurement
Arterial oxygen content (CaO_2)	12–16 mL/dL	(Hgb × 1.39 × SaO_2) + (PaO_2 × 0.003)
Oxygen delivery (DO_2)	900–1,100 mL/min	CaO_2 × CO × 10
Oxygen consumption (VO_2)	250–350 mL/min	(SaO_2 − SvO_2) × Hgb × 13.9 × CO

- “Normal” differs somewhat from resource to resource. Those values listed in Table 6-1 are generally accepted.

- You do not need to memorize formulas for the Adult CCRN exam.

☆ Table 6-2. Hemodynamic Profiles for Select Abnormal Conditions

Condition	BP	RAP (CVP)	PAP	PAOP	CO/CI	SV/SI*	SVR	PVR	SvO_2**	Comments
Shock States										
Cardiogenic	↓	↑	↑	↑	↓	↓	↑	~ or ↑	↓	
Hypovolemic	↓	↓	↓	↓	↓	↓	↑	~	↓	
Septic, early	↓	↓	↓	↓	↑	↑	↓	~	↑	Lactate may ↑ before BP ↓
Septic, late	↓	↓	↓	~ or ↑	↓	↓	↑	~	↓	
Cardiogenic pulmonary edema (left ventricular failure)	~ or ↑	↑	↑	↑	↓	↓	~	~ or ↑	↓	
Noncardiogenic pulmonary edema (ARDS)	~ or ↓	~ or ↑ or ↓	↑	~ or ↓	~	~	~	↑	~ or ↓	PAP↑ due to hypoxemia
Pulmonary hypertension (PE, COPD, hypoxemia)	~	↑	↑	~	~	~	~	↑	↓	BP, CO may ↓ if PE
Cardiac tamponade	↓	↑	↑	↑	↓	↓	↑	~ or ↑	↓	Pressures equalize

Key: ↓ = decrease; ↑ = increase; ~ = no change
*May decrease before CO/CI or BP if compensation is adequate, e.g., increase in heart rate
**May decrease or increase before CO/CI or BP change since it represents change at the tissue level

☆ Table 6-3. Hemodynamic Effects of Various Cardiovascular Agents

Drug	BP	RAP (CVP)	PAP	PAOP	CO/CI	SV/SI	SVR	PVR	Heart Rate
Dopamine									
• Low dose (1–3 mcg/kg/m)	~	~	~	~	~	~	~	~	~
• Medium dose (4–10 mcg/kg/m)	~ or ↑	~	~ or ↑	~ or ↑	↑↑	↑	~	~	↑
• High dose (11–20 mcg/kg/m)	↑	~ or ↑	↑	↑	↑	↑	↑	↑	↑↑
Norepinephrine (Levophed)	↑	↑ or ~	↑	↑	↑	↑	↑↑	~ or ↑	~
Phenylephrine (Neo-Synephrine)	↑	~	~	~ or ↑	~	↑	↑	~	~
Epinephrine drip	↑	↑ or ~	↑	↑	↑	↑	↑↑	~ or ↑	↑
Nitroglycerin									
• Doses up to 1 mcg/kg	~ or ↓	↓	↓	↓↓	~	~	~	~ or ↓	~ or ↑
• Doses > 1 mcg/kg*	↓	↓	↓	↓	~ or ↑	~ or ↑	↓	↓	~ or ↑
Nesiritide (Natrecor)*	~ or ↓	↓	↓	↓	~ or ↑	~ or ↑	↓	↓	~
Nitroprusside (Nipride)*	↓↓	↓	↓	↓↓	~ or ↑	~ or ↑	↓↓	↓	~ or ↑
ACE inhibitors	↓	~	~	~ or ↓	~ or ↑	~ or ↑	↓	~	~
Dobutamine (Dobutrex)	~ or ↑ or ↓	~	~	↓	↑	↑	~ or ↓	~	↑
Milrinone (Primacor)	~ or ↓	~ or ↓	~ or ↓	↓	↑	↑	↓	~	~
Labetalol (Normadyne)	↓	~	~	~	↓	↓	~ or ↓	~	↓
Morphine	↓	↓	↓	↓	~	~	~	↓	~

Key: ↓ = decrease; ↑ = increase; ~ = no change
*High-dose NTG, nesiritide, and nitroprusside are afterload reducers, not positive inotropes, but they may increase CO indirectly by decreasing afterload.

There will be questions that test your knowledge on appropriate interventions for various abnormal hemodynamics and the significance of various oxygenation parameters. Table 6-4 summarizes therapies for alterations in hemodynamics, and Table 6-5 outlines oxygenation parameters.

☆ Table 6-4. Therapies for Alterations in Hemodynamics*

PRELOAD Therapies	
Increases	Decreases
Volume expanders • Crystalloids • Colloids Pressors	Diuretics Dilators • Nitrates • Nitroprusside • Nesiritide Morphine
AFTERLOAD Therapies	
Increases	Decreases
Norepinephrine Phenylephrine High-dose dopamine (11–20 mcg/kg/min) Epinephrine drip	Nitroprusside ACE inhibitors Hydralazine Calcium channel blockers IABP Nitroglycerin (high doses)
CONTRACTILITY Therapies	
Increases	Decreases
Positive inotropes • Dobutamine • Dopamine 5–10 mcg/kg/min • Primacor • Epinephrine drip	Negative inotropes • Beta blockers • Calcium channel blockers Metabolic problems (e.g., metabolic acidosis, endotoxins of sepsis)

*For all abnormalities, attempt to identify the underlying cause(s) and, if able, correct them.

Table 6-5. Hemodynamic Oxygenation Parameters

Parameter	Normal	How it's Calculated/ Measured	Clinical Relevance
Mixed venous oxygen saturation (SvO_2)	60–75%	Direct measurement, intermittent or continuous (pulmonary artery)	Most sensitive indicator of cellular oxygenation
Central venous oxygen saturation ($ScvO_2$)	> 70%	Direct measurement, intermittent or continuous (superior vena cava)	Used to monitor therapy for septic shock
Oxygen delivery (DO_2)	900–1,100 mL/min	$CaO_2 \times CO \times 10$	Pump problems (heart) will decrease DO_2
Oxygen consumption (VO_2)	250–350 mL/min	$(SaO_2 - SvO_2) \times Hgb \times 13.9 \times CO$	Low with septic shock
Oxygen extraction	~ 50% of O_2 delivery	$(CaO_2 - CvO_2)$	Myocardial oxygen extraction is > than that of any other muscle; increases with a drop in CO

- You do not need to memorize formulas for the Adult CCRN exam, but you should know the normal values and their clinical relevance.
- SvO_2: normal is 60% to 75% (too low or too high is **BAD**)
- Sustained changes, not brief changes (e.g., during position change), are significant (Table 6-6).

Table 6-6. SvO_2 Changes

Increased	Decreased
Septic shock Hypothermia Paralysis	Low cardiac output Decreased PaO_2 Increased O_2 demand (fever, shivering, seizures, increased WOB)
If SvO_2 is increased: • Assess for sepsis, septic shock • Assess for hypothermia	If SvO_2 is decreased: • Assess for hypoxemia, increased WOB • Assess for hypotension • Assess for hypovolemia • Assess hemoglobin (drop) • Assess temperature (fever) • Assess for arrhythmias

☆ For the Adult CCRN exam, concentrate on studying the normal and abnormal hemodynamic ranges. Also, study the therapies that affect hemodynamics. Note that printed waveform strips, such as the one in Figure 6-2, are seldom included on this exam.

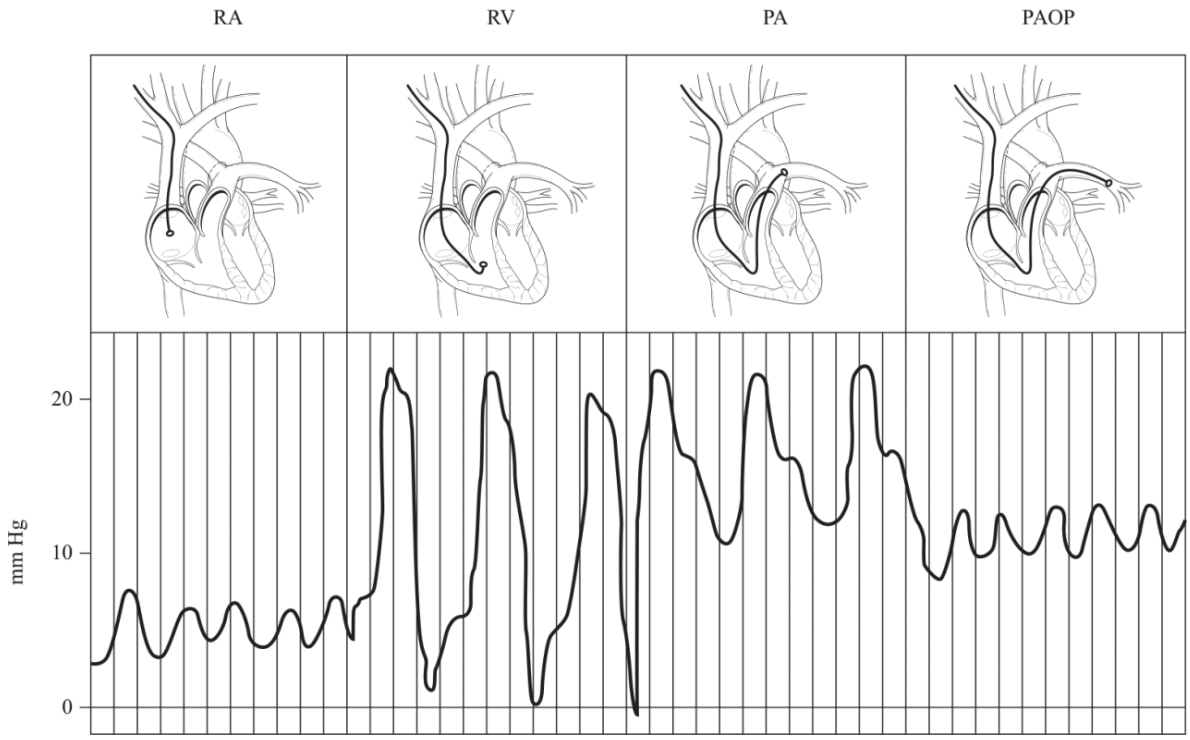


Figure 6-2. Normal pulmonary artery waves

Acute mitral valve insufficiency: In the presence of mitral valve insufficiency, the PAOP waveform changes appearance. When the PA catheter balloon is inflated, **giant V-waves** appear on the PAOP tracing (see Figure 6-3).

- The PAOP is read at the A-wave, not at the V-wave.

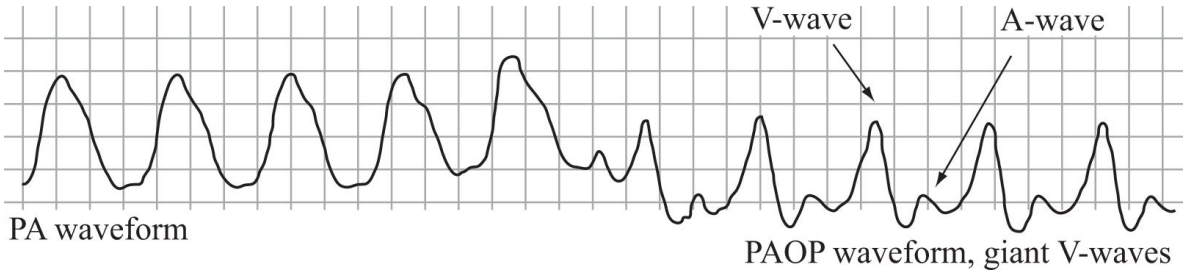


Figure 6-3. Giant V-waves on the PAOP waveform

If you see the term “giant V-waves” on the exam, the problem is mitral valve insufficiency (regurgitation). As you should recall from the Cardiovascular Concepts chapter, this is often associated with acute inferior wall myocardial infarction/papillary muscle dysfunction/rupture.

Square Wave Test (Dynamic Response Test)

- The **dynamic response test**, also called the “square wave test,” is performed to assess the accuracy of the hemodynamic monitoring system. This is done immediately after catheter insertion, at the beginning of each shift after zeroing the system and whenever values are questionable. The strip recorder is started. Then, the flush device is squeezed and immediately released. The dynamic response that is documented on the strip is then examined.
- For the exam, you will only need to know what the square wave test is and the implications if it is overdamped or underdamped. You will not be given strips to assess.
- Overdamped wave response:
  - Results in a falsely decreased systolic pressure and a falsely high diastolic pressure as well as poorly defined components of the pressure tracing, such as a diminished or absent dicrotic notch
  - May be due to air or a blood clot in the system, loose connections, loss of air in the pressure bag, or kinking of the catheter/tubing system
- Underdamped wave response (less common clinically):
  - Results in a falsely high systolic pressure (overshoot), a possibly falsely low diastolic pressure, and “ringing” artifacts on the waveform
  - May be due to pinpoint air bubbles in the system, add-on tubing, or a defective transducer

Now that you have reviewed the key hemodynamics concepts, go to the Practice Questions. Answer the questions, and then check your

answers. Continue to review the information until you answer 80% of the practice questions correctly.

Practice Questions

For questions 1–3

The following hemodynamic profile has been obtained for a patient:

BP	112/60 mmHg
PA	44/24 mmHg
SVR	1,600 dynes/s/cm^{-5}
SVI	23 mL/m^2
RA	3 mmHg
PAOP	22 mmHg
CI	1.9 L/min/m^2
SvO_2	0.58

1. Based on this hemodynamic profile, which of the following conclusions is CORRECT?

   (A) The patient is hypovolemic.
   (B) The patient has evidence of left ventricular failure.
   (C) The patient seems to have sepsis.
   (D) The patient has developed ARDS.

2. Shortly after obtaining the values above, the nurse inflated (wedged) the catheter balloon and observed giant V-waves on the PAOP tracing. What is the most likely problem?

(A) The patient has evidence of mitral valve regurgitation.
(B) The PA catheter has fallen back into the RV.
(C) The PA catheter needs to be advanced.
(D) The PAOP seems to be 40 mmHg.

3. What would be the most appropriate intervention for this patient?

(A) Call the physician for possible catheter repositioning.
(B) Document the strip, administer the PRN order for furosemide (Lasix) for a PAOP > 16 mmHg, and reassess in 1 hour.
(C) Label the balloon “do not wedge.”
(D) Document the strip, interpret the pressure, and notify the physician.

4. A patient with continuous SvO_2 monitoring has sustained a decrease in SvO_2 to 0.50. Priority interventions would include each of the following EXCEPT:

(A) checking the urine output.
(B) checking the BP and the CO/CI.
(C) checking O_2 sat with a pulse oximeter (SpO_2).
(D) checking the temperature.

For questions 5–8

It is important to integrate hemodynamic parameters with each other and not evaluate parameters individually in isolation. Assess the following hemodynamic profiles. Match each profile with the clinical problem listed below that is most likely associated with it.

(BP = blood pressure; HR = heart rate; CVP = central venous pressure; PAOP = pulmonary artery occlusion pressure; SVR = systemic vascular resistance; CI = cardiac index; SV = stroke volume)

	Profile 5	**Profile 6**	**Profile 7**	**Profile 8**
BP	78/40	78/40	78/40	78/40
HR	120	120	120	120
CVP	5	2	15	1
PAOP	19	4	5	4
SVR	1,697	453	1,300	1,387
CI	2.0	5.5	2.5	3.0
SV	29	75	27	25
	5._____	6._____	7._____	8._____

5. (A) hypovolemic shock
   (B) cardiogenic shock
   (C) acute right ventricular failure
   (D) septic shock

6. (A) hypovolemic shock
   (B) cardiogenic shock
   (C) acute right ventricular failure
   (D) septic shock

7. (A) hypovolemic shock
   (B) cardiogenic shock
   (C) acute right ventricular failure
   (D) septic shock

8. (A) hypovolemic shock
   (B) cardiogenic shock
   (C) acute right ventricular failure
   (D) septic shock

For questions 9–20

Match the drug with its **primary** hemodynamic effect. Consult Table 6-3.

9. ____ Dopamine 5–10 mcg/kg/min

10. ____ Dopamine > 10 mcg/kg/min

11. ____ Dobutamine

12. ____ Nitroglycerin 20 mcg/min

13. ____ Norepinephrine

14. ____ Beta blockers

15. ____ Nitroprusside (choose 2)

16. ____ ACE inhibitors

17. ____ Furosemide

18. ____ Fluid bolus

19. ____ Milrinone

20. ____ Morphine

(A) increase preload
(B) decrease preload
(C) increase afterload
(D) decrease afterload
(E) increase contractility
(F) decrease contractility

For questions 21 and 22

Mr. A, a 66-year-old male, presents S/P colectomy for colon cancer. He has the following results upon examination:

MAP of 58 mmHg (↓ from 70) after 2 L of 0.9 normal saline; heart rate of 112 beats/minute; respiratory rate of 34 breaths/minute;

temperature of 38.9°C; urine output < 0.5 mL/kg for the past 2 hours; lungs are clear; skin is warm and dry; WBC is 20,000; 66% segs; 24% bands; 7% lymphs; blood cultures are positive for gram-negative organisms

21. This patient does not have a pulmonary artery catheter. If he did, and his systemic vascular resistance was measured, what SVR should the nurse anticipate this patient would have?

    (A) 2,550 dynes/s/cm^{-5}
    (B) 1,550 dynes/s/cm^{-5}
    (C) 550 dynes/s/cm^{-5}
    (D) 900 dynes/s/cm^{-5}

22. What hemodynamic changes are consistent with this patient's clinical status?

    (A) increase in preload and decrease in afterload
    (B) decrease in preload and increase in afterload
    (C) decrease in preload and decrease in afterload
    (D) decrease in CO due to impaired contractility

23. Ms. B, a 78-year-old female, is admitted with the following clinical findings:

    - Chief complaint of SOB and fatigue
    - Bibasilar crackles noted with S3 gallop
    - Chest radiograph shows venous congestion and cardiomegaly
    - Weight increase of 20 pounds over the last 2 weeks

Which of the following hemodynamic alterations is found with her presenting problems, and what treatment and rationale for that treatment are indicated?

(A) increased afterload, decreased contractility, and decreased preload; nesiritide to increase contractility
(B) decreased afterload, decreased contractility, and increased preload; furosemide (Lasix) to increase afterload
(C) decreased afterload, increased contractility, and increased preload; amiodarone to decrease preload
(D) increased afterload, decreased contractility, and increased preload; dobutamine to increase contractility

For questions 24 and 25

Mr. C, a 54-year-old male, presents with near-syncope. He has no significant past medical history. He sought medical help today after almost fainting in the shower. Clinical findings include:

- Blood pressure of 98/58 mmHg; heart rate of 108 beats/minute with S1 and S2 heart sounds; respiratory rate of 18 breaths/minute; temperature of 37.1°C
- Alert and orientated × 3; lungs are clear; skin is cool and dry; neck veins are flat
- Thirsty with dry oral mucous membranes; abdomen is soft/non-tender with hyperactive bowel sounds

24. The next vital sign assessment reveals a blood pressure of 88/60 mmHg. Which of the following does Mr. C seem to need?

(A) Volume expansion is needed to increase preload and increase myocardial stretch.
(B) Pressors are needed to increase afterload and increase myocardial stretch.
(C) Volume expansion is needed to decrease afterload and increase myocardial stretch.
(D) Pressors are needed to decrease preload and decrease myocardial stretch.

25. The nurse suspects that the arterial line waveform appearance has changed. The recommended method for assessing the adequacy of the catheter/tubing system is to:

(A) perform a square wave test.
(B) zero balance.
(C) check a cuff pressure.
(D) calibrate the monitor.

Answer Key

1. **B**
2. **A**
3. **D**
4. **A**
5. **B**
6. **D**
7. **C**
8. **A**
9. **E**
10. **C**
11. **E**
12. **B**
13. **C**
14. **F**
15. **B**, **D**
16. **D**
17. **B**
18. **A**
19. **E**
20. **B**
21. **C**
22. **C**
23. **D**
24. **A**

25. **A**

Answers and Explanations

1. **(B)** Although there are several hemodynamic abnormalities, the key here is the PAOP of 22 mmHg. Left heart failure is the only problem of those listed that would result in an elevated PAOP. Since the LV is in failure and is not emptying normally, there is a backup of volume, reflected as a higher pressure on the left side of the heart. This, in turn, causes the pulmonary capillary pressures to exceed 18 mmHg and cause pulmonary edema. The low CI causes the elevated PAOP. However, other problems, such as hypovolemia, may also result in a low CI. The SVR is high due to compensatory vasoconstriction, which in this case seems to be maintaining the blood pressure.
2. **(A)** The giant V-waves are due to mitral valve insufficiency or regurgitation, which may be acute or chronic. The PAOP is measured, however, at the A-wave.
3. **(D)** If the patient develops acute mitral valve insufficiency, the physician needs to be notified. There is no problem with the PA catheter. The patient does not automatically require a diuretic (but he might). The balloon is OK, so it doesn't need to be labeled.
4. **(A)** The SvO_2 is low; therefore, the reason for that needs to be determined. A drop in blood pressure or cardiac output, a decrease in the arterial oxygen saturation, or a fever are possible causes. Urine output is not directly associated with a decrease in the SvO_2.

5. **(B)** The key indicator is the elevated PAOP with the low CI. The SVR is elevated as a compensatory response.
6. **(D)** The key indicator is the low SVR (massive dilation) with the higher-than-normal CI.
7. **(C)** The key indicator is the high CVP with a low PAOP due to poor RV output. Since volume is not getting to the left heart, the left heart pressure (PAOP) is low and the CI is low.
8. **(A)** The key indicator is the low CVP and PAOP with the elevated SVR as a compensatory response to the hypovolemia.
9. **(E)** “Midrange” dopamine stimulates the beta-1 receptors in the heart to increase contractility.
10. **(C)** High-dose dopamine stimulates the alpha receptors in the arteries to cause vasoconstriction and to increase afterload.
11. **(E)** Dobutamine stimulates the beta-1 receptors in the heart and increases contractility.
12. **(B)** Nitroglycerin at lower doses causes venodilatation and results in a decrease in preload. Only at higher doses does it dilate the arterial vessels.
13. **(C)** Norepinephrine is a potent vasoconstrictor as it stimulates the alpha receptors of the arteries and increases afterload.
14. **(F)** Beta blockers block the beta-1 receptors of the heart, blocking the adrenergic effects of the autonomic nervous system from affecting the heart. This results in decreased contractility as well as a decrease in heart rate.
15. **B**, **D** Nitroprusside dilates both the venous and arterial sides of the vascular system, resulting in a decrease in both preload and afterload.

16. **(D)** ACE inhibitors (or angiotensin-converting enzyme inhibitors) block the conversion of angiotensin I to angiotensin II. Angiotensin II is a potent constrictor. By blocking its formation, the arterial vessels dilate more, resulting in a decrease in afterload.
17. **(B)** Furosemide is a potent loop diuretic and venodilator. The resulting diuresis and venodilation cause a reduction in preload.
18. **(A)** A fluid bolus increases volume in the vasculature, thereby increasing the preload.
19. **(E)** Milrinone, in the class of phosphodiesterase inhibitors, stimulates cardiac muscle contraction and increases contractility.
20. **(B)** Morphine mildly dilates the venous bed, thereby reducing preload. It may be used in low doses to treat heart failure.
21. **(C)** The scenario describes a patient with septic shock as evidenced by an infection and hypotension, despite 2 L of fluid. The endotoxins cause massive vasodilation, which results in a loss of vascular tone, which leads to a low SVR.
22. **(C)** Septic shock causes massive vasodilation, which decreases afterload. It also causes capillary leakage with a loss of volume in the vasculature, resulting in a decrease in preload.
23. **(D)** The patient scenario reflects heart failure. To compensate for the reduced cardiac output, a patient with heart failure vasoconstricts. Therefore, afterload is high. The heart muscle loses contractility, so contractility is decreased. Due to a low ejection fraction, the left heart pressure increases, resulting in lung crackles, S3 heart sound, and increased preload. A

positive inotropic drug, such as dobutamine (as well as other drugs), may be indicated.

24. **(A)** The patient scenario is one of hypovolemia. Fluid administration will increase preload (fill up the tank), which will in turn improve myocardial stretch. This increase in stretch to increase output is the Frank-Starling law of the heart. Since the patient is volume depleted, the systemic vascular resistance (SVR) is most likely high because vasoconstriction occurs as a compensatory response to maintain the arterial blood pressure. Pressors should not be used in the presence of hypovolemia.
25. **(A)** The square wave (or dynamic response) test provides information as to whether the catheter/tubing system is optimally damped, overdamped, or underdamped. Then, troubleshooting needs to be done accordingly. Cuff pressures should not be used to verify the accuracy of intra-arterial pressures.