25. Support of the Failing Circulation Shock

Michael Wall, M.D., F.C.C.M.

Shock is a syndrome of impaired tissue oxygenation and perfusion where oxygen demands exceed oxygen supply. Shock can result from a decrease in oxygen delivery, decreased tissue perfusion or impaired utilization of oxygen. This chapter will outline the important considerations for the diagnosis and management of shock.

I. Clinical Presentation
   A. History
   B. Physical exam, signs, symptoms
      1. Neurologic
         a) Level of consciousness
         b) Paralyzed ?
      2. CV
         a) Heart rate
         b) Pulse pressure
         c) Blood pressure
         d) Valvular disease ?
         e) CHF?
      3. Pulmonary
         a) Oxygen saturation
         b) Work of breathing
      4. Renal
         a) Oliguira/anuria
         b) Acute renal failure
      5. GI
         a) Exam
         b) Shock liver
      6. Skin/extremities
         a) Temperature
         b) Pulses
         c) Edema
   C. Laboratory
      1. Arterial blood gas
      2. Liver function, bilirubin
      3. Lactate
      4. $ScvO_2/SvO_2$
      5. CBC
      6. Coagulation tests
      7. Electrolytes, BUN, Cr, Ca^{++}, Mg^{++}
   D. Imaging: role of:
      1. CXR
      2. Abdominal films
      3. Chest/abdominal CT
      4. Echocardiogram
      5. CT angiogram (pulmonary)

II. Types of Shock
   A. Understand initial physical exam, differential diagnosis and hemodynamic profiles of
      1. Hypovolemic shock
      2. Distributive shock
      3. Cardiogenic shock
      4. Obstructive shock

III. Physiologic Principles
   A. Oxygen delivery

1. $DO_2 = CaO_2 \times CO$
   a) $CaO_2 = (1.38 \times Hb \times SaO_2) + (0.003 \times PaO_2)$

B. Perfusion
1. Determinants of blood pressure
   a) Ohm's law (V=ri)
   b) MAP = CO x SVR
2. Determinants of cardiac output
   a) CO = HR (rhythm) x SV
   b) Stroke volume determined by
      (1) Preload (Starling's law)
      (2) Afterload
      (3) Contractility
3. Perfusion pressure
   a) Cerebral perfusion pressure
   b) Coronary perfusion pressure
   c) Renal perfusion pressure
   d) Autoregulation

C. Oxygen supply and demand
1. Determinants of $ScvO_2$ and SvO_2
   a) SaO_2
   b) VO_2
   c) CO
   d) Hb
2. $ScvO_2$ vs SvO_2
3. Causes of increased and decreased $ScvO_2/SvO_2$
4. Pitfalls of $ScvO_2/SvO_2$ monitoring

IV. Initial Management
A. Restore perfusion rapidly while looking for and treating initial cause
B. Support airway and breathing
1. Role of intubation, mechanical ventilation and noninvasive ventilation
2. Work of breathing (WOB)
3. Goals
   a) Decrease WOB
   b) $SaO_2 > 95\%$

C. Restore perfusion
1. Goals
   a) MAP ≥ 65 mmHg
   b) Adequate cardiac output
      (1) Rate, rhythm, preload, afterload, contractility
   c) Adequate Hb
   d) $ScvO_2 > 70\%$ or $SvO_2 > 60\%$
   e) Normal lactate, pH, base deficit
   f) Urine output > 0.5 cc/kg/hr

D. Identify cause

V. Specific Management
A. Hypovolemic shock
1. ATLS algorithm
2. Crystalloid
3. Colloid
4. Blood and blood products

B. Distributive shock
1. Early goal directed therapy
2. Steroid use
3. Surviving sepsis guidelines
4. Antibiotic use
5. Diagnosis and treatment of:

a) Anaphylactic shock
b) Adrenal crisis, thyroid storm
c) Neurogenic shock
d) Toxic/drugs

C. Cardiogenic shock
1. Diagnosis and treatment of:
a) Acute myocardial dysfunction
(1) Ischemia/infarction
(2) Cardiomyopathies
(3) Structural defects (valve dysfunction, septal defects, etc.)
(4) Acute right heart failure

D. Obstructive shock
1. Diagnosis and treatment of:
a) Tamponade
(1) Post cardiac surgical tamponade vs traditional tamponade
b) Restrictive myocarditis and pericarditis
c) Obstructive cardiomyopathies
d) Pneumothorax

VI. Monitoring
A. Indications, contraindications, placement, maintenance and complications of:
1. Arterial lines
2. Central lines
3. Pulmonary arterial catheters

B. Role of echocardiography in the diagnosis of shock
1. TEE vs TTE

C. Understand the following monitoring devices and laboratory tests:
1. Noninvasive blood pressure
2. ECG
3. Noninvasive CO monitors
a) Li^{++} dilution
b) Esophageal doppler
c) Pulse contour analysis
4. Pulse pressure variation
5. Tonometry
6. Tissue oxygen tension
7. Lactate
8. Base deficit
9. Near infrared spectroscopy

VII. Pharmacologic Therapy in Shock
A. Antiobiotics
B. Steroids
C. Vasodilators
1. Nitroglycerin
2. Nitroprusside
3. Nesiritide
4. Nicardipine
5. Nitric Oxide
6. Epoprostenol (Flolan)

D. Vasoconstrictors
1. Phenylephrine
2. Norepinephrine
3. Vasopressin

E. Inotropes
1. Epinephrine
2. Dopamine

F. Inodilators

1. Dobutamine
2. Milrinone/inamrinone

G. Miscellaneous
1. Digoxin

VIII. Mechanical Support Options
A. Indications, contraindications, complications for:
1. Intraaorta balloon pump
2. Right/left ventricular assist devices
3. Extracorporeal membrane oxygenators (ECMO)

This chapter is a revision of the original chapter authored by Robin J. Hamill-Ruth, M.D.

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12. Eastern Association for the Surgery of Trauma. http://www.east.org/tpg.html. Best site for evidence-based trauma care guidelines
13. Surviving Sepsis Campaign. www.survivingsepsis.org. This Web site contains guidelines for the management of severe sepsis and septic shock, information on the Surviving Sepsis Campaign, and sepsis-related information and resources for healthcare professionals, patients, and the general public
14. Trauma.org. http://www.trauma.org. Image bank and links

QUESTIONS

25.1. A PA catheter is placed in a critically ill trauma patient in the supine position with good wave tracing and PA occlusion at 52 cm. The patient is then placed head up because of a high intracranial pressure. The pulmonary artery tracing is noted to be damped several minutes later. What is the most likely cause for the damped wave form?

A. PA catheter tip has moved to West lung zone 1 from zone 3
B. Tricuspid regurgitation
C. Congestive heart failure
D. Transducer zeroed at the hip
E. Increased ICP

25.2. You are called to evaluate a patient just admitted from the emergency room after a "head on" MVC. A CVP catheter was placed in the ER. The patient currently has a CVP of 24. Auscultation of the chest reveals bilateral breath sounds without rales. What is the most likely diagnosis?

A. Chordae tendinae rupture
B. Tension pneumothorax
C. Anterior myocardial infarction
D. Myocardial contusion
E. Aortic transection

25.3. A 70kg 65 year-old intubated female is admitted to the ICU with hypotension, altered mental status, hypoxemia, and anuria. Vitals: BP = 60/30, HR = 145, Temp = 35.5° C. No history is available. ECG = tachycardia with lateral ST depression. A PA catheter placed by the ER resident reveals CVP = 1 mmHg, PAOP = 7 mmHg, CI = 1.8 L/min/m2 with SVRI = 3200 dyne/sec/cm-5. Arterial blood gas results show pH = 7.15, $PaCO_2$ = 40, PaO_2 = 89, HCO3 = 14.

A. Rapid infusion of 1000 cc normal saline
B. Start a dobutamine infusion
C. Start a milrinone infusion
D. Increase the mechanical ventilation to normalize the pH
E. Start inhaled nitric oxide

25.4. A 45 year-old diabetic make is admitted to the ICU with hypotension, nausea, diaphoresis, severe dyspnea and hypoxemia of 4 hours duration. His PMH is significant for 70 pack-year history of cigarette abuse. After receiving 2 liters of crystalloid, his BP = 82/30, HR = 130, RR = 36, arterial O_2 saturation = 88% on 100% face tent. ECG shows sinus tachycardia with a left bundle branch block. CVP = 18, PAOP = 24, CI = 1.3 L/min. Which of the following therapies is most appropriate?

A. Metoprolol 5g IV
B. 1 liter NS bolus
C. Start nitroglycerine infusion
D. Start phenylephirine infusion
E. Start dobutamine infusion

25.5. Which of the following should decrease the mixed venous O_2 saturation?

A. Fluid resuscitated, early sepsis
B. End-stage liver disease
C. Malignant neuroleptic syndrome
D. Dobutamine
E. Neuromuscular blockade

25.6. The greatest increase in oxygen delivery will be accomplished by:

A. Increasing the PaO_2 from 79 mmHg to 250 mmHg
B. Adding dobutamine to increase to cardiac output from 4.5 to 6.0 L/min
C. Transfusing to increase hemoglobin from 6.5 to 11.0 gm/dL
D. Increasing the PAOP from 18 to 22 mmHg
E. Increasing the patients core temp from 33.5 to 35.2 degrees

26. Diagnosis and Treatment of Dysrhythmias

Michael Wall, M.D., F.C.C.M.

Dysrhythmias are extremely common in critically ill patients. An understanding of the pathophysiology and treatment of common dysrhythmias is essential for the ICU clinician.

I. BLS and ACLS Algorithms

II. Bradycardia and Sinus Node Dysfunction
  A. Pathophysiology, diagnosis and treatment of:
    1. Bradycardia
      a) Non-pathologic
        (1) Sinus bradycardia
        (2) Nocturnal bradycardia
        (3) Sinus arrhythmia
        (4) Wandering pacemaker
      b) Pathologic conditions
        (1) Multiple etiologies, for example; increased ICP, sepsis, following MI, and heart transplantation, drugs, etc.
    2. Sinus node dysfunction
      a) Persistent sinus bradycardia
      b) Sinus pause or arrest
      c) Sinus atrial exit block
      d) Bradycardia/tachycardia syndrome

III. Vagally Medicated Bradycardias
  A. Pathophysiology, diagnosis and treatment of:
    1. Sinus arrest
    2. Bradycardia
    3. Heart block

IV. Conduction Disturbances
  A. Pathophysiology, diagnosis and treatment of:
    1. Left bundle branch block (LBBB)
    2. Right bundle branch block (RBBB)
    3. Bifascicular block
    4. First-degree AV block
      a) Normal QRS
      b) Wide QRS
    5. Second-degree AV block
      a) Type I (Wenckebach)
      b) Type II
      c) Third-degree AV block
        (1) Acquired
        (2) Congenital

V. Premature Beats
  A. Pathophysiology, diagnosis and treatment of:
    1. Atrial
    2. Junctional
    3. Ventricular

VI. Supraventricular Tachycardia
  A. Pathophysiology, diagnosis and treatment of:
    1. Paroxysmal Supraventricular Tachycardia
      a) AV nodal reentry tachycardia (AVNRT)
      b) AV reentry tachycardia (AVRT)

c) Intraatrial reentry
d) Automatic atrial tachycardia
e) Sinus nodal reentry tachycardia (SNRT)
2. Wolff-Parkinson-White and variants
3. Nonparoxysmal AV junctional tachycardia
4. Paroxysmal atrial tachycardia with block
5. Automatic AV junctional tachycardia
a) AKA junctional ectopic tachycardia
6. Multifocal atrial tachycardia
7. Sinus tachycardia
8. Atrial flutter
9. Atrial fibrillation
a) Prophylaxis for cardiac surgery

VII. Ventricular Arrythmias
A. Pathophysiology, diagnosis and treatment of:
1. Premature ventricular contractions
2. Nonsustained ventricular tachycardia
3. Sustained monomorphic ventricular tachycardia
4. Sustained polymorphic VT
5. Ventricular fibrillation
6. Idiopathic ventricular tachycardias
a) RV outflow tract tachycardia
b) Fascicular ventricular tachycardia
(1) AKA Verapamil - sensitive ventricular tachycardia, Belhassen's ventricular tachycardia
c) Arrhythmogenic right ventricular dysplasia/cardiomyopathy (AVRD)
d) Congenital long QT syndrome
e) Acquired QT prolongation
f) Brugada syndrome
g) Catecholaminergic polymorphic VT
h) Short coupled torsade de pointes
i) Short QT syndrome

VIII. Radiofrequency Ablation
A. Atrial fibrillation
B. Atrial flutter
C. AVNRT
D. Accessory pathways (WPW)
E. Ectopic atrial tachycardia
F. AVRD

IX. Pacemakers and Implantable Cardiac Defibrillators (ICD)
A. Pacemakers
1. 5 letter code
2. Temporary
a) Transvenous
b) Transcutaneous
c) Transesophageal
d) Epicardial
3. Permanent
4. Indications
5. Contraindications
6. Electromagnetic interference
7. Effect of magnet application
B. ICD
1. Indications
2. Electromagnetic interference

3. Effect of magnet application

C. Biventricular pacing
1. Indications

This chapter is a revision of the original chapter authored by Stuart M. Lowson, M.B., B.S.

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11. American Heart Association. http:www.americanheart.org. Guidelines for management of acute coronary syndromes and related cardiovascular topics

Phone Numbers

Boston Scientific: CRM Division (formerly Guidant)	1-800-cardiac
Medtronic	1-800-medtron
St. Jude	1-800-paceicd

QUESTIONS

26.1. A 30 year-old female with a history of Wolff-Parkinson-White Syndrome goes into atrial fibrillation with a wide QRS in the PACU. Her HR is 150, BP 92/63. SaO_2 100%. Spontaneously breathing 2L nc. Which of the following is the most appropriate initial therapy?

A. Verapamil 5mg IV
B. Metoprolol 5mg IV
C. Digoxin 0.5 mg IV
D. Cardioversion
E. Amiodarone 150mg IV

26.2. A VOO pacemaker
- A. Paces the ventricle
- B. Paces both the atria and ventricle
- C. Senses the ventricle
- D. Senses both the atria and the ventricle
- E. Is inhibited by both the atria and ventricule

26.3. A 75 year-old male acutely goes into atrial fibrillation 2 days following CABG. His ejection fraction was 25% on postoperative TEE. His HR is 150, BP 88/62, SaO_2 90% on 2L nc. He is confused. The most appropriate initial therapy is:
- A. Verapamil 5mg IV
- B. Metoprolol 5 mg IV
- C. Digoxin 0.5 mg IV
- D. Cardioversion
- E. Amiodarone 150 mg IV

26.4. A 50 year-old male with COPD and intubated for severe pneumonia has 3 blocked P waves in a row immediately following endotracheal suctioning. This spontaneously resolves. What is the most appropriate therapy?
- A. Tilt table testing
- B. Dopamine infusion
- C. Transvenous pacing
- D. Permanent pacemaker placement
- E. Observation

26.5. A 70 year-old male patient is in the ICU for alcohol withdrawal and is being sedated with haloperidol. He develops hemodynamically stable torsade de pointes. Which of the following is the most appropriate initial therapy?
- A. Magnesium
- B. Procainamide
- C. Ibutilide
- D. Sotalol
- E. Cardioversion

26.6. A 70 year-old patient is intubated and undergoing CPR with chest compressions for VF. What is the most appropriate way to ventilate this patient?
- A. 2 breaths following every 30 compressions
- B. 4 breaths following every 30 compressions
- C. 20 breaths per minute continuously
- D. 8-10 breaths per minute continuously

26.7. Which of the following pacemaker modes would have the least interference from electrocautery?
- A. VOO
- B. DDD
- C. AAI
- D. VVI
- E. DVI

27. Diagnosis and Treatment of Myocardial Ischemia

Ruben J. Azocar, M.D.

A 66-year-old male presents for elective shoulder surgery. Nine months before he suffered an acute myocardial infarction in his left anterior descending coronary artery and underwent emergent cardiac catheterization and placement of 3 drug eluting stents. He stopped his clopidogrel and aspirin 8 days prior to his current surgery as instructed. A recent dobutamine echocardiogram demonstrated no evidence of ischemia and he has excellent exercise tolerance.

Coronary artery disease (CAD) remains one of the leading causes of death in the industrial world and it is the most common cause of death in the US. Clinically, a continuum ranges from a temporary imbalance between coronary oxygen consumption and delivery representing myocardial ischemia (that may not result in myocardial damage) to myocardial infarction and death. As anesthesiologist and intensivists we might encounter patients arriving to the emergency department with myocardial ischemia/myocardial infarction but also with patients developing such changes in the perioperative period. As there are some differences between the two clinical presentations, notes describing pertinent issues in regards to the perioperative ischemia/infarction are included in the chapter.

Classification

There are many classifications based on the extent of the infarction. In a transmural MI the full thickness of myocardium, from endocardium to epicardium, is involved. In a nontransmural MI the damage to the myocardium is limited to the endocardium. The endocardial /subendocardial zones are poorly perfused compared to rest of heart making them more vulnerable to ischemia. Clinically the use of the terms Q Wave MI (indicating the presence of Q waves on ECG) or non Q Wave MI are used as descriptors. A differentiation between the presence of ST segment elevation on ECG (STEMI) or no ST segment elevation on ECG (NSTEMI) has been also used. Although it does not indicate the degree of myocardial involvement, STEMI patients have higher early morbidity and mortality. However this classification does not predict long term sequelae. Finally, emerging data suggest that ischemia/infarction occurring in the perioperative period (PMI) may differ from non-perioperative injury.

I. Factors that determine myocardial oxygen delivery/supply
   - A. Heart rate
     1. When the HR increases, it is the diastolic time which decreases. Significant coronary blood flow occurs during diastole, particularl;y to the left heart
   - B. Coronary perfusion pressure
     1. The aortic diastolic BP is the primary determinant of coronary perfusion pressure
     2. Ventricular end diastolic pressure (EDP) (the higher the EDP the larger the compromise to the subendocardial circulation)
   - C. Arterial oxygen content
     1. Primary determinants:
        a) Arterial oxygen saturation
        b) Hemoglobin concentration
   - D. Coronary vessel diameter
     1. Thrombus superimposed on ulcerated or unstable atherosclerotic plaque; most common cause of MI
     2. High Grade (>75%) stenosis with coronary vasospasm

II. Factors that determine myocardial oxygen demand/consumption
   - A. Basal requirements
     1. Increased metabolic demands such as physical exertion, severe HTN or severe aortic stenosis elevates the demand
   - B. Heart rate
     1. Increases in HR increase oxygen demand
   - C. Wall tension
     1. Increases in wall tension increase oxygen demand

D. Contractility
   1. Increases in contractility increase oxygen demand

III. Pathophysiology of Myocardial ischemia/infarction
   A. Occlusion of Coronary Circulation
      1. Disruption of Vascular Endothelium with Unstable Plaque that Stimulates Intracoronary Thrombus.
      2. 20-40 min to irreversible cell damage.
      3. Most common cause on the patient coming to ED
      4. Plaque forms over many years to decades
         a) Fibromuscular Cap & Underlying Lipid-Rich Core
         b) Shoulder Region – Cap meets Vessel Wall
         c) Platelet-Mediated Thrombus formation on any disruption of vessel wall
   B. "Demand ischemia"
      1. Imbalance between myocardial oxygen consumption and delivery. Appears to be the most likely cause in the early Perioperative period

IV. Extent of Myocardial Damage
   A. Level of Occlusion in Coronary Artery
      1. More proximal thrombus, more myocardium at risk
   B. Length of Time
   C. Collateral Circulation

V. PMI Mechanisms
   A. Risk of PMI peaks within first 3 postoperative days (Days 0/1 highest)
      1. Mobilization of fluids, resulting in an increase on preload
      2. Pronounced thrombotic risk by activation of coagulation cascade during surgery
      3. Increases in HR/BP associated with catecholamines/ postoperative pain worsens myocardial supply/demand mismatch
      4. Recent data suggest ischemia starts at end of surgery and emergence from anesthesia.
      5. It might possible that most "early" PMI are related to demand ischemia and as time passes to plaque rupture and thrombosis similar to the general population

VI. Risk Factors for Arteriosclerosis/MI
   A. Hyperlipidemia
      1. Major Component of Plaque
      2. High LDL levels associated with higher MI rate
   B. Diabetes Mellitus
   C. Hypertension
   D. Smoking
   E. Family History
   F. Male Gender
   G. Age

VII. Risk factors for PMI
The ACA/AHA guidelines provide a blueprint for risk stratification, determining need for further interventions and management of patients with coronary stents. Basically establishes a relationship between patient risk factors and exercise tolerance and surgery risk factors to determine the need for further interventions prior to surgery and anesthesia.
   A. Patient factors:
      1. Active cardiac conditions characterized by
         a) Unstable coronary syndromes, characterized by unstable or severe angina, or recent MI (less than one month)
         b) Decompensated heart failure
         c) Significant arrhythmias
         d) Severe valvular disease
   B. Clinical risk factors include
      1. History of heart disease

2. History of compensated or prior heart failure,
3. History of cerebrovascular disease,
4. Diabetes mellitus
5. Renal insufficiency
6. Note that other risk factors such age (>70), abnormal ECG , uncontrolled HTN are not listed as risk factors anymore as there is no evidence that they increase risk of PMI independently.

C. Surgery factors
1. Vascular surgery (High risk)
2. Intermediate risk :Intraperitoneal, intrathoracic, CEA, head and neck, orthopedic and prostate surgery
3. Low risk Endoscopic, ambulatory, cataract ,breast and superficial surgery

D. Recent Stent Placement
1. Must know type of stent (bare metal vs drug eluting, length of time since insertion and status of anticoagulation
2. Follow recent ASA practice alert

VIII. Signs & Symptoms
A. Chest pain described as a pressure sensation, fullness, or squeezing in the midportion of the thorax
B. Radiation of chest pain into the jaw/teeth, shoulder, arm, and/or back
C. Dyspnea or shortness of breath
D. Epigastric discomfort with or without nausea and vomiting
E. Diaphoresis or sweating
F. Syncope or near-syncope without other cause
G. mpairment of cognitive function without other cause
H. More common in early morning or around time of physical activity
I. Some MI's are without symptoms (Higher with DM)
J. PMI might be asymptomatic (silent)

IX. Diagnosis
A. Electrocardiography
1. ST segment elevation
2. Nonspecific ST and/or T wave changes

B. Blood Tests
1. Release of specific enzymes and cell wall proteins of myocardial cells that become ischemic or infarted
2. CK-MB, troponin
3. Change of serum levels indicate amount of heart muscle affected

C. Echocardiography
1. Identifies heart region/ coronary anatomy that is affected by MI
2. May be old or new wall motion changes
3. Usefulness for diagnosis is limited

D. Concerns regarding diagnosis of PMI
1. Pain may be masked by analgesics
2. Telemetry misses significant ST changes
3. 12 lead ECG only diagnostic 50% time
4. $V_{2\text{-}V4}$ with majority of 12 lead ECG changes
5. Usually NSTEMI in nature
6. Serum biomarkers
   a) CK-MB, less sensitive/specific in PMI
   b) Troponin T & I, markers of choice; Can be elevated in CHF, PE, Sepsis.
   c) Diagnosis for acute MI requires change from baseline.

X. Therapy
A. Goals:
1. Restoration of Coronary Blood Flow
2. Limit myocardial damage

B. Antiplatelet Agents

1. Aspirin (ASA)
   a) Immediate therapy 160-325 mg upon signs or symptoms of MI; interferes with platelet adhesion and cohesion at disruption site
   b) Greatly reduces AMI Mortality
   c) Long term use once MI diagnosed
   d) Clopidogrel (Plavix) & Ticlopidine (Ticlid): Not superior to ASA; Used in ASA allergy
2. Oxygen
   a) Maximize oxygen carrying capacity of RBCs
   b) No published studies that demonstrates improved morbidity/mortality with supplemental oxygen
3. Nitrates
   a) Nitrates are given in the setting of MI with: CHF, persistent ischemia, long term Use, HTN and Large Anterior Wall MI
   b) Reduce preload and afterload
   c) Decreases myocardial oxygen requirements
   d) Effective in early stages (48hrs) of MI but no long term mortality advantages
4. Beta Blockers
   a) Recommended in MI early and often!
   b) Reduces Mortality in Acute MI setting
      (1) 28% reduction mortality if used in first week post MI
      (2) Indefinite Use
   c) Decrease oxygen demand by lowering heart rate and force of contraction and increases supply by increasing diastolic time
   d) Antiarrhythogenic, Decreases VF threshold
   e) Antagonizes adrenergic effects of catecholamines
5. Unfractionated Heparin
   a) Inhibits additional formation and propagation of thrombus
6. Glycoprotein 2b/3a Antagonists
   a) Inhibit Platelet Aggregation by antagonizing receptors on platelets (glycoprotein 2b/3a) bind fibrinogen
   b) Use during PCI reduces mortality, reinfarction, and need for further revascularization procedures
   c) Abciximab, Eptifibatide, & Tirofiban
7. ACE Inhibitors
   a) Recommended in MI patients within first 24 hours
   b) Reduces Afterload via Vasodilatation
   c) Continued if MI patients have:
      (1) CHF
      (2) LVEF < 40%
      (3) HTN
      (4) DM
   d) Relative contraindication if hypotensive or with declining renal function
8. Lipid Management
   a) New evidence that all MI patients should be on statin therapy regardless of HDL/LDL levels(Schwartz et al. *Am J Cardiol* 2005)
   b) Statins can reduce circulating markers of inflammation within days post MI
   c) Improve coronary endothelium function
   d) Reversal of prothrombotic states
   e) Reduces atherosclerotic plaque volume
   f) In a review of 6 randomized, controlled trials high intensity statin therapy (atorvastatin 80mg) reduced early recurrent ischemic events compared to moderate therapy (40mg) or placebo
9. Fibrinolytics
   a) Indicated for MI and ST elevation > 0.1mV in 2 contiguous leads or new bundle block
   b) Restore coronary blood flow in 50-80% of cases
   c) Best when door to needle time is less than 30 minutes
   d) Runs risk of significant bleeding in PMI patients
10. Percutaneous Coronary Intervention (PCI)

a) "Door to balloon " goal of less than 90 minutes
b) Restores coronary flow 90-95% of MI patients
c) Better than fibrinolysis in short term mortality, bleeding rates, and reinfarction rates
d) Better if pt in cardiogenic shock
e) Stents reduce need for subsequent target-vessel revascularization
f) Postoperative MI in Noncardiac Surgery - PCI preferable to Fibrinolysis in immediate postoperative phase due to lower risk major bleeding
g) Berger et al. with 48 postop pts for PCI
   (1) 65% survival rate much better than those untreated
   (2) No significant surgical site bleeding in cath lab
   (3) Pts with sudden onset ST elevation from acute thrombotic occlusion did best with immediate intervention (PCI vs CABG) compared to no intervention in postop pt.

11. Surgical Revascularization (CABG)
    a) Urgent if failed PCI and unstable pt
    b) Must have anatomy amenable for grafting
    c) If Mechanical Complications of MI present
       (1) Ventricular Septal Defect
       (2) Free Wall Rupture
       (3) Acute Mitral Regurgitation
    d) Emergency procedure riskier than elective
       (1) 3-7 days post MI similar risks to elective
    e) Elective CABG improves survival in pts with:
       (1) Left Main Coronary Disease
       (2) 3 Vessel Disease
       (3) 2 Vessel Disease not amenable to PCI
12. Current controversy exists over use of PCI vs CABG. See Reference 8 for more details.

This chapter is a revision of the original chapter authored by David T. Porembka, D.O.

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Questions

27.1. Perioperative myocardial infarction can be characterize by the following EXCEPT:
A. It is usually silent
B. It is usually NSTEMI
C. It is often transmural
D. Enzymes values might be difficult to interpret

27.2. Provision of ACE inhibitors is recommended after acute MI in the following situations EXCEPT
- A. To decrease afterload
- B. In patients developing renal failure
- C. Patients with low LVEF
- D. Patient with HTN

27.3. In relation to β-Blockers the following statements are true, except:
- A. They should be stopped after the acute phase of ischemia is resolved
- B. Decrease incidence of VF
- C. Decrease myocardial oxygen consumption
- D. Improve oxygen delivery

27.4. The goals of managing the patient with coronary ischemia include the following EXCEPT:
- A. Restore coronary blood flow
- B. Minimize extension of the infarct
- C. Decrease oxygen delivery
- D. Decrease oxygen consumption

27.5. In managing patient with coronary stents the following statement are true EXCEPT:
- A. It is important to know the type of stent
- B. The length of time since stent placement is an important piece of information
- C. Once the patient has a stent his risk a new coronary event is close to zero
- D. Management of anticoagulation is of paramount importance to avoid complications

27.6. Patient Risk factors for PMI include
1. Uncontrolled hypertension
2. Age>65
3. Presence of LBBB on EKG
4. Renal failure

28. Valvular Heart Disease

Frank Rosemeier, M.D. and Miguel A. Cobas, M.D.

A 64-year-old female with acute coronary syndrome develops pulmonary edema in the intensive care unit and becomes acutely dyspneic and hypoxemic. A new soft pansystolic murmur is audible at the apex. Despite emergent intubation, ventilation and inotropic support, her cardiorespiratory status continues to deteriorate. A bedside transthoracic echocardiogram reveals severe mitral regurgitation and a left ventricular ejection fraction of 40%. Hemodynamic stabilization is achieved with an intraaortic balloon pump (IABP). Following coronary angiography, she undergoes emergent coronary bypass surgery with mitral valve repair.

Valvular heart disease in the critically ill patient typically presents either as acute valvular dysfunction with acute heart failure or as chronic, but decompensated valvular disease secondary to increased metabolic demands. Valvular regurgitant lesions are far more common than stenosis in the acute setting. However, patients with previously asymptomatic or compensated stenotic lesions may acutely deteriorate triggered by entities such as infection, sepsis, acute hemorrhage, trauma, anemia, or pregnancy.

The following outline aims to provide a basic framework of the causes, diagnostic approaches and therapeutic interventions of emergent valvular disorder frequently encountered in the critically ill patient. It will first provide a general overview and then discuss selected valvular dysfunction specifically.

I. Causes of Acute Valvular Dysfunction in the Critical Ill Patient
   A. Mitral regurgitation
      1. Acute myocardial infarction
         a) Papillary muscle rupture
         b) Regional wall motion abnormality
         c) Left ventricular dilation and systolic dysfunction
      2. Myxomatous disease with flail leaflet (mitral valve prolapse)
      3. Spontaneous chordal rupture
      4. Endocarditis
   B. Mitral Stenosis
      1. Rheumatic heart disease, especially in younger women
      2. Exacerbation by pregnancy, trauma, shock
   C. Aortic Regurgitation
      1. Endocarditis
      2. Aortic dissection
      3. Spontaneous rupture of a congenital fenestration
   D. Aortic Stenosis
      1. Calcification or bicuspid aortic valve in the elderly
      2. Exacerbation by trauma, shock, infection, anemia
   E. Tricuspid Regurgitation
      1. Endocarditis
      2. Penetrating chest trauma
      3. Blunt chest wall trauma
   F. Prosthetic Valves
      1. Endocarditis
      2. Valve thrombosis
      3. Paravalvular dehiscence
      4. Leaflet tear

II. Diagnosis of Acute Valvular Dysfunction
   A. Physical Examination
      1. Variable accuracy
      2. High index of suspicion: Consider valve dysfunction in all patients with pulmonary edema

and/or cardiogenic shock

B. Transthoracic echocardiography
   1. Accurate diagnosis of disease
   2. Quantification of severity of stenosis or regurgitation
   3. Estimation of ejection fraction (EF)
   4. Estimation of pulmonary artery pressures
C. Transesophageal Echocardiography
   1. Sensitive for detection of valvular vegetations and paravalvular leaks
   2. Detection of paravalvular abscess
   3. Essential for prosthetic mitral valve dysfunction
   4. Useful for prosthetic aortic valve dysfunction
D. Right-Sided Heart Catheterization
   1. Not reliable for diagnosis of valve disease
   2. May be helpful for optimizing loading conditions
E. Chest computer tomography
   1. Sensitive and specific for diagnosis of aortic dissection
F. Coronary Angiography
   1. Identifies coronary artery disease

III. Therapeutic Interventions
   A. Accurate diagnosis with echocardiography
      1. Differentiate acute valve dysfunction from acute decompensation in chronic valve disease
   B. Treat the underlying disease process associated with decompensation (i.e. endocarditis, acute myocardial infarction, and anemia)
   C. Optimize pre- and afterload conditions using diuretics, vasodilators, and other agents guided by hemodynamic monitoring
   D. Consult the cardiac surgery team as soon as the diagnosis is made
   E. Use intra-aortic balloon pump for acute mitral regurgitation
   F. Consider surgical or percutaneous intervention for acute valve dysfunction

IV. Mitral Regurgitation (MR)
   A. Etiology
      1. Distortion or disease of any one component of the mitral valve apparatus (mitral annulus, leaflets, chordae, papillary muscles, and shape and function of the left ventricular walls)
      2. Increased volume load on the left ventricle results in left ventricular (LV) dilation and eventually LV failure
      3. In decompensated heart failure, the geometry of the mitral valve apparatus is further compromised by worsening MR and LV dilation
   B. Clinical presentation
      1. Chronic MR is typically well tolerated, even in the presence of systemic infection, pregnancy or acute hemorrhage
      2. Acute MR presents as pulmonary edema
      3. Chordal rupture in patients with mitral valve prolapse
      4. Superimposed symptoms and signs of endocarditis
      5. Acute ischemic papillary muscle dysfunction or subacute papillary muscle rupture several days after myocardial infarction
   C. Diagnosis
      1. High level of clinical suspicion
      2. Holosystolic murmur at the apex radiating to the axilla
         a) *Caveat:* Loudness of murmur may not correlate with severity. Noisy environment and transmitted sounds from pulmonary edema makes auscultation challenging
      3. Echocardiography
         a) Determines etiology by 2D echo and severity by area and width of the regurgitant jet on color flow Doppler
      4. Blood cultures to exclude endocarditis
      5. Prominent v wave on pulmonary artery catheter occlusion pressure tracing (wedge), but may be absent
   D. Management

1. For chronic, but decompensated MR
   a) Treat the underlying cause such as fluid overload, anemia, infection
   b) Optimize preload and afterload with diuretics, and vasodilators
2. For acute MR
   a) Supportive with careful management of pre- and afterload conditions
   b) Afterload reduction with IABP (which also improves diastolic flow)
3. Coronary revascularization for:
   a) Regional wall motion abnormalities causing mitral valve apparatus dysfunction
4. Surgical considerations:
   a) Mitral valve annuloplasty for annular dilation and regurgitation
   b) Mitral valve repair for leaflet prolapse
   c) Replacement, reimplantation, elongation or shortening of the chordae
   d) Valve resection and replacement for endocarditis with heart failure to avoid further structural damage and paravalvular abscess formation
   e) Partial or complete papillary muscle rupture

V. Aortic Regurgitation (AR or AI)
   A. Etiology
      1. Congenital bicuspid valve
      2. Rheumatic valve disease
      3. Aortic root dilation
         a) Hypertension
         b) Cystic medial necrosis
         c) Marfan's syndrome
         d) Bicuspid aortic valve
   B. Clinical Presentation
      1. Pulmonary edema from acute volume overload of the left ventricle
      2. Cardiogenic shock from decreased forward flow and decreased coronary perfusion
   C. Diagnosis
      1. High index of suspicion (endocarditis, family history of aortic root disease, features consistent with aortic dissection)
         a) *To-and-fro* murmur, narrow pulse as signs of low cardiac output in contrast to bounding pulse in chronic aortic regurgitation
         b) Classical signs of de Musset, Traube, Quincke and Corrigan's pulse unreliable in acute decompensation with low cardiac output
      2. Echocardiography
         a) Transesophageal echocardiography for suspected thoracic aortic dissection
         b) Thickened valve leaflets, flail leaflets, prolapse, vegetation, aortic root dilation
         c) Regurgitant jet across the aortic valve on color flow Doppler
   D. Management
      1. Surgical emergency
      2. Stabilize and support cardiovascular and respiratory status
         a) Ventilatory support and invasive hemodynamic monitoring
         b) Use diuretics, inotropic agents, and vasodilators to optimize pre- and afterload conditions and improve contractility with emphasis of acute afterload reduction and faster heart rate to allow forward flow and reduce regurgitant diastolic time
         c) IABP contraindicated as balloon inflation during diastole will increase the regurgitant volume

VI. Mitral Stenosis (MS)
   A. Etiology
      1. Nearly always due to rheumatic disease
      2. Calcific mitral stenosis of the elderly is rare
      3. 80% of cases are women
      4. Large left-sided atrial myxoma may mimic mitral stenosis
      5. Rare: Congenital, malignant carcinoid, lupus, amyloidosis
   B. Clinical Presentation
      1. Slowly progressive disease with insidious decline of effort tolerance level

2. Acute decompensation with increased systemic hemodynamic demands in asymptomatic or compensated patients with severe stenosis
3. Heart failure in pregnant women is most common emergency presentation
4. Pulmonary edema triggered or exacerbated by atrial fibrillation

C. Diagnosis
1. Diastolic rumble and opening snap difficult to hear in ICU environment
2. ECG with left atrial enlargement, atrial fibrillation
3. Echocardiography
   a) Thickened valve, enlarged left atrium, stenotic flow on color Doppler

D. Treatment
1. Control fever, maintain normal hemoglobin level
2. Rate control for atrial fibrillation
3. Consider beta-blockers to prolong diastolic filling time
4. Hemodynamic monitoring and ventilatory support in decompensated CHF
5. Percutaneous transseptal balloon mitral valvotomy as emergency intervention:
   a) Contraindication: Left atrial thrombus, coexisting moderate to severe mitral regurgitation, or heavily calcified and deformed mitral valve
6. Urgent mitral valve replacement if valvotomy fails or contraindicated)

VII. Aortic Stenosis (AS)

A. Etiology
1. Calcification of a normal or congenital bicuspid valve
2. Rheumatic aortic stenosis less common, but usually associated with mitral valve involvement
3. Restenosis after prior commissurotomy in childhood

B. Clinical Presentation
1. Chronic, slowly progressive disease
2. Syncope, pulmonary edema, angina or heart failure during pregnancy
3. Heart failure in the setting of pneumonia, anemia, or other conditions with increased metabolic demands

C. Diagnosis
1. Classical physical findings of pulsus parvus et tardus with ejection systolic murmur unreliable as decompensated stenosis may coexist with other valvular disorders. Loading conditions may reduce or change the systolic murmur
2. ECG: LV hypertrophy, T-wave inversion and ST-segment depression
3. Echocardiography
   a) Calcified, immobile leaflets
   b) Stenotic flow across the aortic valve on color flow Doppler
   c) Assessment of pressure gradient with Doppler

D. Management
1. Conservative approach; restore normal loading conditions
2. Urgent aortic valve replacement for severe AS and acute CHF
3. Balloon valvotomy or afterload reduction with nitroprusside controversial

VIII. Right-Sided Valve Disease

A. Pulmonic valve disease is nearly always congenital
B. Tricuspid valve stenosis relates to rheumatic mitral valve disease and is rare
C. Tricuspid regurgitation can present acutely due to endocarditis or to blunt or penetrating chest wall trauma
1. Blunt chest wall trauma is often accompanied by cardiac injury
   a) May result in pericardial effusion and tamponade
   b) May result in valve rupture, with associated valve regurgitation

IX. Prosthetic Heart Valves

A. Mechanical Valves
1. Valve thrombosis
   a) Caused by inadequate anticoagulation
   b) May result in:
      (1) Functional valve stenosis if movement of the leaflet(s) is restricted

(2) Valve regurgitation if clot prevents full closure of the valve

2. Perivalvular leak
   a) Early presentation often related to suture dehiscence
   b) Associated with hemolytic anemia
   c) Rule out endocarditis if leak occurs past the early stage of valve replacement
3. Clinical presentation similar to native valve stenosis or regurgitation
   a) Echocardiography to assess valve dysfunction
4. Treatment
   a) Controversial
   b) For small thrombus consider conservative therapy with full dose intravenous anticoagulation for several days
   c) Valve replacement for severe hemodynamic compromise
   d) Operative mortality from 17% to 40%
   e) When surgical risk unacceptably high, consider systemic thrombolytic therapy, but:
      (1) 20 % mortality, 16 % systemic embolism, still need emergency surgery in 20%

B. Tissue Valves
1. Slow degeneration of leaflets with calcification over 10 to 15 years
2. Acute decompensation in chronic prosthetic valve dysfunction by superimposed hemodynamic stress
3. Rule out endocarditis in acute regurgitation
4. Diagnosis with echocardiography
5. Treatment similar to native valve with attempts to stabilize and optimize patients prior to valve replacement surgery

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QUESTIONS:

28.1. An 82-year-old man presents with an acute abdomen for exploration. He has a loud late peaking systolic murmur at the apex radiating to his neck bilaterally. The most important consideration is:

A. Decreasing afterload
B. Avoid volume overload
C. Prophylactic antibiotics
D. Placement of Invasive Monitoring Pre-Operatively
E. Prevent tachycardia

28.2. A patient with known severe mitral stenosis decompensates with rapid ventricular response 72 hours after the onset of new atrial fibrillation. When should electrical cardioversion be attempted?

A. A transthoracic echocardiographic exam reveals no left atrial thrombus
B. There is adequate anticoagulation
C. Transesophageal echocardiography reveals no left atrial appendage thrombus
D. The cross-sectional dimensions are >4.5 cm^2
E. There is normal left ventricular function

28.3. A patient is suspected of having an incompetent prosthetic mitral valve. Which diagnostic tool will best assist in assessing any pathology?

A. Computed tomography
B. Transesophageal echocardiography
C. Transthoracic echocardiography
D. Angiogram
E. Pulmonary artery catheter

28.4. In a patient who has hemodynamic compromise, intraaortic balloon pump (IABP) may be useful in the following conditions EXCEPT:

A. Acute myocardial ischemia
B. Ventricular septal defect
C. Acute aortic regurgitation
D. Acute mitral regurgitation
E. Congestive heart failure