CMC Certification Review Course:
   Handout

Session #: 3
Cardiac Trauma Valvular Heart Disease Cardiogenic Shock

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How to use this module:

CMC Review Course
Session 3:
Cardiac Trauma
Valvular Heart Disease
Cardiogenic Shock
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Pericardial Tamponade

Introduction
- Accumulation of blood in the pericardial sac
- Etiology = blunt and penetrating trauma

Signs and Symptoms
- Dyspnea
- Cyanosis
- Beck's triad
- Pulsus paradoxus
- Shock symptoms

Interventions
- Pericardiocentesis
- Open thoracotomy
- Operative intervention

Cardiac Contusion

Introduction
- Bruising of the heart tissue
- Myocardial ecchymosis to myocardial infarction
- Etiology = blunt trauma

Signs and Symptoms
- Chest pain similar to MI
- Chest wall ecchymosis
- S3, or crackles in lung
- Dysrhythmias, ECG changes
- Elevated CK, troponin
Cardiac Contusion

- Interventions
  - Same treatment as for MI
  - Monitor CO and cardiovascular status
  - Monitor for life-threatening dysrhythmias for 24–48 hours

- Complications
  - Cardiac tamponade
  - Cardiogenic shock
  - Myocardial rupture
  - Valve injuries
  - Constrictive pericarditis

Practice Exam Questions

Question #1 - Answer

One of the key pathophysiologic findings in cardiac tamponade is resistance to right ventricular filling. A key sign of this is:

A. Pulsus paradoxus
B. Decreased ECG voltage
C. Kussmaul’s sign — fall in jugular venous pressure (JVP) with inspiration. Other signs of resistance to RV filling are increased CVP and JVD. Narrowed pulse pressure and pulsus paradoxus are due to reduced CO, and decreased ECG voltage is due to fluid accumulation around the heart.
D. Narrowed pulse pressure
Question #2 - Answer

The hemodynamic parameter consistent with a diagnosis of cardiac tamponade is:

A. PAOP = 12 mm Hg
B. CI = 3.4 L/Min/m²
C. BP = 110/80 mm Hg
D. CVP = 12 mm Hg. Classic hemodynamic findings in cardiac tamponade include low CI, elevated preload (CVP & PAOP), equalization of pressure between right & left, and narrowed pulse pressure.

Question #3 - Answer

The most common sign of cardiac contusion is:

A. Chest pain radiating to the left arm
B. Tachycardia. The symptoms are similar to MI with the most common being tachycardia. The chest pain generally does not radiate as in MI. The patient may have diaphoresis and shortness of breath.
C. Diaphoresis
D. Shortness of breath

Question #4 - Answer

The portion of the heart most often injured in a patient with a cardiac contusion is:

A. Right ventricle. The surface of the heart in the anterior position is the right ventricle. It is injured most often in blunt trauma. The most commonly injured valve is the pulmonic valve.
B. Mitral valve
C. Left ventricle
D. Aortic valve
Valvular Heart Disease

Aortic Stenosis (AS)
- Calcific degeneration is the most common cause of AS
- Can affect:
  - Normal trileaflet valve
  - Congenital bicuspid valve
  - Bioprosthetic valve

Question #5 - Answer

The most significant complication of a cardiac contusion is:

A. Congestive heart failure
B. Myocardial infarction
C. Ventricular dysrhythmias. Due to injury to the myocardium, the tissue becomes irritable. The most significant complication is ventricular dysrhythmias requiring monitoring of the patient for 48 hours.
D. Cardiogenic shock
Calcification typically occurs in patients > 70 years of age.
Degeneration of a bileaflet valve typically presents in young adulthood or middle age.

Stenosis may also occur at the subvalvular level:
- Dynamic left ventricular outflow tract (LVOT) obstruction
- Secondary to a subaortic membrane
- Imposes a pressure load on the left ventricle that results in increased intraventricular pressure
- Systolic wall tension

Over time the pressure overload leads to:
- Concentric LV hypertrophy (LVH)
- Increased end-diastolic pressure (EDP)
- Diastolic dysfunction
  - Systolic function is usually preserved until late in the disease
Valvular Heart Disease

Concentric vs. Eccentric Hypertrophy

- **High risk for myocardial ischemia**
- Myocardial oxygen demands are increased due to increased:
  - Pressure work
  - Muscle mass
  - Wall tension

Coronary blood flow (especially endocardium) is reduced due to increased EDP

- > 50% have significant CAD

- Patients with severe AS have a relatively fixed stroke volume (SV)
  - May develop profound hypotension and myocardial ischemia in response to modest vasodilation or exercise
AS

- Calcific AS has a slowly progressive course
  - Symptoms appear late
  - Indicative of severe disease
    - Angina
    - Exertional syncope
    - Dyspnea

AS: Physical Exam

- Small volume, slow rising pulse
- Reduced pulse pressure
- Sustained apical impulse
- Soft S₁

- Single or reverse split S₂
- Prominent S₄
- Long, late peaking ejection systolic murmur
AS: Diagnosis

- ECG
  - LVH with strain pattern
  - Nonspecific ST segment changes
  - Rarely associated with AV block

- Chest X-ray
  - Frequently normal
  - Calcification of the valve may be evident

- Echocardiography
  - Mandatory to confirm the diagnosis and to assess severity

AS Grading

<table>
<thead>
<tr>
<th>Grade</th>
<th>V_{max} (m/sec)</th>
<th>P_{max} (mm Hg)</th>
<th>P_{mean} (mm Hg)</th>
<th>Valve Area (cm²)</th>
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<tbody>
<tr>
<td>Normal</td>
<td>&lt; 1.5</td>
<td>&gt; 25</td>
<td>&gt; 12</td>
<td>&gt; 3.5</td>
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<td>Mild</td>
<td>2.5–3.5</td>
<td>25–50</td>
<td>12–25</td>
<td>2.5–4</td>
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<tr>
<td>Moderate</td>
<td>3.5–4.5</td>
<td>50–80</td>
<td>25–40</td>
<td>1.2–1.8</td>
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<tr>
<td>Severe</td>
<td>&gt; 4.5</td>
<td>&gt; 80</td>
<td>&gt; 40</td>
<td>0.8–0.8</td>
</tr>
<tr>
<td>Critical</td>
<td>&gt; 5.5</td>
<td>&gt; 100</td>
<td>&gt; 50</td>
<td>&lt; 0.6</td>
</tr>
</tbody>
</table>

V_{max} = Maximum aortic jet velocity, P_{max} = Maximum pressure gradient, P_{mean} = Mean pressure gradient

AS: Diagnosis

- Coronary angiography
  - Required in patients
  - > 40 years of age
  - With significant risk factors for CAD
Assessment of the severity of AS in patients with depressed LV function
- Low transvalvular pressure gradient may represent severe AS with secondary low CO
- A low transvalvular pressure gradient may represent minor aortic valve stenosis in a patient with low CO for another reason
- Alcoholism
- Ischemic cardiomyopathy

Low-dose dobutamine stress echocardiography
- Increase in the transvalvular pressure gradient and a failure to increase the measured valve area in response to dobutamine is suggestive of severe AS as the cause of low CO

Symptomatic AS is a surgical condition
- Beta-blockers
  - Provide some relief from angina
  - May protect from tachydysrhythmias
May present with
- Myocardial ischemia
- Dysrhythmias
- CHF

Initial treatment of ischemia
- Oxygen
- Aspirin
- Beta-blockers (in the absence of heart failure)

IABP may be helpful to augment coronary perfusion pressure (CPP)

ACS present
- Urgent angiography
- Combined surgical revascularization and aortic valve replacement (AVR)

Pulmonary Edema
- Diuretics
- Respiratory support

Low-dose sodium nitroprusside
- Slow increments up to 150 mcg/kg/min
- Shown to increase CO in normotensive patients with CHF and AS

Potent vasodilators can precipitate cardiovascular collapse in patients with severe AS, and the treatment is not routine
AS
- Patients with shock are at high risk for progressive cardiac decline or ischemia
  - Induced malignant dysrhythmias

AS: Treatment
- Fluids
- Inotropic support
- IABP
  - May help stabilize the patient prior to urgent AVR

AS Acute Decompensation: Treatment
- Tachydysrhythmias — AF — are poorly tolerated and must be treated promptly
- Be careful with administration of sedatives for cardioversion
  - Likely to cause hypotension
Hemodynamic state is immediately improved by valve replacement even in the presence of preexisting LV dysfunction. Most patients undergoing AVR have preserved LV systolic function but impaired diastolic function. CO and BP are critically dependent on adequate preload and on the maintenance of sinus rhythm.

AS: Postoperative Issues
- Postoperative hypotension is a common manifestation of hypovolemia.
  - EXCEPTION: Difficulty in myocardial protection resulting in myocardial stunning.
    - Treat the cause.
    - Urgent echo can determine if the pathology is coronary ostial pathology.
    - Requires revision surgery.

AS: Postoperative Issues
- AF is not tolerated well and should be treated aggressively.
  - Rapid pacing, particularly ventricular pacing, may cause hypotension.
  - Damage to the His bundle or bundle branches can cause temporary or permanent bundle branch block or complete heart block.
Systolic anterior motion (SAM)
- Can occur in patients with marked LV hypertrophy (basal anterior septum)
- Develop dynamic LVOT obstruction by the anterior mitral valve leaflet after AVR

SAM is uncommon
- Must be considered in any patient who deteriorates despite escalating inotropic therapy, especially patients with a history of
  - HTN
  - LVH
  - Normal systolic function

Diagnosis of SAM is made by echocardiogram
LVOT obstruction is made worse by interventions that reduce LV systolic dimensions
- Hypovolemia
- Inotropic drugs
LVOT obstruction is made worse by interventions that reduce afterload
- Vasodilators
- IABP

Practice Exam Questions
Valvular Heart Disease

Question #1 - Answer
The most common cause of AS is:

A. Rheumatic aortic valve disease
B. Calcific degeneration. It can affect a native valve, a bicuspid valve, or a bioprosthetic valve. Patients are usually > 70 years of age.
C. Pannus formation over the valve
D. Degeneration of a bicuspid valve

Question #2 - Answer
The AS is graded $V_{max} > 4.5$ m/sec; $P_{max} > 80$ mm Hg, $P_{mean}$ 45 mm Hg, and valve area $0.7$ cm$^2$. The patient’s disease is:

A. Mild
B. Moderate
C. Severe. In severe disease the $V_{max} > 4.5$ m/sec; $P_{max}$ >80 mm Hg, $P_{mean}$ 40–50 mm Hg, and valve area 0.6–0.8 cm$^2$. The patient will have severe CHF symptoms.
D. Critical

Question #3 - Answer
A patient presents with symptomatic AS. The most appropriate intervention is:

A. Beta-blocker to reduce angina
B. IABP to augment coronary perfusion
C. Diuretics to reduce pulmonary congestion
D. Surgical replacement of the valve. When the patient is symptomatic, the therapy of choice is surgical replacement of the valve. Most medical treatments do not relieve symptoms.
Aortic Regurgitation (AR): Causes

- Valvular dysfunction
  - Rheumatic aortic disease
  - Degeneration of the bicuspid valve
  - Degeneration of a bioprosthetic valve
  - Endocarditis (native or prosthetic)
- Aortic disease
  - Aortic root dilation
  - Aortic dissection

AR: Pathophysiology

- Imposes a volume load on the LV → progressive LV dilatation and eccentric LV hypertrophy
- LV compliance is increased → large ventricular volumes accommodated with minimal increase in EDP
- With increased LV diameter the wall tension (afterload) is increased → compensatory systemic vasodilation

AR: Pathophysiology

- Arterial EDP is low because of diastolic runoff into the LV
  - Aortic valve opens at a low pressure, but peak systolic aortic pressure is increased due to the high SV
- Arterial pulse pressure is increased
  - May be a baroreceptor
  - Mediated tachycardia
Severe AR
- Risk of myocardial ischemia
- Decreased delivery
- Increased consumption

Chronic Severe AR
- LV progressively fails
- EF falls
- EDP rises
- Congestive cardiac failure develops
- LV dilatation
  - May be profound → functional mitral regurgitation (MR)
  - Severe AR can impair the diastolic opening of the mitral valve → functional mitral stenosis

Acute Severe AR
- Acute volume overload of the LV → sharp increase in EDP → pulmonary edema and shock
AR: Symptoms

- Severe chronic AR (LV dysfunction) produces symptoms of CHF
  - Exertional dyspnea
  - Orthopnea
  - Wide pulse pressure
- Collapsing pulse
  - Fast upstroke and fast downstroke
  - Also known as water hammer pulse
- Diastolic hypotension
- S3

AR: Diagnosis

- ECG
  - Tachycardia
  - Left anterior descending (LAD) artery
  - LVH with strain
- Chest X-ray
  - Increased cardiothoracic ratio
  - Pulmonary venous HTN
  - Interstitial edema
- Echocardiography
  - Determine the cause and severity of AR
  - Assess LV dimensions and function
- Coronary angiography
  - Patients > 40 years old
  - Patients at risk for CAD
Afterload reduction helps to:
- Maintain CO
- Reduce LV wall stress
- Reduce the regurgitant fraction
- Pulmonary congestion
- Diuretics

Prevent dilatation and assist with symptom relief:
- ACE inhibitors
- Calcium channel blockers
- Nitrates
- Hydralazine

AR: Treatment

Alpha receptor blockers: phenylephrine
Both alpha & beta-blockers
- Carvedilol (Coreg)
- Labetalol (Normodyne)

AR: Acute Decompensation
- Aortic dissection (tamponade)
- Aortic valve endocarditis (sepsis)
- Acute prosthetic valve dysfunction
AR: Treatment
- If normotensive, maintain CO with inodilating drugs
  - Dobutamine (Dobutrex)
  - Milrinone (Primacor)
- Inoconstricting drugs increase the regurgitant fraction but may be necessary in the presence of sepsis
  - Norepinephrine (Levophed)

AR: Treatment
- Aortic dissection
  - Acute control of BP with beta-blockers and vasodilators even in the presence of severe AR
  - IABP is contraindicated in moderate or severe AR
  - Acute, severe AR is unstable and unpredictable and is best managed surgically
  - Cardiac arrest and sudden death can occur

AR: Surgical Treatment
- Indications
  - Symptomatic
  - Ventricular dysfunction
  - EF < 50%
  - Ventricular dilatation
  - End-systolic dimension > 5.5 cm
- Severely depressed LV dysfunction (EF < 20%) imposes high perioperative risk and is a contraindication
AR: Postoperative Issues
- LV systolic dysfunction
- Optimization of preload
- Maintenance of a high normal HR
  - Pacing at 90 beats per minute
- Inotropic support
- Systemic HTN
- Damage to His bundle
- After complex aortic root replacement
  - Myocardial stunning
  - Coagulopathy → bleeding
  - Marked systemic inflammatory response system (SIRS)

Practice Exam Questions

Question #4 - Answer

Treatment of aortic insufficiency to prevent ventricular dilatation and assist in symptom relief includes:

A. ACE inhibitors. Other treatments with this same effect include calcium channel blockers, nitrates, hydralazine, and alpha-receptor blockers.
B. Arterial dilators
C. Diuretics
D. Beta-blockers
Valvular Heart Disease

Question #5 - Answer

The complication seen after a complex aortic root replacement is:

A. Myocardial stunning
B. Coagulopathy → bleeding
C. Marked SIRS response
D. All of the above. The surgery is very complex and can lead to coronary ischemia, bleeding, and hypotension due to a SIRS response.

Severe MR can cause:

- LV dysfunction
- AF
- Pulmonary HTN
- RV failure

MR: Etiology

- Connective tissue disorder
- Ventricular dysfunction
- Endocarditis
- Degeneration of a bioprosthetic valve
- Dehiscence of a mitral valve repair
- Rheumatic mitral valve disease
Chronic MR

- LV volume overload
  - Ventricular dilatation
  - Eccentric hypertrophy

MR

- LV afterload is reduced due to systolic ejection into the low impedance left atrium
- Partially offset by LV dilatation → increase in wall stress (increased afterload)
- High preload + low afterload = preserved EF, despite systolic dysfunction
- AF is common once left atrial diameter > 4.5 cm
- Better tolerated than mitral and AS
MR
- MR becomes worse with:
  - Increased SVR
  - AS
  - LV dilatation
    - Increases the regurgitant orifice

Acute MR
- Acute MR has sudden volume overload of the LV
- LV dilatation has not had time to develop, which causes abrupt rise in:
  - LVEDP
  - Left atrial pressure
- Can rapidly lead to:
  - Pulmonary edema
  - Pulmonary HTN
  - Acute RV dysfunction
  - Shock

MR: Symptoms
- Dyspnea
- Orthopnea
- Tachycardia
- Displaced apical impulse quiet S1
- Widely split S2
- S3
- Murmurs
  - Pansystolic
  - High pitched
  - Varies little with respiration
  - Greatest intensity at the apex
  - Radiate to the axilla
- LV Failure
- Pulmonary HTN
MR: Diagnosis

- ECG
  - Tachycardia or AF
  - Left atrial enlargement with P mitrale
- Chest X-Ray
  - Increased cardiothoracic ratio
  - Pulmonary venous HTN
  - Interstitial edema

MR: Diagnosis

- Echocardiography
  - Severity and mechanism of MR
  - Assess left and right ventricular function
  - Determine the presence of pulmonary HTN
- Angiography indicated in patients:
  - > 40 years of age
  - With risk of CAD
MR: Treatment
- Afterload reduction with ACE inhibitors
- Correction of pulmonary congestion with diuretics
- AF may require rate control and anticoagulation

MR: Acute Decompensation
- Causes
  - Dehiscence of a mitral prosthesis or valve repair
  - A torn chordae
  - Endocarditis
  - Myocardial infarction
- Presentation
  - Shock
  - Pulmonary edema

MR: Acute Decompensation Treatment
- IABP and inodilating drugs to promote forward flow
- Diuretics and ventilator support for pulmonary edema
- Treat underlying cause
MR: Indications for Surgery

- Symptomatic
- Asymptomatic with evidence of mild to moderate LV dysfunction
  - EF 30%–60%
  - LV end-systolic dimension > 4.0 cm

MR: Postoperative Issues

- LV systolic dysfunction
  - Preexisting dysfunction
  - Acute increase in LV afterload 2° to newly competent mitral valve
  - New or recurrent AF
  - Often combined with surgery on other valves or with a Maze procedure → long cardiopulmonary bypass times increase postoperative myocardial stunning

MR: Postoperative Issues

- RV dysfunction as a consequence of preexisting pulmonary HTN and acute increases in PVR associated with bypass
- Unexpected hemodynamic instability or pulmonary edema may indicate a problem with the prosthesis
  - Partially open or closed
MR: Postoperative Issues
- SAM
  - Prolapse of the anterior mitral leaflet into the LVOT during systole, causing outflow tract obstruction and MR
- Small nondilated LV
- Use of an undersized annuloplasty ring
- Excessive posterior leaflet tissue causing anterior displacement

Practice Exam Questions

Question #6 - Answer
The most common dysrhythmia seen in patients with MR is:

A. Sinus tachycardia
B. Atrial fibrillation. The most frequently seen dysrhythmia is atrial fibrillation. Treatment focus is control of ventricular rate and anticoagulation.
C. Ventricular tachycardia
D. 3rd degree heart block
Valvular Heart Disease

Question #7 - Answer

Acute decompensation in a patient with mitral insufficiency is generally due to:

A. A torn chordae
B. Myocardial infarction
C. Endocarditis
D. All of the above. The patient will present in shock and pulmonary edema. Treatment includes IABP, inodilating drugs, and diuretics.

Valvular Heart Disease

Question #8 - Answer

The definitive diagnostic following mitral valve surgery in any patient who develops pulmonary edema is:

A. Cardiac catheterization
B. 12-lead ECG
C. Transesophageal echocardiogram (TEE). In the symptomatic patient the TEE can identify a problem with the prosthesis (partially open or closed) versus LVOT obstruction.
D. Chest x-ray

Valvular Heart Disease

Mitral Stenosis (MS)

- Causes
  - Rheumatic heart disease (most common cause)
  - Calcified degeneration
  - Thrombus or pannus on mechanical valve
**Obstruction of LV filling**
- LA becomes pressure and volume loaded
- LV is underfilled
- Increased LAP → pulmonary congestion + left atrial dilatation
- When LA dimension exceeds 4.5 cm, AF is likely

**Severe pulmonary arterial HTN** can cause pressure and volume overload of the RV → functional tricuspid regurgitation → RV failure
- Shunts from the pulmonary vein to the bronchial veins may develop rupture → hemoptysis
- MS + AF = LA thrombus

**Common due to:**
- Chronic underfilling of the left chamber → atrophy, wall thinning, and reduced systolic function
- Heavy calcification of the mitral apparatus (annulus, chordae, papillary muscle) can restrict longitudinal shortening of the LV, impairing systolic function
Valvular Heart Disease

MS: Symptoms
- Pulmonary Congestion
  - Dyspnea
  - Orthopnea
  - Coughing
  - Wheezing
- Mitral Facies
  - Mild cyanosis of lips and cheeks without clubbing of the fingers

MS: Symptoms
- Small volume, irregularly irregular pulse
- Loud S₁
- Opening snap best heard at the apex
- Murmur
  - Low-pitched diastolic rumble
  - Best heard at the apex with the bell of the stethoscope
  - Left lateral position
  - Holding breath in expiration augments the sound
MS: Diagnosis

- ECG
  - AF, or if sinus rhythm, P mitrale
  - Right ventricular hypertrophy (RVH) may be present and is a marker of severity

- Chest X-ray
  - Characteristic features of LA enlargement
  - In severe disease, evidence of enlargement of the pulmonary artery, RV and right atria
  - Pulmonary venous HTN
  - Pulmonary edema

MS: Diagnosis

- Echocardiography
  - Essential for determining the severity of MS

<table>
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<tr>
<th>Severity of MS Based on Valve Area and Pressure Gradients</th>
<th>Mean Transvalvular Pressure Gradient</th>
<th>Mitral Valve Area</th>
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<tr>
<td>Normal</td>
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<td>&lt; 6 mm Hg</td>
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<tr>
<td>Severe</td>
<td>&gt; 12 mm Hg</td>
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MS: Treatment

- No effective medical treatment for severe MS
- Symptomatic relief of pulmonary congestion
  - Diuretics
  - Manage AF
MS: Acute Decompensation
- Pulmonary edema
- Usually seen with:
  - Pregnancy
  - Onset of AF
  - Sepsis

MS: Treatment
- Diuretics
- Respiratory support
- Management of AF
- Inotropic support
- IABP for hypotension

MS: Surgical Treatment
- Indications
  - Moderate or severe MS
  - Symptomatic
  - Evidence of LA or pulmonary HTN
**Postoperative issues**
- Hemodynamic state is immediately improved
- LA pressure reduced, PA pressure reduced,
  RV function improved
- **Exception:** Patients with severe pulmonary hypertension are at risk for RV dysfunction
- Can have development of systolic LV dysfunction
- New or recurrent AF

**Tricuspid Abnormalities**
- RV volume overload (atrial septal defect)
- RV pressure overload (mitral valve disease)
- RV systolic dysfunction (stunning)
Tricuspid Abnormalities
- RV volume overload, initially well tolerated
- Forward SV is maintained by compensatory RV dilatation
- RV dilatation exacerbates tricuspid regurgitation → RV systolic failure

Tricuspid Abnormalities
- Increased systemic pressure
  - Elevated CVP
  - Pulsatile liver

Tricuspid Abnormalities
- Frank RV failure
  - Fatigue
  - Ascites
  - Systemic edema
  - Weight gain
Tricuspid Abnormalities

- Diagnosis
  - Echo important for assessing:
    - Severity
    - Mechanism of the tricuspid valve pathology

Tricuspid Abnormalities: Postoperative Issues

- RV failure
- LV failure (gross dilatation of the RV)
- High systemic venous pressure and low CO
- Damage to the AV node → complete heart block
- 20% after tricuspid replacement

Practice Exam Questions
Valvular Heart Disease

The patient presents with low-pitched diastolic murmur best heard at the apex of the heart while lying in a left lateral position. The valve abnormality is:

A. Mitral valve regurgitation
B. Mitral valve stenosis. Low-pitched diastolic murmur heard best at apex while lying in a left lateral position and holding breath at expiration.
C. AR
D. AS

Question #9 - Answer

A diagnostic finding in mitral stenosis signifying severity of the disease is:

A. EF of 60%
B. Left atrial enlargement
C. Right ventricular hypertrophy. RVH is a later sign and denotes severe disease.
D. Enlarged pulmonary artery

Question #10 - Answer

Cardiogenic Shock
Cardiogenic Shock

- Reduction in flow of oxygen to the myocardium
- Oxygen demand > supply
- Diminished contractility → diminished CI

Cardiogenic Shock

- 90% mortality without medical treatment
- Predictors:
  - Blood lactate > 5 mmol/L
  - CO
  - Arterial pressure

Shock Definition

- Cell hypoxia related to decreased perfusion
- Potentially fatal if not identified and treated
Etiology
- Myocardial infarction
- Wall motion abnormalities
- Dangerous heart rhythms
- Rupture of heart muscle
- Papillary muscle rupture
- Septal rupture
- Slow heart rhythms
- Severe electrolyte disturbances
- Acid base imbalance
- Aortic stenosis
- Drug toxicity (cocaine)
- Cardiovascular surgery
- Cardiomyopathy
- Cardiac contusion
- Advanced septic shock

Initial Stage
- Cellular changes
- No initial signs or symptoms
- Increased glucose
- Increased lactate level
- Decreased base deficit

Compensatory Shock
- Neural (fight or flight)
- Hormonal
- Chemical
**Cardiogenic Shock**

- Failure of compensatory mechanism
  - Brain
  - GI tract
  - Pancreas
  - Kidneys
  - Liver
  - Lungs
  - Myocardium

**Progressive Shock**

- Failure of compensatory mechanism

**Refractory Shock**

- Progression to multiple organ failure
  - Cardiac failure
  - Acidosis
  - Blood clotting
  - Cerebral ischemia
Goals of Therapy

- Increase CI
- Increase contractility
- Decrease afterload
- Optimize preload
- Optimize heart rate

Interventions

- Limit infarct size
  - Aspirin
  - Beta-blocker
  - ACE inhibitor
  - Thrombolytics
  - PCI
- IV nitroglycerin
- Calcium channel antagonist
- CV surgery
- IABP
- Ventricular-assist device
Cardiogenic Shock

Concepts
- Balloon inflation
- Improved coronary perfusion
- Balloon deflation
  - Decreased afterload

Complications
- Limb perfusion
- Contraindications
- Pulseless patient

Practice Exam Questions
The hemodynamic findings consistent with a diagnosis of cardiogenic shock are:

A. CI < 2.0 L/min/m² & SVR > 2000 dynes. CI < 2.0 l/min/m², SVR > 2000 dynes; BP < 90 mm Hg systolic; PAOP < 8 mm Hg or > 22 mmHg; SVO₂ < 60%
B. PAOP 18 mm Hg & CI 2.8 L/min/m²
C. BP 110/72 mm Hg & PAOP > 22 mm Hg
D. SVR > 2000 dynes & SVO₂ 70%

The patient is in the initial stage of cardiogenic shock. A finding in this stage would be:

A. BP < 90/60 mm Hg
B. Respiratory acidosis
C. Lactate level < 2 mmol/L
D. Base excess −4. During the initial stage (cellular shock) the BP is normal, the patient is in respiratory alkalosis, the lactate level begins to rise (>2 mmol/L), and the base excess is < −2.

Treatment for cardiogenic shock should include strategies to:

A. Improve CI
B. Decrease afterload
C. Optimize preload
D. All of the above. Strategies include increasing CI by increasing contractility, decreasing afterload, and optimizing preload.