Session 2
Cardiovascular & Hematology

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Dopamine Hydrochloride (Dopamine)

*Therapeutic Use*
Naturally occurring catecholamine and precursor to norepinephrine, also serves as a central and peripheral neurotransmitter. First line agent for many types of shocks states. Versatile drug secondary to different actions depending on delivered concentration. The stimulation of dopaminergic receptors is a unique property of this agent.

*Pharmacokinetics*
IV administration only with short half life

*Pharmacodynamics*
a. Central and peripheral nervous system neurotransmitter and precursor of norepinephrine
b. Low concentration: vascular DA₂ – dopaminergic receptors primarily in renal, mesenteric, coronary and cerebral beds – cause vasodilation. D₁ receptors mediate a mild natriuresis. Current research has demonstrated that even with 1mcg most people will also get some alpha or beta stimulation.
c. Moderate concentrations: beta₁ adrenergic receptor agonist – positive inotropic effect
d. High concentrations: alpha₁ adrenergic receptor agonist – potent vasoconstriction

*Hemodynamics (dose dependent)*
a. Low concentration: increase in UO, maybe some increase in HR or SBP (current research has not shown this to be renally protective)
b. Moderate concentrations: increase in HR, SBP, CO (mild)
c. High concentrations: increase in SBP, DBP, SVR

*Mixing and Dosing*
a. Typical 400mg/250 D5W or NS
b. Dosed in mcg/kg/min
c. 1-3mcg/kg/min low dose
d. 3-5mcg/kg/min mid dose
e. 5-10mcg/kg/min high dose
Norepinephrine Bitartrate (Levophed)

**Therapeutic Use**
Endogenous catecholamine with powerful inotropic and peripheral vasoconstriction effects. Typically not utilized as first line drug due to strong vasoconstrictive properties.

**Pharmacokinetics**
IV administration only with short half life

**Pharmacodynamics:**
- Potent $\alpha_1$ & $\alpha_2$ agonist
- Mild $\beta_1$ agonist
- No effect on $\beta_2$
- Systemic arterial and venous constriction
- Coronary flow increases slightly

**Hemodynamics**
- Increase in SBP & DBP
- Increase in SVR and PVR
- Cardiac output unchanged or decreased (increase in afterload)
- Heart rate may slow from compensatory vagal reflex

**Dosing and Mixing**
- Typical 4mg/250 D5W
- Dosed in mcg/min
- 2-10mcg/min

Epinephrine Hydrochloride

**Therapeutic Use**
Endogenous catecholamine with powerful inotropic, peripheral vasoconstriction effects and inotropic properties. Typically not utilized as first line drug due to profound vasoconstrictive and subsequent side effects.

**Pharmacokinetics**
Short half life with rapid onset

**Pharmacodynamics**
- Alpha and Bata agonist
- Increases myocardial contractility
- Vasoconstriction (all beds)
- Increases myocardial $O_2$ consumption
Hemodynamics
a. Increases HR, MAP, CO, SVR, PVR
b. Pro-arrhythmic

Mixing and Dosing
a. Typical 2mg/250 D5W or NS up to 8mg/250
b. Dosed in mcg/min
c. 1-4mcg/min

Vasopressin

Therapeutic Use
Is a naturally occurring antidiuretic hormone. In unnaturally high doses it functions as a non-adrenergic peripheral vasoconstrictor. Major use is as a first line agent in ACLS for pulseless VT/V-fib. Shown to reduce or eliminate the need for catecholamine administration.

Pharmacokinetics
a. IV administration only
b. Half life 10-20 min

Pharmacodynamics
a. Direct stimulation of smooth muscle V₁ receptors
b. Smooth muscle constriction: pallor of skin, nausea, intestinal cramps, desire to defecate, bronchial constriction, uterine contraction
c. Less constriction of coronary and renal vascular beds and vasodilation of cerebral vasculature
d. No skeletal muscle vasodilation or increased myocardial O₂ consumption during CPR because there is no Beta-adrenergic activity
e. May enhance platelet aggregation in septic shock

Hemodynamics
a. Increase in SBP, MAP and SVR
b. Increase UO

Mixing and Dosing
a. Typical 200U/250 D5W or NS
b. Dosed in unit/min
c. 0.2-0.9U/min

Dobutamine

Therapeutic Use
Synthetic catecholamine which has selective beta adrenergic agonist properties. Effective as a positive inotropic for both preload and afterload reduction. Used for its positive inotropic
properties when vasoconstriction is not preferable. Also used commonly as a combination therapy with another catecholamine or vasodilator

**Pharmacokinetics**
a. IV administration only  
b. half life 2 minutes – rapid onset

**Pharmacodynamics**
a. β₁ adrenergic receptor agonists: increases contractility and stroke volume, increases sinus node automaticity and AV conduction, increases in myocardial oxygen demand  
b. Mild β₂ adrenergic receptor agonist: mild vasodilation, increased perfusion  
c. Mild α₁ vasoconstriction properties are counter balanced by β₂ properties  
d. Does not cause release of endogenous norepinephrine  
e. Infusions of > 72 hrs have shown tolerance to down regulation of β adrenergic receptors  
f. Less effective in patients receiving β blocking agents or with chronic heart failure

**Hemodynamics**
a. Increase CO  
b. Mild decrease in SVR  
c. Mild increase in HR (sometimes more than mild)

**Mixing and Dosing**
a. Typical 500mg/250 D5W or NS  
b. Dosed in mcg/kg/min  
c. 2-10mcg/kg/min

**Milrinone (Primacor)**

**Therapeutic Use**
Synthetic noncatecholamine agent that does not stimulate or block adrenergic receptors. Inhibits the phosphodiesterase III enzyme. Effective as a positive inotrope and vasodilator.

**Pharmacokinetics**
a. IV administration only  
b. Hepatically cleared  
c. Half life 2-3 hours

**Pharmacodynamics**
a. Phosphodiesterase III enzyme inhibitor – increases cyclic adenosine monophosphate (cAMP) which enhances calcium entry into the cell and improves myocardial contractility, and inhibiting vasoconstriction (vasodilator).  
b. Increased cardiac output by positive inotropic action and reduction in preload and afterload  
c. Most effective with patients who have over stimulated sympathetic system  
d. Effective in patients with beta receptor down regulation
**Hemodynamics**

a. Increase in CO  
b. Decrease in CVP, SVR, PAOP  
c. No significant effect on HR or BP (unless compensatory)

**Mixing and Dosing**

a. Mix with NS ONLY  
b. Dosed in mcg/kg/min  
c. Loading dose 50mcg/kg over 10 min  
d. 0.375-0.75mcg/kg/min

*Review ALL ACLS drugs and algorithms when studying for the PCCN Exam.*
I. ACUTE PULMONARY EDEMA

Introduction
A change in alveolar-capillary membrane permeability leads to pulmonary interstitial edema. The initiating pathology can be either cardiac or non-cardiac in origin.

Pathogenesis of Pulmonary Edema

Alveolar-Capillary Membrane
a. Capillary Endothelial Layer: Microvascular Barrier
b. Alveolar Epithelial Layer: Alveolar Barrier

Fluid Dynamics of the Alveolar-Capillary Membrane
a. Hydrostatic pressure
b. Osmotic pressure
c. Membrane permeability

Etiology of Pulmonary Edema

Cardiogenic Pulmonary Edema
Cardiogenic pulmonary edema, which is the most common type of acute pulmonary edema, occurs when there is an increase in hydrostatic pressure within the pulmonary capillary bed as a result of heart failure. Causes include:
a. Heart Failure
b. Myocardial Infarction
c. Cardiac Ischemia
d. Acute Mitral Regurgitation
e. Cardiac Tamponade
f. Tachy Dysrhythmias
g. Hypertensive Crisis

Non-Cardiogenic Pulmonary Edema
Non-cardiogenic pulmonary edema results from one of four primary abnormalities (or a combination):
a. Impaired endothelial integrity
b. Decreased colloidal oncotic pressure
c. Elevated capillary hydrostatic pressure
d. Lymphatic obstruction

The impaired endothelial integrity (change in permeability) is typically caused by a direct or indirect injury to the lung tissue. Acute respiratory distress syndrome (ARDS) is a form of non-cardiogenic pulmonary edema.
Diagnosis

History
a. Cardiogenic Pulmonary Edema
b. Acute Cardiac Event
c. Chest Pain
d. Tachy-Palpitations
e. New Dysrhythmia
f. History of Ischemic Heart Disease
g. Acute CP and/or SOB in the Absence of Any Other Pathologies
h. Absence of Cardiac Hx Does Not Rule Out CPE

Physical Exam
a. Dyspnea, Tachypnea & Apprehension
b. Presence of S₃ Heart Sound
c. Jugular Venous Distension
d. Breath Sounds
e. Increased Frothy Sputum Production
f. Laterally Displaced Point of Maximal Impulse (PMI)
g. New or Louder Cardiac Murmur
h. Unilateral Lung Adventitious Sounds: more commonly assessed in non-cardiogenic
i. Diffuse Decreased Breath Sounds: more commonly assessed in non-cardiogenic
j. Peripheral Edema is non-specific

Chest X-Ray
There are chest x-ray changes that are unique to cardiogenic and non-cardiogenic pulmonary edema. If the pattern changes from day to day or significantly after treatment it is more indicative of cardiogenic.

ECG Changes
Tachycardia or acute ST-T segment changes are more commonly assessed in cardiogenic pulmonary edema

Echocardiography
A Transthoracic echo may be helpful to identify myocardial ischemia, wall motion abnormalities, ventricular dysfunction, valvular disease, and LV hypertrophy, all of which will suggest cardiogenic pulmonary edema.

Laboratory Data
Arterial Blood Gas:
a. Low Oxygen Saturation
b. Respiratory Alkalosis
c. Refractory Hypoxemia
Treatment Options

**Cardiogenic Pulmonary Edema**
ACLS algorithm while treating the underlying cardiac condition:

a. **Diuretics:** Furosemide IV 0.5 to 1.0 mg/kg
b. **Analgesics:** Morphine IV 2 – 4mg
c. **Preload Reduction:** Nitroglycerin SL
d. **Oxygen/Intubation**
e. **Afterload Reduction if SBP > 100mmHg**
   - IV Nitroglycerin 10-20 µg/min or consider
   - IV Nitroprusside 0.1 – 5.0 µg/kg/min
f. **Vasoconstriction if Hypotensive**
   - Severe (SBP < 70mmHg) and S&S of shock
     - IV Norepinephrine 0.5 – 30 µg/min
   - Moderate (SBP 70– 100mmHg) with S&S of shock
     - IV Dopamine 5 – 15 µg/kg/min
   - Moderate without shock
     - IV dobutamine 2 – 20 µg/kg/min
g. **Further Diagnostic Considerations**
   - Pulmonary Artery Catheter
   - Intra-Aortic balloon pump
   - Cardiac Angiography

II. RUPTURED OR DISSECTING AORTIC ANEURYSMS

**Definitions**
These conditions can overlap at times with one leading to or increasing the risk of the other.

**Aortic Aneurysm**
A localized dilation of the arterial wall that can be saccular, fusiform or cylindrical. The dilation frequently renders the aorta weak in that region.
a. Complication of aneurysms
b. Management of aneurysms

**Aortic Dissection**
a longitudinal separation of the aortic wall between the intima and the adventitia. An acute dissection is one that is diagnosed within 14 days of the onset of symptoms. The risk of death is greatest during this acute period. A chronic dissection is one that is diagnosed after two weeks of the onset of symptoms.
Classification Systems

Stanford Classification System

Type A Aortic Dissection
The dissecting area involves the ascending aorta. It may be confined to only the ascending aorta or may also involve the descending as well. Typically occur in a younger patient population with a congenital weakening of the ascending aorta. Type A dissection account for 2/3 of all dissections.

Type B Aortic Dissection
The dissecting area involves only the descending aorta distal to the left Subclavian artery. Typically occurs in the older patient population with a history of hypertension and atherosclerosis.

DeBakey Classification System

Type I Aortic Dissection
The dissection involves the ascending aorta but also extends beyond the left subclavian artery.

Type II Aortic Dissection
The dissection involves only the ascending aorta.

Type III Aortic Dissection
The dissection involves only the descending aorta. IIIa limited to the thoracic aorta, IIIb involving various degrees of the thoracic and abdominal aorta

Common Risk Factors

a. Although hypertension does not appear to be the sole contributor to the occurrence of AAD, it plays a major role in the development and/or propagation of a dissection. The etiology of AAD is believed to be a combination of something that has caused a weakening in the vessel that ‘allows’ the original tear to occur and that, in combination of HTN (70-90% of the victims have a history of HTN) triggers the filling of the false lumen and dissection of the arterial layers.
b. Marfan’s Syndrome (a chromosomal mutation with many genotypes and phenotypes)
c. Annuloaortic Ectasia
d. Aortic Dilatation and Wall Thinning
e. Bicuspid Aortic Valve (congenital malformation)
f. Spontaneous Rupture of Vasa Vasorum
g. Aortic Coarctation
h. Trauma (blunt, penetrating or iatrogenic)
i. Aging
j. Arterial Hypertension
k. Cocaine Use
Diagnosis

**Presenting Signs & Symptoms**
AAD is Known as the Great Imitator

a. Sudden Severe Pain Not Relieved with Analgesics
b. Initially Normal or High Blood Pressure
c. Hypotension
d. Acute Aortic Valvular Insufficiency: High-Pitched, Blowing Diastolic Murmur
e. Audible S₃ Heart Sound
f. Abrupt Onset of a Pulseless Extremity
g. Peripheral Vascular Insufficiency
h. End-Organ Ischemia (brain, kidney, intestines, spinal, lower extremities)
i. Pericardial Effusion
j. Cardiac Tamponade
k. Acute Myocardial Ischemia

**Tests**
a. Chest X-ray
   - Wide Mediastinum
   - Wide Aortic Silhouette
   - Pleural Effusion
   - CHF
   - Pericardial Effusion (cardiomegaly)
b. ECG: Nonspecific Changes
   - Left Ventricular Hypertrophy
   - Acute Myocardial Ischemia
d. Transthoracic or Transesophageal (TEE) Echocardiography
e. Magnetic Resonance Imaging (MRI) Scan
f. Aortography

**Treatment**

**Adequate Blood Pressure Management**
a. Antihypertensive Agents: Nipride 0.5µg/kg/min titrate up to maintain SBP below 110mgHg or at a level to maintain perfusion
b. Negative Inotropic Agents: β blockers
c. Pharmacological management may be the primary treatment for a dissection involving the descending aorta

**Pain Relief**
Typically done with Morphine

**Reduction of Environmental and Emotional Stresses**
May need anti-anxiety agents.
**Surgical Repair**

a. All patients with ascending aortic dissection require immediate repair
b. Descending dissections repairs have high mortality and morbidity

**III. HYPERTENSIVE CRISIS**

**Introduction**

**Pathophysiology of Hypertension**
Definition and Current Guidelines

**Definitions**

**Hypertensive Crisis**
A diastolic blood pressure greater than 120mmHg. Global term does not denote physiologic response or need for immediate treatment.

**Hypertensive Emergency**
A diastolic blood pressure of greater than 120mmHg with acute or ongoing end organ (neurological, cardiac or renal) damage. Immediate blood pressure reduction is required within a few hours to prevent or limit target organ damage. The reduction does not necessarily need to be back to normal pressure just out of the dangerous range.

**Hypertensive Urgency**
A diastolic blood pressure of greater than 120mmHg without end organ damage. Reduction of blood pressure is important to limit the risk of potential end organ damage but not emergent. The goal is to bring down the blood pressure within 24 – 48 hours.

**Malignant Hypertension**
Described by Volhard and Fahr in 1914, MHT is characterized by severe accelerating hypertension with evidence of renal, neurological, vascular and retinal damage/dysfunction that can be rapidly fatal ending in heart attack, stroke or heart and renal failure. The modern criteria for MHT are severe hypertension (DBP > 120mmHg) associated with retinal hemorrhages, exudates and papilledema (group 4 Keith-Wagener-Barker retinopathy) (Laragh, 2001). Some authors define it simply as elevated BP accompanied by encephalopathy or nephropathy (Varon, 2000).

**Accelerated Hypertension**
A more ‘mild’ form of MHT without the presence of papilledema and a group 3 Keith-Wagener-Barker retinopathy.

**Post Operative Hypertension**
Defined as systolic blood pressure of greater than 190mmHg and/or diastolic blood pressure of greater than or equal to 100mmHg on two consecutive readings following surgery. Because of
the unique and transient physiological factors following surgery and anesthesia this clinical syndrome is separated from the other hypertensive crises.

**Gestational Hypertension**
There are multiple names for this syndrome. A blood pressure is considered an emergency in a pregnant woman and requires immediate pharmacologic management when the systolic pressure is greater than 169mmHg or diastolic greater than 109mmHg.

**Hypertensive Crisis – Pathophysiology & Management**

**Etiologies**
There is not one cause of HTN Crisis. A history of preexisting hypertension is the common denominator regardless of the secondary causative factor(s).

**Pathophysiology**
Although the exact physiological mechanism(s) of hypertensive crisis are unknown, there appears to be a vicious cycle of increased vasoconstriction which leads to increasing pressure.

**Assessment**
In addition to the blood pressure, the presence and relative degree of end organ damage/dysfunction is important to assess for and essential to identify before selecting the appropriate treatment option.

a. Previous Diagnosis of HTN
   - How long?
   - Prescribed Medications?
   - Adherence to Prescription Medication?
   - General Level of Control or Typical Blood Pressure?

b. All Other Medications: Prescription, Over the Counter, Dietary Supplements and/or Illicit Drugs

c. Cardiac Assessment

d. Renal Assessment

e. Neurological Assessment – Hypertensive Encephalopathy

f. Laboratory Data

g. Evaluate Secondary Causes

**Treatment for Hypertensive Emergency**
The goal is to reduce the mean arterial pressure by 25% within the first two hours (preferably within the first few minutes) in a controlled, predictable and safe fashion and then toward 160/100mmHg within two to six hours.

a. Nitroprusside Sodium (Nipride)

b. Fenoldopam Mesylate (Corlopam)

c. IV Vasodilators

d. IV Adrenergic Inhibitors

e. Diuretics
**Treatment for Hypertensive Urgency**

Blood pressure should be lowered within 24-48 hours and frequently oral agents are adequate in this patient population.

a. ACE inhibitors  
   b. Calcium Channel Blockers  
   c. Alpha$_2$ Adrenergic Stimulators (Clonidine)

**IV. VALVULAR HEART DISEASE**

**Pathophysiology**

a. Congenital Malformations  
   b. Connective Tissue Disorders  
   c. Degenerative Disease  
   d. Rheumatic Heart Disease  
   e. Infective Endocarditis  
   f. Dysfunctional Ruptures

**Specific Valvular Dysfunction**

a. Mitral Stenosis  
   b. Mitral Insufficiency/Regurgitation  
   c. Aortic Stenosis  
   d. Aortic Insufficiency/Regurgitation

**Management of Valve Disorders (pre-op and post-op)**

a. Oxygenation  
   b. Hemodynamic Stability  
   c. Dysrhythmias  
   d. Activity  
   e. Anticoagulation  
   f. Antibiotic Prophylaxis  
   g. Patient/Family Education

**Surgical Management of Valve Defects**

a. Indications  
   b. Valve Repairs  
   c. Prosthetic Valve Replacement  
      • Mechanical  
      • Biological
Septal Defects

a. Locations: Atrial and Ventricular
b. Types: Congenital and Acquired

V. VASCULAR DISEASE

Peripheral Vascular Disease

*Note: DVT is addressed in the Pulmonary section combination with the Pulmonary Emboli*

Pathophysiology

a. Smoking
b. HTN
c. DM
d. Lipid Disorders
e. Hyperhomocysteinemia

Assessment

a. Intermittent Claudication
b. Resting Pain
c. Cool Temperature
d. Diminished Pulses
e. Leg/Skin Changes

Diagnosis

Acute occlusion

Acute Arterial Occlusion

5Ps
a. Pain
b. Pulselessness
c. Pallor
d. Paresthesian
e. Paralysis

Management/Treatment

a. Risk Factor Modification
b. Vasodilators
c. Antiplatelets
d. Exercise
e. Angioplasty
f. Vascular Bypass Surgery
g. Minimally-Invasive Interventions (stents, endografts)
**Compartmental Syndrome**

a. Compartments are closed spaces containing muscles, nerves, and vascular structures
b. Internal and external causes can increase pressure within a compartment
c. Increased pressure can lead to ischemia, injury and necrosis to the contents within the compartment
d. Signs and Symptoms of CS
   - Throbbing Pain (localized)
   - Firmness of area
   - Altered Sensation: Numbness, tingling, sticking feelings
   - Pulselessness
   - Decreased Voluntary Limb Movement
e. Treatment for CS
   - Eliminate the Cause
   - Elevation
   - Pain Management
   - Fasciotomy

**Carotid Artery Disease**

*Pathophysiology*

*Assessment*

*Management/Treatment*

a. Risk Factor Modification
b. Vasodilators
c. Antiplatelets
d. Neuro Monitoring
e. Carotid Endarterectomy
f. Carotid Stents

**VI. CARDIAC TAMPOANDE**

*Introduction*

A cardiac effusion is the accumulation of fluid within the pericardial space. A cardiac tamponade occurs when an effusion causes compression on the heart and the external pressure effects cardiac function.
Etiology

Any pathology that can lead to a pericardial effusion can cause a tamponade. Pericarditis is the most common cause:

a. Any Type of Pericarditis (inflammatory, infectious, immunologic or physical)
   - Neoplastic
   - Renal Insufficiency → ESRD
   - Post Acute Myocardial Infarction
   - Collagen Vascular Disease
   - Autoimmune Diseases
   - Chemotherapy
   - Radiation Therapy
   - Nephrotic Syndrome
   - Tuberculosis
   - Hepatic Cirrhosis
b. Invasive Cardiac Procedures (cardiac surgery or biopsy)
c. Indwelling Cardiac Instrumentation
d. Anticoagulant or Thrombolytic Therapy
e. HIV/AIDS
f. Valvular Heart Disease
g. Trauma
h. Pregnancy
i. Aortic Dissection
j. Chronic Heart Failure

Pathophysiology

Pericardium

The pericardium is a membrane that surrounds the heart. The pericardial space is between the visceral (next to the myocardium) and parietal layers and there is typically 15-35ml of serous fluid in the space. The function of the fluid is to provide a cushion for the heart. Fifty mls or more of fluid (serous fluid, blood, pus, clots or gas) is considered an effusion. Tamponade can be classified as acute vs chronic, surgical vs medical.

Presenting Signs & Symptoms

Early Physical Signs & Symptoms
May occur prior to full tamponade
a. Chest Pain
b. Anxiety
c. Tachycardic
d. Tachpneic
e. Diaphoretic
f. Bibasilar Rales
g. Fever
h. Unstable Blood Pressure
i. S&S of Shock
j. Specific to Pericarditis
   - Diffuse ST elevation
   - Chest pain worse when supine
   - Pericardial friction rub

Beck’s Triad (1935)
a. Muffled Heart Sounds: from the accumulation of fluid.
b. Narrowing of the Pulse Pressure and Hypotension: The pulse pressure is the difference between the SBP & the DBP. Due to the increasing pressure outside the heart there is less dilation during diastole, which causes the DBP to either stay the same or rise. There is a lower EF because of the decreased filling and contractility so the SBP drops.
c. Jugular Vein Distention (JVD): There is ‘back-up’ of blood into the venous system from a combination of the impaired filling and impaired emptying of the heart due to external pressure. Kussmaul’s sign is a pathological increase in jugular venous pressure (JVP) seen during inspiration.

Pulsus Paradoxus
a. The negative pressure created in the thorax during normal inspiration limits cardiac filling, decreases CO and causes a weaker pulse. There is typically a difference of as much as 10mmHg in SBP between inspiration and expiration.
b. During tamponade the ‘normal’ pressure is increased causing more of a compression, which is increased even more during the inspiratory phase of ventilation. A drop of >10mmHg systolic blood pressure heard during inspiration is considered a Pulsus Paradoxus. Commonly (present in 90% of cases) assessed with cardiac tamponade.
c. Also found in other pathologies that cause increased pressure on the heart like COPD. PP was described by Kussmaul in 1873.

ECG Changes
a. Electrical Alternans: beat to beat change in QRS amplitude (seen with late or rapid tamponade)
b. Low Amplitude of the QRS Complex
c. Absence of Ischemic Changes Despite Chest Pain
d. Non-specific ST changes may be Present in Pericarditis
e. In late tamponade there may be decreased coronary flow (typically in the elderly and patients with CAD)

Diagnosis

Chest X-Ray
Enlarged cardiac silhouette with clear lungs

Hemodynamic Parameters:
a. Drop in Cardiac Output
b. Pulsus Paradoxus Visible on Arterial Line. Note that if the patient is on positive pressure ventilation this response will be reversed.
**Echocardiography**
A non-invasive, relatively available test to identify the presence of a pericardial effusion. Occasionally if the effusion is primarily posterior it may be difficult to visualize on a transthoracic echo and a transesophageal echo is necessary. Of all the diagnostic tools for tamponade this is the most sensitive and specific.

**Therapeutic Management Options**

**Pericardiocentesis**
Needle aspiration of effusion. Subxiphoidal approach. Procedure can be performed blind, or with ECG clamp, echo or fluoroscopy guidance. If recurrent effusion is a concern a drainage system should be left in place for a few days.

a. 6 inch, 16-18 gauge, over-the-needle catheter.

**Surgical Drainage**
a. Subxiphoid surgical incision and thoracoscopic drainage  
b. Video assisted thoracoscopy and drainage  
c. Pericardial window and drainage  
d. Pericardectomy: complete stripping of the pericardial sac is performed on some medical patients with chronic effusion/tamponade. This is typically a last resort.

**Medical Management**

**ACLS**
Oxygen, IV, CPR- if needed, Epinephrine, diagnosis and treat cause ASAP

**General Management**
a. Support compensatory and physiologic response  
b. Expand Intravascular volume  
c. Increase or decrease SVR  
d. Support contractility and stroke volume
Dysrhythmias

I. INTRODUCTION

Review basic dysrhythmias. Questions on PCCN exam will be related to rhythm identification, cause or appropriate treatment. ACLS is not listed on the blueprint but identifying and treating life-threatening dysrhythmias is the major reason to place a patient on telemetry.

II. CARDIAC ELECTROPHYSIOLOGY

Impulse Conduction & Pathways
SA Node → Internodal Pathways (atrial contraction) → AV Node (delay) → His-Purkinje System (ventricular contraction)
III. DYSRHYTHMIAS

**Common Causes**

- Decreased Coronary Perfusion (CAD)
- Impaired Myocardial Oxygen Delivery (hypoxia)
- Electrolyte Disturbances
- Cardiac Muscle Injury
- Ischemia or Infarction
- Defects in the Heart Muscle or Electrical System
- Cardiac Surgery
- Electrical Stimulation to the Heart Muscle
- Medications

**Lead Selection for Monitoring**

**AACN Practice Alerts for ECG Monitoring**
- Dysrhythmia Monitoring (4/08)
- ST Segment Monitoring (5/09)

**AHA/ACCF/HRS Recommendations for Standardization & Interpretation of the Electrocardiogram**
- I ECG Technology ‘07
- II ECG Diagnostic Tests ‘07
- Pre-hospital ECG and ACS ‘08
- III Intraventricular Conduction Disturbances ‘09
- ST-Segment, T, and U Wave and QT Interval ‘09
- Hypertrophy Evaluation ‘09
- Acute Ischemia/Infarction ‘09
- Prevention of Torsade de Points 2/8/10

### Best Leads for Monitoring

<table>
<thead>
<tr>
<th>Monitoring Purpose</th>
<th>Lead Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dysrhythmia Detection</strong></td>
<td>• Aberrancy vs Ectopy: V₁ or V₆ (if V₁ not available)</td>
</tr>
<tr>
<td></td>
<td>• AFib/flutter: II, III, aVF : Monitoring of P wave Problems; whichever lead allows best visualization of fib/flutter waves; consider atrial ECG for post-cardiac surgery patients if pacer wires in place, could try Lewis Lead RA electrode at 4th ICS Rt Sternal boarder, LA on back at Rt side of spine read Lead I</td>
</tr>
<tr>
<td></td>
<td>• Junctional Rhythms: Lead II</td>
</tr>
<tr>
<td></td>
<td>• Bundle Branch Blocks V₁ and/or V₆</td>
</tr>
<tr>
<td><strong>ST Segment Monitoring</strong></td>
<td>• Unknown Problem III or V₃</td>
</tr>
<tr>
<td></td>
<td>• Right coronary artery: III or aVF</td>
</tr>
<tr>
<td></td>
<td>• Left anterior descending/Circumflex: V₃</td>
</tr>
<tr>
<td></td>
<td>• Activity-induced ischemia (no specific vessel identified): V₅</td>
</tr>
<tr>
<td></td>
<td>• Pt “Finger Print” from Known Ischemic Changes</td>
</tr>
</tbody>
</table>
Inferior Wall
Anterior Wall
Lateral Wall

<table>
<thead>
<tr>
<th></th>
<th>II, III, AVF</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>V₁-V₄</td>
</tr>
<tr>
<td></td>
<td>V₅₆, I, aVL</td>
</tr>
</tbody>
</table>

QTc

<table>
<thead>
<tr>
<th></th>
<th>Identify 12-lead with most well-defined T wave</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>V₃, V₄, II</td>
</tr>
</tbody>
</table>

**Best Lead Combinations**

| One Channel Recording | V₁ or V₆                     |
| Two Channel Recording |                          |

|          | V₁ and III |
| ST Segment: | V₃ and III |
| Arrhythmia + ST Segment: | V₁ or V₆ + aVF or III |

Table originally developed by Bridges and published in CCN (2008) Rauen et al. Updated for this outline by Rauen 1/13

**Treatment Options**
Identify & Treat the Underlying Cause

**Defibrillation/Cardioversion**
The passage of electrical current through the cardiac muscle (cells) causes a massive depolarization allowing the cells to ‘reset’ themselves and hopefully creating an environment where the SA node can ‘take back’ the pacemaker function.

**Pacing**
For bradycardic rhythms, electrical stimulation of the heart might be necessary with a transcutaneous, transvenous or permanent pacemaker.

**Pharmacology**
Drugs are the primary treatment if the dysrhythmia is NOT life-threatening. Meaning there is not a significant drop in blood pressure or level of consciousness. The Antidysrhythmic agents are classified using the VaughanWilliams classification system.

<table>
<thead>
<tr>
<th>Drug Classification</th>
<th>Primary Action</th>
<th>Drug Options/Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>CLASS I Sodium Channel Blockers</td>
<td>Membrane-Stabilizing</td>
<td>IA</td>
</tr>
</tbody>
</table>

| IA | Slows Conduction Velocity Negative Inotrope Prolong Refractory Period (wide QRS & QT) Suppress Ectopic & Reentry Foci |
| IA | Quinidine (Quinaglute) Procan, Proestyl Disopyramide (Norpace) |
| IA | Atrial Flutter, Atrial Fib, SVT, VT |
### Session 2: Cardiovascular & Hematology

#### Specific Dysrhythmias

**Tachycardias**

The major problem with the tachy dysrhythmias is that the heart chambers do not have enough time to completely fill or empty. This leads to a drop in stroke volume and subsequently cardiac output. Depending on the exact rhythm, there may also be loss of synchrony between atrial and ventricular contractions (A-fib, V-Tach), which causes a loss of atrial kick and up to 30% of cardiac output. Another potential problem is clot formation in a chamber that has incomplete emptying. In clinical terms tachycardic rhythms can cause anything from dizziness to heart failure and cardiac arrest.

<table>
<thead>
<tr>
<th>CLASS I Sodium Channel Blockers (continued)</th>
<th>IB</th>
<th>IB</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Shorten Duration of Action Potential</td>
<td>Lidocaine (Xylocaine)</td>
</tr>
<tr>
<td></td>
<td>Shorten Refractory Period</td>
<td>Phenytoin (Dilantin)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mexiletine (Mexitil)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Tocainide (Tonocard)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ventricular Rhythms</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>IC</th>
<th>IC</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Slow Conduction Velocity (depress phase 0)</td>
</tr>
<tr>
<td></td>
<td>Increase Refractory Period</td>
</tr>
<tr>
<td></td>
<td>Increase QRS Duration</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CLASS II Beta Blockers</th>
<th>Antiadrenergics</th>
<th>Antiadrenergics</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Prolong duration of action potential</td>
<td>Bretylium (Bretylol) dropped from ACLS</td>
</tr>
<tr>
<td></td>
<td>Delay repolarization</td>
<td>Amiodarone (Cordarone)</td>
</tr>
<tr>
<td></td>
<td>Prolong QT interval, AP &amp; effective refractory period</td>
<td>Sotalol (Betapace)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ibutilide (Corvert)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dofetilide (Tikosyn)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ventricular and Supraventricular Rhythms</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CLASS III Potassium Channel Blockers</th>
<th>Calcium Antagonist</th>
<th>Calcium Antagonist</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Decreased SA node firing</td>
<td>Diltiazem (Cardizem)</td>
</tr>
<tr>
<td></td>
<td>Slower conduction → AV node</td>
<td>Verapamil (Isoptin, Calan)</td>
</tr>
<tr>
<td></td>
<td>Decrease myocardial O₂ demand &amp; contractility</td>
<td>Supraventricular Tachycardias</td>
</tr>
</tbody>
</table>
Narrow QRS Complex Tachycardias (supraventricular)

a. Rhythms
   - Sinus Tachycardia (ST)
   - Atrial Fibrillation (A-Fib)
   - Atrial Flutter (AF)
   - Atrial Tachycardia (ectopic and reentrant) (AT)
   - Multifocal Atrial Tachycardia (MAT)
   - Junctional Tachycardia (JT)
   - Accessory Pathway-Mediated
     - Atrial tachycardia w/ accessory pathway
     - AV reentry tachycardia

b. Treatment (remember to evaluate ventricular function) Based on 2010 ACLS guidelines
   - Stable?
     - A-Fib or AF: Identify length of time in rhythm and consider WPW and LV impairment before determining treatment. Control Rate, Convert Rhythm, Provide Anticoagulation
     - Vagal Stimulation
     - Adenosine
     - PSVT: β-Blockers, Ca++ Channel Blockers, Dig, Antiarrhythmics and Cardioversion. If EF < 40% Start with Cardioversion
     - JT: β-blockers, Ca++ Channel Blockers, Amiodarone, NO Cardioversion
     - MAT: β-blockers, Ca++ Channel Blockers, Amiodarone, NO Cardioversion
   - Unstable? Immediate Cardioversion, Followed by Drugs

Wide QRS Complex Tachycardias

a. Criteria for Wide QRS
   - Rate > 120 bpm
   - Uniform QRS > 120 ms
   - No S&S or Δ in Consciousness

b. Rhythms
   - Ventricular Tachycardia (VT)
   - Ventricular Fibrillation (VF)
   - SVT with Aberrancy (identify and treat as SVT)

c. Treatment (remember to evaluate ventricular function) Based on 2010 ACLS guidelines
   - Ventricular Tachycardia Stable w/ Pulse
     - Monomorphic: Procainamide, Sotalol, Amiodarone, Lidocaine. Amiodarone 1st if ventricle impaired
     - Polymorphic: normal QT - β-blockers, Lidocaine, Amiodarone, Procainamide, Sotalol, Amiodarone 1st if ventricle impaired. Long QT – Mg⁺, overdrive pacing, Isoproterenol, Phenytoin, Lidocaine
   - Ventricular Tachycardia Unstable w/ Pulse: Immediate Cardioversion
   - Ventricular Tachycardia Without Pulse – Treat as VF
• **Ventricular Fibrillation/Pulseless VT:**
  - Assess ABCs
  - Basic Life Support
  - Defibrillation: 120-200J biphasic or 360J monophasic (one shock)
  - CPR
  - Defibrillation: 120-200J biphasic or 360J monophasic (one shock)
  - Vasopressin or Epinephrine
  - Defibrillation: 200J biphasic or 360J monophasic
  - Antiarrhythmics: Amiodarone, Lidocaine, Magnesium, Procainamide
  - Defibrillation: 200J biphasic or 360J monophasic

**Long QT Syndrome:**
The QT represents the repolarization of the ventricle. Repolarization is an electrically unstable time. VT is a likely outcome if the next R wave were to fall on the T wave. In situations where the QT interval is long, there is an increased likelihood of an R on T to occur. Conditions that can lead to this situation include:

a. Congenital Long QT Syndrome (genetic)
b. Exercise Induced QT Syndrome
c. Drug Induced QT Syndrome (many drugs lengthen QT)
   - Antiarrhythmic Agents: Class IA, IB & III
   - Tricyclic Antidepressants
   - Phenothiazine
   - Antimicrobials (specifically Erythromycin)
   - Nicardipine (Cardene)
   - Cisapride (Propulsid)
   - Haloperidol (Haldol)
   - Tamoxifen (Nolvadex)

**Bradycardias**
The major problem with slow rhythms is a lack of stroke volume to sustain an adequate cardiac output. Treatment is dependent on rhythm and cause of slow rate. In the unstable patient with a slow rate:

a. ABCs & BLS
b. Atropine
c. Transcutaneous Pacing
d. Dopamine or Epinephrine
e. If the rhythm is Type II 2nd Degree or 3rd Degree HB and the pt is unstable pace ASAP (transcutaneous → transvenous)
Conduction Defects

Normal Parameters:
PR = 0.12-0.20
QRS = < 0.12
QT rate dependent

First Degree AV Heart Block
a. Rate: 60 - 100 bpm
b. Rhythm: Regular
c. P Waves: One P for Every QRS with PRI > 0.20
d. QRS Complexes: Normal
e. Symptoms/Concerns: Symptoms will depend on HR. Concern for reason this is occurring and will it progress to higher level block
f. Tx: Depends on Symptoms, tx rarely required. Rhythm very common in elderly

Second Degree AV Heart Block (two types)
a. Mobitz Type I, also known as Wenckebach
   • Rate: atrial rate 60 - 100 bpm, ventricular rate varies
   • Rhythm: Irregular with Pattern
   • P Waves: All QRSs are preceded by Ps
     o But not all Ps are followed by QRSs
     o The PRI progressively gets longer
     o Until there is a dropped beat (a P wave not followed by a QRS)
     o Pattern then starts over
   • QRS Complexes: Normal
   • Symptoms/Concerns: Symptoms will depend on Ventricular HR
   • Tx: Depends on Symptoms, tx rarely required
b. Mobitz Type II, also known as Classical
   • Rate: atrial rate 60 - 100 bpm, ventricular varies
   • Rhythm: P-P regular, R-R regular or irregular
   • P Waves: All QRSs are preceded by Ps
     o But not all Ps are followed by QRSs
     o The PRI consistent and typically > 0.20
     o More than one P wave for every QRS
     o Typically a consistent pattern ex. 2Ps:1QRS
   • QRS Complexes: Normal
   • Symptoms/Concerns: Symptoms will depend on Ventricular HR
- Tx: Depends on Symptoms, typically treated
  o Consider External Pacemaker
  o Consider Cause
  o Stop Digoxin
  o Atropine or Epinephrine

**Third-degree AV Heart Block/Complete Heart Block aka AV Dissociation**

a. Rate: < 60 bpm
b. Rhythm: P-P regular, R-R regular
c. P Waves: P waves “march” out regular but have no discernible relationship to the QRS
d. QRS Complexes: Slow, Wide > 0.12, “march” out, regular
e. Symptoms/Concerns: Symptoms will depend on Ventricular HR and LOC
f. Tx: Depends on Symptoms, tx typically required
   - External pacemaker
   - Atropine (not typically helpful because it will increase sinus node firing (P waves) but not ventricular conduction
   - Epinephrine

**Bundle Branch Blocks**

QRS Complex

a. Represents: Ventricular Depolarization
b. Shape:
   - Q First Negative Deflection
   - R First Positive Deflection
   - S Second Negative Deflection
c. Duration (time):
   - QRS > 0.12sec (3mm)
   - Q < 0.03sec (< 1mm)

![AV Node → Bundle of His →
1. Septal Depolarization L → R
2. Biventricular Depolarization](image)

The ventricles depolarize simultaneously. Because the LV mass is larger than the RV the mean vector of electrical current is the LV depolarization.

When there is an electrical block in the normal conduction pathway for ventricular depolarization it is a called a bundle branch block. This block can be permanent or intermittent and has a variety of causes. Depolarization occurs because of the principle of conductivity. This depolarization takes longer (QRS duration > 0.12sec) and the configuration is slightly different than the normal QRS pattern. Bundle Branch Block patterns are best evaluated in precordial leads $V_1$ and $V_6$. 
Right Bundle Branch Block

V1 rsR' > 0.12sec
V6 qRs > 0.12sec

Left Bundle Branch Block

V1 rS > 0.12sec
V6 R > 0.12sec
I. INTRODUCTION

PCCN Blueprint

*Heme, Endocrine, Renal & GI: 18%

a. Anemia
b. Cancer
c. Hemostasis Disorders (coagulopathies)
   • Heparin-Induced Thrombocytopenia (HIT)
   • Drug-Induced Overdose (Coumadin, Pradaxa)
d. Immunosuppressive Disorders

II. PHYSIOLOGY OF HEMATOPOIETIC SYSTEM

Purpose

a. Circulate
b. Provide Nutrition
c. Provide Oxygen
d. Remove Waste Products (carbon dioxide and metabolic wastes)
e. Maintain Hemostasis

Location

a. Veins & Venules: 66%
b. Pulmonary Loop: 12%
c. Arteries & Arterioles: 11%
d. Heart: 6%
e. Capillaries: 5%

Composition

4-6 liters of blood
a. Plasma: 55%
b. Cellular Component: 45%
   • Erythrocytes (red blood cells)
   • Leukocytes (white blood cells)
   • Thrombocytes (platelets)

Components

Hematopoiesis – blood cells come from stem cells in the bone marrow
# Complete Blood Count (CBC)

<table>
<thead>
<tr>
<th>Test</th>
<th>Normal findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Red Blood Cell</td>
<td>- Male: 4.6 – 6.0 million/mm³</td>
</tr>
<tr>
<td></td>
<td>- Female: 4.0 – 5.0 million/mm³</td>
</tr>
<tr>
<td>Mean Corpuscular Volume (MCV)</td>
<td>- Men: 78 – 100 cubic micrometers</td>
</tr>
<tr>
<td></td>
<td>- Female: 78 – 102 cubic micrometers</td>
</tr>
<tr>
<td>Mean Corpuscular Hemoglobin (MCH)</td>
<td>- 25 – 35 pg</td>
</tr>
<tr>
<td>Mean Corpuscular Hemoglobin Concentration (MCHC)</td>
<td>- 31 – 37%</td>
</tr>
<tr>
<td>RBC Distribution Width (RDW)</td>
<td>- 11.5% - 14.5%</td>
</tr>
<tr>
<td>Erythrocyte Sedimentation Rate (Sed Rate)</td>
<td>- Male: 0 – 17mm/hr</td>
</tr>
<tr>
<td></td>
<td>- Female: 1 – 25mm/hr</td>
</tr>
<tr>
<td>Hematocrit (Hct)</td>
<td>- Male: 37 – 49%</td>
</tr>
<tr>
<td></td>
<td>- Female: 36 – 46%</td>
</tr>
<tr>
<td>Hemoglobin (Hgb)</td>
<td>- Male: 13 – 18 g/100ml</td>
</tr>
<tr>
<td></td>
<td>- Female: 12 – 16 g/100ml</td>
</tr>
<tr>
<td>Hemoglobin Electrophoresis</td>
<td>- Hgb A₁ = 95-98%</td>
</tr>
<tr>
<td></td>
<td>- Hgb A₂ = 1.5%</td>
</tr>
<tr>
<td></td>
<td>- Hgb F &lt; 2%</td>
</tr>
<tr>
<td>Methemoglobin</td>
<td>- &lt; 1% of total Hemoglobin</td>
</tr>
<tr>
<td>Reticulocyte Count</td>
<td>- 0.5 – 2.5% of total RBC count</td>
</tr>
<tr>
<td>White Blood Cells</td>
<td>- 4,500 – 11,000/mm³</td>
</tr>
<tr>
<td>Polymorphonuclear (PMN) or Granulocytes Leukocytes</td>
<td>%</td>
</tr>
<tr>
<td>Neutrophils</td>
<td>45 – 75%</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>0 – 4%</td>
</tr>
<tr>
<td>Basophils</td>
<td>0 – 3%</td>
</tr>
<tr>
<td>Mononuclear Leukocytes</td>
<td>%</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>25 – 40%</td>
</tr>
<tr>
<td>Monocytes</td>
<td>4 – 6%</td>
</tr>
</tbody>
</table>
### Coagulation Profiles

<table>
<thead>
<tr>
<th>TEST</th>
<th>NORMAL RANGE</th>
<th>PARAMETER MEASURED</th>
</tr>
</thead>
<tbody>
<tr>
<td>Platelet Count</td>
<td>150,000-400,000/mm³</td>
<td># of Circulating Platelets, Measures Amount not Functional Ability</td>
</tr>
<tr>
<td>Prothrombin Time (PT)</td>
<td>11-15 seconds</td>
<td>Extrinsic &amp; Common Coagulation Pathways</td>
</tr>
<tr>
<td>International Normalized Ratio (INR)</td>
<td>0.7 – 1.8</td>
<td>Standardized Method of Reporting the PT</td>
</tr>
<tr>
<td>Partial Thromboplastin Time (PTT)</td>
<td>APTT 20-35 seconds, PTT 60 – 70 seconds</td>
<td>Intrinsic &amp; Common Coagulation Pathways</td>
</tr>
<tr>
<td>Bleeding Time</td>
<td>Depends on system Ivy 1-8, Duke 1-3min</td>
<td>Normal Platelet and Tissue Function with Bleeding</td>
</tr>
<tr>
<td>Activated Clotting Time (ACT)</td>
<td>70 – 120 seconds</td>
<td>Intrinsic &amp; Common Coagulation Pathways</td>
</tr>
<tr>
<td>Fibrinogen</td>
<td>200 - 400mg/dL</td>
<td>Circulating Fibrinogen</td>
</tr>
<tr>
<td>Thrombin Time (TT)</td>
<td>14 -16 sec</td>
<td>Common Coagulation Pathway and Quality of the Functional Fibrinogen</td>
</tr>
<tr>
<td>Fibrin Degradation (Split) Products</td>
<td>2-10mcg/ml</td>
<td>Degree of Fibrinolysis</td>
</tr>
<tr>
<td>D-Dimer</td>
<td>&lt; 2.5mcg/ml</td>
<td>Specific Fibrin Breakdown Product</td>
</tr>
</tbody>
</table>

### III. BLEEDING DISORDERS

#### Causes for Bleeding

a. Vessel Integrity Disruption
   - Surgical
   - Trauma
b. Platelet Disorders
   - Quantitative
   - Qualitative
c. Coagulation Disorders
   - Acquired
   - Congenital

#### Coagulation Disorders

**Acquired**

a. Malnutrition
b. Liver Dysfunction (decrease synthesis of factors)
c. Vitamin K Deficiency
d. GI Dysfunction (unable to absorb Vit K)
e. Uremia
f. Medications (heparin, Coumadin)
g. Massive Transfusions

h. Consumptive Coagulopathies (DIC)

**Congenital**

a. Abnormal Structure or Function of Blood Vessels
   - Rendu-Osler-Weber Disease

b. Platelet Coagulation Abnormality
   - Kasabach-Merrit Syndrome
   - vonWillebrand’s Disease
   - Hemophilia A or B
   - Afibrinogenemia

c. Hyper-Coagulable Disorders
   - Protein C or S Deficiency

**DIC - Disseminated Intravascular Coagulation**

**Definition**

DIC is a secondary disorder resulting from a primary pathophysiologic state or disease. It is complex because it presents as an over stimulation of both bleeding and thrombosis. The victim has microvascular thrombi and bleeding occurring simultaneously. The disorder can be life-threatening, acute or chronic and has a mortality rate of 50%-80%. When DIC is a complication of sepsis or shock the mortality rate can be as high as 90%. It frequently is associated with MODS.

**Risk Factors**

There does not appear to be one common risk factor for this acquired coagulation disorder.
### Risk Factors for DIC

<table>
<thead>
<tr>
<th>General Classifications</th>
<th>Primary Event/Disorder</th>
<th>Primary Event/Disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tissue Damage</td>
<td>Major Surgery</td>
<td>Burns</td>
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<td>Major Trauma</td>
<td>Transplant Rejection</td>
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<td>Heat Stroke</td>
<td>Extracorporeal Circulation</td>
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<td>Head Injury</td>
<td>Snake Bites</td>
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<td>Obstructive Complications</td>
<td>HELLP</td>
<td>NS Abortion</td>
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<td></td>
<td>Amniotic Emboli</td>
<td>Eclampsia</td>
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<td>Abruptio Placenta</td>
<td>Placenta Accreta</td>
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<td>Fetal Demise</td>
<td>Placenta Previa</td>
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<tr>
<td>Shock States</td>
<td>Cardiogenic Shock</td>
<td>Massive blood and volume resuscitation</td>
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<td></td>
<td>Septic Shock (severe infection or inflammation)</td>
<td>Drowning</td>
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<td>Hemorrhagic Shock</td>
<td>Anaphylaxis</td>
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<td>Dissecting Aneurysm</td>
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<td>Neoplasms</td>
<td>Acute &amp; Chronic Leukemia</td>
<td>Solid Tumors</td>
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<td>Acute &amp; Chronic Lymphoma</td>
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<tr>
<td>Hematologic Disorders</td>
<td>Thrombotic Thrombocytopenic Purpura (TTP)</td>
<td>Collagen Vascular Disorders</td>
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<td>Thrombocythemia</td>
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<td>Sickle Cell Crisis</td>
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<tr>
<td>Specific System Dysfunction</td>
<td>Acute &amp; Chronic Renal Dis</td>
<td>Acute Pancreatitis</td>
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<td>Ulcerative Colitis</td>
<td>Liver Dysfunction/Failure</td>
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<td>DKA, Acid Ingestion</td>
<td>SIRS &amp; MODS</td>
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<td>HIV Disease</td>
<td>Pulmonary Embolism</td>
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<td>Cirrhosis</td>
<td>Fat Embolism</td>
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</tbody>
</table>

**Common Physiologic Response**

a. Tissue damage  
b. Platelet damage  
c. Endothelial damage

**Pathophysiology**

a. Tissue Damage Occurs  
b. Healing is Stimulated (Clotting)  
c. Hemopoietic Chaos  
d. Fibrinolytic Mediators Released  
e. Initially Microvascular Thrombi  
f. Consumption Exceeds Synthesis  
g. Ability to Clot is Lost  
h. Fibrinolytic Mediators “Run a Muck”  
i. Lyse all Clots  
j. Bleeding State  
k. Consumption Coagulopathy
Physical Assessment and Findings
The primary problem and pre-existing condition certainly play a major role in the presentation. All systems are at risk for dysfunction. The most common problems occur in the pulmonary, renal and hematopoietic systems. Any bleeding patient who does not have a history of or “reason” to bleed should be suspected of DIC.

<table>
<thead>
<tr>
<th>Laboratory Findings</th>
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<tbody>
<tr>
<td><strong>Test</strong></td>
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<tr>
<td>Hgb</td>
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<tr>
<td>HCT</td>
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<tr>
<td>Platelet Ct</td>
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<tr>
<td>PT</td>
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<tr>
<td>PTT</td>
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<tr>
<td>Fibrinogen</td>
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<tr>
<td>FDP/FSP</td>
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<tr>
<td>D-Dimer</td>
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</table>

Treatment
No definitive treatment exists for DIC. The major goal is to treat primary disorder – stopping the hemopoietic chaos. In addition patient and family emotional support is paramount for quality nursing care.

a. Support/Treat the Primary Problem – Eradicate the Cause of DIC
b. Early Recognition
c. Decrease Bleeding Risk
d. Treat Pain
e. Transfusion Therapy – PRBC, FFP, Platelets, Cyro
f. Vit K
g. Anticoagulation Therapy – Heparin
h. General Critical Care Management

Heparin Induced Thrombocytopenia (HIT) & Thrombus

a. Acquired Allergy to Heparin
b. Antibodies are Produced to Heparin
c. With Heparin Admin the Antibodies ‘attack’ Heparin and Thrombocytes
d. Pt’s Platelet Count Drops (typically by 50% from baseline)
e. Some Patients Will Develop Thrombi
f. Treatment is to Stop all Heparin
g. Admin Non-Heparin Anticoagulant
h. Admin Platelets ONLY if Needed
Thrombotic Thrombocytopenic Purpura (TTP)

a. Drop in Platelet Ct
b. Hemolytic Anemia
c. Classically Presents with Neuro Symptoms or Renal Dysfunction and Fever
d. Difficult Diagnosis
e. Causes: Drugs or BMT, Autoimmune Dis, AIDS, Depressed Bone Marrow, DIC, WCS, Bleeding, Extracorporeal Cir., Medications, Artificial Heart Valve, Hemodilution
f. Treatment
   • Stop Cause
   • Admin Platelets or Neumega
   • Plasmapheresis

Idiopathic Thrombocytopenic Purpura (ITP)

a. Thrombocytopenia < 150,000
b. Unable to Determine Cause

Drug Induced Coagulopathies

The Physiology of Coagulation & Fibrinolysis (review)

Hemostatic Mechanisms
The actual forming of a blood clot is a complex integration of mechanisms of the blood vessels, thrombocytes, erythrocytes, coagulation factors, endothelial cells, leukocytes and a myriad of chemical mediators

Physiological Clotting Process
a. Local Vascular Response - vasoconstriction
b. Activation of Platelets & Formation of a Platelet Plug
c. Formation of a Blood Clot

Fibrinolytic Mechanisms
a. Enzymatic degradation of fibrin clot by plasmin
b. Hemostasis/Fibrinolysis Control Mechanisms

Anticoagulants
Principle indication for anticoagulant therapy is to prevent or decrease the risk of venous thrombosis.

Indirect Thrombin Inhibitors
a. Unfractionated Heparin: Monitor aPTT, reversal agent Protamine
b. Low-Molecular-Weight Heparins: Monitor Antifactor Xa, reversal agent Protamine
c. Arixtra (Fondaparinux) synthetic – no reversal
d. Xarelto (Rivaroxaban) oral – no reversal

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Direct Thrombin Inhibitors
a. Hirudin & Derivatives – IV: leech saliva – no reversal
b. Argatroban – synthetic IV – no reversal
c. Pradax (Dabigatran) oral – no reversal

Vitamin K Antagonist
a. Coumadin (Warfarin), Monitor PT/INR, reversal agent vit K

Antiplatelet Agents
Principle indication for antiplatelet therapy is to prevent or decrease the risk of arterial thrombosis. Bleeding time is the primary monitoring test and no reversal agent has been identified
a. Aspirin
b. Non-Aspirin NSAIDs
c. Adenosine Diphosphate Receptor Antagonists
   • Plavix (Clopidogrel)
   • Prasugrel (Effient)

IV. ANEMIA

The primary problem with anemia, decrease number of Red Blood Cells, is that the body will not have adequate oxygen delivery.

Etiologies
a. Blood Loss
b. Lack of or Underproduction of Red Cells
   • Malnutrition
   • Chronic Illness
   • Cancer or Cancer Treatments
   • Liver or Renal Dysfunction
   • Macrocytic
   • Microcytic
c. Destruction of Red Cells or Hemolysis
   • Cardiopulmonary Bypass Machine
   • Immune Response
   • Sickle Cell Disease
   • TTP
Clinical Presentation

Directly result from lack of oxygen delivery
a. Tachycardia  
b. Rapid Respiratory Rate  
c. Weak Pulses  
d. Orthostatic Hypotension  
e. Decreased Urinary Output  
f. Decreased LOC  
g. Hypovolemic Shock

Treatment Option

a. Identify and Treat the Underlying Cause  
b. Administer Packed Red Blood Cells  
c. Recombinant Human Erythropoietin  
d. Supplemental Vitamins & Minerals  
e. Blood Conservation Procedures  
f. Maintain Hgb 7-9 (non-bleeding patient)

V. IMMUNOLOGY – ONCOLOGY

Etiology of Immunosuppression

a. Primary Neutropenia  
b. Immunosuppressive Agents (chemo, anti-rejection)  
c. Radiation Therapy  
d. Autoimmune Disorders  
e. Viral Infections (HIV/AIDS)  
f. Genetic Disorders  
g. Diseases/Disorders (DM, ETOH abuse)  
h. Chronically Critically Ill and Septic

Care Goal Priorities

a. Safety  
b. Prevention of Opportunistic Infection  
c. Monitoring and Treatment if Infection  
d. General Support
HIV/AIDS

A virus spread by blood and body fluids. Incubation period can be 45 days → 6 months. Pt feels “flu like” with some lymphadenopathy in the early stages. The virus destroys the CD4 lymphocyte causing immunosuppression. The first concern with HIV is prevention and education. The second is caring for the Immunosuppressed individual. Currently infection to AIDS is about 10 – 12 years. In the ED setting assess for opportunistic infections, neutropenic precautions, education and emotional and psychological support.

HIV Testing

The target cells for the virus are the T helper cells (CD4). Antibodies will not be detectable immediately after exposure.

Enzyme-Linked Immunosorbent Assay (ELISA)

Antibody test with 94 – 99% sensitivity. It may take between 6 weeks – 6 months for antibody tests to be positive. Confirmed with a Western blot.

CD4 Levels

The overall T cell count will decrease secondary to the drop in CD4 cells. Levels of CD4 are more valuable than total count, a CD4 below 200/mm³ is reflective of HIV. The CD4 is also used to monitor the effectiveness of therapy or disease progression.

Viral Load

Measurement of the HIV-RNA present in the blood. Levels of < 10,000 are considered low risk for disease progression, > 100,000 are considered high risk for progression to AIDS. This measurement is also used to evaluate the effectiveness of therapy.

HIV/AIDS Education

a. Medication Education
b. Infection Prevention and Early Identification
c. Safe Sex Practices
d. Communicate with Intimate Partners
e. Do Not Share Personal Hygiene Items
f. Community Resources
g. Avoid Smoking and ETOH
h. Encourage Healthy Living (eating, sleeping, exercise...)

Prevention & Early Detection

Patients and Families
a. Smoking Cessation
b. Low Fat Diets
c. Ideal Body Wt
d. Exercise
e. Cancer Screening (colonoscopy at 50yo, mammograms...)
f. Sun Screen Use