Adult CCRN® Certification Review

Think in Questions, Learn by Rationale

Kendra Menzies Kent
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To my wonderful husband, Robby, and to my parents, Sid and Judy, for all the love and support they have given me.
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Introduction

Welcome to the journey toward certification. This book was written to help guide the reader on the pathway of the journey. It is written in a question/answer format to encourage you to think in questions when studying for the examination. When you study, I encourage you to ask yourself, “What can be asked about this particular topic?” “What would be a good question?” “What is important in this disease?” “What makes it different from other disorders?” This prepares you to anticipate the kinds of questions that might be asked and not just attempt to memorize content for the certification examination.

The book also provides multiple-choice questions similar to the questions that are found on the Adult Critical-Care Registered Nurse (CCRN®) examination. These questions allow the nurse to practice taking an examination and also assist the nurse in determining areas that require further study prior to taking the CCRN examination. The answers and rationale, including some test-taking skills, are provided for each question, further preparing the nurse for the real examination.

WHY CERTIFICATION?

The most important reason for certification is to do it for yourself (Box I.1). Certification is viewed as a mark of excellence in an area of specialty. It is an achievement and qualification that can be seen by peers, physicians, leaders of health care institutes, and patients/families. Becoming certified takes a certain dedication to critical care nursing and demonstrates a level of competency. The CCRN examination is developed to verify knowledge in critical care nursing.

**Box I.1 Reasons to Become Certified**

- Validates your knowledge of critical care to your hospital and peers
- Validates your knowledge of critical care to the patients
- Validates your knowledge of critical care to the physician
- Promotes continuing excellence in the nursing profession
- Demonstrates competency
- Assists with hospital credentialing
- Provides monetary benefit (from some hospitals)
ADULT CCRN EXAMINATION INFORMATION

The CCRN examination follows the blueprint developed by the American Association of Critical Care Nurses (AACN). The test is developed and reviewed by experts in critical care. The CCRN Application Handbook can be accessed from the website www.certcorp.org. The examination application may be completed online or can be printed and mailed or faxed to the AACN Certification Corporation. The Adult CCRN provides a 3-year certification for critical care nurses.

EXAMINATION

The Adult CCRN examination consists of 150 multiple-choice questions, of which 25 questions will not count for or against you. These 25 questions are being tested for use in future examinations. You will not know which questions count, so complete all 150 questions as if they count. The test is not arranged per system, and it is randomized. You may have one question on the renal system and the next one is on the cardiovascular system. The time allowed to complete the examination is 3 hours (50 questions per hour).

Eligibility requirements to take the Adult CCRN examination are RN licensure and 1,750 hours of direct bedside care of critically ill patients during the previous 2 years with 875 of those hours obtained in the year preceding application to take the examination. Or, one must have been practicing as an RN for 5 years with a minimum of 2,000 hours of direct care of critically ill patients with 144 of those hours logged during the preceding year. Nurse educators and managers in the adult critical care areas may apply hours spent at the bedside supervising nurses and nursing students.

The Adult CCRN examination is offered year-round as a computer-based test (CBT) and is also given as paper–pencil in certain circumstances. Once AACN receives your application and approves it, they send a confirmation e-mail and postcard. Once confirmed, there is a 90-day window to take the examination. You will need to schedule your examination at an approved testing center. These centers can be found at www.goAMP.com. Immediate test results with score breakdown are available with the CBT. Obtaining scores from a paper–pencil test will take 3 to 4 weeks. Following successful completion of the exam (which everyone will have done), a certificate will be sent by mail within 3 to 4 weeks.

〉 HINT

Do not schedule your exam at the end of the 90-day window. If for some reason you are unable to take the examination on the scheduled date, you will have to pay an extra $100 to reschedule.

Renewal of your CCRN license can be made through continuing education recognition points (CERPs) or retaking the examination. The CERP requirement is 100 hours in various
categories (A, B, & C). For more details on renewal, use the AACN’s website for renewal by the CERPs brochure.

TEST PLAN

The Adult CCRN test plan is a blueprint for the exam content. Each major system is divided into subheadings and topics, and clinical judgment, professional caring, and ethical practice.

The amount of coverage in each of the systems is:

- Cardiovascular, 20%
- Pulmonary, 18%
- Endocrine, 5%
- Hematology/Immunology 2%
- Neurology, 12%
- Gastroenterology, 6%
- Renal, 6%
- Multisystem, 8%
- Behavioral/Psychosocial, 4%
- Professional Caring Practice, 20%

The CCRN blueprint also has a list of “testable nursing actions,” which are nursing actions under each body system that may be tested. Nursing assessment, monitoring, and pharmacology are included in each body system and should be reviewed in preparation for the examination.

For additional, detailed information, please consult the Adult CCRN Exam Handbook (www.aacn.org).

PREPARATION

Be positive!! Avoid any negative thoughts about passing the examination. These thoughts can cause a self-fulfilling prophecy. Set the test date, then establish a realistic schedule for preparing for the examination. Set your priorities; study those areas you are less familiar with first. Look at the percentage of each body system and establish timelines based on the largest to smallest percentage. Know how you study best, by yourself or in study groups. Study in a manner that works best for you. There are flash cards, practice questions, review courses, and study books in outline format and narrative format available for studying. Practice your test questions within a set time limit to familiarize yourself with the time limitations. Allow 2 minutes or less per question (remember the 50-questions-per-hour rule).

When using the practice test questions to study, determine several things when the answers and rationale are being reviewed. Analyze why you missed the question: Did you simply not know the content? Go back and restudy this section. Did you misread the question? Did you misread the answers? Did you miss an important element in the question or scenario? Was there a clue based on age, timeline, or symptoms?
Study those areas that you are least comfortable with, or those that are not in your specialty area. As adult learners, we tend to want to read and study what we like, or what we can use on a daily basis. For this examination, do not spend as much time in your area of specialty (you already know it) but focus on other areas you are not familiar with in your clinical practice.

**DAY OF THE TEST**

Before the examination eat a healthy meal and limit the amount of liquids (to avoid the need for breaks during the exam). Remember, restroom breaks are allowed but the testing time does not stop!

Do not try to cram immediately before the test; this will increase your anxiety. After the exam, make plans to do something special for yourself.

Know where you have to go for the test before the actual day of the test, and also know how long it will take you to get there at the appointed time. Running late and feeling hurried will increase your anxiety and can poorly affect your test-taking skills. Remember, if you are more than 15 minutes late, you will not be allowed to take the examination.

Bring your letter of approval and two forms of identification (one picture ID). You cannot bring any personal effects into the testing room, so leave everything in the car or at home (usually a locker is provided for you to put your personal items in).

You are allowed to do a tutorial on the computer before you start your exam if you need some assistance with CBT. The test time begins as soon as you start the first question of the actual exam. Leaving the testing site without authorization results in an automatic voiding of the test. You will be allowed only 3 hours from the time the test is started.

Results of the examination will be presented onsite at the completion of your exam following a test evaluation.

**TEST-TAKING SKILLS**

Frequently, the difference between pass or fail depends on one’s test-taking skills. An important reminder: Do not read into the questions; take the question and information provided at face value. Answer all questions; do not leave any questions blank. A blank answer will be counted against you. Answering the question, even if it is an “educated” guess, will give you a one out of four chance of being correct.

Key words are important phrases or words used to focus attention on what the question is specifically asking. Examples include *always, earliest, first, on admission, best, least, immediately,* and *initial.*

▷ **HINT**

If the question asks for the “best” response, this is an indication that all answers are probably correct and you will have to determine the best answer for that particular scenario.
Eliminate incorrect options first. Sometimes, you will immediately see an answer that is incorrect. Mark through it to narrow down your choices and improve your odds. Frequently you can get the choices down to two that are more correct than others.

**HINT**
Eliminating options gives a 50/50 chance for an educated guess of the correct answer.

Avoid those answers with words such as “always” or “never.” There is rarely a time in the medical field in which you will always or never take a particular action. If three of the four answers are similar, choose the answer that does not sound similar.

Do not change answers unless absolutely sure. You can “bookmark” a question that you are not sure about and return to it at the end of the test. Sometimes, you will feel more comfortable with the answer after you come back to it.

**HINT**
First impressions are usually good! Do not spend too much time on any one question.

Do not let it worry you if you do not know all the answers. Take a deep breath and keep going. Rejoice in those answers you know and find easy!

**HINT**
You are not really supposed to know all the answers.

Do not try to establish patterns, such as using “two As in a row” for answers.
If there is a long scenario with a large amount of data, read the question first, then read the scenario, then reread the question. Sometimes there will be erroneous data that is not required to answer the question. Too much time may be spent on trying to comprehend the whole scenario.

**HINT**
Do not forget to reread the question to make sure you read it correctly the first time.

Read all answers before you make a choice; there may be more than one correct answer, but one will be the better answer for the question.
HINT
Do not choose the first one that appears to be correct. Use the most correct answer.

Read the question carefully and answer only the question asked. Do not read into the question or think you need more information/data to answer the question.

HINT
The question will provide you with all the information needed to correctly answer the question.

Time frame questions are frequently used in the test. Use the time frame to assist with making the correct choice. Example: Which complication of subarachnoid hemorrhage is seen 7 to 10 days after the bleed?

HINT
All answers may be correct, but only one will occur more commonly during the time frame provided in the question.

Questions may be worded using the lead-in, “What is the gold standard …?” This is not asking what is the most common routine but what is the most reliable and accurate.

Scenarios: Read the patient’s description, word for word. Read the question, then formulate an answer. Read answers and choose the one closest to your formulated answer. Reread the question after answering to ensure you understood the question correctly.

HINT
When the question is answered, you are done. Move on to the next question. Do not second-guess yourself.

Look for answers that facilitate the care of the patient. Facilitative words include nurture, aid, support, reinforce, encourage, and assist.
SUMMARY

Certification is a great path toward personal growth and professionalism. You have taken the first step and are on your way to a great journey. Learning is an amazing thing and you will learn new information, remember things you may have previously learned, and apply this to your practice while studying for this examination. Good luck on your journey, and stay positive and excited about the learning process.
Cardiovascular System Review

In this chapter, you will review:

- Acute coronary syndrome
- Cardiogenic shock
- Arrhythmias associated with acute myocardial infarction (AMI)
- Heart failure
- Dissecting thoracic and abdominal aorta aneurysms
- Cardiac trauma
- Cardiomyopathies
- Hypertensive crisis
- Valvular heart disease
- Peripheral artery disease
- Hypovolemic shock
- Cardiac surgeries
- Hemodynamic monitoring
- Intra-aortic balloon pump counterpulsation
- Pacemakers

**ACUTE CORONARY SYNDROME**

- **What are acute coronary syndromes (ACSs)?**
- Unstable angina (UA), ST-segment elevation myocardial infarction (STEMI), and non-ST-segment elevation myocardial infarction (NSTEMI)

UA, STEMI, and NSTEMI are typically considered to be complications of ACS (Box 1.1).

**Box 1.1 Acute Coronary Syndromes (ACSs)**

<table>
<thead>
<tr>
<th>ACS</th>
<th>Echocardiogram Findings</th>
<th>Cardiac Enzyme Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unstable angina</td>
<td>Normal or nonspecific T-wave changes</td>
<td>Normal</td>
</tr>
<tr>
<td>Non-ST-segment elevation myocardial infarction (MI)</td>
<td>Normal or new onset ST-segment depression</td>
<td>Elevated</td>
</tr>
<tr>
<td>ST-segment elevation MI</td>
<td>Elevated ST-segment in two or more contiguous leads</td>
<td>Elevated</td>
</tr>
</tbody>
</table>

**HINT**

The findings on the 12-lead electrocardiogram (ECG) make the initial differentiation between STEMI and NSTEMI/UA. It is important to obtain an ECG immediately when there is chest pain.
PATHOPHYSIOLOGY

■ What are the characteristics of a “vulnerable” plaque in the coronary artery?
■ Large lipid-rich core, thin fibrous cap over the lipid core, and activated smooth muscle cells and macrophages

Vulnerable plaque, also called “unstable plaque,” is more likely to cause ACS. It is more likely to rupture than a “stable plaque.” The stable plaque has a thinner lipid-rich core and a thicker fibrous cap.

▷ HINT
A patient presents with UA but is ruled out for an AMI. The patient is then typically referred for a cardiac catheterization. The most common finding will be a near-complete obstruction of the involved coronary artery. This is a stable plaque. The patient did not have an AMI.

■ What is plaque erosion?
■ The endothelium erodes, exposing the intimal layer to the components of the circulating blood

Plaque rupture is the most common cause of ACS, but erosion of the plaque can also initiate AMI. Both the rupture and erosion lead to the release of tissue factor, proinflammatory factors, and procoagulants, causing intracoronary thrombosis (Box 1.2).

Box 1.2 Other Causes of AMI

<table>
<thead>
<tr>
<th>Coronary artery spasm</th>
<th>Pulmonary hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary artery embolism</td>
<td>Coronary vasculitis</td>
</tr>
<tr>
<td>Hypertrophic cardiomyopathy</td>
<td>Congenital coronary abnormalities</td>
</tr>
<tr>
<td>Dilated cardiomyopathy</td>
<td>Trauma</td>
</tr>
<tr>
<td>Restricted cardiomyopathy</td>
<td></td>
</tr>
</tbody>
</table>

PREVENTION OF ACS

■ What is a commonly prescribed cholesterol-lowering drug?
■ Statin

Statins are HMG-CoA reductase inhibitors and are commonly prescribed to lower cholesterol. Other cholesterol-lowering therapies include omega-3 fatty acids, fibrates, and lifestyle changes (Box 1.3).

Box 1.3 Prevention of ACS

<table>
<thead>
<tr>
<th>Lifestyle changes</th>
<th>Smoking cessation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>DASH-like diet</td>
</tr>
<tr>
<td></td>
<td>Regular physical activity</td>
</tr>
<tr>
<td></td>
<td>Weight management</td>
</tr>
<tr>
<td>Class I recommendation</td>
<td>Blood pressure control</td>
</tr>
<tr>
<td></td>
<td>Low-density lipoprotein (LDL)-C lowering therapy</td>
</tr>
<tr>
<td></td>
<td>β-blocker</td>
</tr>
<tr>
<td></td>
<td>Angiotensin-converting enzyme (ACE) inhibitor/ angiotensin receptor blocker (ARB)</td>
</tr>
</tbody>
</table>

(continued)
1. CARDIOVASCULAR SYSTEM REVIEW

Box 1.3 Prevention of ACS (continued)

<table>
<thead>
<tr>
<th>Class II recommendation</th>
<th>Glycemic control in diabetes mellitus</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Aspirin (ASA)/antiplatelet agent</td>
</tr>
<tr>
<td></td>
<td>Omega-3 fatty acids</td>
</tr>
<tr>
<td>Other</td>
<td>Influenza vaccination</td>
</tr>
</tbody>
</table>

SYMPTOMS/ASSESSMENT

- **What is a positive Levine sign?**
- Clenched fist held over the chest wall in association with angina chest pain

The chest pain is described as a pressure, heaviness, squeezing, burning, or choking sensation. Typical locations for radiation of pain are the arms, shoulders, and neck. The intensity of angina does not change with respiration, cough, or change in position.

- **What is the most common atypical sign of ACS?**
- **Shortness of breath**

These include epigastric discomfort, nausea and vomiting, diaphoresis, dyspnea, and generalized weakness.

➤ **HINT**
A question on nontypical symptoms would most likely involve a woman in the scenario because women often experience the nontypical symptoms of AMI.

- **Which abnormal heart sound accompanies chest pain caused by AMI?**
- **S₄ gallop**

An S₄ gallop is caused by a resistant ventricle during the late diastolic phase. The S₄ is typically present with chest pain and disappears when chest pain is alleviated. An S₄ gallop is associated with congestive heart failure (CHF) or volume overload. It occurs during the early diastolic phase (Box 1.4).

Box 1.4 Associated Symptoms of Angina

<table>
<thead>
<tr>
<th>Cool, clammy skin</th>
<th>Heart rate changes (tachy or brady)</th>
</tr>
</thead>
<tbody>
<tr>
<td>S₃ gallop</td>
<td>Arrhythmias</td>
</tr>
<tr>
<td>S₄ gallop</td>
<td>Diaphoresis</td>
</tr>
<tr>
<td>Blood pressure changes (hyper- or hypo-)</td>
<td></td>
</tr>
</tbody>
</table>

➤ **HINT**
An S₃ or S₄ may be used in the scenario as a hint for the answer. Remember, S₃ is CHF and S₄ is angina.

- **Which patient population may have a “silent” MI?**
- **Diabetics**

Patients with diabetes mellitus may experience an MI without significant chest pain. This may be attributed to their autonomic neuropathy leading to sensory denervation.

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Which two locations for an AMI may result in bradycardia?

- Inferior wall AMI and posterior wall AMI

The SA node is perfused by the proximal right coronary artery (RCA) in 55% of the population and by the proximal circumflex artery (LCX) in 45% of the population. A loss of blood flow through the RCA or the LCX, depending on the dominance, will result in bradycardia. The presence of a first-degree heart block (prolongation of the PR interval) indicates ischemia at the level of the atrioventricular (AV) node, which is mostly supplied by RCA.

**DIAGNOSIS**

- **What is the initial diagnostic test obtained with the onset of chest pain?**
  - 12-lead ECG

  A 12-lead ECG is performed and interpreted within 10 minutes of arrival in the emergency department (ED) with chest pain. If the initial 12-lead shows ST-segment elevation in two contiguous leads, then reperfusion strategies are initiated. If the initial 12-lead does not show significant finding but if the patient continues to have chest pain, then an ECG may be obtained as often as every 10 minutes.

  › HINT
  
  Remember that a person can be having an AMI without elevating ST segments. The differentiation between UA and NSTEMI is cardiac enzymes.

- **The finding of ST-segment elevation in leads II, III, and a ventricular fibrillation (VF) indicates the need for what follow-up ECG?**
  - Right precordial lead placement (right-sided ECG)

  An inferior wall AMI (leads II, III, and aVF) may also involve the wall of the right ventricle (RV). The best diagnostic for RV involvement is to obtain a right precordial ECG and look for ST-segment changes in V_{4R} and V_{5R}. Another change in lead placement may be to extend the left precordial leads laterally toward the left posterior chest to better view the posterior-lateral infarction.

  › HINT
  
  The best ECG lead placement for the diagnosis of a posterior wall AMI is on the back even though this is not commonly performed. Typically, look for reciprocal changes in the anterior leads. The changes include tall R waves and ST depression in V_{1} and V_{2} (sometimes V_{3}). This is a reciprocal change of ST-segment elevation and Q waves (or loss of R wave height). Posterior infarcts are mirror images of anterior infarcts.

- **Which leads are used to recognize lateral wall ischemia and infarction?**
  - Leads I, aVL, V_{5}, V_{6}

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Leads I, aVL, V5 and V6 all view the lateral aspect of the left ventricle. ST elevation in leads I and aVL only indicate a high lateral STEMI (Box 1.5).

**Box 1.5 Localizing the Infarct**

<table>
<thead>
<tr>
<th>Wall Involvement</th>
<th>Leads of Monitor</th>
<th>Involved Coronary Artery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior wall myocardial infarction (AWMI)</td>
<td>V3–V4 or loss of R-wave progression</td>
<td>Left anterior descending (LAD)</td>
</tr>
<tr>
<td>Septal wall</td>
<td>V1–V2</td>
<td>LAD</td>
</tr>
<tr>
<td>Lateral wall MI (LWMI)</td>
<td>I, aVL, V5, V6</td>
<td>Left circumflex (LCX)</td>
</tr>
<tr>
<td>Inferior wall MI (IWMI) or RV infarct</td>
<td>II, III, aVF</td>
<td>Right coronary artery (RCA)</td>
</tr>
</tbody>
</table>

- **Which ECG change indicates an acute injury?**
- **An elevated ST segment**

An elevated ST segment indicates a potentially reversible injury. An ST-segment elevation of ≥1 mm is considered significant. ST segments return to normal after reperfusion.

➤ **HINT**

Q wave without ST-segment changes indicate a previous MI and are usually noted to be of an “undetermined” age. It is important to compare a patient’s ECG with an old one, if available, to evaluate for old or new changes.

- **What is considered to be a “significant” Q wave?**
- **A Q wave of 2 mm or more in depth or longer than 0.04-second duration or greater than a third of the height of the QRS complex**

Significant Q waves indicate irreversible myocardial damage (infarction). Q waves may develop within several hours of injury or may take up to several days to weeks to occur. Q waves can persist for the lifetime of a patient.

➤ **HINT**

Inferior leads typically have small Q waves but are not significant.

- **Which of the cardiac enzymes is the preferred biomarker for diagnosing an AMI?**
- **Troponin levels**

Troponin levels are more sensitive and specific to AMI than creatine phosphokinase-MB (CPK-MB). There are other causes of elevated troponin levels, which may include chronic heart failure (HF), acute cardiomyopathy, cardiac contusions, myocarditis, and chronic kidney disease.

- **How long after the onset of myocardial injury will an increase in cardiac enzymes occur?**
- **4 to 6 hours**

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CPK-MB and troponin increase within 4 to 6 hours after ischemia. To rule out MI, cardiac enzymes are commonly drawn every 8 hours for 24 hours. CPK-MB levels return to baseline within 36 to 40 hours. Troponin levels remain elevated for up to 10 days.

- **Which cardiac protein elevates first?**
- **Myoglobin**

Myoglobin levels elevate within 1 hour of ischemia and return to normal within 24 hours. Myoglobin is a sensitive marker for muscle damage but is not specific to myocardial muscle.

▷ **HINT**

Elevated myoglobin increases suspicion of AMI in patients presenting with anginal-type chest pain.

**MANAGEMENT OF AMI**

- **Name two emergency treatments for STEMI.**
- **Emergency coronary balloon angioplasty with stenting and intravenous (IV) thrombolytic agent**

Primary percutaneous coronary intervention (PCI) is the recommended therapy for STEMI, but thrombolytic therapy remains an important option for treatment in hospitals without PCI capabilities.

▷ **HINT**

IV thrombolytics is only indicated in a STEMI and is not a treatment for NSTEMI or UA.

- **What is the standard for “door-to-balloon” time?**
- **Less than 90 minutes**

Current standards mandate that the time to primary PCI should be less than 90 minutes. PCI includes angioplasty, aspiration thrombectomy, and/or stent placement. Stents are either bare-metal stents (BMS) or drug-eluting stents (DES).

- **For which types of ACS is PCI indicated?**
- **STEMI, NSTEMI, and UA**

PCI is indicated if ischemic symptoms started less than 12 hours ago, clinical evidence of ongoing ischemia is between 12 and 24 hours after onset of symptoms, or if there is cardiogenic shock/severe HF regardless of time delay from onset.
HINT
PCI should not be performed in a noninfarct artery at the time of primary PCI in patients with STEMI who are hemodynamically stable.

High-risk MIs have better outcomes with primary PCI therapy (Box 1.6).

Box 1.6 High-Risk Myocardial Infarctions

<table>
<thead>
<tr>
<th>Elderly patients</th>
<th>Systolic blood pressure (BP) &lt; 100</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior wall STEMI</td>
<td>Signs of acute heart failure or low cardiac output (CO)</td>
</tr>
<tr>
<td>Serious ventricular arrhythmias</td>
<td>Cardiogenic shock</td>
</tr>
</tbody>
</table>

- **Which drug therapy is recommended to support primary PCI?**
- **Anticoagulant and/or antiplatelet agent**

Aspirin (ASA) and thienopyridine (clopidogrel, prasugrel, or ticagrelor) should be given as early as possible or at the time of the PCI. A GP IIb/IIIa receptor antagonist may also be used at the time of the primary PCI if receiving unfractionated heparin (UFH). These include abciximab, tirofiban, and eptifibatide. Bivalirudin monotherapy may be used instead of the combination of UFH and a GP IIb/IIIa receptor antagonist.

HINT
Prasugrel should not be administered to patients with a history of prior stroke or transient ischemic attack (TIA).

- **What is the minimal time required for dual antiplatelet drug therapy in DES?**
- **1 year**

A dual antiplatelet therapy is recommended for a minimum of 1 year after a DES and 1 month (4–6 weeks) after placement of a BMS. A BMS is recommended for:

1. High risk of bleeding
2. Predicted compliance issues
3. Known need for a surgical procedure

The dual antiplatelet is an aspirin and a thienopyridine (P2Y<sub>12</sub> receptor inhibitor). Thienopyridines include clopidogrel (Plavix), prasugrel (Effient), or ticagrelor.

- **What are the clinical indications for fibrinolytic therapy?**
- **STEMI, hyperacute T wave, posterior infarction (reciprocal ST-segment depression V<sub>1</sub>–V<sub>3</sub>), and new-onset left bundle branch block (LBBB)**

HINT
Peaked T waves are tall and narrow and develop at the onset of the infarction. These are called “peaking” or “hyperacute” T waves. Rule out peaked T waves due to hyperkalemia.

Fibrinolytic therapy is a treatment option and should be given, if not contraindicated, within 12 hours of onset of ischemic symptoms if PCI cannot be performed within 120 minutes.

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What is the recommendation for “door-to-needle” time when administering a fibrinolytic agent?

30 minutes or less

If fibrinolytic therapy is used, the goal for “door-to-needle” time is less than 30 minutes from presentation to medical facility.

When would a fibrinolytic agent be administered to a patient with STEMI beyond the 12-hour window?

Ongoing signs of ischemia

A fibrinolytic agent may be administered when PCI is not available and the patient has clinical evidence of ongoing ischemia (symptoms and/or ECG changes), hemodynamic instability, or when a large area of myocardium is at risk. This should still occur within 12 to 24 hours of the onset of symptoms.

HINT

If the scenario is a STEMI patient in cardiogenic shock or acute severe HF, immediate transfer is recommended to a hospital with PCI capabilities, irrespective of the time of onset of symptoms.

Which ACS patients would not be a candidate for fibrinolytic therapy?

NSTEMI and UA

Fibrinolytic therapy is not indicated in NSTEMI or UA, and transfer to a hospital with PCI capabilities is mandated (Boxes 1.7 and 1.8).

Box 1.7 Absolute Contraindications of Fibrinolytic Therapy

<table>
<thead>
<tr>
<th>Contraindication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prior intracranial hemorrhage</td>
</tr>
<tr>
<td>Known intracranial vascular lesion (e.g., aneurysm, arteriovenous malformation [AVM])</td>
</tr>
<tr>
<td>Known malignant intracranial tumor</td>
</tr>
<tr>
<td>Significant traumatic brain injury within 3 months</td>
</tr>
<tr>
<td>Suspected aortic dissection</td>
</tr>
<tr>
<td>Active bleeding</td>
</tr>
<tr>
<td>Ischemic stroke within 3 months</td>
</tr>
</tbody>
</table>

Box 1.8 Fibrinolytic Agents

<table>
<thead>
<tr>
<th></th>
<th>Streptokinase</th>
<th>Alteplase (tPA)</th>
<th>Reteplase (rPA)</th>
<th>Tenecteplase (TNK)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dose</td>
<td>1.5 million units over 30–60 minutes</td>
<td>15 mg bolus followed by 0.75 mg/kg given over 30 minutes, then 0.5 mg/kg over 1 hour</td>
<td>Two 10 U boluses given 30 minutes apart</td>
<td>Based on kilograms of body weight</td>
</tr>
<tr>
<td>Half-life</td>
<td>20 minutes</td>
<td>4–6 minutes</td>
<td>18 minutes</td>
<td>20 minutes</td>
</tr>
<tr>
<td>90-minute patency</td>
<td>50%</td>
<td>75%</td>
<td>60%–70%</td>
<td>75%</td>
</tr>
<tr>
<td>Fibrin specificity</td>
<td>– –</td>
<td>++</td>
<td>+</td>
<td>+++</td>
</tr>
</tbody>
</table>

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1. CARDIOVASCULAR SYSTEM REVIEW

Following the administration of a fibrinolytic agent, what medications should be given to prevent early reinfarction?

Aspirin (ASA), clopidogrel (Plavix), and an anticoagulation drug

Early reinfarction following thrombolytic therapy may be prevented with the administration of antiplatelet and anticoagulation medications. An ASA (81–325 mg) should continue indefinitely. Clopidogrel (Plavix) should be continued for at least 14 days and up to 1 year. Anticoagulation therapy is recommended for a minimum of 48 hours and may continue throughout hospitalization (up to 8 days). Anticoagulation therapy can include one of the following: UFH, enoxaparin (Levenox), or fondaparinux.

What are the four signs of reperfusion following the administration of a fibrinolytic agent or PCI?

Relief of chest pain, ST-segment return to baseline, abrupt onset of ventricular arrhythmias, and increased levels of cardiac enzymes (washout effect)

When the coronary artery is reperfused, there is a relief of chest pain and return of ST-segment toward baseline due to the reversal of the ischemic injury. Reperfusion of the previously obstructed coronary artery allows the washout of cardiac enzymes accumulated distal to the obstruction with accompanying ventricular arrhythmias and short runs of ventricular tachycardia (VT).

The most important sign is the relief of chest pain and ST-segment improvement of more than 50%.

What is the most sensitive continuous monitor used to recognize MI or efficacy of treatment for STEMI?

ST-segment monitoring

ST-segment monitoring is recommended for patients with ACS. If the patient suffered a STEMI, use the best lead with the ST elevation as the “fingerprint” to monitor for changes in the elevation of the ST segment. If the patient does not have ST-segment elevation, monitor leads III and V5. ST-segment monitoring can also recognize a “silent MI” and may be more sensitive than a patient reporting chest pain in some situations.
HINT
High-risk surgical patients may also benefit from continuous ST-segment monitoring. Lead V_s is the most valuable for identifying demand-related ischemia.

What is the indication of a coronary artery bypass graft (CABG) in a patient with a STEMI?
Coronary artery anatomy not amendable by PCI

An urgent CABG is indicated in patients with a STEMI experiencing ongoing or recurrent ischemia, cardiogenic shock, and severe HF, and when coronary artery anatomy is not amendable by PCI. Another indication is a patient who presents within 6 hours of onset of symptoms and is not considered to be a candidate for PCI or fibrinolytic therapy.

What is the primary intervention for an RV infarction?
Fluid bolus

The RV pumps differently from the left ventricle (LV). The LV has both spiral and circular muscles. The spiral muscles wrap around in such a manner that contribute to the contraction or “wringing” out of the blood of the heart. Volume overload worsens LV HF. In patients with LV involvement, fluid intake is limited, and venodilators such as nitrates and morphine are administered to lower the preload. The RV pumps using a bellows-type mechanism with the free wall moving toward the septum. Volume in the RV is required to produce an adequate stroke volume (SV). With RV involvement, fluid boluses are indicated.

HINT
Nitrates should not be used in RV infarction. Inotropic agents and intra-aortic balloon counterpulsations may also be indicated.

Which class of anticoagulation therapy is not recommended in UA or NSTEMI?
Vitamin K antagonist

An aspirin is recommended immediately and must be continued indefinitely. Clopidogrel (Plavix) may also be used as a substitute or as dual antiplatelet therapy. Glycoprotein IIb/IIa inhibitors may also be administered to patients at higher risk and may be used as a third antiplatelet drug. UFH has been found to be beneficial for patients with UA. Factor Xa inhibitors have been shown to produce favorable outcomes in UA/NSTEMI and direct thrombin inhibitors may be an alternative. Warfarin (Coumadin), a vitamin K antagonist, has shown no benefit and may increase bleeding risk.

What two drugs administered after ACS have been found to reduce LV remodeling?
β-blockers and ACE inhibitors

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β-blockers blunt the effects of the sympathetic nervous system, thereby reducing heart rate, blood pressure (BP), and contractility. They may also reduce the risk of serious arrhythmia by preventing maladaptive remodeling of the LV. ACE inhibitors can also prevent the adverse remodeling of the LV following an MI. They should be initiated during hospitalization and continued long term (unless contraindicated).

**HINT**

β-blockers are contraindicated if signs of cardiogenic shock or severe HF are present.

- Which medication is indicated within the first 24 hours of AWMI with a ventricular ejection fraction (EF) of less than 40%?
- ACE inhibitor

This is an American College of Cardiology (ACC) and American Heart Association (AHA) Class I recommendation for AWMI. An ACE inhibitor may also be indicated for other types of AMI within the first 24 hours in the absence of hypotension. ACE inhibitors function as arterial vasodilators, decreasing LV afterload, lowering BP, and preventing adverse LV remodeling. Following an AMI, ventricular remodeling can result in sudden cardiac death (SCD) from ventricular arrhythmias.

**HINT**

High-intensity statin therapy should be administered in all patients with STEMI (unless contraindicated).

- Which clinical situations would be contraindicated to administer an aldosterone blocker?
- Elevated serum creatinine levels and hyperkalemia

Inspira (Eplerenone) is an aldosterone blocker used in acute MIs and HF with an LVEF less than 40%. Contraindications include serum creatinine levels greater than 2.5 mg/dL in men and greater than 2.0 mg/dL in women. It is also contraindicated to administer if potassium levels are greater than 5.0 mEq/L.

- Acute pulmonary edema following an inferior MI with new onset holosystolic murmur indicates which complication?
- Acute severe mitral regurgitation

Acute severe mitral regurgitation usually occurs within 24 hours of the infarction but may occur up to 3 to 5 days later. Early recognition and management with inotropic therapy, intra-aortic balloon counterpulsation, and surgery can improve outcomes. Urgent surgical repair of the mitral valve is a Class I recommendation by the ACC/AHA.

**HINT**

Mitrail regurgitation causes a holosystolic murmur. Look at the type of murmur given as a hint in the scenario.
What is the recommended treatment for pericarditis after STEMI?

ASA

ASA is the recommended drug therapy following a STEMI. If ASA—even at higher doses—is ineffective, then administration of acetaminophen, colchicine, or opioid analgesics may be ordered.

HINT

If pericarditis is due to STEMI, do not administer glucocorticoids and nonsteroidal anti-inflammatory drugs. These are potentially harmful in this situation.

COMPLICATIONS

Which type of AMI is most likely to result in development of an LV mural thrombus?

AWMI

A large AWMI develops LV regional wall akinesia or dyskinesia with blood stasis. The contributing factors of a thrombus include inflammation of the endocardium and a hypercoagulable state. The presentation is an embolic stroke and symptoms depend on the location of the embolus. An echocardiogram can be used to identify the thrombus in the LV. Anticoagulation therapy is used to manage the thrombus (heparin followed by warfarin for 3–6 months).

HINT

This complication of an LV mural thrombus typically occurs within the first 10 days of AMI.

Following ventricular septal rupture (VSR), what type of murmur does the patient suddenly develop?

Loud, harsh holosystolic murmur

The other symptoms of VSR include shortness of breath, biventricular failure, chest pain, and hypotension. This complication may occur within 24 hours of the AMI and then peaks again 3 to 5 days after the AMI. Very rarely would it present after 2 weeks. Treatment would be to manage the patient with vasodilators (reduce afterload), inotropic agents, diuretics, or mechanical support with an intra-aortic balloon pump (IABP) until the defect can be repaired surgically.

HINT

The complication of cardiogenic shock does not produce a murmur and mitral regurgitation has a soft systolic murmur.

What is the most common arrhythmia or cause of death in an LV free wall rupture?

Pulseless electrical activity (PEA)
LV free wall rupture may occur with large, transmural infarctions. Clinical presentation may include sudden, severe chest pain with abrupt hemodynamic collapse and PEA. This is due to the rapid development of pericardial tamponade (Box 1.9).

**Box 1.9 Complications of AMI**

<table>
<thead>
<tr>
<th>Cardiogenic shock</th>
<th>Arrhythmias</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pericarditis</td>
<td>Ventricular free wall rupture</td>
</tr>
<tr>
<td>Post–myocardial infarction syndrome (Dressler’s syndrome)</td>
<td>Cardiac tamponade</td>
</tr>
<tr>
<td>Left ventricular aneurysm</td>
<td>Ventricular septal rupture</td>
</tr>
<tr>
<td>Papillary muscle rupture/mitral regurgitation</td>
<td>Left ventricular mural thrombus</td>
</tr>
</tbody>
</table>

**CARDOGENIC SHOCK**

- **What is the most severe form of HF?**
- **Cardiogenic shock**

Cardiogenic shock is the most severe form of HF and requires emergency management. Cardiogenic shock and pericardial tamponade are life-threatening conditions.

**PATHOPHYSIOLOGY**

- **What is the primary cause of cardiogenic shock?**
- **Ischemia**

Cardiogenic shock remains the leading cause of mortality in AMI. Ischemic cardiomyopathy is the primary cause of cardiogenic shock. Cardiogenic shock is defined as hypoperfusion due to cardiac failure (Box 1.10).

**Box 1.10 Other Causes of Cardiogenic Shock**

<table>
<thead>
<tr>
<th>Hypertrophied cardiomyopathy</th>
<th>Stress-induced cardiomyopathy (takotsubo cardiomyopathy)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aortic dissection with aortic insufficiency</td>
<td>Acute valvular regurgitation (endocarditis or chordal rupture)</td>
</tr>
<tr>
<td>Aortic or mitral stenosis (increases myocardial stress)</td>
<td>Cardiac tamponade</td>
</tr>
<tr>
<td>Acute myocarditis</td>
<td>Massive pulmonary embolism</td>
</tr>
</tbody>
</table>

- **Which type of AMI is most likely associated with cardiogenic shock?**
- **AWMI**

Risk factors for the development of cardiogenic shock following an AMI include AWMI, multiple-vessel disease, older age, hypertension, prior MI, STEMI, and the presence of LBBB.
HINT
May use these risk factors as hints in a scenario to assist with recognizing cardiogenic shock.

SYMPTOMS/ASSESSMENT

■ What are the characteristic hemodynamic parameters of cardiogenic shock?
■ Low cardiac output (CO)/cardiac index (CI), high systemic vascular resistance (SVR), and high filling pressures

Cardiogenic shock demonstrates persistent hypotension with severe reduction in CO/CI and adequate or elevated filling pressures. Compensatory mechanisms for low CO/CI include vasoconstriction (elevates SVR) and tachycardia, which actually worsen the CO/CI due to high resistance and increased workload of the heart, causing a vicious cycle to develop (Box 1.11).

HINT
Severe reduction of CI is defined as less than 1.8 L/min/m² without support and less than 2.0 to 2.2 L/min/m² with support.

Box 1.11 Other Symptoms of Cardiogenic Shock

<table>
<thead>
<tr>
<th>Tachycardia</th>
<th>Altered mental status</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cool, clammy skin</td>
<td>Tachypnea</td>
</tr>
<tr>
<td>Pale nail beds with</td>
<td>Presence arrhythmias</td>
</tr>
<tr>
<td>delayed capillary</td>
<td></td>
</tr>
<tr>
<td>refill</td>
<td></td>
</tr>
<tr>
<td>Decreased urine</td>
<td></td>
</tr>
<tr>
<td>output</td>
<td></td>
</tr>
</tbody>
</table>

DIAGNOSIS

■ What monitoring device may be used to assist with the diagnosis of cardiogenic shock?
■ Pulmonary artery catheter (PAC)

A PAC provides information on the CO/CI, filling pressures and enables the calculation of SVR. These readings are used to define and recognize cardiogenic shock. Newer hemodynamic monitors that are minimally invasive and that use the arterial waveform may also be used to assist with the diagnosis.

HINT
An echocardiogram may be used to confirm the diagnosis of high filling pressures and to rule out other causes of hypotension following an AMI.

MEDICAL MANAGEMENT

■ What is the greatest concern when administering an inotropic agent to a patient in cardiogenic shock?
■ Increase in myocardial workload and oxygen consumption

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Inotropic agents are frequently needed to increase CO and reduce filling pressures in the RV and LV, but they can increase the oxygen demand in a heart with limited oxygen supply. This may increase the ischemic injuries to the myocardium. Inotropes are recommended in hypoperfusion states with or without pulmonary congestion, but may be initiated at a lower dose in cardiogenic shock to limit complications.

**HINT**
Inotropes can also induce arrhythmias in ischemic hearts and should be closely monitored.

- **What is a first-line intervention in managing hypotension in cardiogenic shock?**
- **Inotropes**

Vasoconstrictors (e.g., norepinephrine) should not be used initially to treat hypotension in cardiogenic shock due to the presence of increased SVR. Other interventions for managing hypotension in cardiogenic shock include a combination of inotropic agents with vasodilators, fluid challenges with inotropic agents, and mechanical assistance (IABP, left ventricular assist device [LVAD]).

**HINT**
If vasoconstrictors are needed, it is recommended that norepinephrine be used with caution instead of dopamine.

**SURGICAL MANAGEMENT**

- **What is the surgical procedure for managing cardiogenic shock?**
- **Revascularization**

Emergency revascularization with a CABG procedure is indicated to improve survival. It may not improve the 30-day survival but it has been shown to improve long-term survival (6-month and 6-year outcome studies).

**HINT**
Mechanical devices (e.g., IABP) may be used as a bridge to stabilize the patient prior to surgery.

**ARRHYTHMIAS ASSOCIATED WITH AMI**

**PATHOPHYSIOLOGY**

- **What is the most common reason for atrial arrhythmias following an AMI?**
- **Left atrial (LA) distension**

LA distention is frequently caused by high pressure in the LV. Atrial arrhythmias (PACs, atrial flutter, atrial fibrillation [AF]) are frequently a result of LA distention following an AMI.
DIAGNOSIS

■ **What is the ECG finding of a first-degree heart block?**
  - PR interval prolonged greater than 0.20

A prolonged PR interval without loss of ventricular conduction indicates that the block is above the bundle of His. Calcium channel blockers and β-blockers may exacerbate the prolonging of the PR interval but should only be stopped if hemodynamically unstable or a higher degree of block occurs.

■ **What ECG change determines whether the block is above or below the nodal area?**
  - Width of the QRS

Supranodal or intranodal blocks produce a narrow QRS pattern. Blocks that occur below the nodal area produce a wide QRS complex.

MEDICAL MANAGEMENT

■ **Are rate-control drugs more effective in atrial flutter or AF?**
  - Atrial fibrillation

Atrial flutter is managed similarly to AF except ventricular rate control in the atrial flutter is not as responsive to rate-control drugs. A patient who develops an atrial flutter and is hemodynamically compromised requires synchronized electrical cardioversion.

■ **What complication of AMI would limit the use of IV diltiazem in the treatment of AF?**
  - Moderate to severe HF

The use of IV diltiazem in managing ventricular rate should be done cautiously following an AMI due to the complication of HF.

➤ **HINT**

Remember that some calcium channel blockers have negative inotropic effects.

COMPLICATIONS

■ **Following an inferior AMI, the patient presents with heart rate less than 40 bpm and hypotension. If BP is unresponsive to atropine, what complication may be the cause?**

■ RV infarction and/or volume depletion

An RV infarction can be present with an inferior wall MI. The presence of RV involvement and/or volume depletion will frequently result in continued hypotension, despite
treatment with atropine. Obtain right-sided 12-lead ECG and administer fluids for RV involvement to correct the hypotension.

› HINT
Bradycardia does not require treatment if the patient is hemodynamically stable (Box 1.12). Review Advanced Cardiovascular Life Support (ACLS) certification.

Box 1.12 Arrhythmias

<table>
<thead>
<tr>
<th>Arrhythmia</th>
<th>Causes</th>
<th>Treatment/Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paroxysmal supraventricular tachycardia (PSVT)</td>
<td>Left atrial (LA) distention from elevated left ventricular (LV) pressures, Inflammation (pericarditis)</td>
<td>Adenosine when hypotension not present, If hypotensive, may use intravenous (IV) diltiazem or β-blocker, If severe hypotension, perform synchronized electrical cardioversion</td>
</tr>
<tr>
<td>Atrial flutter</td>
<td>Sympathetic overstimulation of LA (usually transient)</td>
<td>Similar to AF except not responsive to rate-control drugs, If symptomatic, perform synchronized electrical cardioversion, If refractory medical management, may overdrive atrial pace</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>LV failure, Ischemia to atria, Right ventricular (RV) infarction, Pericarditis</td>
<td>If unstable, immediate synchronized electrical cardioversion, If stable, control the ventricular rate with IV amiodarone or digoxin, β-blocker may be used if not hypotensive, Anticoagulation therapy</td>
</tr>
<tr>
<td>Bradycardia</td>
<td>Inferior or posterior wall myocardial infarction, Vagal stimulation</td>
<td>If unstable, administer atropine 0.5 to 1 mg, External or transvenous pacing</td>
</tr>
<tr>
<td>First-degree atrioventricular block</td>
<td>Inferior wall myocardial infarction</td>
<td>No treatment required</td>
</tr>
<tr>
<td>Second-degree Mobitz Type I AV block</td>
<td>Inferior wall myocardial infarction</td>
<td>No treatment required if hemodynamically stable</td>
</tr>
<tr>
<td>Second-degree Mobitz Type II AV block</td>
<td>Anterior wall myocardial infarction</td>
<td>Transcutaneous or transvenous pacing, Atropine, Possibly permanent demand pacemaker</td>
</tr>
<tr>
<td>Third-degree AV block</td>
<td>Anterior wall myocardial infarction, Inferior wall myocardial infarction</td>
<td>Atropine if inferior wall MI, Temporary transcutaneous or transvenous pacing, Permanent demand pacemaker</td>
</tr>
<tr>
<td>Ventricular tachycardia (VT)</td>
<td>Monomorphic VT most likely caused by myocardial scar, Polymorphic VT most likely caused by ischemia, Electrolyte abnormalities, Hypoxia, Acid–base disturbances</td>
<td>If unstable, unsynchronized cardioversion</td>
</tr>
<tr>
<td>Ventricular tachycardia (VF)</td>
<td>MI, Cardiogenic shock</td>
<td>Unsynchronized electrical countershock</td>
</tr>
</tbody>
</table>
HEART FAILURE

During admission history, a patient with HF tells you that he is comfortable at rest but becomes short of breath during activities of daily living. In which New York Heart Association (NYHA) class is this patient?

NYHA Class II

NYHA Class II is described as a person that is asymptomatic at rest but becomes dyspneic during normal activities of daily living.

The NYHA classification system was devised to classify the extent of HF based on functional capacity. It lists four categories of cardiac disease (Classes I-IV) ranging from mild to severe with progressively increasing symptoms of physical limitation associated with each class. For more information, please see www.my.americanheart.org.

At which stage is a patient with normal EF, but a history of mitral regurgitation, according to the ACC/AHA stages?

Stage B

The ACC/AHA stages of HF were developed for early identification of at-risk patients for HF (Table 1.1). The stages are not meant to replace the NYHA classification system. Patients in stages A and B are at risk for HF but are currently not symptomatic. The staging system includes therapy recommendations for each stage (Table 1.2).

Table 1.1 ACC/AHA Stages in Development of HF

<table>
<thead>
<tr>
<th>Stage</th>
<th>Definition</th>
<th>Patients at risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage A</td>
<td>At high risk for HF but without structural heart disease or symptoms of HF</td>
<td>Hypertension, Atherosclerotic disease, Diabetes, Obesity, Metabolic syndrome, Using cardiotoxins, Family history of cardiomyopathy</td>
</tr>
<tr>
<td>Stage B</td>
<td>Structural heart disease but without signs or symptoms of HF</td>
<td>Previous MI, LV remodeling including LVH and low EF, Asymptomatic valvular disease</td>
</tr>
<tr>
<td>Stage C</td>
<td>Structural heart disease with prior or current symptoms of HF</td>
<td>Known structural heart disease, Shortness of breath, Fatigue, reduced exercise tolerance</td>
</tr>
<tr>
<td>Stage D</td>
<td>Refractory HF requiring specialized interventions</td>
<td>Marked symptoms at rest despite maximal medical therapy</td>
</tr>
</tbody>
</table>

Adapted from the ACC/AHA practice guidelines.
Table 1.2 ACC/AHA-Recommended Therapy by Stage

<table>
<thead>
<tr>
<th>Stage</th>
<th>Goals</th>
<th>Drugs</th>
<th>Devices/Options</th>
</tr>
</thead>
</table>
| Stage A | Treat hypertension  
Encourage smoking cessation  
Treat lipid disorders  
Encourage regular exercise  
Discourage alcohol intake, illicit drug use  
Control metabolic syndrome | ACE inhibitor or angiotensin receptor blocker (ARB) in appropriate patients for vascular disease or diabetes | |
| Stage B | Same as Stage A | ACE inhibitor or ARB in appropriate patients  
β-blockers in appropriate patients | Biventricular pacing  
Implantable defibrillators |
| Stage C | All measures under Stages A and B  
Dietary salt restriction | Diuretics for fluid retention  
ACEI  
β-blockers  
Selected patients  
Aldosterone antagonist  
ARBs  
Digitalis  
Hydralazine/nitrates | |
| Stage D | Appropriate measures under Stages A, B, and C. Decision regarding appropriate level of care | | Compassionate end-of-life care/hospice  
Extraordinary measures  
Heart transplant  
Chronic inotropes  
Permanent mechanical support  
Experimental surgery or drugs |

Adapted from the ACC/AHA practice guidelines.

PATHOPHYSIOLOGY

- **Which type of HF causes a difficulty in filling the ventricles?**
- **Diastolic dysfunction**

HF caused by diastolic dysfunction is a result of a difficulty with the filling of the ventricles. The difficulty with the ventricular filling may be caused by an incomplete ventricular relaxation, increased stiffness, pericardial restraint, or high intrathoracic pressure. The most common cause is hypertrophy of cardiac muscle.

- **What is the most common underlying etiology of a diastolic dysfunction HF?**
- **Hypertrophic cardiomyopathy**

Hypertrophic cardiomyopathy is the most common cause of diastolic dysfunction HF. It may be congenital or caused by chronic hypertension or aortic stenosis.

- **Which commonly used drug classification in HF is contraindicated if the underlying abnormality is a diastolic dysfunction?**
- **Inotropic agents**

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The problem is not with the contractility of the pump (most patients with diastolic dysfunction actually have normal EF), but with a small ventricular chamber. An inotropic agent will constrict the chamber, limiting the filling even more. Calcium channel blockers are typically used to manage diastolic dysfunction. The goal is afterload reduction and relaxation of the ventricles.

› HINT
Diastolic dysfunction HF frequently exhibits symptoms of failure but has a high EF.

■ What type of HF has a decreased EF?
■ Systolic dysfunction

Systolic dysfunction is caused by poor contractility or reduction in CO. It is commonly defined as an EF less than 35% to 40%. It is the most common cause of HF (Box 1.13).

Box 1.13 Etiology of Systolic Dysfunction HF

<table>
<thead>
<tr>
<th>Cause</th>
<th>Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myocardial ischemia</td>
<td>Obstructive cardiomyopathy</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>Infection</td>
</tr>
<tr>
<td>Valvular heart disease</td>
<td>Toxin exposure</td>
</tr>
<tr>
<td>Chronic volume overload</td>
<td>Congenital heart defects</td>
</tr>
<tr>
<td>Dilated cardiomyopathy</td>
<td></td>
</tr>
</tbody>
</table>

The goal is to decrease preload and afterload.

› HINT
Remember:
Preload = Volume
Afterload = Resistance

■ Drugs used to manage HF typically inhibit which neurohormonal compensatory mechanism?
■ Renin-angiotensin system (RAS)

Aldosterone blockers, ACE inhibitors, and angiotensin receptor blockers (ARBs) all work on the RAS (Figure 1.1).

■ What compensatory mechanism increases blood flow to the kidneys, thus decreasing the release of renin?
■ Brain natriuretic peptide (BNP)

BNP is a peptide released by stretched myocytes during HF. The BNP will increase glomerular filtration rates, increase renal blood flow, decrease the release of renin, and decrease Na⁺ reabsorption by the kidneys.

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1. CARDIOVASCULAR SYSTEM REVIEW

SYMPTOMS/ASSESSMENT

- Which abnormal heart sound is commonly associated with HF?
  - S₃

S₃ is a gallop that occurs during early diastole and is frequently caused by ventricular overload (Box 1.14).

▷ HINT
  A patient scenario with an S₃ is typically HF, whereas an S₄ is angina.

Box 1.14 Symptoms of HF

<table>
<thead>
<tr>
<th>Left-Sided Failure</th>
<th>Right-Sided Failure</th>
<th>Other Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crackles</td>
<td>Jugular venous distention</td>
<td>Fatigue</td>
</tr>
<tr>
<td>Tachypnea</td>
<td>Hepatomegaly</td>
<td>Weakness</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>Splenomegaly</td>
<td>Decreased exercise tolerance</td>
</tr>
<tr>
<td>Hypoxemia</td>
<td>Elevated central venous pressure (CVP)</td>
<td>Unexplained confusion</td>
</tr>
<tr>
<td>Cough</td>
<td>Peripheral edema</td>
<td></td>
</tr>
<tr>
<td>Pink, frothy sputum</td>
<td>Decreased CO</td>
<td></td>
</tr>
<tr>
<td>Decreased CO</td>
<td>Tachycardia</td>
<td></td>
</tr>
<tr>
<td>Orthopnea</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tachycardia</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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DIAGNOSIS

■ What is the single most useful diagnostic test in the evaluation of a patient with HF?
■ Comprehensive two-dimensional (2-D) echocardiogram coupled with Doppler flow

The 2-D echocardiogram can determine whether the abnormality is with the myocardium, heart valves, or pericardium. SV can be determined with use of echocardiogram by measuring the LV outflow tract and the amount of blood that goes through it (Box 1.15).

Box 1.15 Other Diagnostic Tests

<table>
<thead>
<tr>
<th>Test</th>
<th>Advantage</th>
<th>Disadvantage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Radionuclide ventriculography</td>
<td>Accurate measurements of left ventricular (LV) function and right ventricular ejection fraction (RVEF)</td>
<td>Unable to assess valve abnormalities or cardiac hypertrophy</td>
</tr>
<tr>
<td>MRI</td>
<td>Evaluate chamber size, ventricular mass, right ventricular (RV) dysplasia, and pericardial disease</td>
<td></td>
</tr>
<tr>
<td>Chest x-ray (CXR)</td>
<td>Estimate degree of cardiac enlargement and pulmonary edema</td>
<td>Unable to determine LV function or valvular abnormalities</td>
</tr>
<tr>
<td>12-lead ECG</td>
<td>Demonstrate evidence of prior myocardial infarction, LV hypertrophy, cardiac conduction abnormality, or cardiac arrhythmia</td>
<td>Unable to determine the mechanical function of the ventricles</td>
</tr>
</tbody>
</table>

■ Which laboratory test may be used to determine the severity of HF?
■ BNP levels

BNP levels may also be used to differentiate between HF and pulmonary disease. Elevated BNP levels accurately detect CHF in 95% to 97% of patients (100 pg/mL). The diagnostic “gray area” is between 100 and 500 pg/mL and anything greater than 500 pg/mL is positive.

■ Are there false positives with elevated BNP levels?
■ Yes

There are disease processes that can falsely elevate BNP levels, such as hypertension, LV hypertrophy, renal failure, and brain injury.

■ What is the primary difference between BNP and NT-ProBNP levels?
■ Half-life of NT-ProBNP is longer than BNP

The half-life of NT-ProBNP is 1 to 2 hours versus 20 minutes with BNP. The NT-ProBNP levels correlate to the NYHA classifications:

- NYHA I = mean 1015
- NYHA II = mean 1666
- NYHA III = mean 3029
- NYHA IV = mean 3465

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MANAGEMENT

■ A patient on an ACE inhibitor is noncompliant. Which side effect of the ACE inhibitor is the most likely cause?
■ Dry, nonproductive cough

Some patients who cough while taking ACE inhibitors have this symptom because of CHF rather than ACE inhibitor intolerance and might improve with further diuresis. Others, develop a dry, hacking cough that can interfere with activities of daily living (i.e., eating, talking, sleeping) and may require changing to an ARB.

▷ HINT
Recognize the drug is an ACE inhibitor when it ends in “-pril.”

■ What electrolyte abnormality is common with an ACE inhibitor?
■ Hyperkalemia

ACE inhibitors promote the excretion of Na⁺ and water, thus increasing the reabsorption of K⁺. Hyperkalemia may occur when patients are on ACE inhibitors, ARBs, and aldosterone antagonists. Serum potassium levels should be monitored closely in HF patients. Diuretics are commonly used in HF and can lead to hypokalemia. Hypokalemia may adversely affect cardiac conduction and lead to arrhythmias and sudden death.

▷ HINT
Remember hypokalemia may increase the risk of digitalis toxicity.

■ Which class of drugs is used if a patient is ACE-inhibitor intolerant?
■ ARB

ARBs allow the conversion of angiotensin I to angiotensin II but block the receptor sites of angiotensin II. These drugs have side effects similar to an ACE inhibitor but do not exhibit the cough or angioedema. Following an episode of angioedema, the patient should have the ACE inhibitor changed to an ARB. ARBs may be used as a first-line treatment with mild to moderate HF and reduced LVEF.

■ A person with symptomatic HF on an ACE inhibitor and β-blocker has persistent symptoms. Which combinations of medications are recommended at this time?
■ Hydralazine and nitrate

The addition of a combination of hydralazine and nitrate is reasonable for patients with reduced LVEF, who are already taking an ACE inhibitor and β-blocker, but have persistent symptoms.

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HINT
The combination of hydralazine and nitrate is also recommended to improve outcomes for African Americans with moderate to severe symptoms.

- Which ECG finding indicates the need for cardiac resynchronization therapy?
- QRS duration 0.12 seconds or longer

Other indications include patients with LVEF 35% or less, sinus rhythm, and NYHA functional Class III or ambulatory IV.

DISSECTING THORACIC AND ABDOMINAL AORTA ANEURYSMS

- What is an aortic dissection?
- Lengthwise separation of the medial layer of the aorta

An aortic dissection involves the medial layer of the aorta. There is a lengthwise separation of the medial layer due to a tear in the intima with intramedial extravasation. Blood flows between the intimal and medial layers creating a double lumen, called a false lumen and a true lumen. Dissection can be acute, present within the first 14 days of initial injury, or chronic, with presentation longer than 14 days.

HINT
A traumatic aortic aneurysm is a disruption of the intimal, medial, and adventitial layers. This is called a transection.

- What happens if blood flows into the false lumen, forming a large hematoma?
- Partial to complete obstruction of the true lumen

Following the intimal tear, blood enters the medial layer and can form a large hematoma, obstructing the true lumen and affecting distal perfusion. Shearing forces can also cause further tears, producing exit sites and flow back into the true lumen (Box 1.16).

HINT
Thoracic aortic aneurysm occurs above the diaphragm and abdominal aortic aneurysm occurs below.

Box 1.16 Sites of Aortic Dissections

<table>
<thead>
<tr>
<th>Ascending aorta</th>
<th>Descending aorta</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aortic arch</td>
<td>Thoracoabdominal aorta</td>
</tr>
</tbody>
</table>

- What genetic disorder of connective tissue resulting in above-average height causes risk for an aortic dissection?
- Marfan’s syndrome

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Marfan’s syndrome is inherited as a dominant trait and symptoms vary from mild to severe. People with Marfan’s syndrome tend to be very tall, with long limbs and fingers. They are at risk for aortic aneurysms and dissections (Box 1.17).

**HINT**

Other connective tissue diseases that may be used in the scenario include Ehlers-Danlos or Loeys-Dietz.

### Box 1.17 Causes/Risk Factors of Aortic Dissention

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Cause/Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension, especially uncontrolled</td>
<td>Chronic corticosteroid or immunosuppressive use</td>
</tr>
<tr>
<td>Pheochromocytoma</td>
<td>Congenital factors (bicuspid valve)</td>
</tr>
<tr>
<td>Cocaine or other stimulant use</td>
<td>Inflammatory vasculitis</td>
</tr>
<tr>
<td>Weight lifting or other Valsalva maneuvers</td>
<td>Atherosclerosis (e.g., peripheral vascular disease [PVD], coronary artery disease)</td>
</tr>
<tr>
<td>Pregnancy</td>
<td>Infections of the vascular wall</td>
</tr>
<tr>
<td>Genetic</td>
<td>Iatrogenic (e.g., aortic valve manipulation)</td>
</tr>
<tr>
<td>Polycystic kidney disease</td>
<td></td>
</tr>
</tbody>
</table>

### PATHOPHYSIOLOGY

- **What leads to the complication of organ ischemia with an aortic dissection?**
  - Obstruction of arterial branches off the aorta

The dissection can propagate through the arteries that branch off of the aorta, leading to stenosis or obstruction. The obstruction of flow leads to end-organ ischemia and failure. Dissections can extend retrograde and antegrade (Box 1.18).

### Box 1.18 Branch Arteries of the Aorta

<table>
<thead>
<tr>
<th>Artery</th>
<th>Branch Arteries</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary</td>
<td>Renal</td>
</tr>
<tr>
<td>Brachiocephalic (e.g., subclavian, carotid)</td>
<td>Visceral (e.g., superior and inferior mesenteric arteries)</td>
</tr>
<tr>
<td>Intercostal</td>
<td></td>
</tr>
</tbody>
</table>

**HINT**

May present with symptoms of end-organ ischemia from loss of blood flow through these arteries.

- **What part of the aorta is involved in a DeBakey Type II dissection?**
  - Ascending aorta

There are two major classification systems used in aortic dissections: DeBakey and Stanford systems. The DeBakey system uses Type I to Type III, whereas the Stanford system uses Type A and B (Box 1.19).
Box 1.19 Classifications of Aortic Dissections

<table>
<thead>
<tr>
<th>DeBakey System</th>
<th>Type I</th>
<th>Type II</th>
<th>Type III</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ascending and also involvement of descending aorta</td>
<td>Ascending aorta only</td>
<td>Only the descending is involved: IIIA: distal to the left subclavian artery to the diaphragm IIIB: descending aorta below the diaphragm</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Stanford System</th>
<th>Type A</th>
<th>Type B</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ascending involved</td>
<td>Descending involved</td>
<td></td>
</tr>
</tbody>
</table>

**SYMPTOMS/ASSESSMENT**

- **How is the pain of an aortic dissection frequently described?**
  - Ripping or tearing sensation

  The quality and severity of pain is frequently used to assist with the diagnosis of a dissection. The pain is initially described as a “ripping or tearing” sensation. It is of abrupt onset and severe in intensity. There is sometimes a “latency” period during which the pain will get better after the initial onset, but then it returns as a “knife-like” severe pain.

  > **HINT**
  
  These patients typically report 10/10 pain despite pain management and will typically be agitated.

- **What specific physical examination should be performed in a suspected thoracic aortic dissection?**

  - Obtain BP in both arms

  Compare BPs obtained in each arm. A high-risk feature would be a discrepancy of systolic BP greater than 20 mmHg. Another assessment includes assessing pulses in the upper limbs compared to the lower limbs. A significant pulse deficit in the lower extremity should also increase level of suspicion (Box 1.20).

  > **HINT**
  
  An associated new-onset diastolic murmur (aortic regurgitation) may indicate dissection of the ascending aorta.

**Box 1.20 Signs of Aortic Dissection**

<table>
<thead>
<tr>
<th>Sign</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest or abdominal pain (refers to the back)</td>
<td>Bruit (carotid, brachial, femoral)</td>
</tr>
<tr>
<td>Tracheal compression</td>
<td>Focal neurological deficits</td>
</tr>
<tr>
<td>Laryngeal hoarseness (pressure on recurrent laryngeal nerve)</td>
<td>Hemothorax</td>
</tr>
<tr>
<td>Dysphagia</td>
<td>Anxiety and premonition of death</td>
</tr>
<tr>
<td>Abdominal mass (pulsating)</td>
<td>Fever</td>
</tr>
<tr>
<td>Diastolic murmur (high-pitched blowing)</td>
<td>Hypertension</td>
</tr>
<tr>
<td>Pulse deficit</td>
<td>Manifestations of pericardial tamponade</td>
</tr>
<tr>
<td>Syncope</td>
<td></td>
</tr>
</tbody>
</table>
What are the three signs of Horner’s syndrome?
Ptosis, miosis, and anhidrosis

Horner’s syndrome is caused by an interruption in the cervical sympathetic ganglia and manifests as ptosis, miosis, and anhidrosis. This can occur with a dissection of the aortic arch.

HINT
Horner’s syndrome is the loss of sympathetic nervous system innervation.

DIAGNOSIS

What diagnostic test is best used in an unstable patient with suspected aortic dissection?
Echocardiogram

A chest x-ray (CXR) is frequently the initial evaluation but may not reveal any significant findings. A computed tomography (CT) scan is used in hemodynamically stable patients, whereas an echocardiogram is preferred if unstable. It is performed at the bedside, is rapid and noninvasive (unless a transesophageal echocardiogram [TEE] is used, which is minimally invasive). An emergency CT angiography with 3-D reconstruction is being used to obtain a view of the aorta without the potential complications of the more invasive aortogram.

HINT
The gold standard is still considered to be the aortogram for diagnosis of aortic dissection.

The aortogram can pinpoint the site of intimal tear, the true and false lumen entry site, appearance of dye outside of the aorta, and bulging of aorta.

What is the disadvantage of a TEE in diagnosing aortic dissection?
Limited ability to visualize the distal ascending aorta

TEE has limited ability to visualize the distal ascending aorta and proximal arch because of the air-filled trachea and main stem bronchus.

MEDICAL MANAGEMENT

What class of drugs is recommended for the initial management of a thoracic aortic dissection?
β-blocker

The initial treatment goal is to decrease the aorta wall stress by slowing the heart rate and lowering the BP. Esmolol (Brevibloc) is frequently used in acute management. If contraindicated to use a β-blocker, the second choice of drugs is nondihydropyridine calcium channel-blocking agents, which should be utilized as an alternative for rate control. β-blockers should be used cautiously in acute aortic regurgitation due to the block on compensatory tachycardia.

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Vasodilator therapy, such as Nipride, should not be used until after heart rate control has been achieved to avoid reflex tachycardia.

What is the target BP and heart rate in an acute aortic dissection?
- Systolic rate between 110 and 120 mmHg and heart rate between 60 and 80 bpm

During an acute aortic dissection, aggressive management of BP and heart rate should be initiated to lower the intraluminal pressure to limit extension of dissection. The end-organ perfusion also needs to be evaluated when managing BP to prevent hypoperfusion.

Most patients will be hypertensive; a hypotensive presentation may indicate pericardial tamponade or hemorrhage into the pleural or retroperitoneal space.

SURGICAL MANAGEMENT
- When is surgical repair recommended in an asymptomatic patient with a descending aortic aneurysm?
- Aneurysm greater than 5.5 cm

A patient with a stable chronic dissection may be observed for signs of progressive enlargement of the aorta. Surgical intervention is recommended with an aneurysm in the descending aorta greater than 5.5 cm. It is reasonable to follow up with CT scans or ultrasonography every 12 months to evaluate the size of the aneurysm, if less than 5.5 cm. Other indications for surgery include a saccular aneurysm or postsurgical pseudoaneurysm.

If the patient is symptomatic with acute dissection of any size, surgical intervention may be recommended.

What technique is used in the operating room to protect organs from ischemia during repair of the aorta?
- Cardiopulmonary bypass (CPB)

Other correct answers include partial left heart bypass, deep hypothermic circulatory arrest, and retrograde perfusion. Goals of surgery include repair of aorta as well as prevention of ischemic insult to distal organs. Surgical procedures on aorta can include intimal flap repair, removal of thrombosis and false lumen, replacement of dilated aorta with a graft, repair of aortic root, and replacement of aortic valve. Endovascular repair may also be used in descending aortic dissections through femoral access.
HINT
CPB requires heparinization. Postoperative management includes assessing for signs of coagulopathy and continued reversing of heparin.

What technique may be used to protect the spinal cord during surgical or endovascular repair of descending aorta?
Cerebrospinal fluid (CSF) drainage

CSF drainage is recommended in patients with high risk for spinal cord ischemia during surgical repair of the descending aorta. They may also use other spinal cord perfusion techniques such as proximal aortic pressure maintenance and distal aortic perfusion to optimize spinal cord perfusion (Box 1.21).

Box 1.21 Other Treatments Used in the Prevention of Spinal Cord Ischemia

<table>
<thead>
<tr>
<th>Intraoperative systemic hypothermia</th>
<th>Intrathecal papaverine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epidural irrigation with hypothermic solution</td>
<td>Metabolic suppression with anesthetic agents</td>
</tr>
<tr>
<td>High-dose glucocorticosteroids</td>
<td>Reimplantation of intercostal arteries</td>
</tr>
<tr>
<td>Osmotic diuretic (i.e., Mannitol)</td>
<td>Distal perfusion</td>
</tr>
</tbody>
</table>

HINT
A postoperative patient should be assessed for any motor or sensory abnormalities.

What is the priority in the postoperative care of an aortic repair with graft placement?
BP management

BP management is important in postoperative (as well as preoperative) care to prevent disruption of the graft, dissection, and hemorrhage. Antihypertensive agents used preoperatively may be continued in the postoperative period. Hemodynamic monitoring, prevention of fluid overload, correct coagulopathy, and administration of antibiotics to prevent graft infections are components of postoperative management.

Where does an embolism from catheter manipulation during interventional graft placement typically travel?
Legs and viscera

The internal surface of the aorta can be covered by atheromas. Manipulation of the catheter during interventional stent placement can break loose debris and become a distal embolism. This may involve the lower extremities or the abdominal viscera. Another complication can be migration of the graft.
HINT
May involve the renal, mesenteric, or iliac arteries. Assess for signs of organ hypoperfusion postprocedure.

COMPLICATIONS

What part of the aorta is at the highest risk for aortic rupture?

Ascending aorta

A high-risk aortic injury for rupture involves the ascending portion of the aorta and should be referred for emergent surgery to prevent life-threatening complications.

HINT
Ascending aortic dissections can dissect through the aortic valve resulting in acute aortic regurgitation and HF.

Three days after the repair of a descending aortic dissection (Type B) with a graft placement, a patient develops severe abdominal pain and shows occult blood in stool. What is the most likely cause for the abdominal pain?

Bowel ischemia/infarction

Dissection of the aorta can involve the branch arteries. The superior and inferior mesenteric arteries may be involved in a descending aortic dissection. Signs of malperfusion of the bowel with ischemia and infarction include new-onset severe abdominal pain, elevated lactate and CPK levels, and sometimes the presence of occult blood in stool. The diagnostic procedure is typically abdominal CT.

HINT
Most postoperative complications will involve the malperfusion of organs from branch artery occlusion (Box 1.22).

Box 1.22 Complications

| Perioperative MI | Graft occlusion |
| Stroke | Arterial embolism to extremities |
| Hypertension | Aortic thrombosis/stenosis |
| Low cardiac output syndrome | Wound infection |
| Renal failure | Bowel ischemia |
| Dysrhythmias | Paraplegia |
| Coagulopathies/disseminated intravascular coagulopathy (DIC) | Hemorrhage (retroperitoneal, intraperitoneal) |
| Graft infections | |

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PERICARDIAL TAMPONADE

What is the most common mechanism for a traumatic pericardial tamponade?
■ Penetrating injury

Pericardial tamponade is caused by bleeding into the pericardial sac due to a ruptured coronary artery, lacerated pericardium, or an injury to the myocardium.

Pathophysiology

Is pericardial tamponade considered a diastolic or systolic dysfunction?
■ Diastolic dysfunction

The pericardial sac usually contains 25 to 50 mL of fluid. Following an injury, blood accumulates within the pericardial sac, causing a constriction on the heart. The pericardial pressure becomes higher than the ventricular filling pressures, interfering with the ability of the ventricles to fill with blood (diastolic phase). The amount of blood required to impair filling depends upon the rate of the accumulation of blood and the compliance of the pericardial sac.

HINT
Pericardial tamponade is a constrictive cardiomyopathy.

Symptoms/Assessment

What is the first sign of a traumatic pericardial tamponade?
■ Tachycardia

A significant decrease in ventricular filling results in a decrease in SV. Tachycardia is a compensatory mechanism, an attempt to maintain a normal CO.

HINT
Suspect a pericardial tamponade in chest trauma if shock symptoms are unresponsive to fluid administration.

What is the Beck’s triad?
■ Increased jugular venous distention, hypotension, and muffled heart sounds

The pressure caused by the blood in the pericardial sac limits the filling of the heart, backing the blood up into the venous circulation. This produces jugular venous distention. The decreased filling results in a decrease in SV leading to hypotension. The accumulation of blood in the pericardial sac muffles the heart sounds. The presentation of a classical Beck’s triad occurs with an acute cardiac tamponade.

HINT
Look for distended jugular veins as a clue of the elevated central venous pressure. Trauma patients typically have flat neck veins due to hypovolemia (Box 1.23).

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Other Symptoms of Pericardial Tamponade

- Dyspnea
- Cyanosis
- Diaphoresis
- Cold, clammy skin
- Pulsus paradoxus
- Pericardial friction rub
- Agitation
- Feelings of impending doom

HINT
Scenario of a penetrating chest wound in which the patient insists on sitting bolt upright, is agitated and confused, or has air hunger is more likely to be a condition of pericardial tamponade.

What is the most common type of cardiac arrest?
- PEA

Pulseless electrical activity (PEA) occurs in cardiac tamponade due to the constriction interfering with the mechanical activity of the heart but the electrical activity continues.

HINT
Cardiac tamponade would be one of the differential diagnoses of PEA in a trauma patient.

Diagnosis

What CXR changes would you expect to find in a pericardial tamponade?
- Widened mediastinum (enlarged cardiac silhouette)

The heart may also have the appearance of a water-bottle shape. Not all patients with a traumatic pericardial tamponade will demonstrate CXR findings and therefore it should not be used alone to rule out the injury. A CT scan may also be used to identify pericardial fluid but only on a stable patient.

Which diagnostic test is frequently used in the emergency room to screen for a pericardial tamponade?
- Ultrasonography

Focused assessment sonography for trauma (FAST) is used to assess the abdomen and may frequently be used to assess the chest for pericardial tamponade. Ultrasonography is one of the most important tools for recognizing a tamponade. The classical pattern is a “swinging” heart. The heart oscillates within the pericardium side to side (this produces the pulsus alternans seen on the ECG). It may also detect pericardial fluid, thrombus, and collapsing of the ventricular wall during diastole.

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1. CARDIOVASCULAR SYSTEM REVIEW

Medical Management

- **A patient is found to have a pericardial tamponade following a stab wound to the anterior chest. He is hypotensive. What would be the immediate medical management of this patient?**
- **Oxygen, fluid bolus, inotropic agent**

Remember the ABCs of trauma. Oxygen is administered and pulse oximetry should be monitored. The effects of hypovolemia are profound in a cardiac tamponade and fluid bolus may be used to increase SV and perfusion. Volume overload may worsen the ventricular contractility and should be avoided. Passive elevation of the legs may also be used to increase venous return and improve ventricular filling. Positive inotropic drugs (i.e., dobutamine) may improve contractility without increasing SVR.

**HINT**
Avoid positive pressure ventilation, if possible. If intubated and ventilated, minimize positive end-expiratory pressure (PEEP) levels.

Surgical Management

- **What is the definitive care for a pericardial tamponade?**
- **Remove the blood/fluid from the pericardial space**

This can be done with an emergency subxiphoid percutaneous aspiration in an unstable patient in the emergency department. Pericardiocentesis can also be performed using landmarks or guided by echocardiogram and by placing a drain tube. Pericardotomy can also be performed using a balloon to create the pericardial window. Open thoracotomy may be required in some cases. A minimally invasive technique commonly used is the video-assisted thorascopic (VAT) procedure.

- **What is a life-threatening complication of a pericardiocentesis?**
- **Puncture and rupture of the myocardium or coronary arteries**

Other complications include arrhythmias, puncture of the lungs, liver, or stomach. Continuous ECG monitoring to detect ventricular arrhythmias is recommended.

MYOCARDIAL CONTUSION

Pathophysiology

Myocardial contusion (MC) is a hemorrhage within the myocardium, marked by cellular injury and extravasation of red blood cells (RBCs) into the muscle fibers. Contusion severity ranges from subepicardial to intramural hemorrhage into the intraventricular septum. The hemorrhage can extend up to varying depths into the myocardium. During recovery, the healing occurs with scar formation.
Which ventricle is the most susceptible to injury following a blunt chest trauma?

RV

The RV is the most vulnerable because of its location under the sternum. The mitral and aortic valves are more likely to be injured than the tricuspid and pulmonic because of higher pressures in the LV.

Symptoms/Assessment

What is the most common patient complaint of an MC?

Chest pain

The chest pain is precordial and is frequently unrelieved by analgesics. The symptoms and presentation vary following a myocardial contusion. Some patients may present without symptoms.

What is the most common arrhythmia following an MC?

Sinus tachycardia

Following a trauma, most patients will present with sinus tachycardia, which is a sympathetic nervous system response. The most lethal arrhythmias of a myocardial contusion are ventricular tachycardia and VF. Other arrhythmias include AF, heart blocks, right bundle branch block, and right bundle branch block with hemiblock. The severity of the arrhythmia does not correlate with the severity of the contusion (Box 1.24).

HINT

Tachycardia beyond expectation for hypovolemia based upon calculated blood loss is a high suspicion for MC.

Box 1.24 Other Symptoms of MCs

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bruising on chest wall</td>
<td>Pericardial friction rub/murmurs</td>
</tr>
<tr>
<td>Crackles</td>
<td>Presence of associated injuries</td>
</tr>
<tr>
<td>S3 gallop</td>
<td>Hypotension</td>
</tr>
</tbody>
</table>

When does a patient require cardiac monitoring?

Abnormal echocardiogram or ECG

Monitoring is not required if both echocardiogram and ECG are normal in a hemodynamically stable patient.

Diagnosis

Which of the cardiac markers is most specific to injury caused by MC?

Troponin levels
CPK-MB levels will elevate following skeletal muscle trauma and myocardial injury and are nonspecific for myocardium. Troponin levels are more specific to myocardial injury.

- An echocardiogram is used to identify which specific change found in an MC?
- Abnormal wall motion

Transthoracic and esophageal echocardiograms can be used to identify abnormalities in cardiac function that can be used in diagnosing myocardial contusions. It is also used to identify patients requiring cardiac monitoring and the presence of complications, such as pericardial effusion. The abnormal wall motion results in a decrease in SV, CO, and BP.

**Management**

- Within what time period following an MC would arrhythmias most commonly occur?
- Within 24 to 48 hours

A patient sustaining a blunt chest trauma with a high suspicion of MC should have cardiac monitoring for 24 to 48 hours. Other management issues include hemodynamic stabilization and treatment of associated injuries.

**Complications**

- Following a blunt chest trauma, a patient develops a new-onset systolic murmur. Which valve abnormality is most likely the cause?
- Mitral regurgitation

The two most common valves to be damaged following a blunt chest injury are the mitral and aortic valves. A rupture (regurgitation) of the mitral valve results in a systolic murmur, whereas a rupture of the aortic valve would cause a diastolic murmur (Box 1.25).

**HINT**

Valve regurgitation causes a murmur to be heard during the cardiac cycle when the valve should be closed. The mitral valve should be closed during systole (systolic murmur) and the aortic valve must close during diastole (diastolic murmur).

**Box 1.25 Complications of MCs**

<table>
<thead>
<tr>
<th>Arrhythmias</th>
<th>Cardiac rupture</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV dysfunction with CHF</td>
<td>Ventricular thrombosis</td>
</tr>
<tr>
<td>Acute valvular regurgitation (valve rupture)</td>
<td>Chronic constrictive pericarditis</td>
</tr>
<tr>
<td>Ventricular aneurysm</td>
<td>Coronary vasospasm, thrombosis, or rupture</td>
</tr>
<tr>
<td>Pericardial effusion (with or without tamponade)</td>
<td>Atrial fistula</td>
</tr>
<tr>
<td>Intracardiac structural damage</td>
<td></td>
</tr>
</tbody>
</table>
TRAUMATIC AORTIC ANEURYSM

Traumatic aortic aneurysm is a cause of death at the scene due to a completely transected aorta (intimal, medial, and adventitia layers). If the patient survives to the emergency department, he or she will usually have a small tear or a partial-thickness tear of the aorta forming an aneurysm.

Pathophysiology

■ A traumatic aortic aneurysm is most likely to be caused by what mechanism of injury?

■ Sudden deceleration

A sudden deceleration may be either horizontal (i.e., high-speed motor vehicle crash [MVC]) or vertical (i.e., fall). The aorta is relatively mobile in the chest and will continue to travel after the sudden deceleration except where it is secured by a ligament.

➤ HINT

The scenario given will usually be a high-speed MVC with a sudden deceleration on impact.

■ What is the most common site of the aorta for a traumatic aortic aneurysm?

■ Level of the isthmus

The level of the isthmus is distal to the great vessels, where the aorta begins to descend. The ligamentum of arteriosum secures the proximal descending thoracic aorta, just distal to the arch (at the level of the isthmus). During the sudden deceleration, this ligament holds the aorta back while the arch of the aorta continues to travel. This causes the aorta to transect at the level of the isthmus. Other sites of fixation include the ascending aorta, the aortic root, and the diaphragmatic hiatus.

Symptoms/Assessment

■ What is the classic sign of a traumatic aortic aneurysm?

■ Widened mediastinum on CXR

Patients may be relatively asymptomatic for a traumatic aortic aneurysm. The CXR is used as a screening device. The patient may also present as hemodynamically unstable if there is bleeding into the thoracic cavity or in the presence of associated injuries. The patient may present with a cyclic pattern of responding to a fluid bolus, then becoming hypotensive again (Box 1.26).

➤ HINT

Hemodynamic stability needs to be determined to guide management of these patients.
1. CARDIOVASCULAR SYSTEM REVIEW

Box 1.26 Other Symptoms of Aortic Aneurysm

| Retrosternal or intracapsular chest pain | Swelling at base of neck |
| Hoarseness (tracheal compression)       | Paraplegia               |
| Dysphagia (esophageal compression)      | Pseudocoarctation syndrome |
| Systolic murmur over base of neck       |                           |

What are the signs of pseudocoarctation syndrome?

Hypertension in upper extremities and hypotension in lower extremities

The patient may present with bounding pulses in the upper extremities and hypertension. The pulses are diminished in the lower extremities and hypotensive. This is due to the formation of a hematoma, narrowing the lumen of the aorta. The narrowing creates a high pressure above and low pressure below the site. This can occur following a traumatic transection but is not common.

HINT
The question may also have the nurse check BP in both arms, assessing for a significant difference in BP between the two.

A chest tube is placed for left-sided hemothorax in a trauma patient. What finding following chest tube placement is a sign of an aortic injury?

Large volume of bright-red blood from the chest tube

A rupture of the aorta frequently results in bleeding into the left pleural space and a resulting hemothorax. The sign is a large-volume hemorrhage from the chest tube, typically bright-red blood.

Diagnosis

What are three findings on a CXR that would lead to suspicion of a thoracic aortic aneurysm?

Widened mediastinum, loss of aortic knob, or left apical cap

A widened mediastinum is the most classic sign found on a CXR. The loss of the aortic knob (also called superior widened mediastinum) and an apical cap may also be commonly seen in a thoracic aortic aneurysm. Other CXR signs include a deviated nasogastric (NG) tube to the right, an obvious double lumen contour of the aorta, or depression of the left-stem bronchus.

Which radiographic procedure has the most definitive diagnosis for a thoracic aortic aneurysm?

Arteriogram

The CXR is used as a screening device and is used in combination with the mechanism of injury to warrant further workup for an aneurysm. A CT scan is the best screening tool. The arteriogram has been the gold standard for diagnosing an aortic injury but is being
replaced with a multidetector helical CT scan. An older CT scan using a single slice helical view is not adequate to plan surgery by itself. If the multidetector CT scan findings are equivocal or do not visualize branch vessels or surrounding structures, an angiogram is used as a follow-up prior to surgery.

› HINT
If the question asks for the gold standard, “arteriogram” as the answer.

**Medical Management**

- **At what level should the systolic blood pressure (SBP) be maintained in a patient with thoracic aortic aneurysm?**
- **Between 90 and 120 mmHg**

Allowing an increase in SBP above 120 mmHg will increase the risk of free rupture of the aneurysm. Lowering the SBP below 90 mmHg will cause hypoperfusion and may contribute to organ dysfunction. Short-acting antihypertensives may be used in the acute period to maintain BP within the acceptable range.

› HINT
Avoid aggressive fluid resuscitation even in hemodynamically unstable patients due to risk of rupture.

**Surgical Management**

- **What is the definitive management of an aortic aneurysm?**
- **Graft replacement of the aorta**

Open thoracotomy is the most commonly used route to repair an aortic injury from a trauma. The endovascular route has also been used. Surgery may involve placing the patient on CPB or partial left heart bypass to maintain perfusion distal to the area of injury (Box 1.27).

**CARDIOMYOPATHIES**

- **What type of cardiomyopathy has a high incidence of SCD due to lethal arrhythmias?**
- **Hypertrophic cardiomyopathy**

The hypertrophied LV wall places the patient at risk for increased myocardial oxygen consumption and fatal arrhythmias. The thickening of the ventricular wall is called remodeling.
1. Cardiovascular System Review

**HINT**
A hypertrophic cardiomyopathy is an indication for the placement of automatic internal cardiac defibrillator (AICD).

- **What are the two classifications of hypertrophic cardiomyopathy?**
  - Obstructive and nonobstructive

The obstructive hypertrophic cardiomyopathy has an obstruction to outflow from the LV in combination with ventricular hypertrophy. The obstruction to outflow is due to the enlarged septal wall causing the mitral valve to interfere with outflow during mid-systole.

- **Your patient has a history of chronic alcoholism. Which type of cardiomyopathy would you suspect due to his history?**
  - Dilated cardiomyopathy

Dilated cardiomyopathy is the most common non-ischemic cardiomyopathy. It is classified as a systolic dysfunction (Box 1.28).

**Box 1.28 Causes of Dilated Cardiomyopathy**

<table>
<thead>
<tr>
<th>Cause</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peripartum</td>
<td>Volume overload</td>
</tr>
<tr>
<td>Viral infection</td>
<td>Chemotherapeutic agents</td>
</tr>
<tr>
<td>Alcohol-induced</td>
<td>Idiopathic/genetic</td>
</tr>
<tr>
<td>Cocaine-induced</td>
<td></td>
</tr>
<tr>
<td>Ischemia/infarction (previous</td>
<td>myocaridal infarction)</td>
</tr>
</tbody>
</table>

- **Which cardiomyopathy demonstrates a reduced diastolic volume but near-normal wall thickness?**
  - Restrictive cardiomyopathy

A restrictive cardiomyopathy is characterized by restrictive filling and reduced diastolic volume of either or both ventricles with normal to near-normal systolic function and wall thickness. A restrictive cardiomyopathy needs to be differentiated from a constrictive cardiomyopathy, which may be curable with surgical intervention. A restrictive cardiomyopathy is the least common form of cardiomyopathy.

**HINT**
A constrictive cardiomyopathy is caused by constriction around the heart (e.g., pericardial tamponade), whereas a restrictive cardiomyopathy is caused by stiffness of the ventricles.

- **What causes the ventricles to become noncompliant or stiff in a restrictive cardiomyopathy?**
  - Interstitial fibrosis or amyloid deposits

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A restrictive cardiomyopathy is characterized by intracellular accumulation of amyloid material sufficient to impair myocardial function. Deposits of protein fibrils throughout the myocardium create a rubbery consistency of the ventricular wall. Amyloid deposits are the most common cause (Box 1.29).

**HINT**

On autopsy, in a restricted cardiomyopathy, the heart does not collapse when removed from the chest cavity.

**Box 1.29 Causes of Restrictive Cardiomyopathy**

<table>
<thead>
<tr>
<th>Idiopathic Eosinophilic fibrosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart muscle disease</td>
</tr>
<tr>
<td>Amyloidosis</td>
</tr>
<tr>
<td>Hemochromatosis</td>
</tr>
<tr>
<td>Malignancy</td>
</tr>
</tbody>
</table>

**PATHOPHYSIOLOGY**

**In an obstructive hypertrophic cardiomyopathy, what causes the obstruction to outflow tract from the LV?**

**Leaflet of the mitral valve**

During systole, there is an anterior motion of the mitral valve toward the hypertrophied septal wall. This abnormal motion of the mitral valve results in further narrowing of the outflow tract of the aortic valve. The outflow tract may already be narrowed from the hypertrophied septal wall.

**Which cardiomyopathy is associated with a high EF?**

**Hypertrophic cardiomyopathy**

Hypertrophic cardiomyopathy is characterized by increased contractility due to increased ventricular muscle mass. This increase in contractility results in near emptying of the LV at the end of systole.

**HINT**

Hypertrophic cardiomyopathy is the only cardiomyopathy with a high EF, whereas the others have a low EF.

**Is hypertrophied cardiomyopathy a diastolic or systolic dysfunction?**

**Diastolic dysfunction**

The LV chamber is small, decreasing the preload capability, and limiting the filling of the ventricle during diastole. The hypertrophic LV has increased contractility without significant systolic dysfunction.

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1. CARDIOVASCULAR SYSTEM REVIEW

What effect does a dilated cardiomyopathy have on the CO?

- Decreases the CO

Dilated cardiomyopathy has a decrease in CO and an increase in pulmonary pressures caused by abnormal contractility, volume overload, and HF. It is characterized by an increase in end-diastolic and end-systolic volumes with a low EF.

HINT

Dilated cardiomyopathies are classified as a systolic dysfunction in HF.

Progressive dilation of the LV can lead to which valve abnormalities?

- Mitral or aortic regurgitation

The dilation of the LV stretches the leaflets of the valve, resulting in loss of integrity of the aortic and mitral valves. The regurgitation across these valves contributes to volume overload and further dilation of the ventricle (Box 1.30).

Box 1.30 Comparison of LV Dysfunction Cardiomyopathies

<table>
<thead>
<tr>
<th>Type of Dysfunction</th>
<th>Hypertrophy Cardiomyopathy</th>
<th>Dilated Cardiomyopathy</th>
<th>Restricted Cardiomyopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic dysfunction</td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diastolic dysfunction</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
</tbody>
</table>

SYMPTOMS/ASSESSMENT

What is the most common presenting symptom of a hypertrophied cardiomyopathy?

- Dyspnea

Dyspnea is caused by elevated diastolic pressures (impaired diastolic compliance) and may occur in about 90% of people with hypertrophied cardiomyopathy.

What is the most common cause of SCD in hypertrophied cardiomyopathies?

- VF

VF accounts for about 80% of SCDs of hypertrophied cardiomyopathies. Atrial arrhythmias (i.e., AF, paroxysmal supraventricular tachycardia, Wolff–Parkinson–White [WPW] syndrome) may degenerate to VF as well.

Which cardiomyopathy frequently produces an S4 gallop?

- Hypertrophy cardiomyopathy

An S₄ is produced during atrial contraction against a noncompliant hypertrophied ventricle. A dilated cardiomyopathy is more likely to produce an S₄ due to volume overload. Atrial contraction against a noncompliant ventricle can also produce a double apical impulse and a double carotid arterial pulse.
HINT
Remember, an S₃ occurs during passive filling of the ventricle and an S₄ occurs during the atrial kick. An S₃ is commonly caused by volume overload and an S₄ occurs with ventricle noncompliance.

What is a common 12-lead ECG finding in hypertrophied cardiomyopathy?

Left-axis deviation

The left-axis deviation is caused by the thicker LV wall mass. Other ECG changes include ST-T-wave changes, prolonged PR interval, sinus bradycardia, and atrial enlargement.

HINT
A LBBB with a right-axis deviation is suggestive of a dilated cardiomyopathy.

What is the most common arrhythmia associated with a restrictive cardiomyopathy?

Complete heart blocks and AF

The amyloid and fibrous deposits within the SA and AV node can result in a complete heart block. Amyloid deposits within the bundle branches are rare. Ventricular arrhythmias are not as common as atrial arrhythmias (Box 1.31).

HINT
SCD with restrictive cardiomyopathy is usually caused by PEA.

Box 1.31 Comparative Chart: Symptoms of Cardiomyopathies

<table>
<thead>
<tr>
<th>Hypertrophic Cardiomyopathy</th>
<th>Dilated Cardiomyopathy</th>
<th>Restrictive Cardiomyopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical findings</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sudden cardiac death</td>
<td>Atrial fibrillation</td>
<td>Fatigue</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>Dyspnea</td>
<td>Shortness of breath</td>
</tr>
<tr>
<td>Presyncope/syncope</td>
<td>Chest pain</td>
<td>Peripheral edema (pitting)</td>
</tr>
<tr>
<td>Fatigue</td>
<td>Syncope (due to arrhythmia)</td>
<td>Abdominal ascites</td>
</tr>
<tr>
<td>Angina</td>
<td>Jugular venous distension (JVD)</td>
<td>Chest pain</td>
</tr>
<tr>
<td>Palpitations</td>
<td>Tachycardia (loss of parasympathetic control)</td>
<td>Syncope (due to low CO syndrome)</td>
</tr>
<tr>
<td>Orthopnea and paroxysmal nocturnal dyspnea</td>
<td></td>
<td>Pleural effusions</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td></td>
<td>Hepatomegaly (splenomegaly rare)</td>
</tr>
<tr>
<td>Dizziness</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Heart sounds</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Split S₃</td>
<td>S₃ gallop</td>
<td>Loud S₃</td>
</tr>
<tr>
<td>S₃ and S₄ gallop</td>
<td></td>
<td>Rare S₄</td>
</tr>
<tr>
<td>Systolic murmur</td>
<td></td>
<td>Systolic murmur</td>
</tr>
<tr>
<td>(mitral regurgitation)</td>
<td></td>
<td>(mitral and tricuspid regurgitation)</td>
</tr>
<tr>
<td>Diastolic decrescendo murmur (aortic regurgitation)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
What abnormal lab value is a predictor of poor outcomes in dilated cardiomyopathy?

Hyponatremia

Hyponatremia parallels severity of HF. It is due to release of antidiuretic hormone (ADH) and volume overload (dilutional hyponatremia).

**DIAGNOSIS**

Which lab value is found elevated in a dilated cardiomyopathy?

BNP

BNP levels are increased in dilated cardiomyopathy due to the overstretched ventricle. Restrictive cardiomyopathy will also significantly elevate BNP levels.

▶ HINT

BNP levels will be normal in constrictive cardiomyopathy but grossly elevated in a restrictive cardiomyopathy.

In a restrictive cardiomyopathy, how would the atria appear on an echocardiogram?

Dilated bilateral atria

The ventricles are noncompliant and restrictive to filling so blood backs up in the atria, causing dilation of both the right and left atria. The echocardiogram would also show bilateral ventricular thickening with restrictive filling patterns, normal systolic function and EF (until later in the disease), and abnormal myocardial texture (amyloid deposits).

What is the confirmation diagnosis of a restrictive cardiomyopathy due to amyloid deposits?

Cardiac biopsy

A cardiac biopsy is used to confirm the diagnosis. A fine-needle aspiration of abdominal fat may also be used and is easier and safer for the diagnosis of amyloidosis. A liver biopsy is performed to diagnosis hemochromatosis (another cause of restrictive cardiomyopathy).

**MEDICAL MANAGEMENT**

Which antiarrhythmic agent has been found to lower the incidence of arrhythmogenic SCD in hypertrophied cardiomyopathy?

Amiodarone (Cardarone)

Amiodarone is the only agent proven to reduce the incidence and risk of SCD, with or without obstruction to LV outflow. It is very effective at converting AF and flutter to sinus rhythm and at suppressing the recurrence of these arrhythmias. Disopyramide (Norpace) may be used to raise the atrial and ventricular arrhythmia threshold but is not recommended without concomitant β-blockade.

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HINT
If Norpace is used, monitor QTc interval.

Is the goal in managing hypertrophic cardiomyopathy to increase or decrease the inotropic state of the LV?

Decrease the inotropic state

Hypertrophied cardiomyopathy is a diastolic dysfunction with a small LV chamber. An increase in the inotropic state of the LV will further limit the size of the LV chamber and filling capabilities. Agents that decrease the inotropic state of the LV and result in relaxation of the LV chamber are indicated. First-line agents include β-blockers to titrate the heart rate to 60 to 65 bpm.

HINT
β-blockers have also been shown to reduce the gradient across the LV outflow tract.

Which type of calcium channel blocker would be contraindicated in managing hypertrophied cardiomyopathy?

Dihydropyridine calcium channel blockers

An example of a dihydropyridine calcium channel blocker is nifedipine (Procardia). Cardiac glycosides (Digoxin) and other positive inotropic agents should also be avoided in patients with hypertrophic cardiomyopathy. The positive inotropic effect will increase the contractility and make the LV chamber size even smaller. This will worsen the filling capability of the ventricles, thus worsening CO and HF.

HINT
Verapamil (Calan) is an L-type calcium channel blocker and is indicated if β-blockade is not effective.

What is the indication for anticoagulation therapy in dilated cardiomyopathy?

Severe LV dysfunction or at risk of AF

Dilated cardiomyopathy presents as LV systolic dysfunction. As the LV dysfunction becomes severe with a low EF, a thrombus can form in the LV. Anticoagulation therapy is recommended to prevent a cardioembolic stroke or pulmonary embolism (from the RV). Dilated cardiomyopathy patients are at risk for the development of AF and should also be anticoagulated (Box 1.32).
1. CARDIOVASCULAR SYSTEM REVIEW

Box 1.32 Pharmacological Management of Cardiomyopathies

<table>
<thead>
<tr>
<th>Hypertrophic Cardiomyopathy</th>
<th>Dilated Cardiomyopathy</th>
<th>Restricted Cardiomyopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amiodarone (Cordarone)</td>
<td>ACE inhibitor</td>
<td>Diuretics</td>
</tr>
<tr>
<td>β-blocker</td>
<td>β-blocker</td>
<td>Nitrates</td>
</tr>
<tr>
<td>Ca²⁺ channel blocker (L-type only)</td>
<td>Angiotensin receptor blocker</td>
<td>Anticoagulation therapy (AF)</td>
</tr>
<tr>
<td>Anticoagulation therapy (AF)</td>
<td>Cardiac glycosides</td>
<td>Antiplasma cell therapy</td>
</tr>
<tr>
<td></td>
<td>Diuretics</td>
<td>Corticosteroids</td>
</tr>
<tr>
<td></td>
<td>β-blocker</td>
<td>Interferon</td>
</tr>
<tr>
<td></td>
<td>Nitrates</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Diuretics</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Angiotensin receptor blocker</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cardiac glycosides</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Diuretics</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Antiarrhythmic</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Vasodilator</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Aldosterone antagonist</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Inotrope agents</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Anticoagulation therapy (AF)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Nesiritide (Natrecor)</td>
<td></td>
</tr>
</tbody>
</table>

SURGICAL MANAGEMENT

- **What is the surgical option for a restrictive cardiomyopathy?**
- **Cardiac transplant**

A cardiac transplant may be considered if symptoms are refractory to treatment in idiopathic, familial, and amyloidosis cases of restrictive cardiomyopathy.

- **What is the surgical option for an obstructive hypertrophic cardiomyopathy?**
- **LV myomectomy**

An LV myomectomy is a procedure to remove the septal muscle, thus managing the obstruction to outflow in a hypertrophic cardiomyopathy. LV myomectomy is indicated for patients with severe symptoms refractory to therapy and outflow gradient greater than 50 mmHg. It is usually successful in abolishing the outflow gradient and can provide symptomatic relief for at least 5 years. The gradient outflow may increase gradually over time and return to the same level as before, requiring a repeat procedure. Other options to manage the outflow obstruction include mitral valve replacement, transcatheter septal alcohol ablation, and a dual-chamber pacemaker.

> **HINT**

A patient with hypertrophic cardiomyopathy will have an implanted cardioverter-defibrillator (ICD) to prevent SCD.

COMPLICATIONS

- **What is the primary complication of all of cardiomyopathies?**
- **HF**

Whether the cardiomyopathy is hypertrophied, dilated, restrictive, or constrictive, HF is the common complication. AF can also complicate all types of cardiomyopathies (Box 1.33).
Box 1.33 Other Complications of Cardiomyopathies

<table>
<thead>
<tr>
<th>Hypertrophic Cardiomyopathy</th>
<th>Dilated Cardiomyopathy</th>
<th>Restrictive Cardiomyopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCD (due to arrhythmias)</td>
<td>Hypertrophy (remodeling)</td>
<td>MI</td>
</tr>
<tr>
<td></td>
<td>Mitral and tricuspid regurgitation</td>
<td>Low output syndrome</td>
</tr>
<tr>
<td></td>
<td>Pulmonary embolism</td>
<td></td>
</tr>
</tbody>
</table>

HYPERTENSIVE CRISIS

■ What BP is considered to be a hypertensive crisis?
■ Systolic greater than 180 mmHg and diastolic greater than 110 mmHg

This definition of hypertensive crisis is derived from the seventh report of the Joint National Committee (JNC) on prevention, detection, evaluation, and treatment of high blood pressure in 2003. About 1% of people with hypertension will experience a hypertensive crisis.

■ What makes the hypertensive crisis a “hypertensive emergency” instead of “hypertensive urgency”?
■ Evidence of acute damage of organs

Acute hypertension can cause acute end-organ damage. When there is evidence of acute or ongoing injury to target organs, rapid reduction of BP is recommended. Hypertensive urgency, without evidence of organ involvement, can be treated less aggressively. It is not defined by the absolute BP, but by the presentation (Box 1.34).

▷ HINT
   The risk of a hypertensive emergency may not be the absolute BP but how rapidly the BP increased.

Box 1.34 Clinical Presentation of Organ Damage for Hypertensive Emergencies

<table>
<thead>
<tr>
<th>Hypertensive intracranial hemorrhage</th>
<th>Aortic dissection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertensive encephalopathy</td>
<td>Acute kidney injury</td>
</tr>
<tr>
<td>Angina/myocardial ischemia</td>
<td>Eclampsia/pre-eclampsia</td>
</tr>
<tr>
<td>Left ventricular failure with pulmonary edema</td>
<td></td>
</tr>
</tbody>
</table>

PATHOPHYSIOLOGY

■ What is the most common cause of a hypertensive emergency?
■ Pre-existing hypertension

The most common history following a hypertensive emergency is noncompliance with antihypertensive medications or uncontrolled hypertension.
1. CARDIOVASCULAR SYSTEM REVIEW

❯ HINT
The test question may focus on a complete medication history assessment by the nurse on admission.

- Which hemodynamic change initiates hypertensive crisis?
- Increase in SVR

An abrupt increase in SVR initiates a hypertensive crisis. There is a loss of autoregulation causing greater sympathetic nervous system involvement. The vasoconstriction may be a result of release of vasoactive substances from the endothelium.

❯ HINT
Remember that the diastolic pressure is the resistance the heart is pumping against (afterload).

SYMPTOMS/ASSESSMENT

- What is a common presentation of hypertensive emergency?
- Chest pain, dyspnea, and neurological deficits

The three most common presentations of hypertensive emergencies include chest pain, dyspnea, and neurological deficits. Other symptoms may include fatigue, nasal congestion, or a new-onset cough. The cardiac evaluation includes signs of ACS. If the presentation is consistent with aortic dissection, immediate evaluation is required.

- What is the usual presentation of hypertensive encephalopathy?
- Headache and altered level of consciousness

A focused neurological examination is required to assess for any focal neurological changes such as motor involvement. If present, it indicates potential hemorrhagic stroke. A sudden onset, “worst headache of my life” presentation may indicate an aneurysm rupture and subarachnoid hemorrhage.

❯ HINT
Ocular/fundal examination finding of advanced retinopathy, hemorrhages, or papilledema assist with the recognition of hypertensive encephalopathy.

DIAGNOSIS

- What type of hemodynamic monitoring is recommended during the management of hypertension?
- Arterial BP monitoring

Arterial BP monitoring allows for continuous monitoring of BP during the management of hypertensive emergencies. It is recommended when titrating infusions for targeted BP.

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MANAGEMENT

- What is the percentage of decrease of mean arterial pressure (MAP) typically used as a goal in managing hypertensive emergencies?
- 25% decrease in MAP

Multiple organizations have developed guidelines for the management of hypertension. The typical recommendation is to decrease MAP by 25% within 2 to 6 hours or to decrease diastolic pressure by 10% to 15% or below 110 mmHg within 30 to 60 minutes.

- What is a commonly used goal for systolic pressure in a hypertensive emergency?
- Less than 160 mmHg

The goal in a hypertensive patient should be to control the lowering of BP but should not be to “normalize” the BP. Chronic hypertension resets the autoregulation range for organ perfusion. A normal BP can cause hypoperfusion to vital organs (i.e., brain, heart, kidneys) in a chronic hypertensive patient.

▷ HINT
Continue to monitor for signs of organ hypoperfusion while managing the BP.

- What is the mechanism of action of Labetalol?
- Nonselective β-blocker and α₁-blocker

Labetalol is frequently used in hypertensive emergencies and can be administered as a bolus or continuous IV infusion. It is a nonselective β-blocker (blocks both β₁ and β₂ receptors) as well as a selective α₁-blocker. It is not a pure β-adrenergic blocker, so does not decrease CO like other β-blockers. Potential adverse effects are AV nodal dysfunction (heart block) and bronchospasm.

▷ HINT
If the hypertensive scenario presents a patient with a significant history of asthma, avoid labetalol (or any β-blockers) as the answer for the appropriate antihypertensive therapy due to bronchodilation.

- Which antihypertensive, administered as a continuous infusion, has a rapid onset of 1 minute and a potential side effect of bradycardia?
- Esmolol

Esmolol is a cardioselective β-blocker. It has a very rapid onset of 1 minute with a duration of 10 to 20 minutes (ultra-short acting). It is administered as a continuous infusion, titrated to a goal BP. It is a β-blocker, thus it decreases rate and contractility of the heart. It is frequently used in acute aortic dissection patients as an antihypertensive agent. It is contraindicated in patients with decompensated HF and bradycardia.
1. **CARDIOVASCULAR SYSTEM REVIEW**

- **HINT**
  Esmolol would be a good answer for a hypertensive patient with acute pulmonary edema and underlying diastolic dysfunction.

- **How is esmolol metabolized?**
  - By hydrolysis

  Esmolol is metabolized by hydrolysis of ester linkages by RBC esterases. This agent may be administered to patients in hepatic dysfunction and kidney failure. This accounts for esmolol’s rapid metabolism and ultra-short half-life.

- **HINT**
  Esmolol is a good answer for antihypertensive agent in hepatic or renal failure patients.

- **Which antihypertensive has a vasodilatory effect in both coronary and cerebral vasculature?**
  - **Nicardipine**

  Nicardipine is a dihydropyridine-derivative calcium channel blocker. It produces anti-hypertensive effects by blocking the influx of calcium, thus resulting in relaxation of smooth muscle in the vasculature. It also a coronary and cerebral vasodilator and has been shown to increase coronary and cerebral blood flow. Nicardipine is used as a continuous infusion with onset of 5 to 15 minutes, half-life of 1 hour, and duration of action of 4 to 6 hours.

- **HINT**
  Nicardipine is frequently used as an antihypertensive agent in a hemorrhagic stroke or in patients with coronary artery disease (CAD).

- **What is the advantage of using fenoldopam in hypertensive emergencies?**
  - **Produces naturesis**

  Fenoldopam (Corlopam) is an IV antihypertensive used to manage hypertensive emergencies. It is a dopamine-1-receptor agonist producing vasodilation by acting on dopamine type 1 receptors in the peripheral. It also activates dopaminergic receptors in the renal tubules, resulting in sodium excretion (naturesis). This agent improves creatinine clearance, urine output, and sodium excretion in patients with and without normal renal function. The onset is within 5 minutes and has a duration of 30 to 60 minutes.

- **HINT**
  Fenoldopam is a good answer as an antihypertensive agent in patients with acute renal failure.

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What is the greatest risk of using fenoldopam in hypertensive emergencies?

Hypersensitivity reactions

Fenoldopam’s solution contains sodium metabisulfate. Patients with sulfite allergies may have an allergic reaction to fenoldopam. It is metabolized rapidly and extensively in the liver.

▶ HINT
Look at the scenario. If the patient has an allergy to sulfite, avoid fenoldopam as an appropriate antihypertensive agent.

Which drug may be used as an adjunctive agent in a hypertensive patient with MI?

Nitroglycerin

Nitroglycerin, at high doses, is an arterial dilator. It is both a venodilator and an arterial dilator. It is not considered a first-line drug for a hypertensive emergency, but it can be used as an adjunctive agent in a myocardial ischemic patient experiencing a hypertensive emergency.

▶ HINT
Remember that the mechanism of nitroglycerin is dose dependent. Lower doses affect preload and higher doses affect afterload.

What is the toxin that can accumulate with Nipride at higher doses?

Cyanide

Sodium nitroprusside (Nipride) can lead to cyanide poisoning. The potential amount of cyanide accumulation depends on the dose and duration of the Nipride infusion. Typically, infusions more than 4 mcg/kg/min for 2 to 3 hours have led to toxic levels of cyanide. If higher infusions of Nipride are required, thiosulfate may be administered to prevent accumulation of cyanide.

Does Nipride affect preload, afterload, or both?

Both preload and afterload

Sodium nitroprusside (Nipride) dilates both the arterial and venous circulation, thus producing an effect on both preload and afterload. It has a rapid onset of 1 to 2 minutes and a half-life of 3 to 4 minutes. Due to the quick onset and short duration, Nipride is easily titrated. It should be avoided in patients with renal and hepatic failure due to dependence on these organs for metabolism. Nipride can cause a reduction in coronary blood flow to areas of ischemia (steal phenomenon), an increase in intracranial pressure, and may worsen hypoxia in acute respiratory failure.

▶ HINT
Avoid Nipride as an answer in patients with ACS, acute respiratory failure, and in neurological patients with increased intracranial pressure (ICP).
1. CARDIOVASCULAR SYSTEM REVIEW

- Which of the IV dihydropyridine calcium channel blockers has the shortest half-life?
  - Clevidipine (Cleviprex)

Clevidipine’s half-life is 1 to 2 minutes with rapid onset within 2 to 4 minutes and a duration of 5 to 15 minutes. Nicardipine, in comparison, has a half-life of 1 hour and a duration of 4 to 6 hours. They are both administered as a continuous infusion and are titrated to target BP. Clevidipine is more titratable due to its shorter half-life. It is metabolized by esterases in the blood and accounts for the ultra-short half-life of the drug. Clearance of the drug should not be affected by renal or hepatic impairment but requires more research.

▶ HINT
  When titrating a drug for a targeted BP, clevidipine may be used as the answer over nicardipine, due to the shorter half-life.

- Clevidipine is contraindicated in patients with what allergies?
  - Allergies to soy products and egg or egg products

Clevidipine is a milky, white emulsion that is high in lipids. It is contraindicated in patients who are allergic to soy products, eggs, or egg products. The lipid solutions provide 2 kcal/mL of clevidipine, so it needs to be counted in caloric intake.

- What antihypertensives should be avoided in hypertensive emergencies?
  - Nifedipine and hydralazine

Nifedipine used by oral or sublingual route can cause a sudden, uncontrolled, and severe drop in BP and should not be administered for hypertensive emergencies. Hydralazine is a direct-acting vasodilator. It has a half-life of 3 hours and a half-life of approximately 10 hours. Hydralazine should be avoided because of its prolonged, unpredictable effects and difficulty in titration to target BP.

COMPLICATIONS

- What is a potential complication of antihypertensive therapy?
  - Hypotension and hypoperfusion of organs

The targeted BP is 160/110 to prevent hypoperfusion to vital organs due to the resetting of autoregulation in chronic hypertensive patients. A shorter acting antihypertensive is recommended to prevent long periods of hypotension and is more titratable to targeted BP’s.

VALVULAR HEART DISEASE

- Would a rapid onset of symptoms with valvular structural defect be a stenosis or regurgitation?
  - Regurgitation

The stenosis of a valve occurs over time and is a more gradual process. Regurgitation can present with a sudden onset of symptoms of HF. In acute regurgitation, the heart has not
compensated for the added volume in the ventricles or atria, so atrial and ventricular pressures can rise drastically in a short period of time. This results in HF.

**HINT**
If the scenario provides a “sudden onset” of HF, look for valvular abnormality of regurgitation. The most common is acute mitral regurgitation.

**PATHOPHYSIOLOGY**

- **What is the most common cause of aortic stenosis?**
- **Calcification of the aortic valve**

The most common cause of an aortic valve stenosis in adults is calcification of the valve, eventually causing reduction in leaflet motion.

- **Which cardiomyopathy can be caused by an aortic stenosis?**
- **Hypertrophied cardiomyopathy**

Over time, the LV has been contracting against greater resistance as the aortic valve area decreases and there is a reduction in leaflet motion. The heart compensates by increasing the wall thickness.

- **What are the complications that a patient with acute aortic regurgitation can typically develop?**
- **Pulmonary edema and cardiogenic shock**

There is a sudden large increase in blood volume in the LV due to the incompetence of the aortic valve. This results in a rapid increase in LV pressure with blood backing up into the LA. The elevated LA pressures cause congestion in pulmonary arteries. The LV is unable to compensate with the rapid volume overload and will decrease the CO. So, a patient with acute aortic regurgitation typically presents with either pulmonary edema or cardiogenic shock.

**HINT**
A dissection of the ascending thoracic aorta can damage the aortic valve, causing sudden onset of pulmonary edema.

- **What is the compensatory mechanism of the LV during chronic aortic regurgitation?**
- **Dilated followed by hypertrophy of LV**

The initial response of the ventricles is to dilate to hold the increase in end-diastolic volume following the onset of aortic regurgitation. The LV then begins to hypertrophy to maintain the high EF (due to the large volume or preload). There is an increase in systolic wall stress causing an increase in afterload, thus causing further hypertrophy.
EF is maintained. Eventually, the hypertrophic response may become inadequate and the preload reserve fails, resulting in a low EF and signs of dyspnea.

HINT
Chronic aortic regurgitation occurs over time so the heart compensates and patients may remain asymptomatic for a time period.

What is the most common cause of mitral stenosis?
Rheumatic carditis

Rheumatic heart disease and a history of rheumatic fever may be found in 40% to 60% of the patients diagnosed with mitral stenosis. Rheumatic disease causes thickening and calcification of the mitral valve leaflets, resulting in a funnel-shaped mitral apparatus that narrows the orifice.

Does mitral valve prolapse (MVP) always result in mitral regurgitation?
No

MVP is the billowing of one or both the mitral valve leaflets back up in the LA. It may or may not be associated with mitral regurgitation. An echocardiogram is used to identify the MVP and determine the presence of mitral regurgitation. Other causes of mitral regurgitation include rheumatic heart disease, CAD, infective endocarditis, and ruptured chordae tendineae or papillary muscle.

HINT
The tricuspid valve may also have prolapse in 40% of patients with MVP.

What is the most common arrhythmia induced by mitral regurgitation?
AF

During mitral regurgitation, blood is pushed back up into the LA during systole. This causes LA overload and LA dilation. The dilating of the LA triggers the onset of AF. Pulmonary hypertension may also occur with CHF in later signs of mitral regurgitation.

DIAGNOSIS
What is the most widely used screening for valvular heart disease (VHD)?
Auscultation for murmurs

Murmurs are produced by three mechanisms:
1. High flow through normal or abnormal valve
2. Forward flow through a narrowed orifice (stenosis)
3. Backward flow through incompetent valve (regurgitation)
HINT
Know the physiology of which valves are opened and closed during systole and diastole. This will help you determine whether a valve abnormality causes a systolic or diastolic murmur (Box 1.35).

Box 1.35 Valvular Murmurs

<table>
<thead>
<tr>
<th>Questions to Ask Yourself</th>
<th>Answer</th>
<th>Potential Valve Abnormalities</th>
</tr>
</thead>
<tbody>
<tr>
<td>Which two valves open during diastole?</td>
<td>Mitral and tricuspid</td>
<td>Mitral or tricuspid stenosis</td>
</tr>
<tr>
<td>Which two valves close during diastole?</td>
<td>Aortic and pulmonic</td>
<td>Aortic or pulmonic regurgitation</td>
</tr>
<tr>
<td>Which two valves are open during systole?</td>
<td>Aortic and pulmonic</td>
<td>Aortic or pulmonic stenosis</td>
</tr>
<tr>
<td>Which two valves close during systole?</td>
<td>Mitral and tricuspid</td>
<td>Mitral or tricuspid regurgitation</td>
</tr>
</tbody>
</table>

What are the characteristics that describe a cardiac murmur?

Timing, quality, loudness, location, and radiation

The timing is in relation to the cardiac cycle and may use exact descriptions (i.e., holosystolic or throughout systole). It is also described on the configuration of the murmur (i.e., crescendo, decrescendo, and crescendo–decrescendo). The loudness is written with the bottom number being the scale used and the top number being the loudness of the murmur itself (i.e., 3/6 murmur—murmur is rated a 3 out of 6 for loudness). The location of the chest sounds heard best assists with determining the valve producing the murmur (Box 1.36).

Box 1.36 Location of Auscultation

<table>
<thead>
<tr>
<th>Chest Wall Landmarks</th>
<th>Referred Valve Sounds</th>
</tr>
</thead>
<tbody>
<tr>
<td>Second intercostal space (ICS) right sternal border</td>
<td>Aortic valve</td>
</tr>
<tr>
<td>Second ICS left sternal border</td>
<td>Pulmonic valve</td>
</tr>
<tr>
<td>Fifth ICS midclavicular left side</td>
<td>Mitral valve</td>
</tr>
<tr>
<td>Fourth ICS left of sternal border</td>
<td>Tricuspid valve</td>
</tr>
</tbody>
</table>

HINT

Use the location and timing of the murmur to determine the cause of the murmur. For example, a systolic murmur heard best at the second ICS right of the sternal border is most likely caused by aortic stenosis. Typically, the loudness of the murmur indicates the severity of the structural defect (Box 1.37).
Box 1.37 Abnormal Valve Characteristics

<table>
<thead>
<tr>
<th>Valve Abnormalities</th>
<th>Murmur Characteristics</th>
<th>Other Associated Abnormal Heart Sounds</th>
<th>Hemodynamic Changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aortic stenosis</td>
<td>Crescendo-decrescendo systolic murmur</td>
<td>Paradoxical splitting of the $S_2$</td>
<td>Decreased CO</td>
</tr>
<tr>
<td>Aortic regurgitation</td>
<td>Short and/or soft diastolic murmur</td>
<td>$S_1$ due to increased volume</td>
<td>Tachycardia Decreased CO Widened pulse pressure Exaggerated “A” wave in pulmonary artery occlusive pressure (PAOP) tracing</td>
</tr>
<tr>
<td>Mitral stenosis</td>
<td>Mid-diastolic murmur</td>
<td></td>
<td>Large “V” wave in PAOP tracing</td>
</tr>
<tr>
<td>Mitral regurgitation</td>
<td>Late systolic or holosystolic murmur</td>
<td>$S_3$ systolic clicks</td>
<td>Exaggerated “A” waves</td>
</tr>
</tbody>
</table>

- **Which diagnostic test is recommended for patients with cardiac murmurs and signs of HF?**
  - Echocardiogram

The presence of murmurs in symptomatic patients requires further evaluation of the cardiac structures. This includes patients with signs of HF, ACS, syncope, infectious endocarditis, and other evidence of structural heart disease. Other diagnostics include cardiac catheterization and exercise testing.

- **HINT**
  - A suspected aortic root dissection with aortic valve involvement may require a thoracic CT scan for more rapid diagnosis.

- **Which arrhythmia can trigger the onset of symptoms in a patient with mitral stenosis?**
  - AF

In mitral stenosis, the narrowed orifice decreases the speed of filling the LV during diastole. The LV diastolic volume is dependent on the gradient pressure caused by the atrial contraction. AF causes a significant decrease in LV end-diastolic volume due to loss of the atrial kick and onset of dyspnea. Exercise-induced dyspnea may also be found in patients with mitral stenosis due to the tachycardia and decreased ventricular filling time that occurs with exercise.

Aortic stenosis can also become symptomatic with new-onset AF. The hypertrophied ventricle has a greater resistance to filling and requires a strong atrial contraction to fill the chamber. A person may be asymptomatic until the onset of AF. The atrial contraction is lost in AF, resulting in less filling of the LV during diastole and a decrease in CO and clinical deterioration in symptoms.
Symptoms of HF with exercise or new-onset AF should be red flags for the diagnosis of mitral stenosis but aortic stenosis may also be a differential diagnosis.

While obtaining BP, you notice that Korotkoff sounds continue down to zero. Which valve abnormality would be the cause of loss of diastolic pressure?

Aortic regurgitation

The aortic valve does not close completely during diastole, allowing for the equilibration of pressures in the aorta and LV during diastole. This frequently presents with a loss of diastolic pressure being obtained when auscultating BP. Arterial BP may demonstrate a widened pulse pressure (very low diastolic pressure).

Which ECG change may contribute to SCD in patients with MVP?

Prolongation of QT interval

SCD is not common in mitral regurgitation but is more likely in familial versus nonfamilial forms of MVP. They have been frequently found to have prolongation of the QT interval, which may result in ventricular tachycardia (VT) and SCD.

MEDICAL MANAGEMENT

What is the drug therapy of choice in severe acute aortic regurgitation?

Vasodilator and positive inotropic support

Treatment of severe acute aortic regurgitation is surgery. The goal for medical management temporarily before surgery is to augment the forward flow by increasing contractility and decreasing LV end-diastolic pressures. Nipride with dobutamine may be used in combination. Vasodilator therapy may also be used in severe chronic aortic regurgitation to lower LV resistance (afterload).

Which drug therapy should be avoided in severe acute regurgitation?

β-blockers

β-blockers will block the compensatory tachycardia, which may benefit the patient by increasing CO. β-blockers are typical drugs for managing an aortic dissection but should be avoided if the dissection involves the aortic root and aortic valve.

The scenario may use an ascending thoracic dissection with aortic valve involvement, giving esmolol as a potential answer. In this scenario, Nipride may be the better answer.
What is the heart rate goal in managing mitral stenosis?
Slow the heart rate

Medical management involves the use of drugs, which may slow the heart rate, allowing for greater time for the LV to fill during diastole. Agents with negative chronotropic effects, including β-blockers and heart rate regulating calcium channel blockers, are recommended.

HINT
Avoid any medication that will increase the heart rate and worsen diastolic filling.

What are the treatment recommendations in patients with mitral stenosis and acute-onset AF or atrial flutter?
Anticoagulation and control of heart rate response

Anticoagulation is to prevent systemic or pulmonary embolism during AF. Control of rapid ventricular response to AF includes the use of IV digoxin, heart rate-regulating calcium channel blockers, and β-blockers. If unable to use these previous agents, the second-line drug is an IV or oral amiodarone. If hemodynamically unstable, urgent electrical cardioversion is recommended.

HINT
If the scenario indicates a need for electrical cardioversion, IV heparin is recommended before, during, and after the procedure.

What is the drug of choice to stabilize acute mitral regurgitation in preparation for surgery?
Nitroprusside (Nipride)

Medical management of acute mitral regurgitation is limited and is typically used to stabilize and prepare the patient for surgery. Nitroprusside increases forward flow and reduces pulmonary congestion.

HINT
Nipride should not be used alone if the patient is hypotensive. May combine with positive inotropic agent.

SURGICAL MANAGEMENT

What would be the indication for aortic valve replacement in aortic stenosis?
Presence of symptoms

The decision to replace the aortic valve in aortic stenosis depends upon the severity of symptoms more than on the actual aortic valve area (size of the orifice). Some people have very severe stenosis but are asymptomatic. Others have mild stenosis but are symptomatic.
What symptoms would indicate the need to evaluate a patient with chronic aortic regurgitation for surgery?

Dyspnea, angina, or syncope

It is recommended that symptomatic patients with severe aortic regurgitation undergo aortic valve replacement instead of long-term medical management.

What echocardiogram finding would be a contraindication for percutaneous mitral balloon valvotomy?

LA thrombus

An echocardiogram is recommended before percutaneous mitral balloon valvotomy to assess for the presence of an LA thrombus. If an LA thrombus is found, the patient should be anticoagulated with warfarin for 3 months for the resolution of the thrombi.

What can be used as a “bridge” for a hypotensive patient with acute mitral regurgitation caused by papillary muscle rupture?

IABP counterpulsation

An acute mitral regurgitation may be treated with Nipride, but if hypotensive, an IABP may be beneficial. The IABP will increase forward flow and MAP while decreasing regurgitant volume and LV filling pressures.

COMPLICATIONS

What is the primary complication of acute valvular dysfunction in left-sided valves?

CHF

Acute aortic and mitral valve dysfunction result in higher pressures within the LV and/or LA. The high pressures result in congestion and pulmonary edema. Other complications include cardiomyopathy, low CO syndrome, and thrombus formation.

PERIPHERAL ARTERY DISEASE

PATHOPHYSIOLOGY

What is the primary cause of peripheral arterial disease (PAD)?

Atherosclerosis

PAD is atherosclerosis of the extremities causing ischemia. The risk factors are the same as for CAD (Box 1.38).
Box 1.38 Risk Factors of PAD

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>Male</td>
</tr>
<tr>
<td>Diabetes</td>
<td>Obesity</td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td>High homocysteine levels</td>
</tr>
<tr>
<td>Cigarette smoking</td>
<td></td>
</tr>
</tbody>
</table>

What is the most common location of a thrombus in the peripheral vascular system of the lower extremities?

Popliteal bifurcation

Obstruction of the thrombus occurs at arterial bifurcations just distal to the common femoral bifurcation and at the popliteal bifurcation.

SYMPTOMS/ASSESSMENT

What is the most classic symptom of PAD in the lower extremities?

Intermittent claudication

Intermittent claudication is painful cramping or ache in the legs with exercise that is alleviated by rest. The most common site of claudication is the calves but it can also occur in thighs, hips, buttocks, or feet. Claudication is exercise-induced reversible ischemia, similar to a stable angina. Some have atypical pain, including exercise intolerance, hip pain, and other joint pain.

HINT

Pain at rest may indicate irreversible muscle ischemic injury and requires immediate intervention.

What aggravates the pain at rest?

Elevating the leg

Pain worsens when the leg is elevated and improves when lowered (below the level of the heart). The pain may be described as cramping, burning, aching, or tightening (Box 1.39).

Box 1.39 Other signs of PAD in the Lower Extremities

<table>
<thead>
<tr>
<th>Sign</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diminished or absence of peripheral pulses</td>
<td>Cyanotic</td>
</tr>
<tr>
<td>Dependent rubor</td>
<td>Increased sweating in extremities</td>
</tr>
<tr>
<td>Prolonged capillary refill (dependent)</td>
<td>Extremity cool to touch</td>
</tr>
<tr>
<td>Edema (if immobile)</td>
<td>Leg ulcers</td>
</tr>
<tr>
<td>Thin, pale (atrophic) skin</td>
<td>Erectile dysfunction (Leriche’s syndrome)</td>
</tr>
</tbody>
</table>

DIAGNOSIS

What is the noninvasive test used to recognize PAD?

Ankle–brachial index (ABI)
A low ABI indicates the presence of PAD. A normal index is between 1.00 and 1.40. ABI values of 0.91 to 0.99 are considered “borderline” and values greater than 1.40 indicate noncompressible arteries (Boxes 1.40 and 1.41). Ultrasonography is also used to evaluate noninvasively by determining pressure gradients and pulse–volume waveforms.

**HINT**
The lower the index, the more severe the PAD.

<table>
<thead>
<tr>
<th>Box 1.40  How to Measure ABI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obtain SBP in bilateral arms</td>
</tr>
<tr>
<td>Obtain SBP in bilateral ankles (may use a Doppler probe)</td>
</tr>
<tr>
<td>Calculate ankle-to-arm ratio (divide SBP of ankle by SBP of brachial)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Box 1.41  ABI Severity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
</tr>
<tr>
<td>0.71–0.90</td>
</tr>
</tbody>
</table>

- **What is the diagnostic test obtained prior to surgery for PAD?**
- **Arteriogram**

An arteriogram provides details of the location and extent of arterial occlusion. It is typically performed before surgery or percutaneous transluminal angioplasty (PTA). It does not provide information about the functional significance of the abnormal findings.

- **When measuring transcutaneous oximetry (TcO₂), what level is predictive of poor wound healing?**
- **Less than 40 mmHg**

TcO₂ may also be used to evaluate peripheral arterial insufficiency. A value less than 40 mmHg is predictive of poor wound healing and less than 20 mmHg indicates critical limb ischemia.

**MEDICAL MANAGEMENT**

- **What is the primary pharmacological management of PAD?**
- **Antiplatelet therapy**

Antiplatelet therapy is used to modify atherogenesis and reduce the risk of CAD, stroke, and vascular death. In lower extremity PAD, it may also lessen the symptoms and improve the walking distance. Antiplatelet therapy is also used after lower extremity revascularization (endovascular or surgical bypass). Aspirin is the recommended antiplatelet therapy and clopidogrel (Plavix) is an effective alternative. The combination of aspirin and clopidogrel may be considered in a high-risk patient for CAD or loss of a limb who is not at increased risk of bleeding.
1. CARDIOVASCULAR SYSTEM REVIEW

SURGICAL/INTERVENTIONAL MANAGEMENT

What is the nonsurgical intervention for the treatment of PAD?

PTA

Angioplasty, with and without stent placement, is recommended in patients with severe PAD that is amendable by the nonsurgical route. Stents may keep the arteries open with a lower restenosis rate over angioplasty alone.

HINT

Stents work best in larger arteries with a higher flow, such as iliac and renal arteries (Box 1.42). PTA is not as useful in diffuse disease or long occlusions (typically > 3–5 cm).

Box 1.42 Indications for PTA

<table>
<thead>
<tr>
<th>Indications</th>
<th>Suitable Lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Claudication inhibiting daily activities</td>
<td>Short iliac stenosis (&lt; 3 cm)</td>
</tr>
<tr>
<td>Rest pain</td>
<td>Short, single, or multiple lesions that are superficial on the femoropopliteal segment</td>
</tr>
<tr>
<td>Gangrene</td>
<td>Complete occlusions superficial on the femoral artery</td>
</tr>
<tr>
<td></td>
<td>Iliac stenosis proximal to bypass femoropopliteal artery</td>
</tr>
</tbody>
</table>

What is a complication of PTA?

Loss of blood flow distal to the extremity

Complication following a PTA is loss of blood flow distal to the site of angioplasty and stent placement. Postprocedural assessment includes frequent neurovascular checks on the involved extremity. The loss of blood flow may be due to a thrombosis at the site of dilation, distal embolization, or the dissection of intimal lining causing an obstruction to flow.

HINT

Sudden arterial occlusion may require immediate revascularization surgery or thrombolytic therapy.

What is the recommended conduit in surgical bypass procedures of the lower extremities?

Autogenous vein

Surgical bypass procedures with an autogenous vein conduit are recommended in severe PAD with critical limb ischemia in patients with life expectancies longer than 2 years. Outcomes using a prosthetic bypass are poor and balloon angioplasty with stent placement may be recommended over the use of a prosthetic conduit.
COMPLICATIONS

- What is a common complication of PAD?
- Foot or heel ulcers

Lower extremity ulcers that are not healing can indicate PAD. Skin ulcerations and presence of gangrene in the extremities meet the criteria for critical limb ischemia. Amputation of the extremity is also a resulting complication of severe PAD and critical limb ischemia.

HYPOVOLEMIC SHOCK

- What is the major component in defining shock?
- Hypoperfusion

Shock is the pathophysiological state in which there is defective vascular perfusion of tissues and organs. It is a state of inadequacies between delivery of oxygen and the removal of end products of metabolism from peripheral tissues. This results in widespread reduction in tissue perfusion, hypoxia, and conversion of cellular respiration to an anaerobic form of metabolism, which produces lactate as a by-product. Rapid restoration of oxygen delivery can be a major factor in preventing the development of multiple organ dysfunction syndrome.

› HINT
Remember, shock is defined by hypoperfusion not hypotension.

PATHOPHYSIOLOGY

- During hypovolemic shock, which compensatory mechanism decreases urine output in an attempt to restore circulating blood volume?
- RAS

During periods of hypovolemia and hypoperfusion, the kidneys release renin, which converts angiotensin I to angiotensin II. Angiotensin II is a potent vasoconstrictor, shunting blood away from nonvital organs. Angiotensin II stimulates the release of aldosterone, which results in reabsorption of sodium and water. This decreases the urine output while increasing vascular volume. The sympathetic nervous system is another compensatory system activated during hypovolemic shock. It results in tachycardia, increased myocardial contractility, and vasoconstriction.

› HINT
Vasoconstriction may maintain a BP during hypovolemic shock. Vital signs may not reflect the presence or severity of shock.

- What are the hemodynamic findings of hypovolemic shock that differentiate it from other types of shock?
- Low filling pressures and high SVR

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Hypovolemic shock is due to a decrease in circulating blood volume causing a low SV/CO ratio. Hypovolemia can be caused by blood loss, poor intake, increased fluid losses, or redistribution of fluid (third spacing).

**HINT**
Both the central venous pressure (CVP) and pulmonary artery occlusive pressure (PAOP) are low.

- **What compartmental fluid shift occurs with hemorrhagic shock?**
- **Extravascular to intravascular**

In hemorrhagic shock, fluid shifts from the extravascular space into the intravascular space in an attempt to replace volume due to acute blood loss. In disease states in which plasma volume is lost, the fluid shifts from intravascular to the interstitial space. This is frequently called third spacing and can result in hypovolemic shock. Examples include peritonitis, burns, and crush injuries.

**SYMPTOMS/ASSESSMENT**

- **Following a trauma, a patient presents with the following vital signs on admission: HR 124, RR 32, BP 94/60, and UO 15 cc/hr. Based on these vital signs, what is the class of hemorrhagic shock?**
- **Class III hemorrhagic shock**

The American College of Surgeons has developed a classification of hemorrhagic shock based on vital signs to indicate the severity of blood loss. This is not exact and patient presentation can vary.

- **A Class III hemorrhagic shock would indicate what percentage of blood loss?**
- **30% to 40%**

The classification is based on the percentage of total blood loss (TBV). The estimated amount of blood volume loss is based on a 70-kg male (TBV approximately 5 L). A 30% to 40% TBV loss (Class III) would be approximately 1,500 to 2,000 mL (Box 1.43).

**Box 1.43 American College of Surgeons Classification of Hemorrhage**

<table>
<thead>
<tr>
<th>Class</th>
<th>Blood loss (mL)</th>
<th>Blood loss (%)</th>
<th>Systolic blood pressure</th>
<th>Heart rate (bpm)</th>
<th>Respiratory rate (breaths/min)</th>
<th>Mental status</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>&lt;750</td>
<td>&lt;15</td>
<td>Normal</td>
<td>&lt;100</td>
<td>14–20</td>
<td>Anxious</td>
</tr>
<tr>
<td>II</td>
<td>750–1,500</td>
<td>15–30</td>
<td>Normal</td>
<td>&gt;100</td>
<td>20–30</td>
<td>Agitated</td>
</tr>
<tr>
<td>III</td>
<td>1,500–2,000</td>
<td>30–40</td>
<td>Decreased</td>
<td>&gt;120</td>
<td>30–40</td>
<td>Confused</td>
</tr>
<tr>
<td>IV</td>
<td>&gt;2,000</td>
<td>&gt;40</td>
<td>Decreased</td>
<td>&gt;140</td>
<td>&gt;35</td>
<td>Lethargic</td>
</tr>
</tbody>
</table>

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HINT
Young patients may have normal BP/HR in the presence of significant blood loss due to the effectiveness of their compensatory mechanisms. Elderly patients may be hypotensive with minimal blood loss.

What classification of drugs limits tachycardic response that occurs during hemorrhagic shock?
β-blockers

Blocking β-receptors of the heart results in limited ability to respond to the sympathetic nervous system with tachycardia. The lack of tachycardia does not rule out hemorrhagic shock in patients taking β-blockers.

Other signs of hypovolemic shock include pale, cool, clammy skin. The urine output will progressively decrease as the shock worsens.

HINT
Hypovolemic/hemorrhagic shock patient may narrow the pulse pressure before decreasing systolic BP.

DIAGNOSIS

Which laboratory studies may be used to identify the presence of shock in a normotensive patient?
Lactate and base deficit

Vital signs are not reliable in identifying all patients in shock. Cellular metabolism is limited by inadequate tissue hypoperfusion and results in mandatory changes from an aerobic to an anaerobic metabolism. In anaerobic metabolism, the production of lactic acid is an end product that creates lactic acidosis. Elevated lactate levels and the presence of a base deficit are used to identify anaerobic metabolism.

What is a base deficit?
Amount of base needed to titrate 1 L of whole blood to pH 7.40

The base deficit reflects the extent of anaerobic metabolism and the severity of the metabolic acidosis. This value is obtained from an arterial blood gas. The normal base is +2 to −2 mEq/L with positive numbers indicating a base excess and negative numbers indicating a base deficit.

HINT
Base deficit is used as an end point of resuscitation.

Why does the hgb/hct not accurately reflect the RBC mass during an acute hemorrhage?
Equal loss of all blood components
Hematocrit and hemoglobin concentration are indices of balance between loss of blood and movement of extravascular fluid to intravascular space. During an acute hemorrhage, there is loss of whole blood with a decrease in all blood components in a similar ratio. If the initial hgb is low, it is caused by fluid administration and hemodilution. The rate of change in hgb over time is more predictive of the severity of bleeding.

› HINT
A normal hgb/hct does not rule out active bleeding in acute situations.

MANAGEMENT

■ What is the primary treatment for hypovolemic/hemorrhagic shock?
   ■ IV fluids

IV fluids are the mainstay treatment for hypovolemia. In the case of trauma or acute bleeding, finding the source of blood loss and stopping the bleeding surgically may be required. If the patient is hypothermic, the resuscitation fluids should be warmed prior to or during infusion.

› HINT
Remember airway and breathing are still priority of care in all hemorrhagic shock patients.

■ What is the greatest disadvantage of resuscitating with crystalloids?
   ■ Fluid shifts from intravascular to interstitial space

Crystalloids are electrolyte solutions with small molecules, which can shift across the spaces. A large amount of infused crystalloids will shift from the intravascular to the interstitial space within minutes of administration. This requires larger volumes of fluids to be administered to replace vascular losses. Frequently used crystalloids for resuscitation include lactated Ringer’s (LR) and normal saline (NS). These fluids are both isotonic solutions.

› HINT
A 3:1 replacement rule has been used to determine the amount required for crystalloid resuscitation (3 L of crystalloids for every 1 L of blood loss).

■ Which acid–base imbalance is caused by large-volume infusions of NS?
   ■ Metabolic acidosis

A 1-L bag of NS contains 154 mEq/L of sodium and chloride. Large amounts of NS administered during resuscitation can cause hyperchloremic metabolic acidosis. An LR solution is a more balanced salt solution and may be used in large-volume resuscitations to prevent metabolic acidosis (Box 1.44).
**HINT**
The patient’s respiratory rate may be rapid to compensate for metabolic acidosis.

**Box 1.44 Crystalloids Versus Colloids**

<table>
<thead>
<tr>
<th></th>
<th>Crystalloids</th>
<th>Colloids</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Advantages</strong></td>
<td>Replaces interstitial fluid losses that may have occurred</td>
<td>Uses less fluid to resuscitate</td>
</tr>
<tr>
<td></td>
<td>Cheaper</td>
<td>May draw fluid into the vascular space from interstitial space</td>
</tr>
<tr>
<td></td>
<td>Easier to store</td>
<td>Albumin may have anti-inflammatory effects</td>
</tr>
<tr>
<td><strong>Disadvantages</strong></td>
<td>Uses larger amounts of fluid to resuscitate</td>
<td>During periods of increased capillary permeability, albumin will third-space into the extravascular space.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Synthetic colloids (i.e., Dextran) activate immune response</td>
</tr>
<tr>
<td></td>
<td></td>
<td>May cause hypersensitivity reaction</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Synthetic colloids increase bleeding tendencies</td>
</tr>
<tr>
<td></td>
<td></td>
<td>More expensive</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Difficult to store</td>
</tr>
</tbody>
</table>

- **Which crystalloid is used to increase serum osmolality and rapidly expands the intravascular space?**

**Hypertonic saline**

Small amounts of hypertonic saline (4–5 mL/kg) can decrease the total amount of crystalloids used during resuscitation. Hypertonic saline increases serum osmolality and draws fluid from the extravascular space into the intravascular space. It may improve blood flow to organs and has been found to lower intracranial pressure.

**HINT**

Metabolic acidosis and hypernatremia are complications of a hypertonic saline because of large amounts of chloride, even greater than an NS.

Na⁺ in 3% NS is 513 with Cl⁻ of 513  
Na⁺ in 7.5% NS is 1,283 with Cl⁻ of 1,283

- **What is a benefit of “hypotensive resuscitation” in a bleeding patient?**

**Limited blood loss**

Avoiding aggressive fluid resuscitation to increase BP may limit the amount of blood volume loss in a bleeding patient prior to surgery. Hypotensive resuscitation aims to maintain the systolic BP between 80 and 90 mmHg with smaller boluses of fluid (200-mL bolus). Higher systolic BPs increase intravascular hydrostatic pressure, worsening blood loss in a bleeding patient. The risk of this strategy is hypoperfusion.

**HINT**

The exception is traumatic brain injured patients. They require a SBP greater than 90 mm Hg. Maintain the systolic BP more than 90 for those with traumatic brain injury.
1. CARDIOVASCULAR SYSTEM REVIEW

When giving multiple units of packed red blood cells (PRBCs), what other blood products need to be administered?

Fresh frozen plasma (FFP) and platelets

Administering PRBCs and fluid causes a dilutional coagulopathy. PRBCs are void of clotting factors and platelets. Transfusion practice is changing by adding more FFP and platelet transfusions into the resuscitation. Some practitioners are using the 1:1 replacement rule. For every one unit of blood, one unit of FFP is administered.

A patient without a history of cardiac problems was given PRBCs for acute blood loss. Within 4 hours of the transfusion, the patient became hypoxic, febrile, showing pulmonary edema on a chest x-ray, requiring intubation. What is the most likely cause of this change in clinical status?

Transfusion-related acute lung injury (TRALI)

TRALI is the most common cause of transfusion-related deaths. The theory behind TRALI is a “two hit” insult. The first hit is a stressful situation (such as trauma, sepsis, massive transfusion, cardiopulmonary bypass [CPB] surgery), which causes the neutrophils to be “primed” and adhere to the pulmonary endothelial bed. The second hit is the actual transfusion of the blood. The transfused blood contains donor antibodies against neutrophil antigens and human leukocyte antigens. These antibodies activate the “primed” neutrophils and monocytes resulting in increased capillary permeability and noncardiogenic pulmonary edema.

Hint
Think of TRALI if there is a sudden onset of hypoxia, fever, and cough within 1 to 6 hours after a blood transfusion.

What is the laboratory value that can be used as an end point of resuscitation?

Base deficit

The base deficit has been found to be a better prediction of metabolic dysfunction during hypovolemic shock and correlates with lactate levels and SVO₂ (guide to determine magnitude of volume deficit). Lactate levels are used to determine the presence of anaerobic metabolism but do demonstrate rapid adjustments to identify the return to aerobic metabolism.

Hint

A base deficit of –3 to –5 may be seen on a postoperative patient and may indicate the need for further fluids (Box 1.45). A base deficit of more than –15 may indicate an ongoing blood loss.

Box 1.45 Base Deficit Determines Severity Hypovolemia

<table>
<thead>
<tr>
<th>Severity</th>
<th>Hypovolemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>–3 to –5</td>
</tr>
<tr>
<td>Moderate</td>
<td>–6 to –14</td>
</tr>
<tr>
<td>Severe</td>
<td>&gt; –15</td>
</tr>
</tbody>
</table>

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COMPLICATIONS

- **What is the “lethal triad” that can occur with hemorrhagic shock and resuscitation?**
- **Coagulopathy, hypothermia, acidosis**

A worsening of one of these can lead to a cycle that results in rapid deterioration and ultimately death in a bleeding patient. All IV fluids should be warmed during the resuscitation, adequate replacement of clotting factors and platelets must be done to limit the coagulopathy, and maintaining perfusion of tissues and organs to help prevent these complications.

▷ **HINT**
Remember hypothermia worsens coagulopathy, and tissue hypoxia (shifts oxyhemoglobin dissociation curve), and decreases CO (decreasing myocardial contractility).

- **What are the electrolyte abnormalities commonly found after a massive resuscitation?**
- **Hypocalcemia, hypomagnesium, hypo- or hyperkalemia**

Blood transfusions contain citrate to increase the shelf-life of stored blood. Citrate binds calcium and magnesium, lowering the ionized levels of both. Multiple transfusions of blood can also increase potassium levels due to cell lysis, but frequently potassium levels are low after resuscitation. The low potassium may be caused by the release of aldosterone. The kidneys hold on to sodium and excrete potassium.

▷ **HINT**
Signs of low magnesium are similar to that of low calcium. Look for either answer if the scenario provides symptoms of muscle spasms, Chvostek’s sign, or Trousseau’s sign following blood transfusions.

- **What is the abdominal complication associated with aggressive fluid resuscitation?**
- **Abdominal compartment syndrome**

Excessive fluid administration increases the third spacing, resulting in compartment syndromes (cranium, thoracic, abdominal). The elevated pressure in the abdominal cavity results in pulmonary and renal complications, elevated intracranial pressure, and decreased venous return. Abdominal compartment syndrome affects almost all organ functions.

▷ **HINT**
Bladder pressure measurements are used to monitor abdominal compartment syndrome.
1. CARDIOVASCULAR SYSTEM REVIEW

CARDIAC SURGERIES

■ What is an indication for a CABG surgery?
■ Left main coronary artery stenosis

Other indications include severe triple vessel disease, recurrent HF due to ischemia, multiple coronary artery occlusions, and any contraindication to angioplasty/stent procedures (Box 1.46).

Box 1.46 Cardiac Surgeries

<table>
<thead>
<tr>
<th>CABG</th>
<th>Repair or replacement of aorta root</th>
</tr>
</thead>
<tbody>
<tr>
<td>Valve repair or replacement</td>
<td>Intracardiac tumors</td>
</tr>
<tr>
<td>Repair congenital or acquired defects (atrial septal defect [ASD] and ventricular septal defect [VSD])</td>
<td>LV aneurysmectomy</td>
</tr>
</tbody>
</table>

■ What is the purpose of the use of cardioplegic hyperkalemic solution in open heart procedures?
■ Induce asystole

The induction of asystole with cardioplegic hyperkalemic solutions on the heart during the surgery decreases myocardial metabolism and oxygen consumption. This potentially protects the heart during the period of ischemia.

PATHOPHYSIOLOGY

■ What aspects of CPB stimulate the release of the inflammatory system?
■ Nonpulsatile flow and exposure to bypass circuit

CPB can cause a systemic reaction with the release of inflammatory mediators similar to sepsis. This inflammatory response is responsible for many of the adverse effects that can occur with CPB, including multisystem organ failure.

■ What are potential advantages of “off-pump” CABG?
■ Lowers risk of bleeding and multisystem organ failure

The development of off-pump or “beating heart” CABG procedures lower the incidence of complications attributed to CPB. This technique does not require CPB to be used.

❯ HINT
The greatest risk of off-pump CABG is requiring early revascularization procedures.
SYMPTOMS/ASSESSMENT

What is the major focus of hemodynamic monitoring on a postoperative cardiac patient?

Ventricular function

Ventricular function must be continuously assessed postoperatively in a cardiac surgery patient. Hemodynamic monitoring is used to determine ventricular function. Even after the heart abnormality is repaired, the ventricular function may continue to be affected for a period of time. Cardiac function has been found to be depressed postoperatively, peaking at 4 to 6 hours after surgery and typically improving within 24 hours.

› HINT
Interventions will be aimed at improving ventricular function pharmacologically or mechanically.

DIAGNOSIS/POSTOPERATIVE MONITORING

Following a CABG procedure in which the left internal mammary artery (LIMA) was grafted to the left anterior descending (LAD) artery, what would ST elevation in all anterior leads indicate?

LIMA spasm

ST elevation in all anterior leads following the procedure of grafting the LIMA to the LAD indicates spasm of the LIMA. This 12-lead ECG finding should be reported to the physician immediately. It is important on a post-CABG patient to perform ST-segment monitoring. The lead frequently used for continuous monitoring is a lead in the territory of the graft.

› HINT
ST elevation in two or more contiguous leads in a territory that was grafted indicates acute graft failure.

Which electrolytes are commonly monitored closely in a postcardiac surgery patient?

Potassium and magnesium

Hypokalemia and hypomagnesium are frequent electrolyte abnormalities encountered in a postcardiac surgical patient and require careful monitoring to treat. Hypokalemia and hypomagnesium can significantly increase the likelihood of postoperative arrhythmias.

› HINT
Remember, to effectively treat the low potassium, the magnesium needs to be corrected first.
MANAGEMENT

■ What is the hemodynamic complication that occurs during the rewarming of a postcardiac surgery patient?

■ Hypotension

Hypothermia causes vasoconstriction and an increase in SVR. Most postcardiac patients have been cooled in the operating room (usually < 34°C). Vasodilation occurs during the rewarming process resulting in hypotension. Patients are rewarmed with the use of air convection that blows warm air over the patient.

▶ HINT
Rewarming is important in the management of patients to prevent complications of hypothermia (Box 1.47).

Box 1.47 Complications of Hypothermia

<table>
<thead>
<tr>
<th>Complication</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myocardial contractility depression</td>
<td>Causes shivering and increased oxygen consumption</td>
</tr>
<tr>
<td>Predisposes to ventricular arrhythmias</td>
<td>Decreases CO₂ production</td>
</tr>
<tr>
<td>Increases afterload and myocardial workload</td>
<td>Causes coagulopathy</td>
</tr>
</tbody>
</table>

■ Which of the inotropic agents is a phosphodiesterase inhibitor?

■ Milrinone (Primacor)

Inotropic agents are used in postcardiac surgery patients to increase the contractility of the ventricles. Milrinone, being a phosphodiesterase inhibitor, does not rely on either α or β stimulation for inotropic effects. Milrinone increases the levels of cyclic adenosine monophosphate (CMP) and intracardiac calcium, which promote increased contractility. The influx of calcium in the vascular beds leads to vasodilation and lowering of SVR.

▶ HINT
If the patient develops tolerance due to catecholamine depletion, milrinone is the drug of choice (Box 1.48).

Box 1.48 Other Inotropic Agents

<table>
<thead>
<tr>
<th>Inotropic Agent</th>
<th>Inotropic Agent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dopamine</td>
<td>Epinephrine</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>Norepinephrine</td>
</tr>
</tbody>
</table>

■ What hemodynamic profile needs to be assessed before administering a vasopressor?

■ Fluid status

Vascular fluid status needs to be assessed before administering a vasopressor. The vascular volume should be replaced before administering a vasoconstricting drug. The vasoconstriction and increased afterload can result in organ hypoperfusion in a hypovolemic patient. Adequate volume status during the administration of a vasoconstrictor will limit the hypoperfusion.
HINT
Do not attempt to squeeze an empty vessel. Replace fluid volume first, then vasoconstrict with pharmacological agents to increase MAP.

Which vasoconstrictor is a pure $\alpha$-agonist drug?
Phenylephrine

Phenylephrine is a pure $\alpha$-agonist that has no $\beta$-receptor effects. Norepinephrine is both an $\alpha$- and $\beta$-receptor agonist. Vasopressin may also be used as a vasoconstrictor. It is an exogenous production of antidiuretic hormone (ADH).

COMPLICATIONS

Why is bleeding a common postoperative complication of open heart surgery with the use of CPB?
Heparinization

During the period of CPB, the patient must be heparinized and the activated clotting times (ACT) must be maintained at more than 400 seconds to prevent clotting in the bypass circuit. Longer CPB times result in greater incidence of bleeding complications, even with the reversal of heparin at the end of the surgery with protamine. If the ACT is elevated, administer protamine to further reverse the heparin. The patient may not have been completely reversed (inadequate dose) or received additional heparin at the time of discontinuation from the pump (Box 1.49).

HINT
An ACT test should be performed on admission to the intensive care unit (ICU) to assure adequate reversal of heparin (normal values between 100 and 120 seconds). Always consider a surgical source of bleeding with sudden onset of fresh, rapid bleeding from mediastinal chest tubes.

Box 1.49 Other Causes of Postoperative Bleeding

<table>
<thead>
<tr>
<th>Thrombocytopenia and abnormal function of platelets</th>
<th>Bleeding from small arteries or veins</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypothermia</td>
<td>Leaks at vascular anastomosis</td>
</tr>
<tr>
<td>Preoperative anticoagulation or antiplatelet drugs</td>
<td></td>
</tr>
</tbody>
</table>

What are the signs of a protamine reaction?
Hypoxia and hypotension

A protamine reaction can occur with any administration of protamine, even if the patient had tolerated the drug previously. Pulmonary hypertension with resulting hypoxia and systemic hypotension are the signs of a protamine reaction. Monitor for this adverse reaction when administering protamine.

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1. CARDIOVASCULAR SYSTEM REVIEW

HINT

Excessive use of protamine can also cause coagulopathy.

- Administration of five units of platelets should increase the platelet count by how much?
- 25,000 to 50,000

Correction of the bleeding complication includes administration of platelets, FFP, cryoprecipitate, and PRBCs. Monitoring PT/PTT will guide the replacement of FFP. Cryoprecipitate contains fibrinogen and factor VIII. Other methods to control small venous bleeding in the thoracic cavity include raising the head of the bed and adding PEEP on the ventilator to increase pleural and mediastinal pressures. There are no definitive studies on these methods.

HINT

The main purpose of giving PRBC in a postcardiac bleeding patient is to improve oxygen delivery. Otherwise, a stable patient may tolerate hgb of 7.0 g/dL.

- When would aminocaproic (Amicar) be indicated in a postoperative cardiac patient?
- Actively bleeding

Rescue Amicar may be used in a postoperative cardiac patient who is actively bleeding when all other causes of bleeding have been addressed. Amicar and tranexamic acid (TXA) are frequently administered to cardiac surgical patients in the operating room to reduce the amount of blood loss and limit the amount of required blood transfusions. They are antifibrinolytic agents that inhibit conversion of plasminogen to plasmin thus preventing activation of fibrinolysis. The major risk of both drugs is thrombosis, including complete occlusion of the new graft.

HINT

Amicar may cause renal failure and tranexamic acid may increase the risk of seizures.

- How does desmopressin acetate potentially work to prevent bleeding complications?
- Improves platelet function

Desmopressin acetate elevates the levels of factor VII and von Willebrand’s factor. This may improve the platelet function following cardiac surgery.

- Following an aortic valve replacement, a patient suddenly becomes hypotensive and decreases output from the mediastinal chest tubes. What complication would you suspect?
- Cardiac tamponade

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Cardiac tamponade can be a complication of cardiac surgeries. It is more common in open heart procedures such as valvular surgeries. The classic signs of pericardial tamponade may not be present but the presence of hypotension needs to be evaluated for potential cardiac tamponade. Decreasing or abrupt cessation of output from the mediastinal chest tubes should increase the suspicion of a cardiac tamponade. TEE may be used to diagnose the cardiac tamponade. Volume resuscitation, inotropes, and vasopressors may be temporary measures until surgery.

HINT
Cardiac tamponade can have a regional effect such as compression of RV with the onset of symptoms of RV failure.

Which arrhythmia commonly presents in elderly patients 2 to 3 days post–cardiac surgery?

AF

AF is common following cardiac surgeries, in particular, valve surgeries. It may be seen in patients of all ages but occurs most frequently in the older population. Other common arrhythmias that occur after cardiac surgery are heart blocks and ventricular arrhythmias.

HINT
New onset AF should be immediately converted to prevent the formation of intracardiac thrombi.

What type of stroke is more common following CPB surgeries?

Watershed stroke

Watershed strokes are ischemic strokes, which occur between major cerebral vascular territories. Periods of hypotension or hypoperfusion during the nonpulsatile flow state of CPB leads to the ischemia. Other causes of stroke following CPB include showing of atherosclerotic emboli mobilized by surgical manipulation of the aorta and embolic strokes with the development of AF.

What is the primary symptom of an air embolus following CPB surgeries?

Seizures

An air embolus is a risk when the surgery requires aortotomy or when an open heart procedure is performed. The symptoms include seizures, delayed emergence from anesthesia, and focal neurological deficits. In contrast to strokes, an air embolus rarely shows changes on CT or magnetic resonance imaging (MRI) (Box 1.50).

HINT
A scenario with valve replacement in a postop patient who is not waking up, but shows nothing on CT/MRI, would most likely be caused by an air embolus.
1. CARDIOVASCULAR SYSTEM REVIEW

**Box 1.50 Neurological Complications of CPB**

<table>
<thead>
<tr>
<th>Stroke</th>
<th>Seizures</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coma</td>
<td>Memory deficits</td>
</tr>
<tr>
<td>Paralysis</td>
<td></td>
</tr>
</tbody>
</table>

- **Following a mitral valve replacement, what signs would indicate an acute dehiscence of the valve repair?**
- **New systolic murmur and new “V” wave**

Acute dehiscence of a valve repair is rare. The signs would be a new regurgitant murmur. A mitral regurgitation produces a systolic murmur whereas an aortic valve regurgitation produces a diastolic murmur. A new “V” wave develops in the PAOP waveform due to high pressures in the LA during ventricular systole due to the backflow of blood into the LA.

**TESTABLE NURSING ACTIONS**

**HEMODYNAMIC MONITORING**

See Table 1.3.

- **A patient develops hypotension. The CVP is 14 and PAOP is 7. What is the most likely cause of these hemodynamic findings?**
- **RV failure**

RV failure will result in an elevation of CVP due to backward flow and a lower PAOP due to less blood flow to the left side of the heart. This may be caused by RV infarct, pulmonary hypertension, or pulmonary embolism.

**Table 1.3 Hemodynamic Profiles**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Method Calculation</th>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>MAP</td>
<td>(Systolic BP – diastolic BP/3) + Diastolic</td>
<td>70–105 mmHg</td>
</tr>
<tr>
<td>CO</td>
<td>Liters per minute</td>
<td>4–8 L</td>
</tr>
<tr>
<td>Cardiac index</td>
<td>CO/body surface area</td>
<td>2.5–4.0 L/min/m²</td>
</tr>
<tr>
<td>Stroke volume (SV)</td>
<td>CO/Heart rate (HR)</td>
<td>50–100 mL/beat</td>
</tr>
<tr>
<td>Stroke index (SI)</td>
<td>SV ÷ BSA</td>
<td>25–45 mL/m²/beat</td>
</tr>
<tr>
<td>Systemic vascular resistance (SVR)</td>
<td>(MAP – CVP) × 80 ÷ CO</td>
<td>800–1200</td>
</tr>
<tr>
<td>Pulmonary vascular resistance (PVR)</td>
<td>(Mean PA – PAOP) × 80 ÷ CO</td>
<td>50–250</td>
</tr>
<tr>
<td>Left Ventricular Stroke Work Index (LVSWI)</td>
<td>SI × (MAP – PAOP) × 0.0136</td>
<td>40–65 g·m/m² 2</td>
</tr>
<tr>
<td>Right Ventricular Stroke Work Index (RVSWI)</td>
<td>SI × (Pam – CVP) × 0.0136</td>
<td>5–12 g·m/m² 2</td>
</tr>
<tr>
<td>Pulmonary artery pressure (PA)</td>
<td>(MPAP = 9–18)</td>
<td>25/10</td>
</tr>
<tr>
<td>Pulmonary artery occlusion pressure (PAOP)</td>
<td></td>
<td>4–12</td>
</tr>
<tr>
<td>CVP</td>
<td></td>
<td>2–6</td>
</tr>
</tbody>
</table>

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**HINT**

If both the CVP and PAOP are elevated, it could be because of LV failure or volume overload (see Figure 1.2).

![Hemodynamic interpretation diagram]

**FIGURE 1.2** Hemodynamic interpretation.

- **Which valve abnormality interferes with the ability to obtain an accurate pulmonary artery occlusive pressure?**
  - **Mitral stenosis**
    
    For the LA pressure to be equal to the LV end-diastolic pressure (LVEDP), the mitral valve must be open for the pressures to equalize. In mitral stenosis, the valve does not open completely, thus limiting the ability of the LA pressure to accurately reflect LV pressure. After obtaining a CVP reading that is the same on the right side of the heart, the tricuspid valve must be open completely to obtain an accurate reading of the RV end-diastolic pressure (RVEDP).

  **HINT**
  
  An accurate pressure reading can be obtained in mitral regurgitation if read during the A wave only (when the mitral valve is open).

- **PAOP and CVP readings in patients with hypertrophied cardiomyopathy are falsely high or low?**
  - **High**

  PAOP and CVP are readings of pressures in the heart chambers. Pressure is not always equal to volume and can cause an erroneous reading. Hypertrophied cardiomyopathies will read the pressures higher than the actual volume. This is due to the high pressure of the thickened myocardial muscle placed on the heart chambers. Dilated cardiomyopathies exert little pressure but are dilated chambers that hold large volumes of blood. In dilated cardiomyopathy, CVP and PAOP readings are lower than the actual volume.
HINT
Pressure is not always equal to volume. Read the scenario and interpret findings based on the patient case study presented in the question.

What is SV variance (SVV) an indicator of?
Preload responsiveness

SVV is frequently used to determine the need for fluid. SVV is not an indicator of actual preload but of relative preload responsiveness. The goal is to maintain SVV less than 13%.

HINT
The patient must be intubated for accuracy of measurement (Box 1.51).

Box 1.51 Limitations of SVV

- Requires mechanical ventilation (improves accuracy)
- Arrhythmias (affect accuracy)
- Positive end-expiratory pressure (PEEP; SVV)
- Vascular tone (vasodilation may increase SVV)

What maneuver could be used instead of SVV to determine fluid responsiveness in a spontaneous breathing patient?
Passive leg raising

Without the availability of SVV, raising the legs has proven clinically to act like a “self volume challenge” to indicate the patient’s status on the Fran–Starling curve. This provides a physiologic fluid bolus and may be performed if the patient has arrhythmias or spontaneous breathing.

What are the three components of oxygen delivery (DO₂)?
Oxygen saturation, hemoglobin (hgb), and CO

DO₂ is the amount of oxygen leaving the heart per minute and delivered to the tissue level. The components of DO₂ include oxygen saturation, hgb, and CO. If DO₂ decreases, the body compensates by offloading more oxygen from the hgb to the tissues. CO is the most important component of this equation.

HINT
If the tissues take more oxygen from the hgb, then less oxygen is returned to the right side of the heart.

What is oxygen consumption (VO₂) equal to in a normal situation?
Oxygen demand

In a normal situation with adequate oxygen delivery, an increase in oxygen demand will increase oxygen consumption. VO₂ will vary based on the metabolic needs of the tissues.
HINT

Intervention may be to increase the delivery of oxygen or decrease the demand of oxygen (Box 1.52).

Box 1.52 Causes of Increased VO₂

<table>
<thead>
<tr>
<th>Fever</th>
<th>Anxiety</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shivering</td>
<td>Hyperthermia</td>
</tr>
<tr>
<td>Increase in work of breathing</td>
<td>Response to major illness or surgeries</td>
</tr>
<tr>
<td>Pain</td>
<td>Seizures</td>
</tr>
</tbody>
</table>

Are critically ill patients usually supply independent or supply dependent?

Supply dependent

VO₂ is a good measurement of the overall aerobic metabolism but by itself is an unreliable indicator of adequacy of tissue perfusion. Adding DO₂ in relation to VO₂ tells more about oxygen use. Supply independency occurs when the VO₂ remains constant during a period of increased demand or decreased supply. This indicates that DO₂ is sufficient for the demand, indicating adequate tissue perfusion. In critically ill patients, it is difficult to achieve this supply independency. Supply dependency occurs as the tissue demand increases and requires an increase in blood flow. This indicates that the DO₂ was not sufficient for the tissue demands.

HINT

An increase in VO₂ after increasing DO₂ indicates an oxygen debt (see figure below).

What is a normal SvO₂ obtained from a PAC?

60% to 80%

SvO₂ monitoring is used to allow for continuous monitoring of O₂ supply and demand. SvO₂ measures oxygen saturation of the venous blood from the right side of the heart (it is the amount of oxygen returning to the right side after tissue extraction). A change in SVO₂ may indicate a change in either DO₂ or VO₂ or both.

HINT

If the question gives a change in the SvO₂, look at both the demand and delivery to determine the cause.

What is the difference between SvO₂ and ScvO₂ monitoring?

SvO₂ is a true mixed venous gas

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ScvO₂ is obtained from a central line (central venous circulation), whereas SvO₂ is obtained from the PAC (pulmonary artery). ScvO₂ is not a true “mixed” venous gas but can be used to determine oxygen consumption. The goal is to maintain ScvO₂ at greater than 70%.

**INTRA-AORTIC BALLOON PUMP COUNTERPULSATION**

**Where is the balloon positioned in the aorta?**
- Descending thoracic aorta

The tip of the catheter should be positioned just distal to the left subclavian artery and above the renal and mesenteric arteries.

› **HINT**
Sudden cessation of urine may indicate migration of the balloon distally, obstructing the renal arteries.

**When does inflation of the balloon occur?**
- Beginning of diastole

The inflation of the balloon just after the closure of the aortic valve, the beginning of diastole, increases the aortic pressure and augments perfusion. It elevates the diastolic pressure in the aorta, thus improving coronary perfusion.

› **HINT**
Remember coronary perfusion pressure (CPP) is the difference between aortic diastolic pressure (ADP) and right atrial pressure (RAP):

\[
\text{CPP} = \text{ADP} - \text{RAP}
\]

**What is the purpose of balloon deflation immediately prior to systole?**
- Decreased afterload

The balloon should deflate just before the opening of the aortic valve, immediately prior to systole. This results in a sudden decrease in aortic pressure and decreases the afterload or resistance of the LV.

› **HINT**
If the balloon deflates after the onset of systole, it will increase the workload of the heart. It is important that deflation occurs immediately prior to systole.

**Which of the following provides the most support to the heart: the 1:1, 1:2, or 1:3 cycle?**
- 1:1
A 1:1 cycle indicates that the balloon inflates and deflates with every heartbeat. It provides support with every contraction. A 1:2 cycle provides support with every second cardiac cycle, and the 1:3 cycle every third.

**HINT**
Weaning the IABP is typically performed by decreasing the cycle frequency from 1:1 to 1:2, then to 1:3 before removing the balloon.

**Which valve abnormality would be a contraindication for the use of IABP?**

- Aortic insufficiency

Inflates after closure of the aortic valve. An aortic insufficiency would cause the blood to flow back into the LV during balloon inflation, increasing the LV volume. This would increase the workload of the heart and worsen LV failure. Other contraindications include aortic dissection and severe peripheral vascular disease (due to placement of the catheter in femoral location).

**HINT**
IABP is indicated for mitral valve insufficiency to lower the resistance of the LV (Box 1.53).

**Box 1.53  Indications for an IABP**

<table>
<thead>
<tr>
<th>Cardiogenic shock: medically refractory</th>
<th>Acute ventricular septal rupture</th>
</tr>
</thead>
<tbody>
<tr>
<td>AMI: medically refractory</td>
<td>Bridge to cardiac transplantation</td>
</tr>
<tr>
<td>Acute mitral regurgitation</td>
<td></td>
</tr>
</tbody>
</table>

**A common complication of IABP is lower extremity ischemia. What should be performed routinely to assess for this potential complication?**

- Check distal pulses hourly

The catheter is placed in the femoral artery and can diminish or occlude blood flow distally. It is recommended to assess distal pulses hourly for as long as the balloon is in place. Hematomas can occur at the insertion site contributing to a decrease in distal flow. Assessment of the insertion site hourly for the presence of hematoma is recommended (Box 1.54).

**Box 1.54  Other Complications of Using an IABP**

<table>
<thead>
<tr>
<th>Occlusion of the renal, superior mesenteric, or subclavian artery</th>
<th>Thrombocytopenia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute aortic dissection or perforation with retroperitoneal hemorrhage</td>
<td>Thromboembolism</td>
</tr>
<tr>
<td>Wound infection</td>
<td></td>
</tr>
</tbody>
</table>

**PACEMAKERS**

- According to the pacemaker code, where does a DVI pacemaker sense and pace?

  - Senses in ventricles, paces in both atrium and ventricles
A DVI pacemaker paces in both the atrium and ventricles and senses in the ventricles only. The first letter in the code is chamber paced, and the second letter is chamber sensed (Box 1.55).

Box 1.55 Inter-Society Commission for Heart Disease (ICHD) Pacemaker Codes

<table>
<thead>
<tr>
<th>I Chamber Paced</th>
<th>II Chamber Sensed</th>
<th>III Mode of Response (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>V = Ventricle</td>
<td>V = Ventricle</td>
<td>T = Triggered</td>
</tr>
<tr>
<td>A = Atrium</td>
<td>A = Atrium</td>
<td>I = Inhibited</td>
</tr>
<tr>
<td>D = Double</td>
<td>D = Double</td>
<td>D = Double</td>
</tr>
<tr>
<td>O = None</td>
<td>O = None</td>
<td>O = None</td>
</tr>
</tbody>
</table>

HINT
A fourth letter would indicate whether the pacemaker is rate responsive. For example, VVIR.

Following placement of a permanent pacemaker, the patient's BP decreased from 142/76 to 99/40 on admission to the ICU. What is the most likely cause of the hypotension?

Cardiac tamponade

During placement of lead, manipulation or fixation of the screw into the wire causes bleeding into the pericardial space. The most common symptom is hypotension. Emergency pericardiocentesis is required to manage pericardial tamponade (Box 1.56).

Box 1.56 Potential Complications of an Implanted Pacemaker

<table>
<thead>
<tr>
<th>Rejection phenomena</th>
<th>Surgical complications (hematoma, infection, thrombosis)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin erosion</td>
<td>Lead problems (fractured, compressed, dislodgement)</td>
</tr>
<tr>
<td>Muscle or nerve simulation</td>
<td>Cardiac perforation, cardiac tamponade</td>
</tr>
</tbody>
</table>

Which pacemaker would be contraindicated in a patient with a complete AV heart block?

Atrial pacemaker (AAI)

An atrial pacemaker senses and paces in the atrium only. The impulse travels to the AV junction and requires an intact pathway to travel down to the ventricles. A patient with an AV block requires either a ventricular (VVI) or dual chamber pacemaker (DDD) to pace below the level of the block.

HINT
Ventricular pacemakers (VVI) sense and pace only in the ventricles. Without any coordination with the atrium, the atrial kick is lost and can decrease the SV.

If the patient does not know what type of pacemaker he or she has, what test must be ordered?

CXR
A CXR can identify the types of leads, the number of leads, and their positions. This indicates what system the patient has implanted. The shape of the pacemaker and manufacturer can assist with further identifying the type of pacemaker.

› HINT
Two wires would indicate a dual-chamber pacemaker.

- **If CXR reveals three leads, what type of pacemaker does the patient have?**
- **Biventricular pacemaker (three-chamber pacemaker)**

A biventricular pacemaker is also called cardiac resynchronization therapy (CRT). Single- and dual-chamber pacemakers pace in the right side of the heart. Depolarization of the LV occurs after the RV, resulting in dyssynchronization of the ventricles and can decrease CO. Pacing both LV and RV will resynchronize the ventricular contraction and improve CO. It is indicated for the reduction of the symptoms of moderate to severe HF (NYHA Functional Class III or IV) in those patients who remain symptomatic despite stable, optimal medical therapy and have an LVEF of 35% or less and a prolonged QRS duration.

› HINT
The paced QRS complex on a triple-chamber pacemaker has a normal width compared to the wide QRS complex on a paced beat with ventricular or dual-chamber pacemaker.

- **What pacemaker problem can lead to competition between the pacemaker and the heart?**
- **Undersensing**

Failure of the heart to sense the underlying electrical activity (P wave or QRS complex) is called undersensing. The pacemaker sends electrical impulses when the heart does not need it. This results in competition.

› HINT
Competition can cause an R-on-T phenomenon resulting in deadly arrhythmias, such as ventricular tachycardia (torsades de pointes).

- **What is it called when the pacemaker detects other activities besides the intended P wave or QRS complex?**
- **Oversensing**

Oversensing results in the chamber not being paced when indicated because the pacemaker sensed other activity. Unwanted signals commonly sensed can be T wave (sensed as a QRS), skeletal muscle myopotentials, and signals from the pacemaker (cross-talking) in which the pacemaker senses the pacemaker spike as intrinsic activity.

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1. CARDIOVASCULAR SYSTEM REVIEW

❯ HINT
On an ECG strip, this can look like a failure to pace.

- **What does a “failure to capture” look like on an ECG strip?**
- **Pacer spike without electrical activity**

Capture is the depolarization of the paced chamber. This is influenced by the amplitude and duration of the stimulus.

❯ HINT
To correct failure to capture, the milliamps (mA) are increased.

**BIBLIOGRAPHY**


Questions

1. Mr. M begins to complain of chest pain. A 12-lead ECG is obtained and shows new-onset ST-segment elevation in leads II, III, and aVF. Which of the following would be the most appropriate assessment following this finding?
   A. Measure the QTc interval
   B. Place the precordial leads on the back of the patient
   C. Perform an ECG with right precordial leads
   D. Extend the left precordial chest leads

2. A patient is admitted to the ICU for a syncopal episode and is being evaluated. A 12-lead ECG and cardiac enzymes are obtained. His troponin levels are elevated and myocardial ischemia is suspected. Which of the following disorders can also elevate troponin levels and would need to be ruled out?
   A. Acute cardiomyopathy
   B. Scleroderma
   C. Sepsis
   D. Glioblastoma

3. ECG findings of ST-segment elevation may be found in other disorders and mimic STEMI. Which of the following disorder may elevate the ST segment?
   A. Hypovolemic shock
   B. Brugada’s syndrome
   C. Dilated cardiomyopathy
   D. Mitral regurgitation

4. A preoperative cardiology consult was obtained on a patient admitted for a hip repair. The cardiologist recommended a cardiac catheterization procedure for evaluation. Significant coronary artery narrowing was found in two vessels. Which of the following is the most likely intervention to be performed?
   A. BMS placement
   B. CABG surgery
   C. DES placement
   D. Thrombolytic therapy

5. You have received report of a patient with a stent placed for a STEMI. During the report, the nurse tells you that there has been a recent increase in ST elevation, which occurred after the patient was bathed. Which of the following would be the most appropriate response?
   A. Ask the patient whether he wishes to be resuscitated.
   B. Ask the nurse whether the ECG patches were replaced during the bath.
   C. Call the physician stat and prepare for cardiac catheterization.
   D. Tell the nurse that it is normal to have an increase in ST segment in the acute period after a stent is placed.
6. Which of the following medications has been found to lower the efficacy of clopidogrel (Plavix) by reducing the formation of the active metabolite?
   A. Proton-pump inhibitors (PPIs)
   B. Calcium channel blockers
   C. β-blocker
   D. Antihistamine

7. A patient presents to the hospital with ACS within a month of a previous STEMI and stent placement. On assessment, the patient’s home medications were found to include aspirin, clopidogrel (Plavix), and a statin. Which of the following is the most accurate statement regarding the restenosis?
   A. This is very common following placement of a BMS.
   B. The patient may have a CYP2C19 deficiency, resulting in a lowering of the efficacy of clopidogrel.
   C. The early restenosis indicates that the patient is noncompliant with the medications.
   D. Following a restenosis, the patient will require a CABG procedure to reopen the stenosis.

8. A β-blocker is frequently ordered following an ACS. In which of the following situations would you expect the β-blocker to be discontinued?
   A. Cardiogenic shock
   B. CHF
   C. An elderly patient with cardiovascular disease
   D. Chronic lung disease without active bronchospasm

9. A patient received eplerenone (Inspra) for LV failure following an AMI. Which of the following laboratory values should be followed closely?
   A. Calcium
   B. Sodium
   C. Potassium
   D. Magnesium

10. A patient presents with an acute inferior wall MI with hypotension, clear breath sounds, and elevated jugular distention. Which of the following diagnosis would be the most likely based on the findings?
    A. CHF
    B. Dilated cardiomyopathy
    C. Septal wall infarction
    D. RV infarction

11. Which of the following class of medications should be avoided in patients with HF?
    A. Antidepressants
    B. Anticonvulsants
    C. Opioids
    D. Nonsteroidal anti-inflammatory drugs (NSAIDs)
12. A cardiologist ordered eplerenone, an aldosterone antagonist, to be started on a patient with HF in the ICU. Which of the following lab values would be a contraindication to administering the aldosterone antagonist?
   A. Potassium < 3.4 mEq/L
   B. Serum creatinine > 2.5 mg/dL
   C. Sodium < 135
   D. pH > 7.45

13. Mr. M presents with symptoms of HF. An echocardiogram finds an EF of 30%. The ECG demonstrates an abnormally wide QRS complex measuring 0.16 seconds. Which of the following therapeutic interventions would be recommended?
   A. Cardiac resynchronization therapy
   B. Implantable cardioverter–defibrillator
   C. Atrial ventricular pacemaker
   D. IABP

14. A patient presents with STEMI and has a history of stroke. Which of the following drugs or drug combinations may be administered to this patient during the PCI?
   A. ASA and prasugrel
   B. Fondaparinux alone
   C. Prasugrel
   D. ASA and clopidogrel (Plavix)

15. A patient presents to the ICU with angioedema after being on captopril (Capoten) for 1 year. Which of the following drugs would you expect the physician to order for this patient instead of captopril?
   A. Lisinopril (Prinivil)
   B. Losartan (Cozaar)
   C. Eplerenone (Inspra)
   D. Nesiritide (Natrecor)

16. A patient with parasternal penetrating chest wounds is sitting bolt upright, agitated, confused, and demonstrating air hunger. What is the most likely cause?
   A. Pericardial tamponade
   B. Flail chest
   C. Bronchial tear
   D. Aortic dissection

17. A patient is suspected of having a pericardial tamponade following a trauma. Which of the following is the best diagnostic test to rapidly identify excess pericardial fluid?
   A. CXR
   B. CT scan
   C. MRI
   D. FAST
18. Which of the following is a symptom of the Beck’s triad?
   A. Increased jugular distention
   B. Agitation
   C. Hypertension
   D. S₃ gallop

19. Which of the following findings on an ultrasonography is a classic sign of pericardial tamponade?
   A. Dilated RV
   B. New-onset mitral regurgitation
   C. Dyskinesia
   D. “Swinging” heart

20. A common finding in a pericardial tamponade includes which of the following?
   A. Water Hammer’s sign
   B. Pulsus alternans
   C. Auscultatory gap
   D. Levine’s sign

21. Following a traumatic pericardial tamponade, which of the following statements would be most accurate in managing the patient?
   A. The priority of care is to intubate and ventilate the patient with positive pressure
   B. Maintain ventilation with bilevel positive airway pressure (BiPAP)
   C. Use higher levels of PEEP to manage the hypoxia
   D. Apply oxygen and avoid intubation if possible

22. Following an MVC, a patient was found to have rib fractures, pneumothorax, and a tibial/fibula fracture. The following vital signs are obtained on admission to the ICU:
   BP 126/78
   HR 138
   RR 22

Which of the following would you suspect based on the above findings?
   A. Pericardial tamponade
   B. Myocardial contusion
   C. Hypovolemic shock
   D. Septic shock

23. Mr. M was involved in a high-speed MVC. Upon presentation to the emergency department, his vital signs are:
   BP 90/40
   HR 98
   RR 32
Fluid bolus is administered with BP increasing to 120/82. A left hemothorax and widened mediastinum is noted on CXR. A CT is placed on the left to drain the hemothorax. BP begins to decrease within 30 minutes of the initial bolus. Which of the following is the most likely cause of this patient’s hemodynamic instability?

A. Aortic aneurysm
B. Myocardial contusion
C. Pericardial tamponade
D. Tension pneumothorax

24. Which of the following would be the most appropriate intervention in managing a hemodynamically unstable traumatic aortic aneurysm?

A. Provide aggressive fluid resuscitation
B. Maintain systolic BP < 120 mmHg
C. Perform passive leg raise procedure
D. Administer dopamine

25. A patient is admitted to the ICU with acute pulmonary edema. He has no history of HF. He had received mediastinal irradiation treatments about 2 years ago. An echocardiogram is ordered. Which of the following forms of cardiomyopathy would be the most likely cause of his congestion?

A. Hypertrophic cardiomyopathy
B. Dilated cardiomyopathy
C. Restrictive cardiomyopathy
D. Constrictive cardiomyopathy

26. A patient presents with HF and dyspnea. He is diagnosed with a hypertrophied cardiomyopathy based on the echocardiogram. Which of the following medications will most likely be used as a first-line management for his HF?

A. Digoxin
B. Nifedipine
C. Verapamil
D. Metoprolol (Lopressor)

27. Mr. M is admitted from the emergency room to the ICU with a diagnosis of hypertensive emergency. His initial BP in the emergency room was 225/122. The following are his admitting vital signs in the ICU:

BP 195/104
HR 86
RR 22

Which of the following is the most correct statement?

A. Hypertensive urgency requiring rapid decrease in systolic BP.
B. Hypertensive emergency is defined as a BP greater than 220 systolic or 120 diastolic.
C. Hypertensive urgency and hypertensive emergency are most commonly caused by acute kidney injury.
D. Hypertensive emergency is defined as hypertension accompanied by organ dysfunction.
28. A patient presents in a hypertensive crisis with a BP of 220/125. Which of the following medications would be the most appropriate for this patient?
   A. Enaloprit IV  
   B. Esmolol IV  
   C. Nifedipine SL  
   D. Clonidine orally

29. Which of the following statements is the most correct regarding esmolol (Brevibloc)?
   A. It should be avoided in renal-failure patients due to excretion through the kidneys.  
   B. The metabolism is via rapid hydrolysis and can be used in hepatic dysfunction.  
   C. The half-life is 4 to 6 hours and may result in accumulation of the drug.  
   D. It should be avoided in tachycardic patients due to reflex tachycardia.

30. An antihypertensive is ordered for a patient experiencing acute pulmonary edema and hypertension. On review of the patient’s chart, it is noted that the patient is allergic to sulfites. Which of the following antihypertensive agents should be avoided in this patient?
   A. Fenoldopam (Corlopam)  
   B. Esmolol (Brevibloc)  
   C. Nicardipine (Cardene)  
   D. Hydralazine

31. A physician ordered an antihypertensive agent to maintain systolic BP between 130 and 150 mmHg in a patient with hemorrhagic stroke. Which of the following antihypertensive agents is the most likely choice by the physician for this patient?
   A. Nimodipine (Nimotop)  
   B. Sodium nitroprusside (Nipride)  
   C. Nitroglycerin  
   D. Nicardipine (Cardene)

32. Which of the following medications is contraindicated in patients with allergies to soy products?
   A. Nimodipine (Nimotop)  
   B. Clevidipine (Cleviprex)  
   C. Nicardipine (Cardene)  
   D. Sodium nitroprusside (Nipride)

33. In a hypertensive emergency, what is the recommended reduction in MAP during initial treatment in the first hour?
   A. 10%  
   B. 15%  
   C. 20%  
   D. 25%
34. Which of the following is the best drug in managing a dissecting thoracic aortic aneurysm?
   A. Sodium nitroprusside (Nipride)
   B. Nicardipine (Cardene)
   C. Hydralazine
   D. Esmolol (Brevibloc)

35. Which class of antihypertensives is preferred in the perioperative period in high-risk cardiac patients?
   A. β-blockers
   B. α-antagonists
   C. Calcium channel blockers
   D. Dopaminergic-1 receptor agonist

36. A patient is admitted with a cocaine overdose. He is tachycardic and hypertensive. Which of the following drugs should be avoided when initially managing this patient?
   A. β-blockers
   B. α-antagonists
   C. Calcium channel blockers
   D. Dopaminergic-1 receptor agonist

37. Which drug therapy is recommended in patients with mitral stenosis to improve LV filling?
   A. α-antagonists
   B. Diuretics
   C. β-blockers
   D. Digitalis

38. A patient presents with a history of intermittent claudication. His femoral pulse is strong but his popliteal and dorsalis pedis are absent. At what level is the obstruction?
   A. Aortoiliac
   B. Femoropopliteal
   C. Distal veins
   D. Popliteal dorsalis

39. A patient is admitted for severe claudication in lower extremities. ABI was noted to be 0.38. The physician has discussed the possibility of a bypass procedure with the patient. What is a normal ABI?
   A. Greater than 1.40
   B. 1.00 to 1.40
   C. 0.80 to 1.00
   D. Less than 0.40

40. This question refers to the patient in Question 39. In the previous patient scenario, which of the following would you expect the physician to order at this time?
A. Cardiac catheterization  
B. MRI  
C. Doppler ultrasonography  
D. Arteriogram

41. Which of the following strokes is more common in patients following CPB surgeries?  
A. Lacunar stroke  
B. Vasospasms  
C. Watershed stroke  
D. Subarachnoid hemorrhage

42. A patient in your unit is hypotensive following a cardiac procedure. He has a significant history of hypertrophied cardiomyopathy. Which of the following vasoconstrictors would be the most appropriate for this patient?  
A. Phenylephrine  
B. Norepinephrine  
C. Dopamine  
D. Epinephrine

43. Your patient has RV failure. Which of the following agents can be used to decrease pulmonary vascular resistance to improve RV function?  
A. Nitroglycerin  
B. Nipride  
C. Epoprostenol  
D. Nicardipine

44. Which of the following potential side effects is the biggest concern when administering recombinant factor VIIa (rVIIa) to a bleeding patient?  
A. A hypersensitivity reaction  
B. Thrombocytopenia  
C. Arterial thromboembolic events  
D. Hemolytic blood transfusion reaction

45. Which of the following statements best describes the clinical significance of a new right bundle branch block (RBBB) following cardiac surgery?  
A. It is usually temporary and of little clinical significance  
B. It requires an external pacemaker until it resolves  
C. Shifts of the axis are uncommon and should be reported immediately  
D. It indicates the need for a biventricular pacemaker

46. While monitoring a post-CABG patient, the ST-segment monitor alarms due to a change in the ST segment. What finding on a 12-lead ECG would indicate acute graft failure?  
A. ST elevation in all the leads  
B. ST elevation in two or more contiguous leads in the territory of the grafted artery  
C. ST elevation in all the anterior leads  
D. ST depression in the inferior leads
47. While turning your post-CABG patient, there is a sudden increase in dark bloody output from the mediastinal tubes. Which of the following would be the most appropriate response?
   A. Page the cardiovascular surgeon stat
   B. Continue watching the patient for any signs of hemodynamic compromise
   C. Prepare the patient immediately for return to the operating room
   D. Strip the mediastinal chest tubes

48. On admission to the ICU from open heart surgery, a patient’s ACT level was 250 seconds. Protamine 25 mg IV was ordered. After administering the protamine, saturations decreased to 89% and the patient became hypotensive. What is the most likely cause for these sudden changes?
   A. Severe bleeding
   B. Pulmonary embolism
   C. Cardiac tamponade
   D. Protamine reaction

49. You have a patient with an implanted pacemaker. The patient does not know what type of pacemaker she has and is not carrying the pacemaker ID card with her. Which of the following is the best method of determining the type of pacemaker this patient has?
   A. Echocardiogram
   B. 12-lead ECG
   C. CXR
   D. Right-sided 12-lead ECG

50. Your patient has an AAI implanted pacemaker. The patient has developed a second-degree Mobitz Type II block. What would you expect the physician’s actions to be at this time?
   A. Watch the patient for signs of development of third-degree heart block
   B. Place an external pacemaker and prepare to replace the current pacemaker
   C. Place a magnet over the pacemaker to assure pacing
   D. Nothing; the patient already has a pacemaker

51. An implantable pacemaker was placed due to a third-degree AV block. One week after placement, it was noted that the pacemaker was not consistently capturing the ventricles. What would be the most likely cause for the change in pacing threshold?
   A. Lead maturation
   B. Fracture of lead
   C. Loss of lead contact with the heart
   D. Kinked lead

52. Your patient has been diagnosed with a dissecting abdominal aortic aneurysm. He is awaiting surgery and during your assessment, you note that his BP is 190/98 and HR is 108. Which of the following medications is most likely to be ordered?
   A. Nitroprusside (Nipride)
   B. Esmolol (Brevibloc)
C. Norepinephrine (Levophed)
D. Dobutamine (Dobutrex)

53. Your patient has an SvO₂ PAC, and the physician has ordered for oxygen delivery (DO₂) and oxygen consumption (VO₂) to be calculated. You note an increase in VO₂ after the CO was improved with dobutamine (Dobutrex). What does this indicate?
   A. Tissues are adequately perfused
   B. The patient was supply independent
   C. DO₂ was not sufficient for the tissue demands
   D. VO₂ was not calculated correctly

54. When caring for a patient after abdominal surgery, you note that SvO₂ has decreased from 68% to 55%. On assessment, you find the following:
   BP 124/64
   HR 88
   RR 20
   SₜO₂ 97%
   Temperature 35.0°C
   CO/CI 4.5 L/min/3.4 L/min/m²
   Hgb/hct 9.5/28%

   Which of the following would be the most likely cause of the decrease in SvO₂ reading?
   A. Shivering
   B. Agitation
   C. Anemia
   D. Hypoxia

55. A patient is admitted to the critical care unit (CCU) following an anterior wall MI. He is hypotensive (82/55) and tachycardic (102 bpm). The echocardiogram found severe LV dysfunction, high LV filling pressures, and aortic regurgitation. The patient was diagnosed with cardiogenic shock. Which of the following interventions would be indicated at this time?
   A. Dopamine (Intropin)
   B. Dobutamine (Dobutrex)
   C. IABP counterpulsation
   D. Norepinephrine (Levophed)

56. While caring for a patient with an IABP, you note that the urine output has significantly decreased in the past hour. What potential complication of IABP would be of concern at this time?
   A. Balloon rupture and air embolus
   B. Timing problems with balloon inflating during systole
   C. Balloon migration
   D. IABP is no longer effective, and the patient is hypoperfused
57. Passive leg raising may be used to determine which of the following:
   A. Presence of hypovolemia
   B. Fluid responsiveness
   C. Presence of jugular venous distention
   D. CVP

58. A patient is admitted for severe pneumonia. It is noted that she has a history of MI. The most current echocardiogram determined LV EF to be 55%. She is classified as a Stage B in HF, according to the ACCF/AHA staging system. Which of the following would be an appropriate drug therapy for this patient to have received?
   A. Ramipril (Altace)
   B. Digitalis (Digoxin)
   C. Isosorbide (Isordil)
   D. Furosemide (Lasix)

59. A patient is in the ICU for respiratory failure and is found to have a significantly elevated BNP level. An echocardiogram shows hypertrophied cardiomyopathy. The patient is diagnosed with CHF. Which of the following medications would you expect the physician to order at this time?
   A. Dobutamine (Dobutrex)
   B. Nifedipine (Procardia)
   C. Furosemide (Lasix)
   D. Digitalis (Digoxin)

60. Which of the following statements best describes the role of BNPs?
   A. BNPs initiate the rennin–angiotensin system and increase the release of aldosterone.
   B. A decrease in BNP levels indicates HF.
   C. BNPs block the release of renin and decrease reabsorption of sodium.
   D. Elevation of BNP works as a vicious cycle in worsening HF.

61. Which type of cardiomyopathy is associated with a normal to high EF?
   A. Restrictive
   B. Constrictive
   C. Dilated
   D. Hypertrophied