Hematology/Immunology/Oncology

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I. INTRODUCTION

PCCN Blueprint

_Heme, Endocrine, Renal & GI: 18%

- Anemia
- Cancer
- Hemostasis Disorders (coagulopathies)
  - Heparin-Induced Thrombocytopenia (HIT)
  - Drug-Induced Overdose (Coumadin, Pradaxa)
- Immunosuppressive Disorders

II. PHYSIOLOGY OF HEMATOPOIETIC SYSTEM

Purpose

- Circulate
- Provide Nutrition
- Provide Oxygen
- Remove Waste Products (carbon dioxide and metabolic wastes)
- Maintain Hemostasis

Location

- Veins & Venules: 66%
- Pulmonary Loop: 12%
- Arteries & Arterioles: 11%
- Heart: 6%
- Capillaries: 5%

Composition

4-6 liters of blood

- Plasma: 55%
- 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

### Assessment

#### Complete Blood Count (CBC)

<table>
<thead>
<tr>
<th>Test</th>
<th>Normal findings</th>
</tr>
</thead>
</table>
| Red Blood Cell                            | Male: 4.6 – 6.0 million/mm³  
Female: 4.0 – 5.0 million/mm³  |
| Mean Corpuscular Volume (MCV)             | Men: 78 – 100 cubic micrometers  
Female: 78 – 102 cubic micrometers  |
| Mean Corpuscular Hemoglobin (MCH)         | 25 – 35 pg                                           |
| Mean Corpuscular Hemoglobin Concentration (MCHC) | 31 – 37%                                           |
| RBC Distribution Width (RDW)              | 11.5% - 14.5%                                        |
| Erythrocyte Sedimentation Rate (Sed Rate) | Male: 0 – 17mm/hr  
Female: 1 – 25mm/hr                                           |
| Hematocrit (Hct)                          | Male: 37 – 49%  
Female: 36 – 46%                                           |
| Hemoglobin (Hgb)                          | Male: 13 – 18 g/100ml  
Female: 12 – 16 g/100ml                                           |
| Hemoglobin Electrophoresis                | Hgb A₁ = 95-98%  
Hgb A₂ = 1.5%  
Hgb F < 2%                                           |
| Methemoglobin                             | < 1% of total Hemoglobin                             |
| Reticulocyte Count                        | 0.5 – 2.5% of total RBC count                        |
| White Blood Cells                         | 4,500 – 11,000/mm³                                    |
| Polymorphonuclear (PMN) or Granulocytes Leukocytes | % | Absolute count |
| Neutrophils                               | 45 – 75%  | 2,000 – 7,000 |
| Eosinophils                               | 0 – 4%   | 0 – 400       |
| Basophils                                 | 0 – 3%   | 0 – 200       |
| Mononuclear Leukocytes                    | % | Absolute count |
| Lymphocytes                               | 25 – 40% | 1700 – 3500  |
| Monocytes                                 | 4 – 6%   | 200 – 600     |
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>
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<tr>
<td>International Normalized Ratio</td>
<td>0.7 – 1.8</td>
<td>Standardized Method of Reporting the PT</td>
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<tr>
<td>(INR)</td>
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<tr>
<td>Partial Thromboplastin Time (PTT)</td>
<td>APTT 20-35 seconds</td>
<td>Intrinsic &amp; Common Coagulation Pathways</td>
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<tr>
<td>Activated Partial Thromboplastin</td>
<td>PTT  60 – 70 seconds</td>
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<tr>
<td>Time (APTT)</td>
<td></td>
<td></td>
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<tr>
<td>Bleeding Time</td>
<td>Depends on system</td>
<td>Normal Platelet and Tissue Function with Bleeding</td>
</tr>
<tr>
<td>Ivy 1-8, Duke 1-3min</td>
<td></td>
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<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>
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<tr>
<td>D-Dimer</td>
<td>&lt; 2.5mcg/ml</td>
<td>Specific Fibrin Breakdown Product</td>
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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
   • von Willebrand’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.
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<th>Risk Factors for DIC</th>
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<td><strong>General Classifications</strong></td>
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<td>Tissue Damage</td>
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<td>Obstetric Complications</td>
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<td>Hematologic Disorders</td>
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<td>Specific System Dysfunction</td>
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**Common Physiologic Response**

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

d. **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
Hematology/Immunology/Oncology

**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.

| Laboratory Findings |
|---------------------|------------------|
| Test                | Elevated | Decreased | |
| Hgb                 |          |          | |
| HCT                 |          |          | |
| Platelet Ct         |          |          | |
| PT                  |          |          | |
| PTT                 |          |          | |
| Fibrinogen          |          |          | |
| FDP/FSP             |          |          | |
| D-Dimer             |          |          | |

**Treatment**
No definitive treatment exists for DIC. The major goal is to treat primary disorder – stopping the hemapoietic 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
**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 Underlining 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/mm$^3$ 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