Checklist for Success

We will guarantee your success on the CCRN certification exam!
-- If you study the right things in the right ways --

Success Checklist:

☐ Attend the entire CCRN: Test Prep program (or use the entire A/V package).

☐ Study 5 hours per week for 90 days using the handout, pocket study guide, and flash cards.

☐ Listen to all of the audio CDs or watch all of the video-enhanced CDROMs.

☐ Identify areas of weakness that need additional study.

☐ Review the audio CDs (or CDROMs) of the topics you identified as requiring additional study.

☐ Participate in the Nurses’ Success Network on-line study groups and post at least one comment or question per week.
  Login at: www.Nurses-Success-Network.com
  User: ccrn
  Password: excellence

☐ Achieve a passing grade of at least 80% on the “Challenge Exam” on-line at the Nurses’ Success Network.

☐ Use the on-line resources recommended in the “Challenge Exam” results.

The CCRN: Test Prep is a 90-day program to guarantee your success on the certification exam. You must use this program and take the exam within 90 days of registering for the guarantee for us to assure your success.

Register for the guarantee on-line at :www.Nurses-Success-Network.com
**CCRN: Test Prep**

Description:
This unique two-day program presents the content of the CCRN exam in a question and answer format. By the conclusion of the program the participant will have answered 150 questions in the format and distribution of the actual exam. In-depth explanations will be presented for rationale behind correct and incorrect answers, along with the theoretical underpinnings of essential concepts.

This unique, informative and fun seminar is perfect for CCRN preparation, or a comprehensive critical care review.

Objectives:
1. Examine strategies for successful completion of the CCRN exam.
2. Describe common hematologic and immunologic dysfunction in the critical care patient.
3. Describe the process of coagulopathy in DIC.
4. Compare and contrast common GI disorders.
5. Plan care for the patient suffering from abdominal trauma.
6. Compare and contrast septic, hypovolemic, and cardiogenic shock.
7. Describe hemodynamic changes that occur with shock.
9. Compare and contrast acute and chronic renal failure.
10. Describe clinical symptoms of electrolyte disturbances.
12. Explain the benefits of several treatment options for acute respiratory failure.
14. Describe a simple assessment plan for patients with increased intracranial pressure.
15. Evaluate nursing interventions for increased intracranial pressure.
17. Compare and contrast diabetic ketoacidosis and hyperosmolar, hyperglycemic syndrome.
18. Define professional and ethical nursing care using AACN definitions.
**DAY 1**

8:00  Introduction and Test Overview

8:30  Hematologic / Immunologic (3%)
     A&P Blood Products & Plasma
     Organ Transplantation
     Life-threatening coagulopathies
     Immunosuppression-Acquired
     Sickle Cell Crisis

9:45  Break

10:00 Gastrointestinal (6%)
     GI Bleed
     Hepatic Failure
     Acute Pancreatitis
     Bowel infarction/obstruction/perforation
     Abdominal Trauma

11:30 Multisystem (8%)
     Sepsis / Septic Shock / MODS
     Toxic Ingestions
     Toxic Exposures

12:00 Lunch

1:00  Multisystem (con’t)

1:30  Cardiovascular (32%)
     Acute Coronary Syndromes
     Cardiac Inflammatory Disease
     Conduction System Defects
     Acute Heart Failure & Pulmonary Edema
     Aortic Aneurysm
     Pericarditis

2:15  Break

2:30  Cardiovascular (continued)
     Cardiac Trauma
     Hypertensive Crisis
     Shock

4:30  Adjourn
DAY 2
8:00  Renal (5%)
   Acute & Chronic Renal Failure
   Renal Trauma
   Electrolyte Imbalances
9:00  Pulmonary (17%)
   Acute Respiratory Failure
   Pulmonary Pharmacology
9:45  Break
10:00 Pulmonary (con’t)
   ARDS
   Pneumonia
   Pulmonary Embolus, Fat Embolus
   Asthma / COPD
   Chronic Lung Disease
   Thoracic Trauma / Thoracic Surgery
12:00 Lunch
1:00  Neurologic (5%)
   Aneurysm
   Encephalopathy
   Stroke (ischemic, hemorrhagic)
   Intracranial Hemorrhage
   Seizures
   Head Trauma
   Neurosurgery / ICP Monitoring
2:00  Break
2:15 Endocrine (4%)
   Diabetes Insipidus
   Diabetes Ketoacidosis & Hyperosmolar Coma
   Acute Hypoglycemia
   Hormones and Endocrine A&P
3:00  Professional Caring and Ethical Practice (20%)
   Advocacy
   Collaboration
   Caring Practice
4:00  Adjourn
Today’s speaker:

David W. Woodruff, MSN, RN, CNS

David began his healthcare career as a paramedic. After years of treating patients “in the field”, David obtained his nursing degree. His extensive experience includes trauma nursing at a level-I trauma center, and staff positions in Neurological, Coronary, Medical and Surgical Intensive Care Units. David holds a Master’s degree in Adult Health nursing and is a Clinical Nurse Specialist in Critical Care Nursing. He is a member of AACN, The Society of Critical Care Medicine, and Sigma Theta Tau. He has served as an Instructor of Nursing, Unit Manager, Nursing Expert Witness, and President of a private nursing consulting firm. David presents seminars throughout the country on a variety of topics including critical care and medical-surgical nursing, and has published articles in Nursing, RN, and Image. He is widely regarded as a knowledgeable and thorough instructor who can make even the most difficult content material understandable.

I would be happy to hear from you and answer any additional questions you may have. Feel free to contact me at:
Phone: (330) 467-2629
e-mail: askdavid@ed4nurses.com
web: www.ed4nurses.com
Introduction and Test Overview

1. Why Become Certified?

A study conducted by the Nursing Credentialing Research Coalition found that certification has a profound impact on the personal, professional and practice outcomes of certified nurses. Overall, nurses in the study stated that certification enabled them to experience fewer adverse events and errors in patient care than before they were certified. Additional results revealed that certified nurses:

- expressed more confidence in detecting early signs of complications;
- reported more personal growth and job satisfaction;
- believed they were viewed as credible providers;
- received high patient satisfaction ratings;
- reported more effective communication and collaboration with other health care providers; and
- experienced fewer disciplinary events and work-related injuries.

2. What is “CCRN”?
   a. Registered service mark of AACN.
   b. Credential for certified critical care nurses.

3. What to Expect from “The Test”

<table>
<thead>
<tr>
<th>AACN – Certification Corporation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fees: $300 non-member</td>
</tr>
<tr>
<td>$220 member of AACN</td>
</tr>
<tr>
<td>Test dates: Year-round</td>
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<tr>
<td>Requirements: RN license</td>
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<tr>
<td>1750 hours of clinical experience with acute and critical care patients within the previous 2 years (875 within the past year).</td>
</tr>
</tbody>
</table>

If you join AACN ($78 fee) at the time you register, you pay $298 and get member benefits.

Exam is computer-based, 150 questions, with a 3-hour time limit
Paper-based testing is offered at the NTI
Certification is for 3 years.
Recertification can be by CERPs or re-testing.
Cost of recertification is: $250 non-member, $170 member
4. Testing Dates, Places and Times

AACN Certification Corporation
101 Columbia
Aliso Viejo, CA  92656-4109
Phone: (800) 899-2226
E-mail: certcorp@aacn.org
Web: www.certcorp.org

Applied Measurement Professionals Inc. (AMP)
8310 Nieman Road
Lenexa, KS 66214-1579
Phone: (800) 345-6559
Fax: (913) 541-0156
Business Hours: 8:30 am - 5:00 pm CST Monday-Friday
E-mail: info@goamp.com
Web: http://www.goamp.com/

Over 100 testing centers nationwide

5. What to bring with you:
   a. Photo ID
      i. Driver’s license
      ii. State ID card
      iii. Military ID card
   b. Second ID without photo
   c. Do not bring any personal items with you
   d. 

**Please Note:**
This is a focused 90-day program designed to assure your success on the ANCC Med-Surg certification exam. You must register for the guarantee and complete the “Certification Checklist” within 90 days to be eligible for the guarantee.

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**You can do this!**

- If you are qualified
- And you study the right stuff in the right way
- You will pass!

*I guarantee it!*
Hematologic / Immunologic (3%) 4 questions

1. The nurse is caring for a 32-year-old experiencing organ rejection after a kidney transplant. Which of the following signs will the patient exhibit?
   a. Decreased BUN/Creatinine
   b. Increased transaminase level
   c. Increased urine output
   d. Increased BUN/Creatinine

2. A primary chemical mediator in anaphylactic reaction is?
   a. Myocardial Depressant Factor
   b. Histamine
   c. Complement
   d. Interferon

3. Which of the following laboratory diagnostic findings will most likely be seen in DIC?
   a. PT and PTT prolonged
   b. Fibrinogen increased
   c. Platelet count increased
   d. D-dimer normal

4. The beneficial effects of heparin in DIC are thought to be due to its:
   a. Stimulating effect on platelet manufacture
   b. Neutralizing of free-circulating thrombin
   c. Antifibrinolysin activity
   d. Inhibition of platelet factor XII release
Hematology

1. Functions:
   a. Medium for transport of O₂ and CO₂ and nutrients
   b. Maintains hemostasis
   c. Maintains internal environment
   d. Immune
   e. Inflammation
   f. Stress Response
      i. Impaired skin barrier or irritated mucous membrane
      ii. Impaired gag, cough or swallow
      iii. Increased gastric pH, colonization = aspiration
      iv. Acute Stress Reactions
         1. Catabolism
         2. Decreased healing
         3. Inhibit immune response
         4. Inflammatory Response
   g. Hemostasis
      i. Termination of bleeding
      ii. Vascular response
      iii. Platelet response
      iv. Coagulation
         1. Platelets
         2. Thrombocytopenia
         3. HITT response

Disseminated Intravascular Coagulation (DIC)

1. Definition
2. Factors Triggering DIC
3. Etiology:
   a. Bleeding
   b. Trauma
   c. Sepsis
   d. Abrupto Placenta
4. Clinical Presentation
   a. Bleeding
   b. Signs of Thrombosis
   c. Clinical Presentation
      i. Petechiae
      ii. Ecchymosis
      iii. Purpura
   d. Labs in DIC
      i. Platelets
      ii. PTT
      iii. PT
      iv. Fibrinogen
      v. FDP/FSP
      vi. D-dimer
      vii. Antithrombin III

5. Medical Management
   a. Maintain ABC’s
   b. Careful or oral and mucosal bleeding
   c. Treat stimuli
   d. Correct hypovolemia, hypotension, hypoxia, and acidosis
   e. Stop microclotting to maintain perfusion
   f. Stop Bleeding
   g. Stop Thrombosis
   h. Administer IV Heparin
   i. Plasmapheresis
   j. Nursing Management
   k. Nursing Care of the Bleeding Patient
   l. Blood Products
      i. Risks of transfusion
      ii. PRBC’s
      iii. Platelets
      iv. FFP
      v. Cryoprecipitate
      vi. Adverse Reactions

6. Complications of DIC
   a. Mortality
   b. Hypovolemic Shock
   c. Acute Renal Failure
   d. Infection
   e. Acute Respiratory Distress Syndrome
   f. Stroke
   g. GI dysfunction
7. Nursing
   a. Administer Vitamin K and Folic Acid
   b. Treat Ischemic Pain
   c. Maintain skin integrity

**Acquired Immunodeficiency Syndrome (AIDS)**

1. Etiology
   a. HIV, CD4 retrovirus
   b. High-risk groups
      i. High-risk sexual behavior
      ii. Infected sex partners
      iii. IV drug users
      iv. Recipients of blood products before 1985
   c. Pathophysiology
      i. Invasion and destruction of T4 (helper) cells
      ii. Incubation 6 months to 10 years
      iii. Decreased immune response
      iv. Opportunistic infection

2. General principles for management
   a. Universal precautions
   b. Protect from infection
   c. Inflammatory response will be muted

**Transplantation**

Criteria for organ transplantation

1. Recipient criteria
   a. End-stage organ disease
   b. Absence of:
      i. Infection
      ii. Malignancy
      iii. Other failing organs
      iv. Substance abuse

1. Donor criteria
   a. Free of sepsis, cancer, prolonged hypotension
   b. Free of communicable disease
### Anti-rejection medications

<table>
<thead>
<tr>
<th>Drug</th>
<th>Major Effects</th>
<th>Side Effects</th>
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<tbody>
<tr>
<td>Corticosteroids</td>
<td>↓ Inflammation</td>
<td>↑ Risk of infection</td>
</tr>
<tr>
<td></td>
<td></td>
<td>GI bleed</td>
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<tr>
<td></td>
<td></td>
<td>Hyperglycemia</td>
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<td></td>
<td></td>
<td>Adrenal suppression</td>
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<tr>
<td>Cyclosporine</td>
<td>↓ Immune and inflammatory responses</td>
<td>Potentiates other immunosuppressives</td>
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<tr>
<td></td>
<td></td>
<td>Hepatotoxicity</td>
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<td></td>
<td></td>
<td>Nephrotoxicity</td>
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<td></td>
<td></td>
<td>Hyperkalemia</td>
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<tr>
<td></td>
<td></td>
<td>Hypomagnesemia</td>
</tr>
<tr>
<td>ATGAM (antithymocyteglobulin)</td>
<td>Reduces T-cell production</td>
<td>↑ Risk of infection</td>
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<tr>
<td></td>
<td></td>
<td>Thrombocytopenia</td>
</tr>
<tr>
<td>Imuran (azathioprine)</td>
<td>↓ Immune response</td>
<td>↑ Risk of infection</td>
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<tr>
<td></td>
<td></td>
<td>Oral and gastric erosion</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hepatotoxicity</td>
</tr>
<tr>
<td>OKT3 (muromonab-CD3)</td>
<td>Alters T-cell recognition of antigens</td>
<td>↑ Risk of infection</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Symptoms of infection</td>
</tr>
<tr>
<td></td>
<td></td>
<td>↓ WBC, platelet levels</td>
</tr>
<tr>
<td>Prograf (tacrolimus)</td>
<td>↓ Inflammatory response</td>
<td>GI distress</td>
</tr>
<tr>
<td></td>
<td></td>
<td>HTN, chest pain</td>
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<tr>
<td></td>
<td></td>
<td>Hyperkalemia</td>
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<td></td>
<td></td>
<td>Hepatotoxicity</td>
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<tr>
<td>CellCept (mycophenolate)</td>
<td>↓ Immune response</td>
<td>GI distress</td>
</tr>
<tr>
<td></td>
<td></td>
<td>↓ WBC, platelet levels</td>
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<tr>
<td></td>
<td></td>
<td>Hypertension</td>
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<tr>
<td></td>
<td></td>
<td>Hypokalemia</td>
</tr>
</tbody>
</table>

1. General patient care
   a. Support transplanted organ
      i. Heart Transplant
      ii. Lung
      iii. Liver
      iv. Pancreas
      v. Kidney
   b. Watch for signs of infection
      i. May be ↓ due to ↓ immune response
**Leukemia’s**

<table>
<thead>
<tr>
<th>Acute</th>
<th>Incidence</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute Lymphocytic (ALL)</td>
<td>Age 2-4</td>
<td>Anemia, Bleeding, Infection, ↓ RBC, H&amp;H, ↑ WBC, Joint and bone pain</td>
</tr>
<tr>
<td>Acute Myelogenous (AML)</td>
<td>Age 12-20</td>
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<thead>
<tr>
<th>Chronic</th>
<th>Incidence</th>
<th>Characteristics</th>
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<tbody>
<tr>
<td>Chronic Lymphocytic (CLL)</td>
<td>Age 50-70</td>
<td>↑ WBC, ↓ RBC, Enlarged spleen, Hepatomegaly, Swollen glands</td>
</tr>
<tr>
<td>Chronic Myelogenous (CML)</td>
<td>Age 30-50</td>
<td></td>
</tr>
</tbody>
</table>

- a. Diagnostics
  - i. Bone marrow aspiration
- b. Treatment
  - ii. Chemotherapy
  - iii. Stem cell transplant
  - iv. Transfusion

3. Multiple Myeloma
   - a. Plasma cells invade bone marrow, and lymph system
   - b. Bones become weak and painful
   - c. Diagnostics
     - i. X-rays
     - ii. Bone marrow aspiration
     - iii. Hypercalcemia
   - d. Treatment
     - i. Chemotherapy
     - ii. Interferon
     - iii. Bone marrow transplantation
     - iv. Plasmapheresis
     - v. Management of Hypercalcemia

4. Non-Hodgkin’s Lymphoma
   - a. Malignant neoplasm of the lymphatic system
   - b. Results in overgrowth of premature and ineffective cells
   - c. Diagnostics
     - i. Fever, swollen glands, night sweats, weight loss
   - d. Treatment
     - i. Chemotherapy
     - ii. Radiation therapy
     - iii. Stem cell transplant
Sickle-Cell Crisis

1. Etiology
   a. More common in black males
   b. Presence of Hemoglobin S

2. Precipitating factors
   a. Dehydration
   b. Stress or strenuous exercise
   c. Infection
   d. Fever
   e. Bleeding
   f. Acidosis
   g. Hypoxia (smoking)
   h. Cold weather
   i. Pregnancy

3. Presentation
   a. Bone crisis
      i. Long bone pain
   b. Acute chest syndrome
      i. Chest pain
      ii. Dyspnea
      iii. Tachycardia
      iv. Bloody sputum
      v. Pulmonary fibrosis
   c. Abdominal crisis
      i. Sudden, constant abdominal pain
      ii. Not usually associated with N/V/D
   d. Joint crisis
      i. Stiff, painful joints
   e. Jaundice, bruising, blood in urine may occur with any

4. Management
   a. Oxygen
   b. Fluids
   c. Folic acid
   d. Hydroxyurea (Hydrea)
   e. Pain control
      i. Mild: Tylenol or NSAIDs
      ii. Moderate: Codeine, Oxycodone
      iii. Severe: Morphine, Dilaudid
   f. Transfusion

5. Complications
   a. Renal dysfunction
   b. Stroke
   c. Blindness
   d. Infection (spleen becomes clogged)
**Gastrointestinal (6%) 9 questions**

1. Nursing interventions for the patient with hepatic failure include:
   a. Restrict protein in diet
   b. Avoid use of narcotics, sedatives and tranquilizers
   c. Administer lactulose and neomycin
   d. All the above

2. The most common cause of upper GI bleeding is:
   a. Peptic ulcer disease
   b. Esophageal varices
   c. AV malformation
   d. Gastric tumor

3. Octreotide is often used to control bleeding from esophageal varices. The primary action of Octreotide is to:
   a. Increase platelet aggregation
   b. Increase clotting factors
   c. Decrease venous return
   d. Decrease blood flow

4. The administration of vasopressin should be most carefully monitored in patients who have:
   a. Diabetes Insipidus
   b. Coronary artery disease
   c. Hypotension secondary to GI bleed
   d. Diabetes Mellitus

5. The inability of the liver to conjugate what substance is the primary contributor to hepatic coma?
   a. Ammonia
   b. Urea
   c. Fatty Acids
   d. Bilirubin

6. Ecchymosis around the umbilicus indicative of peritoneal bleeding is called
   a. Chvostek’s sign
   b. Grey Turner’s sign
   c. Cullen’s sign
   d. Trousseau’s sign
7. Pulmonary complications of acute pancreatitis may include:
   a. Adult Respiratory Distress Syndrome
   b. Elevation of the diaphragm and bilateral basilar rales
   c. Atelectasis, especially of the left base
   d. All of the above

8. Which of the following laboratory findings is most specific for pancreatitis?
   a. Leukocytosis
   b. Elevated serum and urinary amylase
   c. Hyperglycemia and hypokalemia
   d. Decreased serum albumin and total protein

9. Another diagnostic finding seen in the patient with pancreatitis would include:
   a. Increased Hct
   b. Hypocalcemia
   c. Hyperalbuminemia
   d. Decreased potassium
GI Bleed

Etiology:
1. Peptic Ulcer Disease (55%)
2. Esophageal varices (14%)
3. Arteriovenous malformations (6%)
4. Mallory-Weiss tears (5%)
5. Tumors & erosions (4% each)
6. Other (12%)

H. Pylori infection or NSAID use is responsible for >98% of upper GI bleeds.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Mechanism of injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>Caffeine</td>
<td>↑ acid production</td>
</tr>
<tr>
<td>Vasopressors</td>
<td>↓ mucosal blood flow</td>
</tr>
<tr>
<td>ASA, alcohol, indomethacin, steroids</td>
<td>H+ back diffusion</td>
</tr>
<tr>
<td>Corticosteroids</td>
<td>↓ mucous secretion</td>
</tr>
<tr>
<td>Chemotherapy, steroids</td>
<td>↓ cell renewal</td>
</tr>
</tbody>
</table>

Prevention:
1. Helicobacter pylori
   a. Pathogenesis
      i. Transmitted by fecal-oral route
      ii. Renders mucosa vulnerable to acid damage
      iii. Inflammatory response
   b. Treatment (80-90% eradication rate)
      i. Antibiotics
      ii. Antisecretory agent

2. NSAIDS
   a. Affects local and systemic prostaglandin inhibition
   b. Majority are uncomplicated and asymptomatic

3. Stress
   a. Common cause of UGI bleeding (1.5% of all ICU pts.)
   b. Higher mortality than pts. admitted with 1° dx. Of UGI bleeding
   c. Independent risk factors:
      i. Respiratory failure
      ii. Coagulopathy
4. Esophageal varices
   a. Secondary to portal hypertension
   b. Bleeding stops spontaneously in >50% of cases
   c. Mortality 70-80% in those who continue bleeding
   d. Treatment
      i. Blood pressure management
         1. Propanolol, nadolol
      ii. Vasopressin, NTG
      iii. Octreotide
         1. ↓ gastrin production
         2. Local vasoconstriction
      iv. Esophageal balloon tamponade (Blakemore / Linton tubes)
      v. Injection sclerotherapy
     vi. Variceal band ligation (↓ rebleeding rate, mortality)
     vii. Transjugular intrahepatic portosystemic shunt (TIPS)
        1. ↓ portal pressure
        2. Complications:
           a. ↑ encephalopathy
           b. Shunt occlusion and rebleeding
           c. Shunt migration

5. GI prophylaxis
   a. H₂ receptor antagonists
      i. Block gastric acid output by blocking histamine receptors
   b. Sucralfate
      i. Inhibits pepsin secretion
   c. Proton pump inhibitors
      i. Inhibits Hydrogen ion formation regardless of source of stimulation
   d. ↑ risk of pneumonia in mechanically ventilated patients (??? ↑ risk of aspiration)

Early Detection
1. Bloody nasogastric aspirate (10-15% false negative)
2. Hemoglobin / Hematocrit
3. Melena / occult blood monitoring
4. Nausea / vomiting / hyperactive bowel sounds
5. Coagulation abnormalities
6. Shock
7. Risk scoring for intervention:
   a. Hemoglobin
   b. Systolic B/P
   c. Syncope / melana
   d. Tachycardia
   e. Cardiac disease
   f. Hepatic disease
Management of Acute Crises

1. ICU admission
   a. Aspiration is a major risk with active bleeding

2. Management of coagulopathies

3. Blood product replacement (most transfusion physicians recommend only component therapy)
   a. PRBCs (to HCT of 30)
   b. FFP
   c. Platelets

4. Hemodynamic support
   a. Fluids
   b. Vasopressors
   c. Monitoring

5. Gastric acid reduction
   a. H2 blockers
   b. Proton pump inhibitors

6. Endoscopy
   a. Diagnostic intervention of choice
   b. Allows treatment

7. Angiography
   a. Cauterization

8. Surgery
   a. Gastric resection
   b. Shunt surgery
   c. Liver transplantation

References:

Internet sites:
American Gastroenterological Association: www.gastro.org
American College of Gastroenterology: www.acg.gi.org
Society of Gastroenterology Nurses and Associates: www.sgna.org
Hepatic Failure

1. Etiology
   a. Viral hepatitis
   b. Acetaminophen overdose
      i. Chronic alcohol use increases susceptibility
   c. Alpha1-antitrypsin deficiency
   d. Autoimmune disease

2. Diagnostic testing
   a. CBC
   b. PT
   c. AST / ALT
   d. Bilirubin
   e. Ammonia
   f. Glucose
   g. Lactate

3. Symptoms
   a. Jaundice
   b. ↓ level of consciousness
   c. Ascites
   d. Hypotension & tachycardia (SIRS)

4. Management
   a. Supportive:
      i. ↑ ICP: mannitol
      ii. Renal failure: dialysis
      iii. Coagulopathy: platelets, FFP
   b. Liver transplant

Acute Pancreatitis

1. Etiology
   a. Alcoholism
   b. Biliary tract disease
   c. Drugs
      i. Thiazides
      ii. Acetaminophen
      iii. Tetracycline
      iv. Oral contraceptives
   d. Infection
   e. Hyperlipidemia, hypertriglyceridemia
   f. Structural abnormalities of bile or pancreatic ducts

2. Pathogenesis
   a. Edema
   b. Necrosis
c. Hemorrhage
d. Pancreatic enzyme release
e. Inflammation
   i. Enzymes and toxins enter the peritoneum
   ii. ↑ permeability of blood vessels, third spacing
   iii. Enzymes enter systemic circulation ↑ capillary permeability
   iv. Shock from ↓ circulating volume

3. Symptoms
   a. Abdominal pain
      i. ↑ after eating or alcohol ingestion
      ii. Severe, persistent, penetrating
      iii. Radiates to back or neck
   b. Fever
   c. Nausea / Vomiting without ↓ pain
   d. Sweating

4. Physical exam
   a. Appears acutely ill
   b. Tachycardia, tachypnea, hypotension
   c. ↑ temperature
   d. LUQ abdominal tenderness with guarding
   e. ↓ or absent bowel sounds
   f. Signs of dehydration
   g. Signs of necrosis (50% mortality)
      i. Grey Turner’s sign
      ii. Cullen’s sign

5. Hemodynamics
   a. ↓ preload (CVP, PAOP)
   b. ↓ CO
   c. ↓ afterload (SVR)

6. Diagnostic tests
   a. Labs
      i. ↑ Serum and urine amylase
      ii. ↑ Lipase
      iii. Amylase:creatinine clearance ratio
      iv. ↑ Glucose
      v. ↓ Calcium 2° to ↓ albumin
Ranson’s Criteria

<table>
<thead>
<tr>
<th>On Admission</th>
<th>During 1st 24 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Age &gt; 55</td>
<td>• HCT ↓ &gt;10%</td>
</tr>
<tr>
<td>• WBC &gt; 16</td>
<td>• BUN ↑ &gt; 5mg/dl</td>
</tr>
<tr>
<td>• Glucose &gt; 200</td>
<td>• Ca++ &lt; 8</td>
</tr>
<tr>
<td>• LDH &gt; 350</td>
<td>• pO2 &lt; 60 mmHg</td>
</tr>
<tr>
<td>• SGOT &gt; 250</td>
<td>• Base deficit ↑ &gt; 4</td>
</tr>
<tr>
<td></td>
<td>• Fluid sequestration ↑ &gt; 6L</td>
</tr>
</tbody>
</table>

> 3 criteria require supportive care
> 7 are critically ill with close to 100% mortality

7. Treatment
   a. NPO
   b. NG drainage
      i. Does not decrease pancreatic enzyme secretion
      ii. Helpful in managing:
         1. Vomiting
         2. Gastric distension
         3. Ileus
         4. Aspiration from ↓ mental status
   c. IV fluids
   d. Hemodynamic support
   e. Pain relief
      i. Demoral or Dilaudid
      ii. Morphine may cause biliary colic or spasms of the sphincter of Oddi
   f. Antibiotics for necrotizing pancreatitis
      i. Imipenem
      ii. Ciprofloxin
      iii. Cefotaxime
   g. TPN nutrition (low lipids)

8. Complications
   a. Death from cardiovascular instability
   b. Infection
   c. Pseudocyst
      i. Collection of blood, necrotic tissue, inflammatory debris encapsulated in fibrotic tissue
   d. Hypovolemic shock
   e. Respiratory failure / ARDS
   f. Pleural effusion
   g. Renal failure 2° to hypovolemia
Bowel infarction

1. Pathogenesis
   a. Acute mesenteric ischemia (AMI)
   b. Insufficient blood flow due to:
      i. Arterial occlusion
      ii. Venous occlusion
      iii. Non-occlusive processes

2. Symptoms
   a. Pain
   b. N/V
   c. Bloody diarrhea
   d. Hypovolemia
   e. Metabolic acidosis

3. Diagnostic tests
   a. Labs:
      i. ↑ H/H
      ii. ↑ Amylase
      iii. ↑ WBC
   b. KUB
   c. CT or MRI
   d. Ultrasound
   e. Guaiac stools

4. Treatment
   a. Medical
      i. Volume replacement
      ii. Correct underlying condition
      iii. Improve mesenteric blood flow
      iv. NG tube
      v. ATB
   b. Surgical
      i. Bowel resection
      ii. Embolectomy
      iii. Revascularization

5. Complications
   a. Perforation
   b. Strictures
   c. Infection
Abdominal Trauma

1. Esophagus
   a. Penetrating injury more common than blunt
   b. Early diagnosis is important, gastric acid erodes tissues, and contaminates the wound
   c. Mortality is as high as 27%, mostly due to infection
   d. Areas at risk for injury
      i. At the cricoid cartilage
      ii. At the arch of the aorta
      iii. As it passes through the diaphragm
   e. Manifestations
      i. Look for abrasions, contusions, lacerations
      ii. Pain
      iii. Fever
      iv. Dysphagia
      v. Bloody emesis
      vi. Mediastinal crepitus
   f. Diagnosis
      i. CXR, KUB
      ii. Esophagogram
   g. Treatment
      i. NG decompression
      ii. Surgical repair
   h.Leaks are common

2. Diaphragm
   a. Fairly well protected
   b. Most often injured by penetrating trauma of the lower chest
   c. 15% of patients with stab wounds
   d. 46% of patients with GSW
   e. Manifestations
      i. Have a high degree of suspicion in pts. with trauma to the abdomen or as high as T4
      ii. Chest pain
      iii. Dyspnea
      iv. Peristalsis heard in the chest
      v. Difficulty passing an NG tube
      vi. Persistent air leak from a chest tube
   f. CXR
   g. Evidence on exploratory lap
   h. Treatment
      i. Herniation can occur weeks to years later
      ii. Therefore, surgical repair is necessary
   i. Complications
      i. Intra-abdominal hypertension increases risk of herniation
3. Stomach
   a. Most is penetrating
   b. Accounts for about 19% of abdominal injuries
   c. Can result from CPR
   d. Good prognosis with prompt recognition and treatment
   e. Manifestations
      i. Epigastric pain and tenderness
      ii. Peritonitis
      iii. Bloody drainage from NG
      iv. Abdominal free air
   f. Treatment
      i. NG tube
      ii. Surgical resection
      iii. H2-blockers
   g. Complications
      i. Peritonitis
      ii. Intra-abdominal abscess
      iii. Gastric fistula
      iv. Prolonged healing or breakdown of the repair may result in contamination or hemorrhage

4. Liver: size and location make it vulnerable to injury
   a. Most common abdominal organ to be injured
   b. Highest mortality with direct blunt trauma (about 70%) and shotgun injuries: (10-15% from hemorrhage)

Liver Injury Scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Hematoma Subcapsular, nonexpanding, &lt;10% surface area</td>
</tr>
<tr>
<td>I</td>
<td>Laceration Capsular tear, non-bleeding, &lt; 1 cm depth</td>
</tr>
<tr>
<td>II</td>
<td>Hematoma Subcapsular, nonexpanding, 10-50% surface area</td>
</tr>
</tbody>
</table>

   c. Manifestations
      i. Have a high degree of suspicion with patients with persistent unexplained hypotension
      ii. Evidence of peritonitis with bile leakage
      iii. RUQ pain or tenderness
   d. CXR
   e. Diagnostic peritoneal lavage may be helpful
   f. CT is preferred, if stable
   g. Treatment
      i. If bleeding is small, serial CT scans
      ii. Liver resection is indicated if:
          1. Bleeding is extensive, or on-going
          2. Signs of sepsis
3. Deterioration of liver function tests

h. Complications
   i. Uncontrolled hemorrhage
   ii. Sepsis
   iii. Decreased albumin
   iv. Hypoglycemia
   v. Drug toxicity
   vi. Bleeding from loss of clotting factors

i. Post-operative follow-up
   i. Labs:
      1. Coagulation profile
      2. Ammonia level
      3. Liver profile
      4. Serum protein and glucose
      5. Replace blood products as needed
   ii. Major complications secondary to liver damage
      1. Blood loss
      2. PRBCs, platelets, FFP
      3. Assess for DIC
      4. Pulmonary insufficiency
      5. Atelectasis, pleural effusion, pneumonia are common
         a. Related to position of the liver, pleural irritation, and pain
      6. Infection
      7. Tissue debris, necrosis, bile
      8. Abscess or sepsis
      9. Assess for signs of infection
     10. CT for abscess formation

5. Spleen: most commonly injured organ in blunt trauma
   a. Isolated splenic injury occurs in about 20% of all cases and is associated
      with a very low mortality
   b. Overall mortality 11%, with associated injury 25%
   c. Assessment
      i. LUQ injury
      ii. Pain or tenderness
      iii. Ballance’s sign: dullness to percussion that disappears with
           position change
   d. Manifestations
      i. Graded from I to V depending on injury severity
      ii. KUB may show changes in splenic outline
      iii. CT scan
      iv. Fractures of ribs 8-10 associated with a 20% chance of injury
      v. ↑ WBC, ↓ H/H
      vi. Hypovolemia, shock
e. Treatment
   i. Localized bleeding control to preserve spleen, if damage is superficial and localized
   ii. Partial splenectomy, when wound is deep
   iii. Splenectomy, when blood supply is interrupted, spleen is destroyed, or hemorrhage cannot be stopped
f. Complications
   i. OPSI: Overwhelming Postsplenectomy Infection: due to loss of immune actions of the spleen
   ii. Hemorrhage
   iii. Infection, abscess

6. Pancreas: mostly from penetrating wounds
   a. Associated with multi-organ injury
   b. Pancreatic enzymes may not elevate due to inactivation during injury
   c. Manifestations
      i. Mechanism of injury
      ii. Epigastric pain & tenderness
      iii. ↑ amylase, lipase
      iv. Nausea, vomiting
d. Treatment
   i. Drainage of enzymes
   ii. Surgical repair
   iii. Wound drainage
e. Complications (due to inadequate drainage during surgery)
   i. Pseudocyst
   ii. Abscess

7. Bowel
   a. Penetrating, blunt or shearing trauma
   b. Duodenal and Ileum Injuries
   c. Rarely single organ injuries
   d. Alkalinity of contents produces immediate irritation
   e. Often difficult to diagnose since contents are sterile, peritonitis does not occur immediately
   f. Fever, jaundice, bowel obstruction, abdominal pain, edema
   g. Graded I-V by severity
   h. Octreotide to decrease secretions
   i. Complications
      i. Sepsis with MODS and duodenal fistula can be lethal

The American Gastroenterological Association: www.gastro.org
8. Small Bowel
   a. Look for contusions, wounds over abdomen
   b. Abdominal pain and tenderness
   c. ↓ bowel sounds
   d. Hypovolemia
   e. Delayed rupture is possible
   f. CT scan, KUB for free air
   g. Treatment is surgical intervention
   h. Fluid / nutrition deficiency common post-op
   i. Fistula formation is possible post-op

9. Abdominal assessment
   a. History
      i. Prior surgeries
      ii. Nutritional deficits
      iii. Absorption problems
   b. Inspection
   c. Auscultation
   d. Percussion
   e. Palpation
   f. Diagnostic studies
      i. X-rays
      ii. CT
      iii. Arteriography
      iv. Diagnostic Peritoneal Lavage

10. Intra-abdominal Hypertension
    a. Caused by fluid volume resuscitation
    b. Results in renal dysfunction and respiratory compromise
    c. Measured in bladder with T-piece catheter
    d. Hypertension is defined as >18 mmHg
    e. Temporary abdominal closure reduces abdominal pressure and improves lung dynamics, but does not improve renal function or oxygenation.
GI Surgeries:

1. Whipple (Pancreaticoduodenectomy)
   a. Used for:
      i. Resectable pancreatic cancer
      ii. Pancreatic cancer
      iii. Chronic pancreatitis
   b. Removal of:
      i. Head of the pancreas
      ii. Duodenum
      iii. Part of the common bile duct
      iv. Gallbladder
      v. Sometimes a portion of the stomach
   c. Complications:
      i. Peritonitis
      ii. Sepsis, SIRS, MODS
      iii. Pancreatic fistula
      iv. Uncontrolled blood sugar in diabetics

2. Esophago-gastrectomy
   a. Used for:
      i. Esophageal cancer
   b. Removal of:
      i. Part of the esophagus
      ii. Part of the stomach
      iii. Anastomose with intestine
   c. Complications:
      i. Anastomotic leak
      ii. Stricture formation
      iii. Diarrhea

3. Gastric bypass (Roux-en-Y)
   a. Used for:
      i. Surgical treatment of obesity
   b. Bypass of:
      i. Part of the stomach
      ii. Duodenum
   c. Complications:
      i. Dumping syndrome
      ii. Peritonitis
      iii. Gallstones
      iv. Nutritional deficiency

Resources:
Multisystem (8%) 12 questions

1. As a result of multisystem trauma, edema can occur in the peritoneal and retroperitoneal areas and cause intra-abdominal pressure to increase. Intra-abdominal pressure is measured using a urinary catheter and is hypertensive if the pressure exceeds:
   a. 10 mmHg
   b. 50 mmHg
   c. 100 mmHg
   d. 150 mmHg

2. Initial treatment for hypovolemic shock includes:
   a. Vasopressors
   b. Volume resuscitation
   c. Stopping the loss
   d. Antibiotics

3. Death from multisystem trauma that occurs within minutes is usually caused by:
   a. Great vessel laceration
   b. Head injury
   c. Pelvic fracture
   d. Multisystem organ failure

4. The primary purpose of obtaining blood cultures in the septic patient is:
   a. To diagnose sepsis
   b. To guide therapy
   c. To evaluate the level of response
   d. To determine a source

5. A defining characteristic of septic shock that differentiate it from other types of shock is:
   a. Low blood pressure
   b. Wide pulse pressure
   c. Decreased urine output
   d. Tachycardia

6. Corticosteroids are often used in septic shock for:
   a. Inflammation
   b. Adrenal replacement
   c. Immunosuppression
   d. Bronchodilation
7. Septic shock with ARDS and acute renal failure may be treated with activated protein C (Xigris). A major complication of Xigris is:
   a. Hypoxia
   b. Hyperglycemia
   c. Bleeding
   d. Acidosis

8. The systemic inflammatory response syndrome (SIRS) can cause multiorgan dysfunction. The first organ to be involved is:
   a. The heart
   b. The lungs
   c. The brain
   d. The liver

9. Using vasopressors in shock may cause:
   a. Increased splanic perfusion
   b. Decreased cardiac output
   c. Decreased pulmonary perfusion
   d. Increased peripheral perfusion

10. Mr. Jones took 100 tablets of Percocet in a suicide attempt. As his nurse, you should know that treatment of ingested poisoning includes:
    a. Managing the ABCs and administering activated charcoal
    b. Administering ipecac
    c. Hyperbaric oxygen
    d. Prompt transport to a poison control center

11. Ms. Lett is admitted for burns suffered in a house fire. Since she is complaining of shortness of breath, an ABG is drawn. Due to the etiology of the burns, the nurse should be especially concerned about:
    a. pO2 of 83
    b. pCO2 of 50
    c. COHb of 18
    d. pH of 7.32

12. In the initial resuscitation of burns, which treatment is the priority?
    a. Fluid volume replacement
    b. Administration of antibiotics
    c. Management of the airway
    d. All of the above
Multisystem Trauma

1. Decreased intravascular volume
   a. Hemorrhage
   b. Dehydration
   c. Burns
   d. Third spacing

2. Decreased blood pressure
   a. ↓ preload, ↓ SV, ↓ CO

3. Compensatory mechanisms activated r/t ↓ CO

4. Treatment goal is to replace lost volume
   a. RBCs
   b. Colloids
      i. Albumin, Dextran, Hetastarch
      ii. May decrease risk of pulmonary edema
      iii. Osmotic “pull” increases intravascular volume
   c. Crystalloids
      i. NS, Lactated Ringers
      ii. Proven efficacy in traumatic hypovolemia
      iii. Only 20% remains in the blood stream at 1 hour
      iv. Can result in significant hemodilution and ↓ DO2
   d. Hemoglobin substitutes
      i. PolyHeme®
      ii. Oxygent

<table>
<thead>
<tr>
<th>Fluid</th>
<th>Vol. Expansion</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>NS, LR</td>
<td>1 hour</td>
<td>Proven efficacy</td>
<td>May contribute to edema</td>
</tr>
<tr>
<td>Colloids</td>
<td>24 hours</td>
<td>Less edema</td>
<td>Volume limit</td>
</tr>
<tr>
<td>Blood products</td>
<td>Remains</td>
<td>Great colloid, replacement</td>
<td>↑ inflammation.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>↑ mortality</td>
</tr>
<tr>
<td>Hb substitutes</td>
<td>Varies</td>
<td>Immediate oxygen delivery</td>
<td>Multiple side effects</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Not proven effective</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Volume loss</th>
<th>Stage</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>10%</td>
<td>1</td>
<td>↑ HR, normal B/P</td>
</tr>
<tr>
<td>20%</td>
<td>2</td>
<td>↓ B/P, ↓ CO, ↑ HR</td>
</tr>
<tr>
<td>&gt;25%</td>
<td>3</td>
<td>Compensation begins to fail</td>
</tr>
</tbody>
</table>
Hemodynamics in Hypovolemic Shock

General Principles for Managing Multisystem Trauma

1. Primary Survey
   a. Airway, Breathing, Circulation, Disability, Exposure

2. Trimodal Distribution of Death
   a. First Peak
      i. Within minutes
      ii. Due to lacerations of large vessels or of essential organs
   b. Second Peak
      i. Minutes to several hours
      ii. Due to:
         iii. Subdural / epidural hematoma
         iv. Hemothorax
         v. Pelvic fractures
         vi. Ruptured spleen
         vii. Significant blood loss
   c. Third Peak
      i. Several days to weeks
      ii. Due to sepsis or multisystem organ failure
Sepsis / Septic Shock / MODS

1. Maldistribution of blood volume (massive vasodilation)
   a. Sepsis (most common)
   b. Anaphylactic
   c. Neurogenic
   d. Spinal

2. Hyperdynamic stage:
   a. Tachycardia, ↑ CO
   b. ↓ afterload
   c. Flushing
   d. Fever
   e. ↑ blood glucose

3. Shock stage
   a. ↑ HR, ↑ RR
   b. ↑ afterload
   c. Hypothermia
   d. ↓ organ perfusion

4. Sepsis stimulates the Systemic Inflammatory Response Syndrome (SIRS)
5. Compensatory mechanisms activated r/t ↓ B/P
6. Treatment goals:
   a. “Fill” vascular space
   b. Prevent secondary organ damage
      i. Vasopressors
         1. Dopamine
         2. Levophed
         3. Neosynphrine
         4. Vasopressin
      ii. IV fluids
      iii. Colloids
      iv. Blood products
      v. Xigris

Hemodynamics in Sepsis
Poisoning

Ingested
1. Emesis
   a. Serious aspiration risk
2. Gastric lavage
   a. 500-3000cc
3. Activated charcoal
   a. 50-100 grams
4. Specific antidotes
   a. Narcan for opiates
   b. Atropine for organophosphates
   c. Methylene blue for methemoglobinemia
   d. Acetylcystine for acetaminophen
5. Support
   a. Cardiovascular
   b. Pulmonary
   c. Valium or Phenobarbital for seizures
   d. Mannitol and dexamethasone for ↑ ICP

Carbon Monoxide
1. Emitted from gas, charcoal, oil, wood
2. Brain and heart most affected
3. Symptoms:
   a. Low-level exposure
      i. Shortness of breath
      ii. Mild nausea
      iii. Mild headache
   b. Moderate-level exposure
      i. Headache
      ii. Nausea
      iii. Light-headedness
      iv. Dizziness
   c. High-level exposure
      i. Death within minutes
4. Treatment
   a. Oxygen (reduces COHb half-life from 4-5 hours to 1 hour)
   b. Hyperbaric oxygen therapy (↓ half-life to <30 minutes)
Burns

1. Types:
   a. Thermal
   b. Electrical
   c. Chemical
   d. Radiation

2. Zone of injury

3. Assessment
   a. Rule of nines
   b. Classification
     i. First degree
     ii. Second degree
     iii. Third degree

4. Complications
   a. Intra-abdominal hypertension
   b. Pulmonary injury
     a. Smoke inhalation
     b. CO intoxication
     c. Airway burns
   c. Fluid volume deficit
     i. First 24 hours
        (1) 4 ml LR / %TBSA / kg
        (2) ½ volume in 1st eight hours
        (3) ¼ volume next eight hours
        (4) ¼ volume last eight hours
     ii. Second 24 hours
        (1) D5W with 40 mEq KCl to maintain normal electrolyte balance
        (2) Plasma or albumin to maintain hemodynamic balance
   d. Infection
     i. Burn dressing
     ii. Antibiotics
   e. Electrolyte imbalances

5. Pain Control
Cardiovascular (32%) 48 questions

1. Which of the following variables affects cardiac output directly?
   a. Preload
   b. Stroke volume
   c. Afterload
   d. Resistance

2. Coronary artery perfusion is dependent upon:
   a. Diastolic pressure
   b. Systolic pressure
   c. Afterload
   d. SVR

3. Mixed venous oxygen saturation (SvO2) assesses:
   a. Preload
   b. Afterload
   c. Oxygen delivery
   d. Oxygen consumption

4. Chest pain that is not relieved by rest and nitroglycerine is called:
   a. Variant angina
   b. Stable angina
   c. Unstable angina
   d. Prinzmetal’s angina

5. The nurse administering t-PA for acute myocardial infarction must monitor the patient for all of the following except:
   a. Peripheral thrombosis
   b. Myocardial reperfusion
   c. Bleeding complications
   d. Coronary reocclusion

6. Which finding would not indicate coronary reperfusion during t-PA infusion?
   a. Drop in arterial blood pressure
   b. Resolution of ST segment elevation
   c. Ventricular tachycardia
   d. Dramatic reduction in chest pain

7. Which of the following is not an indication for thrombolytic therapy?
   a. An occluded arteriovenous fistula
   b. Non-Q Wave Myocardial Infarction
   c. Peripheral arterial occlusion
   d. Acute Myocardial Infarction
8. The pathologic changes found on 12 Lead ECG to indicate myocardial ischemia are:
   a. ST elevation
   b. ST segment depression and T wave elevation
   c. Q wave formation
   d. ST segment depression and T wave inversion

9. Failure to capture is a complication of pacemakers that may be caused by:
   a. Lead maturation
   b. Lead displacement
   c. Dead battery
   d. Open circuit

10. Automatic implantable cardio-defibrillators (AICDs) may be initiated in the treatment of:
    a. Frequent PVCs
    b. Atrial fibrillation
    c. Narrow-complex SVT
    d. Symptomatic VT

11. The nurse auscultates an S₄ gallop during her assessment. The appearance of an S₄ gallop during an anginal episode may signify:
    a. Congestive heart failure
    b. Decreased compliance of the ischemic myocardium
    c. Aortic stenosis
    d. Increased left ventricular filling volume

12. Heart failure caused by the inability to fully relax is called:
    a. Systolic
    b. Diastolic
    c. Biventricular
    d. Complete

13. The primary function of drug therapy with beta-blockers in heart failure is to:
    a. Increase blood pressure
    b. Block compensatory mechanisms
    c. Increase urine output
    d. Decrease arrhythmias

14. Early symptoms of fluid overload and pulmonary edema are:
    a. Rales and hypoxia
    b. S₃ heart sound and tachycardia
    c. Increased respiratory rate and subjective dyspnea
    d. ST-segment elevation in the chest leads
15. Mechanical ventilation may be helpful to your patient with CHF because it:
   a. Decreases preload
   b. Increases alveolar pressure
   c. Increases oxygenation
   d. All of the above

16. An Intraaortic Balloon Pump (IABP) has the following hemodynamic effects:
   a. Increases left ventricular pressure
   b. Increases wedge pressure
   c. Increases coronary artery perfusion
   d. Increases afterload

17. The IABP is:
   a. Deflated during systole
   b. Inflated during systole
   c. Deflated during diastole
   d. None of the above

18. IABP therapy is contraindicated for which of the following disorders?
   a. Papillary muscle rupture
   b. Incompetent aortic valve
   c. Left ventricular failure
   d. Unstable angina refractory to medical regimen

19. Which coronary artery supplies the atrioventricular (AV) node?
   a. Right coronary artery
   b. Coronary sinus artery
   c. Left anterior descending artery
   d. Nodal artery

20. Coronary perfusion occurs during:
   a. Systole
   b. Diastole
   c. Equally during diastole and systole
   d. Continuously

21. The forth heart sound (S₄) occurs:
   a. After ventricular contraction
   b. Is best heard with the diaphragm of the stethoscope
   c. Is a normal finding in children
   d. During atrial contraction
22. Flotation of a pulmonary artery catheter into a wedge position increases the risk of:
   a. Dysrhythmias
   b. Infection
   c. Pneumothorax
   d. Pulmonary infarction

23. Which of the following pulmonary artery pressures are within normal limits?
   a. PAP 34/24, wedge = 12
   b. PAP 30/20, wedge = 10
   c. PAP 28/18, wedge = 20
   d. PAP 24/14, wedge = 12

24. Which of the following results in an elevated pulmonary artery pressure and a normal wedge pressure?
   a. Cardiac tamponade
   b. Left ventricular failure
   c. Myocardial infarction
   d. Pulmonary embolism

25. Which of the following is the least accurate in diagnosing an acute myocardial infarction?
   a. Patient’s history
   b. Physical examination
   c. Enzyme studies
   d. Serial EKG’s

26. The inferior wall myocardial infarction will show changes in which EKG leads?
   a. V₁ to V₄
   b. V₁, AVL
   c. V₅ and V₆
   d. II, III, AVF

27. The most common complication of an acute myocardial infarction is:
   a. Dysrhythmia
   b. Congestive heart failure
   c. Cardiogenic shock
   d. Pulmonary embolism

28. Which of the following hemodynamic parameters would indicate left ventricular failure in a patient with COPD?
   a. PAP 25/22, wedge = 14
   b. PAP 48/26, wedge = 16
   c. PAP 22/12, wedge = 16
   d. PAP 48/26, wedge = 20
29. Which of the following medications would be most effective in the acute myocardial infarction patient to decrease preload and afterload?
   a. Dopamine
   b. Nitroglycerine
   c. Dobutamine
   d. Digoxin

30. Positive inotropic agents are used to:
   a. Improve tissue perfusion
   b. Decrease water loss through the kidney
   c. Increase heart rate
   d. Vasodilate vessels

31. Which condition would stimulate renin production?
   a. Increased blood supply to the renal tubules
   b. Decreased blood pressure
   c. Decreased sympathetic output
   d. Increased sodium concentration

32. Acute rejection in cardiac transplantation is diagnosed by:
   a. ECG
   b. Chest X-ray
   c. Echocardiography
   d. Endomyocardial biopsy

33. After cardiac transplantation, the patient is placed on cyclosporine (Sandimmune). In assessing for complications related to this drug therapy, the nurse should monitor:
   a. Blood glucose
   b. Serum creatinine
   c. Serum amylase
   d. Serum magnesium

34. You are caring for a patient recently admitted with an IWMI. Which of the following 12 Lead ECG findings would you anticipate?
   a. T-wave inversion I, and AVL
   b. Q wave formation and ST-segment elevation in II, III, and AVF
   c. QRS duration greater than 0.01 in all leads
   d. R-wave taller in V6

35. Your patient with an IWMI also has a RV infarction. He soon develops RV failure. Which of the following data would you expect to see?
   a. PAP 23/8 PAOP 19 CVP 20
   b. PAP 54/28 PAOP 14 CVP 14
   c. PAP 54/18 PAOP 24 CVP 5
   d. PAP 28/10 PAOP 10 CVP 20
36. A sign of a peripheral arterial occlusion is:
   a. Pallor
   b. Swelling
   c. Redness
   d. Dyspnea

37. Your patient has just come to the unit after a Carotid Endarterectomy (CEA). As her nurse, you will assess for all of the following except:
   a. Hypertension
   b. Changes in mental status
   c. Bleeding
   d. Seizures

38. A thoracic aortic aneurysm causes chest pain that:
   a. Radiates to the left arm
   b. Bores through to the back
   c. Is sharp and worse while reclining
   d. Is associated with diminished breath sounds

39. A patient who presents with stabbing chest pain that is worse in the supine position, with fever and chills is probably suffering from:
   a. Myocardial infarction
   b. Pulmonary embolism
   c. Pericarditis
   d. Pneumothorax

40. Primary patient care management of pericarditis includes all of the following except:
   a. Monitoring for signs of cardiac tamponade
   b. Evaluating the effectiveness of pain relief strategies
   c. Maintaining the patient’s bowel regimen
   d. Providing emotional support

41. Subacute bacterial endocarditis (SBE) is usually caused by:
   a. Dental procedures
   b. Normal valves
   c. IV drug abuse
   d. Prosthetic valves

42. The valve most often affected by infective endocarditis is:
   a. Mitral
   b. Aortic
   c. Tricuspid
   d. Pulmonary
43. Following a motor-vehicle accident, pericardial tamponade is suspected. Which of the following findings is consistent with traumatic tamponade?
   a. Muffled heart sounds
   b. Pericardiocentesis of 50 cc of blood
   c. ST-segment depression in the limb leads
   d. Rales on auscultation

44. The classic triad (Beck’s triad) of symptoms of cardiac tamponade are:
   a. Tachycardia, hypotension, narrow pulse pressure
   b. Rales, muffled heart sounds, bradycardia
   c. Widened pulse pressure, atrial arrhythmias
   d. Hypertension, flushing, pulses paradoxus

45. Mr. Ford comes to the emergency department (ED) after a motor-vehicle accident. He is complaining of chest pain, dyspnea, and has ST-segment elevation on the anterior leads. Mr. Ford is most likely suffering from:
   a. Pneumothorax
   b. Flail chest
   c. Cardiac contusion
   d. Pulmonary embolism

46. In Cardiogenic shock the initial goal is to:
   a. Increase cardiac output
   b. Increase oxygen supply
   c. Decrease oxygen consumption
   d. Decrease contractility

47. The medication that increases oxygen supply to the heart during Cardiogenic shock is:
   a. Dopamine
   b. Nitroglycerine
   c. Nitroprusside
   d. Dobutamine

48. Calcium-channel blockers have which of the following functions?
   a. Increase vascular tone
   b. Increase velocity of AV conduction
   c. Decrease cardiac oxygen consumption
   d. Increase cerebral oxygenation
Cardiovascular

1. Coronary Perfusion
   a. Cardiac cycle
   b. Aortic pressure
   c. Coronary Artery Perfusion Pressure
      i. CAPP = Diastolic BP – PAOP
      ii. Normal 60-80mmHg

2. Determinants of Ventricular Function
   a. Cardiac Output
      i. Heart Rate X Stroke Volume
      ii. Stroke Volume
         1. Preload
         2. Afterload
         3. Contractility
      iii. Supply and Demand
         1. Supply
            a. Coronary artery patency
            b. Diastolic time
            c. Diastolic pressure
            d. O₂ extraction
            e. Hemoglobin
            f. SaO₂
         2. Demand
            a. HR
            b. Preload
            c. Afterload
            d. Contractility
Flow and Assessment

3. Hemodynamic Monitoring
   a. Uses
      i. Measure hemodynamic waveforms
      ii. Blood samples
      iii. Central venous access
      iv. Perform intracardiac pacing
   b. Indications
   c. Complications

Electrocardiogram (ECG)

1. General Info
2. Electrolyte imbalances
   a. Hypokalemia: ventricular irritability
      i. Flat T wave with prominent U wave
      ii. T-wave + U wave same amplitude
      iii. ST-segment flattening
      iv. Prolonged QT interval
      v. ST-segment depression
      vi. Treatment:
         1. PO or IV replacement
   b. Hyperkalemia: ventricular depression
      i. Tall, narrow peaked T waves
      ii. Widened QRS
      iii. P wave widens
      iv. P-wave barely visible
      v. Treatment
   c. Hypo-hyper-calcemia
   d. Hypo-hyper-magnesemia
RA   3-5 mmHg
RV   25/3-5 mmHg
PA   25/8-12 mmHg
PAOP 8-12 mmHg
LA   4-12 mmHg
LV   120/4-12 mmHg
Ao   120/80 mmHg

(c) Lifeart
Mixed-Venous Oxygen Saturation (SVO₂)

1. CO/CI
2. H+H
3. Oxygenation
4. Metabolic Demand

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**Coronary Artery Disease**

1. **Definition**
   - a. Pathophysiology
   - b. Etiology
   - c. Risk Factors
2. **Clinical Manifestations**

**Stable Angina**

1. **Clinical presentation**
   - a. ECG
3. **Treatment**
   - a. Rest
   - b. Anticoagulants
   - c. Vasodilators
   - d. Beta Blocker
   - e. ACE I

**Unstable Angina**

1. **Clinical Presentation**
2. **Pathophysiology**
3. **New Terminology**
4. **Biochemical Markers**
5. **Treatment management**
   - a. ASA
   - b. Beta Blockers
   - c. Calcium Channel Blockers ???
   - d. Heparin
   - e. NTG
   - f. Morphine
   - g. GP IIb-IIIa drugs
   - h. Assistance for the ventricle
      - i. IABP
         1. Increase coronary perfusion
         2. Decrease afterload
         3. Absolute contraindication: Aortic insufficiency
         4. Monitor
            - a. Vascular Exam
            - b. Timing
      - ii. Interventional
         1. Pre-procedure
         2. Post-procedure
         3. 6 P’s
Acute Myocardial Infarction

1. Etiology
2. Pathophysiology
3. Classifications
4. Clinical Presentation
   a. ECG Changes
      i. Anterior Wall
      ii. Inferior Wall
   b. Enzymes
   c. Diagnosis
5. Manage and Monitor
   a. Reduce Size of Infarction
   b. Door to Diagnosis and Treatment
   c. Diagnosis
   d. Treatment Paradigm
      i. Oxygen, pain management
      ii. Reperfusion therapies
         1. Cath lab (PCI)
         2. Thrombolytics (tPA)
         3. CABG
      iii. Increase Myocardial Oxygen Supply
      iv. Decrease Myocardial Oxygen Demand
   e. ACC/AHA guidelines
      i. Reperfusion Therapy
      ii. ASA
      iii. ACE I
      iv. Beta Blocker
      v. Lipids: Statins
      vi. Smoking Cessation Information
   f. RV Infarction
      i. Assess for clinical signs of RVMI:
         1. Classic triad:
            a. Jugular vein distension
            b. Clear lungs
            c. Hypotension
      ii. Maintain adequate filling pressures
      iii. Avoid diuretics and NTG (highly sensitive)
      iv. Hemodynamics
         1. CVP >10 mmHg
         2. CVP within 5 mm Hg of PAOP
      v. Complications

Plug in the Pump!
Heart Failure

Systolic Dysfunction
1. Dysfunction of contractility
2. Weak contraction $\rightarrow$ ↓ SV $\rightarrow$ ↓ CO $\rightarrow$ ↑ EDP/EDV $\rightarrow$ hypertrophy
3. Etiology:
   a) Ischemic heart disease
   b) Cardiomyopathies
   c) Hypertension
   d) Valvular disease
   e) Pericardial disease
   f) Chronic tachycardia
   g) Connective tissue disease
   h) Neurogenic
   i) Pulmonary disease
4. Primary symptoms
   a) Dyspnea / orthopnea
   b) Exercise intolerance
   c) Edema
   d) Mental status changes
   e) S₃, S₄
   f) Tachycardia
   g) Rales
   h) Hepatomegaly
   i) JVD

Diastolic Dysfunction
1. Dysfunction of relaxation
2. Incomplete relaxation $\rightarrow$ restricted filling $\rightarrow$ ↓ SV $\rightarrow$ ↓ CO $\rightarrow$ ↑ EDP (EDV is normal)
3. Etiology:
   a. LV hypertrophy
   b. Ischemic states
4. Primary symptoms
   a. Dyspnea, fatigue

Compensation
1. Renin-Angiotensin-Aldosterone
2. Clinical Presentation LVF
   a. Tachypnea, Dyspnea, orthopnea, PND
   b. Pulsus alternans
3. Clinical Presentation RVF
   a. JVD, HJR, edema, ascites, CVP elevation
   b. Abnormal liver functions
Management

1. Beta-adrenergic agonists
   a. Dopamine (Intropin), Dobutamine (Dobutrex), Norepinephrine (Levophed)
   b. Short-term exacerbation treatment
   c. Long-term use in HF clinics
   d. ↑ cardiac output (↑ contractility), ↑ VO2

2. Phosphodiesterase inhibitors
   a. Amrinone, (Inocor), Milrinone (Primacor)
   b. Short-term exacerbation treatment
   c. Long-term use in HF clinics
   d. ↑ cardiac output (contractility), vasodilation (↓ afterload), ↑ VO2

3. Diuretics
   a. ↑ cardiac output by ↓ preload
   b. Watch for electrolyte disturbances

4. Vasodilators
   a. Nitrates
      i. ↓ preload, ↑ contractility, ↓ afterload
   b. Ca+ channel blockers
      i. ↑ contractility, ↓ afterload
   c. Natrecor
      i. ↓ preload, ↓ afterload

5. Angiotension-converting enzymes (ACE) inhibitors (ie. Enalapril)
   a. Block the RAS activation that causes vasoconstriction and remodeling
   b. Decrease afterload (vasodilation)
   c. Favorable affects on mortality and morbidity
   d. ACE inhibitors continue to be preferred over Angiotensin II (AT) blockers

6. Beta-blockers (ie. Metaprolol, Carvedilol)
   a. Blocks sympathetic NS compensation that leads to decompensation & remodeling
   b. Improves mortality and morbidity

7. Anticoagulation and antiplatelet drugs
   a. Atrial fibrillation
   b. Venous stasis from ↓ CO

8. Amiodarone
   a. Currently not recommended for primary prevention of death in CHF

9. Automatic Implantable Cardiac Defibrillator (AICD)
   a. Recommended for patients with “sudden cardiac death” syndrome
10. Aldosterone Antagonists (spironolactone)
   a. Blocks aldosterone action on the sympathetic NS

11. Mechanics of positive pressure ventilation (CPAP, BiPAP, MV)
   a. Positive pressure ventilation
   b. Effects:
      i. Pulmonary pressures
      ii. Airflow
      iii. Hemodynamics

![Diagram showing positive pressure ventilation](image)

12. Goals of therapy
   a. Prevent further myocardial remodeling / damage
   b. Prevent reoccurrence of failure
   c. Increase activity tolerance
   d. Relieve symptoms
   e. Improve prognosis

13. Novel Treatments
   a. A-V sequential pacemaker
   b. Biventricular pacing
   c. Ventricular assist devices
   d. Cardiomyoplasty
   e. Enhanced external counterpulsation
   f. Transplant
**Infective Endocarditis**

Infection of the endocardium (inner lining) of the heart that covers the valves and contains the purkinje fibers.

1. Incidence
   - a. Males 3X > females
   - b. > 50 years
   - c. Mitral valve prolapse (30% in younger patients)
   - d. Rheumatic heart disease (<20%)
   - e. Calcific aortic stenosis (50% in older patients)

2. Etiology:
   - a. Subacute bacterial endocarditis (SBE)
     - i. Dental procedures
     - ii. GU or GI tract
     - iii. Abnormal valves
   - b. Acute bacterial endocarditis
     - i. Normal valves
   - c. Prosthetic valvular endocarditis
     - i. Within 1 year of valve replacement
     - ii. After pacemaker or AICD placement
   - d. Right-sided endocarditis
     - i. IV drug abuse
     - ii. Catheter-related infections (CVC, PA cath)

3. Clinical presentation
   - a. Develops on:
     - i. Mitral (most common)
     - ii. Aortic
     - iii. Tricuspid
     - iv. Pulmonary (rare)
   - b. Fever
   - c. Fatigue, night sweats, anorexia
   - d. Weight loss
   - e. Back pain
   - f. Embolism
     - i. MI
     - ii. CVA

4. Diagnosis
   - a. Blood cultures
     - i. 5% will not have positive cultures
     - ii. May take 4 days to grow some organisms
   - b. Murmur
     - i. Aortic insufficiency murmur (most common)
   - c. Widened pulse pressure
   - d. Transesophageal echocardiography (TEE)
     - i. Detects >90% of vegetations
5. Management
   a. Untreated endocarditis is always fatal
   b. Antibiotics
   c. Valvular repair if heart failure present
6. Complications
   a. Heart failure
   b. Emboli
   c. Sepsis

**Trauma**

1. Blunt: myocardial contusion
   a. RV Primary site
   b. Labs
   c. Treat pain
   d. Ventricular rupture, tamponade, CA thrombosis, valve dysfunction, conduction defects, HF, shock, emboli

2. Penetrating
   a. Puncture of heart, or BOX, with sharp object
   b. Etiology: violence, industrial accident, sports, explosion, crush injury
   c. Pathophysiology: loss of blood, tamponade
   d. Presentation: visible wound, bleeding, hypotension, tamponade
   e. Management:
      i. Control hemorrhage
      ii. OR
      iii. Monitor for complications
           1. Hemorrhagic shock
           2. Tamponade
           3. Hemothorax
           4. Pneumothorax

f. Diagnosis
   i. H & H, ECG, CXR, Aortogram, CT scan

g. Overall Management
   i. Control bleeding
   ii. Control BP
   iii. Prepare for exploratory thoracotomy
   iv. Monitor for complications
      1. Hemorrhage shock
      2. Cardiac tamponade
      3. Hemothorax
      4. False aneurysm
3. Tamponade
   a. Etiology
      i. Post-cardiotomy
      ii. Post MI
      iii. Iatrogenic causes
      iv. Post CPR
      v. Anticoagulation
      vi. Rupture of great vessels
      vii. Aortic aneurysms
      viii. Infection
   
   b. Pathophysiology
      i. Accumulation of fluid
      ii. Decreased contractility
      iii. ↓ stroke volume, cardiac output, LV function, RV function, shock
   
   c. Presentation
      i. BECK’s TRIAD
         a. Tachycardia, Hypotension, Narrowed PP
      ii. Hemodynamics
   
   d. Diagnosis
      i. CXR
      ii. ECG
      iii. Echo and/or TEE
      iv. CT Fluoroscopy
   
   e. Management
      i. ABC’s
      ii. Circulating blood volume
      iii. Inotropes
      iv. Pericardiocentesis
      v. Pericardial window
      vi. Emergency thoracotomy
**Hypertensive Crisis**

Diastolic blood pressure >120 mmHg

1. **Etiology**
   a. Pre-existing hypertension (most common)
   b. Renal disease
   c. Scleroderma
   d. Illicit drugs
   e. Pre-eclampsia, eclampsia
   f. Head injury
   g. Autonomic dysreflexia
   h. Tumors

2. **Symptoms**
   a. Chest pain
   b. Headache
   c. Decreased mental status
   d. Diuresis

3. **Diagnostics**
   a. CBC
   b. Electrolytes
   c. Urine
      i. Blood
      ii. Casts
   d. EKG
   e. Chest x-ray

4. **Treatment**
   a. Sodium nitroprusside
   b. Apresoline
   c. Vasotec
   d. Brevibloc
   e. Labetalol

5. **Complications**
   a. MI, CHF
   b. Stroke, cerebral bleed
   c. Aortic dissection
CARDIAC PEARLS

a. ABC’s
b. CO/CI – preservation of function
c. PERFUSION
d. Maintaining HR X SV
   i. PRELOAD
   ii. AFTERLOAD
   iii. CONTRACTILITY
e. ST segment depression = ischemia
f. ST segment elevation = current of injury
g. IABP= increase coronary perfusion, decrease afterload: SO, it increases myocardial oxygen supply and decreases demand
h. Murmurs: systolic = AS, MR
i. Most common systolic murmur in recent MI is mitral insufficiency
j. ST segment elevation in II, III, AVF = inferior infarction
k. ST segment elevation in I, AVL, V leads = anterior infarction
Renal (5%) 8 questions

1. Acute renal failure differs from chronic renal failure in that it:
   a. Results in higher BUN levels
   b. Has a higher mortality rate
   c. Requires peritoneal dialysis
   d. Is associated with diabetes

2. The best dialysis schedule for the patient with acute renal failure is:
   a. Every other day
   b. Weekly
   c. Daily
   d. Bi-weekly

3. The primary etiology of hyperphosphatemia is:
   a. Over-replacement
   b. Hypercalcemia
   c. Renal failure
   d. Hypoalbuminemia

4. Bradycardia, tremors and twitching muscles are associated with which electrolyte disorder?
   a. Hypokalemia
   b. Hyperkalemia
   c. Hypophosphatemia
   d. Hyperphosphatemia

5. Treatment for hypercalcemia includes:
   a. Fluids and diuretics
   b. Amphogel
   c. Kayexelate
   d. Dialysis

6. Hyponatremia is usually associated with:
   a. Fluid overload
   b. Dehydration
   c. Diuresis
   d. Over-administration of normal saline

7. Mr. Smith was involved in a motor-vehicle accident and is experiencing hematuria. The best diagnostic test to evaluate renal trauma is:
   a. Ultrasound
   b. Computed tomography (CT)
   c. Intravenous pyelogram (IVP)
   d. Angiography
8. Which of the following is not an etiology of acute renal failure (ARF)?
   a. Sepsis
   b. Shock
   c. Bladder tumor
   d. Hypertension

**Acute & Chronic Renal Failure**

1. Acute Renal Failure: Sudden loss of renal function
   a. Etiology:
      i. Pre-renal
         1. Most common outside the ICU
         2. Etiology
            a. Low cardiac output
            b. Shock
            c. Renal artery stenosis
         3. ↓ blood flow to kidneys, ↓ pressure in renal artery, ↓ forces favoring filtration, ↓ GFR
         4. Kidney’s response is vasoconstriction
         5. End result is ischemic damage to kidney
      ii. Intra-renal
         1. Most common in the ICU
         2. Causes
            a. Glomerulonephritis
            b. Antibiotics
            c. Myoglobinemia
            d. SLE, Diabetes
         3. Direct damage to glomerulus
iii. Post-renal
   1. Rare
   2. Causes
      a. Urethral calculi
      b. BPH
      c. Urethral stricture
      d. Bladder cancer
      e. Neurogenic bladder
   3. Partial obstruction = ↑ forces opposing filtration = ↓ GFR
   4. Total obstruction = compression and necrosis

   Acute Renal Failure is a secondary disease. Therefore mortality is about 40%

b. Phases:
   i. Oliguria
      1. Sudden onset of oliguria
      2. Symptoms resemble CRF
         a. Nausea & Vomiting
         b. Drowsiness, confusion, coma
         c. GI bleeding
         d. Asterixis
         e. ↑ K+, ↓ Na+, acidosis
         f. Cardiac arrhythmias
         g. Kussmaul’s respirations
         h. Hypervolemic
         i. Edema
         j. HTN

   3. Treatment:
      a. Dialysis
      b. Renal diet
      c. Fluid restriction

ii. Diuretic (10-15 days)
    1. Indicates that nephrons are healing
    2. UO ↑ to 4-5 liters/day
    3. Unable to concentrate urine or filter wastes
    4. Can have excessive excretion of K+ and Na+
    5. Manifestations
       a. Hypovolemic
       b. Hypotension
       c. Electrolyte imbalances
iii. Recovery (lasts 4-6 months)
   1. BUN, Cr slowly return to normal

iv. Treatment:
   1. Hemodialysis
   2. Continuous renal replacement therapy
      a. CAVHD
      b. CVVHD
   3. Renal diet
   4. Fluid restriction

2. Chronic Renal Failure: Progressive loss of renal function
   a. Etiology:
      i. Diabetes
      ii. Hypertension
      iii. Glomerulonephritis
   
   b. Stages:
      i. Decreased renal reserve
         1. ↓ number of functional nephrons
      ii. Renal insufficiency
         1. Asymptomatic ↑ in BUN / Cr.
      iii. Renal failure
         1. Symptomatic ↑ in BUN / Cr.
      iv. End-stage renal disease
         1. Severe ↑ BUN / Cr.
         2. Chronic dialysis is needed
   
   c. Bricker hypothesis
      i. Intact nephrons hypertrophy to compensate for diseased nephrons
   
   d. Signs and symptoms of oliguria

   e. Treatment:
      i. Hemodialysis
      ii. Peritoneal dialysis
      iii. Renal diet
      iv. Fluid restriction
      v. Medications
Renal Trauma

1. Renal injuries
   a. Blunt trauma
      i. Coup, contracoup
      ii. Shearing of renal artery, ureters
      iii. Direct kidney damage is most often accompanied by other abdominal injury
   b. Penetrating
   c. Manifestations
      i. Flank pain
      ii. Gray-Turner’s sign (flank ecchymosis 76%)
      iii. Hematuria
   d. KUB
   e. IVP
   f. Urethrogram
   g. Cystogram
   h. Ultrasound
   i. CT scan
   j. MRI

2. Kidney laceration

3. Treatment
   a. Partial / total nephrectomy
Electrolyte Abnormalities

**Potassium (3.5-5 mEq/L)**

1. Acquired in diet, excreted in urine, must be replaced daily
2. Major intracellular cation
3. Functions:
   a. Maintains osmotic pressure inside cells
   b. Maintains electrical potential
   c. Maintains acid/base balance
   d. Participates in metabolism

4. Hyperkalemia
   a. Common causes:
      i. Renal failure
      ii. Over-replacement
      iii. Cell damage / shift out of cells
         1. Acidosis
         2. Hemolysis
         3. Sepsis
         4. Chemotherapy
      iv. Spironolactone administration
   b. Manifestations
      i. Bradycardia
      ii. Tremors, twitching
      iii. Nausea / vomiting
      iv. EKG changes: (K+ suppresses the SA node)
         1. Peaked T-waves
         2. Shortened ST-segment
         3. Flattened P-wave
         4. Long PR-interval
         5. Blocks
         6. PVCs, ventricular arrhythmias
   c. Treatment
      i. Kayexelate
      ii. Insulin / glucose
      iii. Dialysis
      iv. HCO3, Ca++
      v. Albuterol aerosol

If a patient is NPO, he will require 40 mEq of potassium per day to maintain his potassium level. 200 mEq or more may be required to replace lost stores.
5. Hypokalemia (a **Low K**)
   a. Common causes:
      i. Poor intake
      ii. Renal loss
         1. Diuretics
         2. Renal tubular acidosis
         3. Gent, Ampho
      iii. GI loss
         1. Diarrhea
         2. Vomiting
      iv. Shift into cells
         1. Excessive insulin administration in DKA
         2. Alkalosis
   
   b. Manifestations
      i. Tachycardia
      ii. Hypotension
      iii. Flaccid muscles
      iv. EKG changes:
         1. Flattened T-waves
         2. Long ST-segment
         3. U-waves
         4. Peaked P-wave
         5. Long PR-interval
         6. PVCs, ventricular arrhythmias
   
   c. Treatment
      i. Oral replacement is preferable (allows slower equilibration with intracellular compartment)
      ii. IV: no faster than 20mEq/hour
   
6. Testing Implications:
   a. Potassium levels change inversely to serum pH
   b. Opening and closing the fist with a tourniquet in place ↑ K+ level
   c. ↓ K+ can lead to digoxin toxicity
**Calcium (8.4-10.2 mg/dl)**

1. Ionized (active fraction)
2. Inactive fraction (bound to albumin)
3. Adjusted calcium level
   a. \([(4-\text{Alb}) \times 0.8] + \text{Calcium} = \text{Adjusted calcium}\)
4. Essential for the functioning of:
   a. Neuromuscular activity
   b. Integrity of cell membrane
   c. Cardiac activity
   d. Blood coagulation
5. Increases in PTH, ↑ Ca++ level
6. Hypercalcemia
   a. Etiology:
      i. Hyperparathyroidism
      ii. Paget’s disease
      iii. Excessive Vitamin D intake
   b. Manifestations
      i. Anorexia, nausea, vomiting
      ii. Coma
      iii. ARF
      iv. Flaccid muscles
      v. EKG changes
         (1) Short ST
         (2) Short QT
         (3) Steep drop off of T-wave
   c. Treatment
      i. Fluids / lasix
      ii. Oral or IV Phosphate

---

**Chvostek’s sign:**
- Tap the facial nerve just below the temple
- Twitch of the lip or nose is a positive sign

**Trousseau’s sign**
- Contraction of the hand or fingers when arterial flow is occluded for 5 minutes.
7. Hypocalcemia
   a. Etiology:
      i. Surgical Hypoparathyroidism
      ii. Malabsorption
      iii. Acute pancreatitis
      iv. Renal failure
      v. Vitamin D deficiency
      vi. Hypoalbuminemia
      vii. Excessive administration of citrated (banked) blood
   b. Manifestations
      i. Laryngeal spasm
      ii. Seizures & muscle cramps
      iii. Hypotension
      iv. Hyperactive reflexes
      v. Trousseau’s sign
      vi. Chvostek’s sign
      vii. EKG changes:
           (1) Prolonged QT interval
           (2) Flat ST
           (3) Small T-wave
   c. Treatment
      i. Oral route is safer
      ii. IV: 10-20 mL of 10% calcium gluconate over 5-10 minutes
      iii. Monitor EKG during treatment

8. Implications:
   a. Ionized calcium level is inversely proportional to serum pH
   b. Serum Ca++ levels should be assessed in conjunction with serum albumin levels
Magnesium (1.5-1.95 mEq/L)

1. Intracellular enzymatic reactions and utilization of ATP
2. CNS transmission
3. Cardiovascular tone
4. Hypermagnesemia (rare)
   a. Etiology
      i. Renal disease
      ii. Adrenal insufficiency
   b. Manifestations
      i. Flushing and hypotension
      ii. Hypotension & bradycardia
      iii. Respiratory depression
      iv. Hypoactive reflexes
      v. CNS depression
   c. Treatment
      i. IV calcium: 10-20 mL of a 10% calcium gluconate
      ii. Mechanical ventilation
      iii. Temporary pacemaker
      iv. Dialysis

5. Hypomagnesemia (common electrolyte disorder)
   a. Etiology
      i. CRF
      ii. Pancreatitis
      iii. Hepatic cirrhosis
      iv. GI losses
      v. Alcoholism
      vi. Treatment of DKA
   b. Manifestations
      i. Increased reflexes
      ii. + Trousseau’s sign
      iii. + Chvostek’s sign
      iv. Tachycardia
      v. EKG changes:
         1. PR & QT prolongation
         2. Widened QRS
         3. ST depression
         4. T-wave inversion
      vi. ↓K+, ↓Ca++, ↓PO4

Magnesium is cardio-protective, and may be given to a patient with myocardial infarction even if the Mg++ level is normal.
c. Treatment:
   i. Dietary replacement
   ii. IV magnesium acts as a vasodilator (expect flushing and hypotension)
      1. Acute hypomagnesemia
         a. 1-2 grams over 60 minutes
      2. During a code for VT/VF
         a. 1-2 grams IV push (over 1-2 minutes)

6. A 24-hour urine magnesium level may be helpful in assessing deficiency
Phosphorus (2.5-4.7 mg/dl)

1. Phosphorus is an important part of all body tissue
2. Phosphate has a marked diurnal variation; therefore single measurements are of little use.
3. Mostly stored intracellularly
4. Phosphate is cleared by the kidney; therefore renal function must be monitored as well.
5. Hyperphosphatemia
   a. Etiology
      i. Renal failure
      ii. High PO4 intake
      iii. Chemotherapy
      iv. Lactic acidosis
   b. Manifestations
      i. Most often is asymptomatic
      ii. Numbness, tingling of hands and mouth
      iii. Muscle spasms
      iv. Precipitation of Ca++ salts can lead to hypocalcemia
   c. Treatment
      i. Treat underlying disorder
      ii. Phosphate-binding agents (Amphogel)
      iii. IV fluids
      iv. D50 & insulin
      v. Dialysis
6. Hypophosphatemia
   a. Etiology
      i. Refeeding syndrome (refeeding after severe malnutrition)
      ii. Calcium and magnesium deficiency
      iii. Acute respiratory disorders
      iv. Alcoholism
      v. DKA, insulin administration

Acute Respiratory Disorder

Hypophosphatemia

Acute Respiratory Distress
b. Manifestations
   i. Hemolysis & anemia
   ii. Muscle pain & weakness
   iii. Respiratory muscle weakness
   iv. ↓ LOC, paresthesias

c. Treatment
   i. Treat the primary disorder
   ii. Nutrition
   iii. Oral or IV replacement

7. Sudden ↑ in serum PO4 level during treatment can cause hypocalcemia
8. Introduce nutrition gradually to the malnourished patient
9. Phosphorus levels are inversely related to Ca++ levels
Imbalances in Sodium and Water

Sodium (135-145 mEq/L)

- Most important ion in maintaining extracellular fluid balance
- Balance is controlled by CNS & endocrine systems
- Imbalance will result in fluid shifts and edema or dehydration

\[
\text{Osmolality} = 2 \times \text{Na} + \text{Glu} / 18 + \text{BUN} / 2.8
\]

- Blood osmolality is normally 280-300 mOsm/kg H2O
- Maximum daily sodium load is 400 mEq/day (NS @125ml/hr provides 465 mEq/day)
- Hyponatremia is more common
- Hypernatremia has 40-60% mortality

Fluid shifts from low osmolality to high!

Sodium & Water Imbalances

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<th>Water Balance:</th>
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2. Normal volemic states
   a. Hypernatremia (↓ TBW, near normal TBNa)
      i. Etiology:
         1. Diabetes insipidus (lack of response to ADH)
         2. ↑ insensible losses without replacement of water
      ii. Signs and symptoms:
         1. Thirst
         2. CNS depression
      iii. Treatment:
         1. Water replacement
         2. ADH for diabetes insipidus
   b. Hyponatremia
      i. Etiology:
         1. Water ingestion > 25L/day
         2. Defect in renal diluting ability
         3. Post-operative fluid administration / non-osmotic ADH release
         4. Drugs:
            a. NSAIDS
            b. Oxytocin
      ii. Signs and symptoms:
         1. Edema
      iii. Treatment:
         1. Water restriction
         2. Sodium replacement

3. General Management Principles:
   a. Hyponatremia:
      i. Mild (Na+ <120)
         1. Usually asymptomatic
         2. Treat underlying cause
      ii. Moderate (Na+ <115)
         1. CNS depression
         2. Replace with NS
         3. Fluid restriction (<1000cc/day)
      iii. Severe (Na+ <110)
         1. Coma, seizures, and death
         2. Replace with NS or hypertonic saline (3%)
         3. Do not ↑ serum Na+ by more than 1 mEq/L/h or 10 mEq/L/day.

\[ \text{Free Water Deficit} = (\text{kg wt.} \times 0.6) \times \left[ \frac{(\text{Na}/140) - 1}{\text{Na}} \right] \]
**Pulmonary (17%) 25 questions**

1. Mr. Smith (57) is one-day post abdominal aortic aneurysm (AAA) repair. This morning he develops atrial fibrillation with subjective dyspnea. His heart rate is 121 but otherwise his vital signs are normal. What pulmonary complication is Mr. Smith suffering from?
   a. Pneumonia
   b. ARDS
   c. Shock lung
   d. Pulmonary embolism

2. The most common EKG changes that occur during pulmonary embolus are:
   a. Q-waves in AVR and Lead I
   b. Tachycardia and atrial fibrillation
   c. Bradycardia and ST-segment depression
   d. High-degree AV blocks

3. How does the D-Dimer lab test help to diagnose pulmonary embolism (PE)?
   a. A positive test indicates PE
   b. A negative test rules out PE
   c. A positive test rules out PE
   d. A negative test indicates PE

4. Thrombolytic therapy (t-PA) is indicated for pulmonary embolism when:
   a. The patient has more than one embolism
   b. The patient is alert
   c. The patient is hemodynamically unstable
   d. The embolism is confirmed on CT scan

5. The major symptoms of Fat Embolism Syndrome (FES) are:
   a. Petechiae, hypoxia, pulmonary edema
   b. Tachycardial, rales
   c. Fever, purulent sputum
   d. Chest pain and dyspnea

6. Your patient, Mr. Winston comes to the Emergency Department (ED) with an exacerbation of COPD. He is hypoxic and hypercapneic. He does not wish to be intubated and mechanically ventilated. What criteria are necessary to initiate bilevel positive-pressure ventilation (BiPAP)?
   a. Must be able to slow his breathing down and not fight the machine
   b. Must be able to maintain his own airway
   c. Must be less than 75 years-old
   d. Must quit smoking first
7. Pressure-control ventilation differs from volume-control ventilation in which of the following ways:
   a. Volume remains constant
   b. High peak pressures are delivered
   c. Pressure is limited
   d. Inspiratory time is shorter

8. Heliox is a combination of helium and oxygen that is used in asthmatics to:
   a. Reduce airway resistance
   b. Bronchodilate
   c. Reduce carbon dioxide
   d. Improve oxygenation

9. Nursing interventions that decrease the incidence of hospital-acquired pneumonia include:
   a. Placing gastric tubes through the nose
   b. Brushing the patient’s teeth
   c. Administering systemic antibiotics
   d. Keeping the patient NPO

10. The most common etiology for Acute Respiratory Distress Syndrome (ARDS) is:
    a. Sepsis
    b. Multiple trauma
    c. Pancreatitis
    d. Shock

11. Treatment for ARDS includes ventilatory support and oxygen. In addition, which of the following interventions has been shown to reduce mortality in ARDS?
    a. Hyperbaric oxygenation
    b. Prone positioning
    c. Lung-protective ventilation
    d. Artificial surfactant

12. The nurse’s role during chest tube removal is to:
    a. Provide adequate analgesia
    b. Clamp the tube
    c. Suture the insertion site
    d. Have the patient inhale during removal

13. Your patient Mr. Jones is admitted to the emergency department with acute pulmonary edema. His pO2 is 48, and his pCO2 is 57. Vital signs: B/P-158/90, P-122, RR-36. The most appropriate initial intervention is:
    a. Bi-level positive airway pressure (BiPAP)
    b. Continuous positive airway pressure (CPAP)
    c. Pressure-control ventilation
    d. Inverse-ratio ventilation
14. Appropriate interventions for the patient with hospital-acquired pneumonia include:
   a. Assure adequate fluid intake, endotracheal suction q 1-hour
   b. Antibiotics, percussion and vibration
   c. Turning, positioning, and ambulation
   d. Saline lavage, mechanical ventilation

15. Ms. Kelly (42) is recovering from an asthma attack, when she suddenly starts complaining of substernal chest pain that is worse with inspiration and radiates to the neck, along with dyspnea. Which respiratory condition is most likely causing her discomfort:
   a. Bronchospasm
   b. ARDS
   c. Pneumomediastinum
   d. Chronic bronchitis

16. What intervention is most appropriate for the asthma patient who develops hypoxia and hypercapnia:
   a. 100% oxygen by non-rebreather mask
   b. CPAP
   c. BiPAP
   d. Mechanical ventilation

17. Warning signs of a severe asthma attack include:
   a. Decrease in FEV₁
   b. Daily inhaler use
   c. Nocturnal bronchodilator use
   d. Family history of COPD

18. Magnesium sulfate may be given to the asthmatic who:
   a. Looks toxic
   b. Is unresponsive to traditional therapy
   c. Has a FEV₁ of less than 60%
   d. Is hypoxic

19. Chronic obstructive pulmonary disease (COPD) is characterized by:
   a. Airway overinflation and atelectasis
   b. Airway smooth muscle degeneration
   c. Bronchoconstriction of the terminal bronchi
   d. Chronic CO₂ retention and hypoxia
20. Pulmonary Sarcoidosis is characterized by:
   a. Productive cough and alveolar distention
   b. Dry cough and granulomas on CXR
   c. Hypoxia and CO2 retention
   d. Chest pain and weight loss

21. Your patient has developed severe subcutaneous emphysema (from neck to groin and in both arms) after a traumatic tension pneumothorax. The most appropriate nursing interventions are:
   a. Assessing for patency of the chest tube and limiting airway pressures
   b. Assessing for atelectasis and assuring adequate PEEP
   c. Employing recruitment maneuvers to re-expand the lung
   d. Instilling talc into the chest tube

22. Severe pulmonary contusion with hypoxia is treated with:
   a. Fluids and pressors
   b. 100% non-rebreather mask
   c. Mechanical ventilation and PEEP
   d. CPAP with 100% oxygen

23. Treatment of a flail chest includes stabilization with:
   a. A rib belt to hold the chest tight
   b. Rods and pins during surgery
   c. A large pillow taped to the chest
   d. Tape applied to the affected side

24. The most common cause of a traumatic hemothorax is:
   a. Rib fracture
   b. Aortic laceration
   c. Liver laceration
   d. Pulmonary bleeding

25. A patient with pulmonary hypertension who is receiving Flolan (epoprostenol) is at risk for developing which complication if therapy is interrupted?
   a. Bronchoconstriction
   b. Cardiac arrest
   c. Pulmonary embolism
   d. Pneumonia
Pulmonary Embolus

1. Brief Pathophysiology
   a. Sudden obstruction of pulmonary vasculature
   b. Altered ventilation / perfusion
   c. Increased alveolar dead space
   d. Pneumoconstriction
   e. Bronchoalveolar hypocapnia
   f. Hypoxemia → vasoconstriction
   g. Increased PVR, ↓ CO, tachycardia
   h. Inflammation
   i. Loss of surfactant
   j. Atelectasis
   k. Edema

2. Diagnostic Tests
   a. VQ scan (89% sensitivity, 92% specificity)
   b. Magnetic resonance angiography (MRA)
   c. Pulmonary angiography (the “gold standard”)
      i. Visualize embolus
      ii. Embolectomy
      iii. Thrombolysis
   d. Spiral CT
      i. Much faster than V:Q scan
      ii. Similar sensitivity/specificity
   e. Doppler-Ultrasound
      i. Positive U/S, high clinical probability = probable PE
      ii. Negative U/S, low clinical probability = low probability
   f. EKG
      i. Non-specific findings (20%)
      ii. Classic changes
         1. S1 s-wave in lead I
         2. Q3 q-wave in lead III
         3. T3 t-wave inversion in lead III
         4. Sinus tachycardia
         5. New onset atrial fibrillation

3. Labs
   a. CBC: WBC, H/H
   b. Electrolytes
   c. PT/PTT
   d. D-Dimer

4. Hemodynamics
   a. ↓ preload
   b. ↓ CO
   c. ↑ afterload
5. Symptoms
   a. Many are asymptomatic
   b. Dyspnea (73%)
   c. Pleuritic chest pain (66%)
   d. Cough (37%)
   e. Hemoptysis
   f. Restlessness / apprehension
   g. Calf pain

6. Signs
   a. Tachycardia (70%)
   b. Rales (51%)
   c. Tachypnea
   d. Hypocapnia, hypoxemia
   e. JVD
   f. Pulsus paradoxus
   g. Hypotension
   h. Diaphoresis
   i. + Homan’s sign

7. Treatment
   a. Initial management strategies for suspected PE
      i. Ventilatory support
      ii. Oxygen therapy
      iii. Circulatory support
   b. Conservative treatment
      i. Prevention
         1. Prophylactic anticoagulation
         2. TED hose
         3. Sequential compression devices
         4. Early ambulation
      ii. Anticoagulant therapy
         1. Heparin
         2. Coumadin
         3. Low-molecular-weight heparin
         4. Inferior vena cava filter
   c. Treat right-sided heart failure
   d. Invasive intervention
      i. Thrombolytic therapy (t-PA)
         1. Systemic or localized
         2. Relative contraindications same as for MI
8. Fat Embolus Syndrome (FES)
   a. Risks:
      i. Long-bone fractures
      ii. Hip replacement surgery
   b. Lipid distribution causes ARDS-like syndrome
   c. Onset within 24-48 hours
   d. Symptoms:
      i. Major:
         1. Axillary / subconjunctival petechiae
         2. Hypoxia
         3. CNS depression
         4. Pulmonary edema
      ii. Minor:
         1. Tachycardia
         2. Fever
         3. Retinal fat emboli
         4. Urinary fat globules
         5. ↓ platelet count / HCT
         6. ↑ ESR
         7. Fat globules in sputum

Internet site:
Preventing Deep Vein Thrombosis and Pulmonary Embolism: www.dvt.org
ARDS

ARDS was discovered in 1967 by Dr. Ashbaugh’s group. They studied 12 patients who had serious acute illnesses, but no direct lung damage. These patients all developed symptoms similar to those found in infants with infant respiratory distress syndrome, hence the investigators named the disorder Adult Respiratory Distress Syndrome.

1. Characterized by:
   a. Compatible history
   b. Acute onset of severe respiratory impairment (PO2:FiO2 < 200)
   c. Decreased lung compliance
   d. Refractory hypoxia
   e. Bilateral, diffuse lung infiltrates (white out)
   f. Tachypnea

2. Etiology (Risk factors)
   a. Sepsis (most common)
   b. Aspiration (2nd most common)
   c. Pneumonia
   d. Pulmonary trauma
   e. Multiple trauma
   f. Shock
   g. Multiple transfusions
   h. DIC
   i. Acute hemorrhagic pancreatitis

3. Additive result of predisposing factors
   a. One risk factor = 25% chance of ARDS
   b. Two risk factors = 42% chance of ARDS
   c. Three risk factors = 85% change of ARDS
4. Pathophysiology
   a. Can be caused by local inflammation or as the result of systemic inflammation (SIRS)
   b. Risk factors cause an abnormal accumulation of cells in the pulmonary vasculature.
   c. Neutrophils: phagocytosis, adherence, degranulation, oxygen radical production.
      i. Bronchoalveolar lavage = 1-3% neutrophils in healthy lungs
      ii. BL = 76-85% neutrophils in ARDS
   d. Polymorphonuclear leukocytes (PMN): releases mediators
   e. Platelets: causes microclotting
   f. Endothelial cells: causes adherence, releases neutrophil chemotactic factor.
   g. These cells are activated by the underlying risk factor condition and cause inflammation.

5. Inflammatory process causes:
   a. Direct microvascular and alveolar injury
   b. Disrupt normal pulmonary blood flow and coagulation
   c. Results in pulmonary hypertension, bronchoconstriction & ↑ vascular permeability
   d. Fluid leaks out of the pulmonary vasculature, into the interstitial spaces and into the alveoli resulting in diffuse pulmonary edema
   e. Release of oxygen free radicals
      i. Directly damages lung epithelium
      ii. Bronchoalveolar lavage in patients with ARDS indicates oxidant activity

**Inflammation in ARDS**

- Vasodilatation → Maintains O2
- Capillary Permeability → Edema
- Clotting → ↓ Perfusion → Hypoxia
- Vasoconstriction

[c] 2002 David W. Woodruff
6. Clinical presentation
   a. Latent phase
      i. Beginning changes in the structure of the a-c membrane
      ii. Symptoms will be from the underlying illness
   b. Acute interstitial edema phase
      i. Beginning of alveolar edema
      ii. Decreased lung compliance
      iii. Symptoms:
         1. Apprehension, restlessness
         2. Tachypnea, subjective dyspnea
         3. Usually normal chest x-ray
         4. Acute respiratory alkalosis 2° to hyperventilation
   c. Acute intra-alveolar edema phase
      i. Alveolar flooding occurs
      ii. Damage to alveolar epithelial cells
      iii. Changes in surfactant composition
      iv. Severe ↓ in lung compliance
      v. Decreased pulmonary perfusion
      vi. Intrapulmonary shunting
      vii. Symptoms:
         1. Agitation
         2. Dyspnea, tachypnea
         3. Profound hypoxemia
         4. ↑ peak inspiratory pressures
         5. Fine, diffuse crackles, diminished BS
         6. Diffuse, bilateral “white out” on chest x-ray
         7. Acute respiratory alkalosis, hypoxemia on ABGs
         8. Differential diagnosis from cardiogenic pulmonary edema
            a. Lack of acute cardiac event
            b. Presence of risk factors
            c. PAOP <18
            d. No JVD or gallop
   d. Subacute-chronic phase
      i. Formation of hyaline membranes
      ii. Alveolar septum thickens
      iii. Loss of functional units
      iv. Slow recovery of respiratory structures
      v. Death often results from infection
7. Medical & nursing intervention goals
   a. Reverse underlying pathophysiology
   b. Block mechanism of a-c membrane injury
      i. Corticosteroids
      ii. Monoclonal antibodies to endotoxins
      iii. Antioxidants
      iv. NSAIDS
      v. Nitric Oxide
      vi. Xigris
   c. Minimize consequences of acute injury
      i. Minimizing pulmonary edema
      ii. Maintain acceptable CO, while ↓ PAOP
      iii. Budget fluid administration
      iv. Maintain tissue oxygenation
      v. Consider alternative methods of mechanical ventilation
         1. Consider extracorporeal gas exchange (ECMO, AVCO₂R)
      vi. Maintain nutrition
   d. Collaborative interventions to prevent or limit complications
      i. Maintain the lowest possible PAOP that will maintain an acceptable CO
      ii. Use the lowest possible FiO₂, without risking hypoxemia
      iii. Use care in administering high levels of PEEP

ARDS Internet Sites:
ARDS Net: www.ardsnet.org
ARDS Support Center: www.ards.org
ARDS Foundation of Illinois: www.ardsfoundationil.com
Managing Acute Hypoxic Respiratory Failure

Pulmonary Hygiene
1. Turning & positioning
2. Coughing and deep breathing
3. Forced expiration
4. Incentive spirometry
5. Hydration
6. Equipment

Oxygen Delivery

FiO2

Tissues

Hemoglobin
Cardiac Output

Mechanical Ventilation
1. Ventilator Terminology
   a. Tidal volume (TV): Volume of air passing into and out of the lungs with each normal breath, usually set at 10cc/kg (IBW).
   b. Inspiratory reserve: Maximal inspiration
   c. Expiratory reserve: Maximal expiration
   d. Residual volume: Volume that cannot be exhaled, where most gas exchange occurs.
   e. Fraction of inspired air (FiO2): Percentage of oxygen delivered in inspired air.
   f. I:E Ratio: Inspiratory:Expiratory Ratio
   g. Peak airway pressure: Maximum pressure in airways.
   h. PaO2: Partial pressure of oxygen dissolved in the blood.
   i. PaCO2: Partial pressure of carbon dioxide dissolved in the blood.
   j. SaO2: Percentage of hemoglobin saturated with oxygen.

2. Hemodynamic effects
   a. Decreased venous return to the heart
   b. Decreased cardiac output
   c. Increased afterload

3. Continuous Positive Airway Pressure (CPAP)
   a. Positive pressure at end-expiration to prevent alveolar collapse
   b. Increases residual volume
   c. Improves gas exchange
4. Bilevel Positive Airway Pressure (BiPAP)
   a. CPAP with additional inspiratory pressure
   b. Improves gas exchange
   c. Decreases work of breathing

5. Modes of Mechanical Ventilation
   a. Assist Control (AC)
      i. Every breath is supported
      ii. Used to rest pulmonary musculature
         1. Post-cardiac or respiratory arrest
         2. Pulmonary edema
         3. ARDS
         4. Anxiety or apprehension

   b. Intermittent Mandatory Ventilation (IMV or SIMV)
      i. Machine initiated breaths are supported
      ii. Allows patient effort
      iii. Minimizes barotrauma and hemodynamic effects
      iv. Less likely to hyperventilate
      v. Used for initial weaning efforts in some patients
c. Positive End-Expiratory Pressure (PEEP) / Continuous Positive Airway Pressure (CPAP)
   i. Prevents alveolar collapse
   ii. Increases pO2 without increasing FiO2
   iii. CPAP used in spontaneously breathing patients
   iv. Normal starting PEEP: 5 cmH2O
   v. Can use 8-12 cmH2O with low Vt
   vi. Adverse effects of PEEP
       1. Respiratory
       2. Hemodynamic

d. Pressure Support Ventilation (PS)
   i. Overcomes resistance of tubing
   ii. Used for weaning

e. Volume Control Ventilation
   i. Preset volume is delivered
   ii. Develops high peak pressures

f. Pressure Control Ventilation
   i. Preset pressure is delivered
   ii. Peak pressure is limited
   iii. Pressure is maintained longer

**Volume vs. Pressure Control**
Ventilatory Adjuncts

1. Aerosol treatments
   a. Bronchodilators
      i. Any patient that may have bronchoconstriction
      ii. Helps to mobilize secretions
   b. Mucolytics
      i. Hydrate the patient
      ii. Hydrate the airways
      iii. Then use a mucolytic

2. Nitric oxide
   a. Pulmonary vasodilator
   b. Increases oxygenation
   c. Does not improve mortality

3. Helium
   a. Promotes oxygen transport to alveoli
   b. Used in asthma and COPD to improve oxygenation

4. Prone positioning
   a. Redistributes lung fluid
   b. Relieves heart weight on lower lobes
   c. Improves oxygenation
   d. ↓ in CO2 is associated in improved mortality
   e. Complications can be avoided by:
      i. Limiting prone time to less than 2 hours
      ii. Seeking assistance in proning from anesthesia
      iii. Using adequate staff to prone

5. Rotational beds
   a. Move the patient!
      i. Ambulation
      ii. Turning and positioning
      iii. Rotational beds

6. Vibration & percussion
   a. Helps to mobilize secretions
   b. Rotational beds will do this for you
Pneumonia

Brief Pathophysiology

1. Hospital-acquired (HAP)
   a. Affects 10-25% of ICU patients
   b. Ventilator-associated pneumonia (VAP) incidence is 6-21 times higher than in non-ventilated patients.

2. Factors that lead to colonization of the respiratory tract
   a. ↓ salivary flow rate
   b. Poor oral hygiene
   c. Systemic antibiotics
   d. No oral fluids or food

3. How colonization takes place
   a. Nosocomial pathogens are transmitted from one patient to the next
   b. Subglottic secretions pool above the endotracheal tube cuff: within 24 hours 95% of ETs were partially covered with bacteria, and 84% were completely covered.
   c. Nasally placed NG tubes lead to colonization of the nasopharynx
   d. Gut failure leads to translocation of bacteria: early enteral feedings have been shown to decrease the risk of pneumonia and other secondary infections.
   e. Metabolic acidosis increases the risk of colonization

4. Risk factors
   a. Advanced age
   b. Pre-existing chronic disease
   c. Immunosuppression
   d. Medications
      i. Steroids
      ii. Antibiotic therapy
      iii. Antacids
      iv. Aerosol treatments
   e. Mechanical ventilation
   f. Endotracheal intubation / tracheostomy
   g. Surgery

5. Diagnostic Tests
   a. Bronchoscopy
   b. Bronchial lavage

6. Labs
   a. ↑ WBC
   b. Cultures
      i. Sputum
      ii. Bronchial lavage
7. Symptoms
   a. Dyspnea
   b. Productive cough
   c. Change in sputum amount and color
   d. Fatigue, weakness, malaise
   e. Friction rub

   a. Fever (average 38.5°C)
   b. ↑ RR (average 30)
   c. WBC > 10,000/mm3 (82%)
   d. Evidence on CXR (80%)
   e. Need for oxygen therapy (76%)
   f. ↓ pO2:FiO2 (69%)
   g. Intubation (46%)
   h. Sputum production & abnormal breath sounds (64%)

9. Treatment:
   a. Prevention
      i. NIPPV
      ii. Pulmonary hygiene
      iii. Care of equipment
      iv. Hydration
   b. Antibiotics
   c. Consider antibiotic rotation to prevent resistant nosocomial infections
   d. Enteral feeding

Outcomes:
   • Favorable 72%
   • Complications 5%
   • Death 20%
Air-leak Syndromes (pneumothorax/pericardium/mediastinum, PIE)

1. Types:
   a. Pneumothorax
   b. Spontaneous
   c. Traumatic
   d. Iatrogenic
   e. Tension
   f. Open
   g. Mediastinal drainage
      i. Post-CABG
      ii. Air
   h. Pleural
      i. Effusion
      ii. Empyema

2. Nursing Interventions
   a. Ensure analgesia during placement
   b. Sterile technique
   c. Sutured in place to prevent dislodgment
   d. Attach to drainage system
   e. Sterile, occlusive dressing
   f. Confirm placement

3. Chest Drainage System
   a. Suction control
      i. Water column
      ii. Dial control
   b. Water seal
   c. Collection chamber

4. Avoiding Complications
   a. Avoid “stripping” to decrease trauma
   b. Report drainage of more than 100 cc/hr
   c. Keep tubing free from obstruction
   d. Maintain sterile, occlusive dressing
   e. Assess
      i. Site
      ii. Tube
      iii. Output
      iv. Patency

5. Critical Situations
   a. Tension pneumothorax
   b. Sudden ↑ or ↓ in drainage
6. Common Problems
   a. System fell over
   b. Patient transfer
   c. Specimen collection
   d. Water level has changed
   e. Clots
   f. Replacing the system

7. Chest Tube Removal
   a. Lung re-expansion
   b. Preparation for removal
      i. Clamping for 2 hours
      ii. Pain control during removal
   c. Dressing
      i. 1st occlusive
      ii. DSD thereafter

Components of a properly functioning chest drainage system:
- Gentle bubbling in the suction chamber
- No bubbling in the water seal
- Gentle rising and falling in the water seal with respiration
1. Circadian influence  
   a. Worst function around 3 am  
   b. Best function around 3 pm  
2. Risk factors for death from severe asthma attacks  
   a. Previous severe asthma attacks  
   b. Hypercapnia  
   c. Airway hyper-reactivity  
   d. Long-term steroid therapy  
   e. Age  
   f. Noncompliance  
   g. Psychiatric illness  
3. Warning signs of a severe asthma attack  
   a. Subjective increase in dyspnea  
   b. Increases in sleep disturbances  
   c. Increase in nocturnal bronchodilator use  
   d. Morning chest stiffness or heaviness  
   e. Increase in cough frequency or severity  
   f. Runny nose or sneezing bouts  
4. Manifestations  
   a. Immediate bronchoconstriction (early-phase reaction)  
   b. Dyspnea, tachypnea (> 30 bpm)  
   c. Tachycardia (> 120 bpm)  
   d. Wheezing  
   e. Cough (sputum can be yellow due to eosinophils)  
   f. Accessory muscle use (retractions & nasal flaring in children)  
   g. Orthopnea
h. Diaphoresis  
i. ↓ FEV1 may reach 30-35% of personal best  
j. ↓ FEV1:FVC  
k. Pulsus paradoxus > 10 mmHg  
l. ↑ PAP due to vasoconstriction and alveolar overdistention  
m. Shunt develops  
n. Hypoxia, hypercapnia will develop as the attack progresses  
o. Delayed airway obstruction, inflammation and hyper-responsiveness (late-phase reaction)  
   i. Symptoms may seem to relapse within 8-24 hours  

5. Treatment  
a. Bronchodilators:  
   i. Beta-agonists  
      1. Low dose 2.5mg every 20 minutes X3 (7.5mg)  
      2. High dose 7.5mg every 20 minutes X3 (22.5mg)  
      3. Intermittent dosing as effective as continuous infusion  
   ii. Anticholinergics  
      1. 0.5mg every 4-8 hours  
   iii. Steroids  
   iv. IV Magnesium  
      1. Acts as a bronchodilator, ↓ inflammation  
      2. Greatest effect in most severe cases  
      3. 2 grams IV  
b. Antibiotics  
   i. Viral infections more common  
   ii. Get sputum sample and treat accordingly  
   iii. Strong link between sinus infections and asthma exacerbations  
c. Assisted ventilation  
   i. BiPAP 5 – 7.5 cmH2O  
   ii. Oral intubation is recommended  
      1. Asthmatics frequently have sinusitis  
   iii. Sedation with Propofol may induce bronchodilation  
   iv. Avoid paralytic agents: can cause myopathies  
d. Anxiety control  
e. The National Asthma Education Program  
   i. Patient education reduces ER visits and hospitalizations.  
   ii. Patients managed by allergists had fewer hospitalizations and ER visits than those managed by the primary physician.  
f. Immune modification  
g. Allergy control  
h. Patients exposed to cats and dogs in the first year of life had less incidence of asthma
COPD

1. Economic impact
   a. More than 14 million Americans are affected to some degree
   b. Second largest financial impact on the Social Security Disability system 
      (second only to heart disease)
   c. Forth leading cause of death
   d. 45% have restrictions on their activity level

2. Etiology
   a. Cigarette smoking (80-90%)
   b. Air pollution
   c. Occupation: Coal miners, firefighters
   d. Genetic link?
   e. Hyper-reactive airways
   f. Alpha-1 antitrypsin deficiency

3. Review of pathophysiology
   a. Emphysema: permanent enlargement of the terminal airspaces with 
      destruction of their walls.
   b. Chronic bronchitis: chronic, productive cough for more than 3 months in 
      two consecutive years.
   c. Inactivation of alpha-1 antitrypsin
      i. Stimulation of alveolar macrophages to attract neutrophils 
         (inflammation)
      ii. Inhibits enzymes that synthesize and repair elastic fibers
      iii. Destruction of the elastic fibers allows small airways to collapse
      iv. Collapse of the small airways causes air-trapping
      v. Inflammation occurs from deposits of irritant substances
      vi. Proliferation of goblet cells
      vii. Enlargement of mucous glands
      viii. Smooth muscle hypertrophy
      ix. Fibrosis
      x. Breaks down alveolar walls, resulting in bulla

4. Manifestations
   a. PFTs
      i. ↑ TLC
      ii. ↑ FRC
      iii. ↓ FEV1 to <1L
   b. Hypercapnia, hypoxia
   c. Dyspnea
   d. Fatigue
   e. Productive cough with changes in amount or color or sputum
   f. Wheezing
   g. Paradoxical respirations
   h. Change in mental status
5. Criteria for ICU admission
   a. Respiratory muscle fatigue
   b. Need for ventilatory assistance
   c. Refractory hypoxemia
   d. Respiratory acidosis (pH <7.30)
   e. Cardiovascular instability

6. Pulmonary care
   a. Bronchodilation
   b. Beta2-agonist
   c. Albuterol: beta2 smooth muscle relaxant
      i. 4 puffs using MDI & spacer = 2.5mg via aerosol
      ii. Some studies show no effect on airway resistance
      iii. Only about 3% is deposited in the airways
      iv. MDI q 30-60 min. until effective or side effects occur
      v. Aerosol 2.5mg
   d. Anticholinergic: inhibits vagal mediated smooth muscle contraction
      i. Atrovent (ipratropium bromide)
      ii. MDI 4 puffs or aerosol 0.5mg q 4-8 hours
   e. Aminophylline: xanthine smooth muscle relaxant
      i. ? bronchodilator effect
      ii. Improves secretion clearance & diaphragm contractility
      iii. Loading dose: 5-6 mg/kg
      iv. Followed by a continuous infusion: 0.5mg/kg/hr
      v. Therapeutic level: 8-12 mg/ml
   f. Steroids: anti-inflammatory agent
      i. 60-125 mg IV for 24 hours, then
      ii. 60-80 mg P.O. tapering dose for 10-14 days
   g. Antipyretics
      i. Fever increases O2 consumption and CO2 production
      ii. Can be as much as 10% for each degree Fahrenheit
   h. Oxygen
      i. Maintain PaO2 >60mmHg
      ii. Maintain O2 Sat >90%
   i. Maintain patency of the airway
      i. Humidification of inspired gases
      ii. Airway adjuncts
      iii. Suctioning
   j. Percussion, vibration, and postural drainage
   k. Ambulation, turning & positioning, forced expiration, incentive spirometry

7. Assisted ventilation
   a. If ↑ PCO2 without ↓ pH, pt. is probably a CO2 retainer
   b. If ↑ PCO2 with ↓ pH, pt. may require mechanical ventilation
8. Non-invasive: CPAP, BiPAP
   a. Must be alert, cooperative, able to handle secretions, and stable

9. Mechanical ventilation
   a. May be needed to rest the respiratory muscles
      i. ↓ WOB
      ii. ↓ Oxygen consumption
   b. Improve gas exchange
   c. Simplify suctioning

10. Antibiotics may be indicated for:
    a. Change in sputum
    b. To prevent complications

11. Lung-volume reduction surgery

12. Goals of therapy
    a. Prevent disease progression
    b. Relieve symptoms
    c. Improve exercise tolerance
    d. Improve health status
    e. Prevent and treat exacerbations
    f. Prevent and treat complications
    g. Reduce mortality
    h. Minimize side effects from treatment

13. Pulmonary rehabilitation

Internet sites:
Asthma:
Global initiative for asthma: www.ginasthma.com
American Lung Association: www.lungusa.org

COPD:
Global initiative for COPD: www.goldcopd.com
COPD Support: www.copd-support.org
COPD: www.ibreathe.com
American Lung Association: www.lungusa.org
Chest Trauma

1. Etiology
   a. Blunt
   b. Penetrating

Level of injury corresponds with specific anatomical injuries:

<table>
<thead>
<tr>
<th>Level of injury</th>
<th>Anatomy</th>
</tr>
</thead>
<tbody>
<tr>
<td>C4</td>
<td>Hyoid bone</td>
</tr>
<tr>
<td>C6</td>
<td>Cricoid cartilage</td>
</tr>
<tr>
<td>T2</td>
<td>Suprasternal notch</td>
</tr>
<tr>
<td>T4</td>
<td>Tracheal bifurcation, aortic arch</td>
</tr>
<tr>
<td>T6</td>
<td>Pulmonary artery</td>
</tr>
<tr>
<td>T8</td>
<td>Vena caval foramen in diaphragm</td>
</tr>
<tr>
<td>T10</td>
<td>Esophageal hiatus in diaphragm</td>
</tr>
<tr>
<td>T12</td>
<td>Aortic hiatus in diaphragm</td>
</tr>
<tr>
<td>L2</td>
<td>Right crus of diaphragm</td>
</tr>
<tr>
<td>L4</td>
<td>Umbilicus</td>
</tr>
</tbody>
</table>

Pulmonary Contusion: bruising of the pulmonary tissues

1. Etiology
   a. Usually blunt trauma

2. Pathophysiology
   a. Bruising causes stimulation of inflammation
   b. ↑ capillary permeability
   c. Fluid leaks out causing pulmonary edema
   d. WBC’s migrate to area
   e. Fluid, inflammatory debris, damaged cells form pus and disrupt the capillary/alveolar membrane
   f. Alveoli collapse and decrease FRC, causing hypoxemia

3. Manifestations
   a. Look for contusions on chest wall
   b. Tachypnea
   c. Dyspnea
   d. Blood-tinged sputum
   e. Increased airway/peak pressures
   f. Decreased PaO2:FiO2

4. Treatment
   a. Airway
   b. Mechanical ventilation with PEEP
   c. Negative fluid balance (if possible) to control pulmonary edema

5. Complications
   a. Infection
   b. ARDS, MODS
Rib Fractures

1. Simple fractures may result in ↓ ventilation due to pain
2. Multiple fractures can result in flail segments
3. Bone fragments can rupture skin or disrupt underlying tissues
4. 1st rib fractures are associated with a higher incidence of great vessel injury and cervical spine injury.
5. Lower rib fractures (7 to 12) are associated with abdominal injuries
6. Manifestations
   a. Look for signs of contusion
   b. Pleuritic pain
   c. ↓ respiratory effort
   d. X-ray, CT scan
7. Treatment
   a. Physiologic splinting
   b. Rib belts
   c. ORIF
8. Complications
   a. Pneumothorax
   b. Hemothorax
   c. Underlying tissue damage

Flail Chest

1. Results from fractures of two or more segments of ribs
2. Allows a free floating segment that moves paradoxically
3. Lung does not expand as usual, resulting in hypoxemia
4. Segment may cause damage to surrounding tissue
5. Manifestations
   a. Pleuritic pain
   b. Dyspnea
   c. Deformity (increases as muscle spasm decreases)
   d. Crepitus
   e. Hypoxemia
6. Treatment
   a. Oxygen
   b. Ventilation
   c. Stabilize with tape (one side only, do not wrap chest)
   d. ORIF
7. Complications
   a. Pneumothorax
   b. Atelectasis
   c. ARDS
Pneumothorax:
Collection of air in the pleural space $\rightarrow$ Loss of negative pressure eliminates pull to open lungs $\rightarrow$ Lung collapses
1. Etiology
   a. Rib fractures
   b. Penetrating trauma
   c. Blunt trauma, without exhalation
2. Types
   a. Simple: air escapes from lung structures into pleural space
   b. Spontaneous: bleb on lung ruptures
   c. Tension: pleural pressure is positive, causing displacement of mediastium and opposite lung.
3. Manifestations
   a. Dyspnea
   b. Hypoxemia
   c. Decreased breath sounds on affected side
   d. X-ray, CT scan
4. Treatment
   a. Observation
   b. Chest drainage
   c. Chest tube
   d. Thoracic catheter
   e. Needle thoracotomy for tension pneumothorax
   f. McSwain dart
5. Complications
   a. Tension pneumothorax

Hemothorax:
Collection of blood in the pleural space
1. Source:

<table>
<thead>
<tr>
<th>Left Hemothorax</th>
<th>Right Hemothorax</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rib fracture</td>
<td>Rib fracture</td>
</tr>
<tr>
<td>Pulmonary parenchyma</td>
<td>Pulmonary parenchyma</td>
</tr>
<tr>
<td>Aorta</td>
<td>Liver</td>
</tr>
<tr>
<td>36%</td>
<td>51%</td>
</tr>
<tr>
<td>35%</td>
<td>27%</td>
</tr>
<tr>
<td>15%</td>
<td>10%</td>
</tr>
</tbody>
</table>

2. Manifestations
   a. Dyspnea
   b. Tachypnea
   c. Cyanosis, hypoxemia
   d. Shock
3. Treatment
   a. Chest drainage
   b. Volume replacement
   c. Thoracotomy
d. Auto-transfusion
   i. Advantages
      1. Blood is readily available
      2. Requires no cross-matching
      3. Avoid the risk of AIDS, HbV
      4. Platelet counts and 2,3 DPG; levels near normal maximizing clotting and oxygenation
      5. May overcome denial based on religious belief
   ii. Contraindications
      1. Known malignancy
      2. Renal / liver failure
      3. Wounds greater than 3 hours old
      4. Bowel or stomach contamination
  e. Complications:
     i. Emergency thoracotomy may be necessary if there is:
     ii. Greater than 1500 ml of blood evacuated on initial removal
     iii. Continued bleeding of >300 ml/hr (150 ml/hr in elderly) for more than 3 hours
     iv. Hemodynamic instability
     v. Tension hemothorax
Neurologic (5%) 8 questions

1. Brief loss of consciousness, followed by a lucid period, followed by a secondary loss of consciousness is characteristic of which traumatic brain injury?
   a. Subdural hematoma
   b. Subarachnoid hemorrhage
   c. Epidural hemorrhage
   d. Concussion

2. The most common cause of subarachnoid hemorrhage is:
   a. Aneurysms
   b. Coagulopathies
   c. Trauma from falls
   d. Ischemia

3. Which of the following statements best describes transient ischemic attacks (TIAs)?
   a. Damage and symptoms resolve
   b. Damage and symptoms are permanent
   c. Damage is permanent, but symptoms resolve
   d. Damage is permanent, there are no symptoms

4. The best indicator of changes in neurological function in the alert patient is:
   a. Changes in behavior
   b. Disorientation
   c. Unresponsiveness
   d. Pupil changes

5. In a patient with increased intracranial pressure, cerebral perfusion pressure should be maintained at:
   a. 40 mmHg
   b. 50 mmHg
   c. 60 mmHg
   d. 70 mmHg

6. The most sensitive indicator of changes in intracranial pressure in patients who are unresponsive is:
   a. Change in systolic blood pressure
   b. Change in pupil response
   c. Blood glucose levels
   d. Response of the cranial nerves
7. Nursing intervention for the neuro patient should include:
   a. Not clustering activities
   b. Performing frequent neuro checks
   c. Administering diuretics
   d. Positioning in the prone position

8. If hyperventilation is used to control increased intracranial pressure it should be directed at achieving the goal of:
   a. Decreasing the CO₂ to 32-35
   b. Increasing the pO₂ to 200
   c. Maintaining a respiratory rate of 30
   d. Lowering the pH

**Bleeds, Aneurysms, AVMs**

1. Subdural
   a. Acute (first 48 hours)
   b. Subacute (2 days to 2 weeks)
   c. Chronic (after 2 weeks)

2. Epidural
   a. Usually arterial
   b. LOC, followed by lucid period, followed again by LOC

3. Subarachnoid
   a. (see aneurysms)

4. Intracerebral
   a. Slow developing
   b. Progressive ↓ in LOC
   c. Poor prognosis
Aneurysms
A saccular outpouching of a cerebral vessel, which can burst and result in SAH. 90% are berry aneurysms, occur in the circle of Willis.

1. Classification
   a. Small: < 10mm
   b. Medium: 10-15mm
   c. Large: 15-25mm
   d. Giant: 25-50mm
   e. Super-giant: > 50mm

2. Unruptured
   a. Most are asymptomatic
   b. Signs / symptoms:
      i. Dilated pupils
      ii. EOM
      iii. Eye pain
      iv. Localized headache
      v. Neck rigidity
      vi. Photophobia

3. Ruptured
   a. Bleeds into subarachnoid or intracerebral space
   b. Signs / symptoms:
      i. “Explosive” headache
      ii. ↓ LOC
      iii. Nausea & vomiting
      iv. EKG changes
Hunt-Hess Classification of Subarachnoid Hemorrhage

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>II</td>
<td>Mild cranial nerve dysfunction</td>
</tr>
<tr>
<td>III</td>
<td>Mild focal deficit, lethargy, confusion</td>
</tr>
<tr>
<td>IV</td>
<td>↓ LOC, hemiparesis, abnormal posturing</td>
</tr>
<tr>
<td>V</td>
<td>Deep coma, posturing</td>
</tr>
</tbody>
</table>

4. Diagnosis:
   a. CT scan
      i. Usually can detect SAH
   b. CTA
      i. Pretty good sensitivity/specificity
   c. MRI
      i. Not helpful in the first 24 hours
   d. Angiography
      i. “Gold Standard”

5. Treatment:
   a. Surgical
      i. Wrapping
      ii. Trapping
      iii. Clipping
   b. Post-op care:
      i. Blood pressure control
         (120-150 systolic)
      ii. Watch for vasospasm!
         1. Gradual ↓ in LOC
         2. Focal
            a. Hemiparesis
            b. Cranial nerve deficit
            c. Aphasia
      iii. Fluid volume control
         1. Triple-H therapy
            a. Hypervolemic
               i. NS, albumin
            b. Hypertensive
            c. Hemodilution
      iv. Medications
         a. Nimodipine
         b. Anticonvulsants
         c. Stool softeners
         d. Steroids
         e. Analgesics
         f. Sedatives
Arteriovenous Malformations

1. Types:
   a. Capillary Telangiectases
   b. Cavernous Malformations
   c. Venous Malformations
   d. Arteriovenous Malformations

2. Signs & symptoms
   a. Intracerebral bleeding
   b. Seizures
   c. Headache
      i. Recurrent, migraine-like
   d. Progressive neurological deficits

3. Treatment:
   a. Surgery
   b. Embolization
   c. Radiosurgery
   d. Conservative medical management
Neurosurgical Complications

Stroke
TIAs
1. Vascular events that result in temporary, focal neurological findings
2. Characteristics:
   a. Maximal dysfunction within 5 minutes
   b. Resolve within 15 minutes (may persist for 24 hours)
   c. If resolution occurs within 21 days termed: Reversible Ischemic Neurological Deficit (RIND).
3. Etiology:
   a. Cardiac & atherosclerotic plaques
   b. Arterial obstruction
   c. Arterial inflammation
   d. Hematologic abnormalities
4. May be a precursor to stroke

Ischemic Stroke
1. Risk factors
   a. Hypertension
   b. Cardiac disease, hyperlipidemia
   c. TIA’s, previous stroke
   d. Diabetes
   e. Asymptomatic carotid bruit
   f. Oral contraceptives
2. Types:
   a. Thrombotic
      1) Atherosclerotic vessel narrowing
      2) TIAs may precede
   b. Lacunar
      1) Thrombus occurs in small arteries of the deep gray or white matter
      2) Occurs frequently in pts. with HTN
   c. Embolic
      1) Accounts for 20% of ischemic strokes
      2) Carotids
      3) Cardiac origin:
          i. A-fib
          ii. Diseased heart valves
          iii. Infectious endocarditis
          iv. Cardiomyopathy
   d. Perioperative
      1) CABG
         i. 8% focal neuro deficits
         ii. 10% diffuse encephalopathy
         iii. 50-80% cognitive deficits
      2) Hypotension
Seizures

1. Etiology:
   a. Bleeding
   b. Infection
   c. Ischemia
   d. Electrolyte disorders
2. Precipitating factors:
   a. Stress
   b. Sleep deprivation
   c. Fever
   d. Alcohol or drug withdraw
3. Types:
   a. Partial
   b. Complex partial
   c. Generalized
4. Phases:
   a. Aura
   b. Sensory or motor
   c. Post-ictal
5. Nursing care
   a. Precautions
      i. Bed low and locked
      ii. Pad side rails
      iii. Airway, oxygen and suction at bedside
   b. Management of the seizure
      i. Protect patient from injury
      ii. Maintain airway
      iii. Documentation
      iv. Antiepileptic medications
         1. Valium
         2. Dilantin
         3. Phenobarbital
         4. Propofol
         5. Tegretol
         6. Valproate
   c. Post-ictal care
      i. Neuro check
      ii. Support airway and breathing
      iii. Monitor EKG
      iv. Assess for cause
Meningitis

1. Intrinsic (bloodborne)
2. Extrinsic (sinus infection, contaminated CSF)
3. Organisms
   a. H-flu
   b. Neisseria meningitis
   c. Streptococci pneumonia
   d. Pneumococcal
   e. Viruses
   f. Fungi
4. Signs and symptoms
   a. Headache
   b. Neck rigidity
   c. Fever
   d. ↑ WBC
   e. Neurologic degeneration
   f. CT: usually negative
   g. CSF analysis
5. Treatment
   a. Supportive
   b. Antibiotics
   c. Steroids
   d. Surgical

Herniation

Abnormal protrusion of the brain
1. Protrudes out of its cavity
2. Movement is:
   a. Lateral
   b. Down
3. Protrusion goes into the midbrain and brain stem
   a. Local signs followed by central signs
      i. ↓ LOC
      ii. Pupil changes
      iii. Motor and reflexes
         1. Flexion
         2. Extension
      iv. Cushing’s triad
      v. Decompensation
4. Treatment
   a. ↓ ICP
   b. Surgical
ICP Monitoring Techniques

**Invasive**

1. Intracranial monitoring
   a. Pressure
      1. Epidural
      2. Subdural
      3. Subarachnoid
      4. Intraparenchymal
      5. Intraventricular
b. Normal waveform

![Normal waveform image]

c. Intracranial Pressure Waveforms
   1. A-waves
   2. B-waves
   3. C-waves

![Intracranial Pressure Waveforms image]

d. Cerebral perfusion pressure (CPP)
e. Cerebral oxygenation
   (1) Jugular venous oxygen saturation (Norm: 60-75%)

*Non-invasive*

1. INVOS® Cerebral Oximeter
   a. Cerebral oxygenation
2. BIS Monitor
   a. Brain activity
   b. Tested only for use in sedation
Management of Increased Intracranial Pressure

Causes of ↑ ICP

1. Vasogenic Edema
   a. Disruption of blood/brain barrier
   b. Allows fluid and proteins to “leak” into brain tissue
   c. Etiology:
      (1) Trauma
      (2) Ischemia
      (3) Tumor
      (4) Infection
      (5) Brain abscess

2. Cytotoxic Edema
   a. Hypoxic injury causes intracellular swelling
   b. Etiology:
      (1) Trauma
      (2) Cerebral hemorrhage
      (3) Hypo-osmolar states

3. Interstitial Edema
   a. Increased CSF production or decreased removal
   b. Etiology:
      (1) Infection
      (2) Cerebral aneurysm rupture
      (3) Brain tumor
Evidence of cerebral edema (increased ICP)

1. Signs / symptoms
   a. Decreased level of consciousness
   b. Alterations in thought process
   c. Headache, nausea, vomiting
   d. Sensory loss, paresthesias
   e. Motor loss, paralysis
   f. Pupil changes
   g. Alteration in body temperature
   h. Seizures

Multisystem effects of increased intracranial pressure

1. Gastrointestinal bleeding
2. EKG abnormalities
   a. T-wave changes
   b. S-T elevation / depression
   c. Q-waves
   d. Arrhythmias

Management of ↑ ICP

1. ↓ ICP
2. Balance oxygen supply and demand using the Ventilation-Perfusion Train

![Diagram of the Ventilation-Perfusion Train]

- **FiO2**
- **Hemoglobin**
- **Cardiac Output**
- **Tissues**
Medical & nursing interventions

1. Cerebral perfusion
   a. Thrombolytics
   b. Anticoagulants
   c. Angiography

2. Oxygenation
   a. Supply and demand
      i. ↑ FiO2 / PO2
      ii. ↑ CO
      iii. ↓ VO

3. Hyperventilation
   a. Effects are temporary
   b. Must be sustained

4. Steroids
   a. ↓ inflammation

5. Mannitol
   a. ↓ volume
   b. Neuroprotective effect

6. Decreasing metabolic activity
   a. ↓ temp
   b. ↓ activity

7. Surgical release

<table>
<thead>
<tr>
<th>Vasodilation</th>
<th>Vasoconstriction</th>
</tr>
</thead>
<tbody>
<tr>
<td>↓ B/P</td>
<td>↑ B/P</td>
</tr>
<tr>
<td>↑ CO2</td>
<td>↓ CO2</td>
</tr>
<tr>
<td>↓ O2</td>
<td>↑ O2</td>
</tr>
<tr>
<td>↓ pH</td>
<td>↑ pH</td>
</tr>
</tbody>
</table>
Endocrine (4%) 6 questions

1. The “cardinal sign” of SIADH is?
   a. Hyponatremia
   b. Urinary output of 10 liters/day
   c. Hypotension
   d. Systemic edema

2. Which of the following are characteristic of diabetes insipidus?
   a. Low urine osmolarity
   b. Serum osmolarity increased
   c. Serum sodium elevated
   d. All of the above

3. The nurse understands that the primary cause of the classic clinical manifestations in HHS is:
   a. Rapid decrease in plasma osmolality
   b. Markedly elevated serum glucose
   c. Intravascular dehydration
   d. Serum electrolyte abnormality

4. The altered mental status in a patient in HHS results from:
   a. Hyperosmolality of plasma
   b. Intracerebral dehydration
   c. Severe osmotic diuresis from hyperglycemia
   d. Intravascular dehydration

5. When plasma glucose falls to 250 mg% in acute DKA, IV fluids should be changed to D5 1/2NS to prevent which of the following?
   a. Hyperglycemia
   b. Hyperkalemia
   c. Cerebral edema
   d. Somogyi effect

6. Nursing care for the patient with hypoglycemia may include which of the following:
   a. Administering D50 IV push
   b. Giving skim milk to the alert patient
   c. Providing additional nutrients with a meal
   d. All of the above
Endocrine

1. Functions
   A. Metabolic functions
   B. STRESS response
   C. Growth and development
   D. Fluid and electrolytes
   E. Adaptation and reproduction

Diabetes Insipidus (DI)

1. Etiology
   a. Neurogenic
   b. Nephrogenic
   c. Psychogenic

2. Clinical Presentation
   a. Polyuria
   b. Thirst
   c. Fatigue
   d. Dehydration
   e. Neurologic
   f. Urine Specific Gravity
   g. Serum Sodium
   h. BUN $\uparrow$
   i. Serum Osmolality
   j. Serum ADH level
   k. Water Deprivation Test

3. Diagnostic
   a. Serum Na
   b. BUN
   c. $\uparrow$ Serum Osmolality

4. Management
   a. Detect clinical indications of DI
   b. Monitor urine output, wt, serum labs, hypovolemia
   c. Correct fluid deficit
   d. Hypotonic solutions
Syndrome of Inappropriate Anti-diuretic Hormone (SIADH)

1. Etiology
   a. Neurogenic
   b. Ectopic tumor
   c. Nephrogenic
   d. Pulmonary
   e. Hypoxia, stress, multifactorial in ICU patient

2. Clinical Presentation
   a. Oliguria: urine output less than 0.5 ml/kg/hr
   b. Urine Specific Gravity: > 1.030
   c. Clinical indications of overhydration
   d. Anorexia, N+V, diarrhea
   e. Dyspnea and pulmonary edema
   f. HA, personality changes, altered LOC
   g. Seizures
   h. Muscle weakness or cramps
   i. Serum Na <120mEq/liter
   j. BUN ↑↑
   k. Serum osmolality ↑↑
   l. Serum ADH level ↑↑ if neurogenic

3. Treatment
   a. Detect SIADH
   b. Monitor urine output, specific gravity
   c. Treat cause
   d. Surgery to remove malignancy
   e. Demeclocycline, phenytoin, lithium to inhibit the effect of ADH on the renal tubule
   f. DC causative drugs
   g. Correct fluid volume excess
   h. Correct electrolyte imbalance
   i. Institute seizure precautions


**Diabetes (DM, DKA, HHNK)**

**Diabetic Ketoacidosis (DKA)**

1. **Presentation**
   a. Glucose
   b. Na, K
   c. Ketones, BUN/creatinine
   d. Serum osmolality
   e. Metabolic acidosis from ketosis
   f. WBC’s
   g. N/V, abdominal pain
   h. Polyphagia, polydipsia, polyuria
   i. Dehydration
   j. Tachycardia, orthostatic hypotension
   k. Kussmaul’s breathing
   l. Lethargy progressing to coma

2. **Treatment**
   a. Identify and treat cause
   b. Correct fluid volume deficit
   c. Normalize serum glucose
   d. Replace electrolytes
   e. Correct acid-base balance
   f. Maintain safety
   g. Treat infection

3. **Complications**
   a. CV
   b. Neurologic
   c. Renal
Hyperosmolar Hyperglycemic Syndrome (HHS)

1. Etiology
   a. Dehydration

2. Clinical Presentation
   a. Tachycardia, orthostatic hypotension, volume deficit, neurologic alterations
   b. Glucose >> 600-2000
   c. Na, K, Serum osmolality
   d. ABG’s: metabolic acidosis from hypotension

3. Treatment
   a. ABC’s
   b. Identify cause
   c. Correct fluid deficit
   d. Normalize serum glucose level
   e. Correct electrolyte imbalance
   f. Safety
   g. Monitor for complications

Hypoglycemia

1. Etiology
2. Mild to Moderate
3. Severe
4. Treatment: restore normal serum glucose

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Serum Sodium</th>
<th>Serum Osmolality</th>
<th>Urine Osmolality</th>
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<tbody>
<tr>
<td>SIADH</td>
<td>↓</td>
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<tr>
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<td>↑</td>
<td>↑</td>
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<tr>
<td>Diabetes Insipidus</td>
<td>↑</td>
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</tr>
</tbody>
</table>
Endocrine Pearls

SIADH = low sodium levels
   Fluid restrict

DI = neurological injury
   Volume replacement

Vasopressin = ADH = Pitressin

Normal serum osmolality = 275-295

Acidosis causes shift of cellular K to serum

Resources:

American Diabetes Association: www.diabetes.org
Endocrine Web: www.endocrineweb.com
Thyroid Today: www.thyroidtoday.com
**Professional Caring and Ethical Practice (20%) 30 questions**

1. Resiliency is the patient’s ability to:
   a. Avoid illness
   b. Adapt to his illness
   c. Accept his illness
   d. Recover from his illness

2. The extended family of a critically-ill patient wants to stay at his bedside around the clock. Hospital policy limits visiting times and number of visitors. The best response from the nurse is to:
   a. Explain the policy and ask them to leave
   b. Bring in cots and chairs for the family to stay
   c. Find a local hotel for the family
   d. Allow one or two family members to stay

3. In communicating with the family of a dying patient it is important to:
   a. Find little improvements to give them hope
   b. Provide accurate information
   c. Direct all questions back to the physician
   d. Reassure them that it is God’s will

4. Your patient is diagnosed with anoxic brain injury. The family overhears a physician stating that dialysis would improve the patient’s condition. Your best response to the family would be:
   a. Explain to them that this physician does not have all the facts
   b. Reinforce the diagnosis, and the limited value of dialysis
   c. Speak to the physician about his comments and the patient’s prognosis
   d. Ignore the concern and ask the family to sign a DNR form

5. Which statement best describes the nursing process?
   a. Assessment, planning, implementation, and evaluation
   b. Planning, implementation, and teaching
   c. Diagnosing and evaluation
   d. Charting and staffing

6. The most common cause for the patient to file a nursing negligence claim is:
   a. Medication errors
   b. Sloppy work by the nurse
   c. Ineffective communication
   d. Poor outcomes
7. Having a responsibility to a patient describes which essential element of litigation?
   a. Duty
   b. Negligence
   c. Causation
   d. Proximal cause

8. A patient can file a negligence claim if:
   a. They perceive a bad outcome as a result of care
   b. You actually made a mistake
   c. Your unit was understaffed
   d. They suffer from residual pain

9. In order to meet the standard of care required during your treatment of a patient, you must:
   a. Deliver exceptional care
   b. Use the most up-to-date equipment and treatments
   c. Act as a reasonable and prudent nurse would
   d. Meet all of the patient’s expectations

10. In error, you give your patient a medication that was meant for another patient. Your best response would be to:
    a. Ignore the error, it probably won’t hurt him
    b. Tell the physician, but not the patient
    c. Tell the patient about the error, chart it, and consult with the physician
    d. Call pharmacy and ask for an antidote

11. The role of the staff nurse in the research process is to:
    a. Test hypotheses
    b. Develop research questions
    c. Perform statistical analysis
    d. Test theories

12. A new research study shows that an intervention would help your critically-ill patient. The best action to take would be:
    a. Implement the strategy, even though it is contrary to hospital policy
    b. Ask the physician to order the intervention
    c. Request a policy change from administration
    d. Bring the study results to the attention of the physician and administration
13. If you feel that one of your hospital’s policies is outdated and ineffective, the best action to take is to:
   a. Complain loudly about it
   b. Learn how to navigate the system to change it
   c. Tell your patients about it
   d. Ridicule it publicly on an internet discussion group

14. If the critical-care nurse has questions about a patient’s response to therapy, it is his responsibility to:
   a. Seek the education to fully understand it
   b. Not let it bother him
   c. Ignore it, this is the physician’s realm
   d. Refuse to treat the patient

15. Mr. Squash has a subdural hematoma with increased intracranial pressure. He is very anxious and wants his wife to stay at the bedside. In order to decrease his stimuli and treat his increased intracranial pressure, the nurse should:
   a. Ask his wife to leave, so he can sleep
   b. Leave his wife at the bedside and decrease the room brightness
   c. Check his pupils often for changes
   d. Have his wife come in frequently for support

16. Ms. Regal was involved in a motor-vehicle accident (MVA) and is in critical condition. Her mother is at the bedside and is found applying a homeopathic cream to her forehead. Your best response would be:
   a. Immediately wash off the cream and ban the mother from unsupervised visits.
   b. Explain that homeopathic treatments are of limited value
   c. Obtain more information about the treatment
   d. Call security

17. During your admission assessment, you find that your patient takes the herbal preparation Ginseng daily. Your assessment should include:
   a. Assessing for hypotension
   b. Watching for bleeding
   c. Analyzing blood lipid levels
   d. Evaluating for depression

18. The physician orders prone positioning for a patient with ARDS. As his nurse you recognize that his safety will be maintained by:
   a. Assuring that adequate personnel are available to position the patient
   b. Disconnecting the ventilator during positioning
   c. Explaining the need for prone positioning to the family
   d. Preparing for CPR in the prone position
19. A nurse new to your unit is having trouble using your monitors. Your best response is to:
   a. Provide the operational manual for the monitor
   b. Assist her with the operation of the monitor
   c. Set up the monitor for her
   d. Answer her questions as she sets up the monitor

20. You are asked to float to a unit you are unfamiliar with. Your responsibility to that unit will be to:
   a. Provide care at the level of the regular employees on that unit
   b. Provide basic nursing care that is consistent with your licensure
   c. Provide care that is consistent with your units standards
   d. Provide only the care that you wish to

21. A float nurse is assigned to your unit. You can best support her by:
   a. Providing her with a brief orientation
   b. Telling her to call on you with any questions
   c. Giving her your policy manual
   d. Assigning her to the least acute patients

22. One of your colleagues is having difficulty with a patient’s family. As a professional nurse, you should:
   a. Offer to take the assignment
   b. Suggest active listening techniques
   c. Tell her to ignore the family
   d. Talk to the family yourself

23. Professional education and development is the responsibility of:
   a. Your hospital
   b. Your manager
   c. Your state nursing association
   d. Yourself

24. Members of the nursing staff are developing written patient education materials for a group of patients with diverse reading abilities. It would be most effective for the staff to:
   a. Design individual handouts for each patient
   b. Develop a computer-based education series.
   c. Write the materials at a fourth-grade reading level.
   d. Limit text and provide color pictures.
25. The best method for assuring patient compliance with changing negative health behaviors is to:
   a. Ask the patient which behaviors he would like to change
   b. Tell the patient which behaviors he needs to change
   c. Emphasize the dangers of negative health behaviors
   d. Provide written materials that tell him where to follow-up

26. The nursing staff is resisting being assigned to a disruptive patient. An appropriate resolution would be to:
   a. Request the physician to transfer the patient
   b. Rotate the patient assignment among staff.
   c. Confront the family and demand an end to the disruptive behavior.
   d. Hold a nursing team conference to discuss possible alternatives

27. A nurse who is able to synthesize multiple data sources and respond to a dynamic situation is at which level of professional practice?
   a. Novice
   b. Advanced beginner
   c. Expert
   d. Retired

28. Your patient’s family has requested to be present during CPR. Your best response is to:
   a. Let them stay if they are out of the way
   b. Explain that they have to leave for legal reasons
   c. Follow your hospital policy
   d. Call security

29. You learned about a new procedure at a nursing conference. The most effective method to assure its implementation at your hospital is to:
   a. Obtain references and present the information to hospital administration
   b. Tell your physicians that they are providing poor care and need to be updated
   c. Ask your administration to look into the subject
   d. Forget it, things will never change around here

30. The most important value of seeking certification is:
   a. To prove that you’re better than your co-workers
   b. The process leads to higher levels of professional conduct
   c. To validate your clinical skills
   d. You will be worth more to your hospital
Basic Information About the AACN Synergy Model for Patient Care

The core concept of the reconceptualized model of certified practice - the AACN Synergy Model for Patient Care - is that the needs or characteristics of patients and families influence and drive the characteristics or competencies of nurses.

All patients have similar needs and experience these needs across wide ranges or continuums from health to illness. Logically, the more compromised patients are, the more severe or complex are their needs. The dimensions of a nurse’s practice are driven by the needs of a patient and family. This requires nurses to be proficient in the multiple dimensions of the nursing continuums. When nurse competencies stem from patient needs and the characteristics of the nurse and patient synergize, optimal patient outcomes can result.

NOTE: The point of the Synergy Model, and its incorporation into the CCRN and CCNS exams, is not to have nurses memorize the various patient or nurse characteristics, or their levels; they are presented here to help you begin to comprehend the model. Test questions will not cover the terminology of the Synergy Model.

The Synergy Model was developed by the AACN Certification Corporation to link certified practice to patient outcomes. The fundamental premise of this model is that patient characteristics drive nurse competencies. When these characteristics and competencies are matched, optimal patient outcomes are realized. The integration of the Synergy Model into AACN CertCorp’s credentialing programs puts an emphasis on the patient, and says to the world that patients come first! Nurses make a unique contribution to the quality of patient care, containment of costs, and patient outcomes.

Patient Characteristics

Each patient and family is unique, with a varying capacity for health and vulnerability to illness. When seeking healthcare, each person brings a set of unique characteristics to the care situation. These patient characteristics span the continuum of health and illness:

Resiliency--the capacity to return to a restorative level of functioning using compensatory coping mechanisms; the ability to bounce back quickly after an insult.
- Level 1 - Minimally resilient - Unable to mount a response; failure of compensatory/coping mechanisms; minimal reserves; brittle
- Level 3 - Moderately resilient - Able to mount a moderate response; able to initiate some degree of compensation; moderate reserves
- Level 5 - Highly resilient - Able to mount and maintain a response; intact compensatory/coping mechanisms; strong reserves; endurance

Vulnerability--susceptibility to actual or potential stressors that may adversely affect patient outcomes.
- Level 1 - Highly vulnerable - Susceptible; unprotected, fragile
- Level 3 - Moderately vulnerable - Somewhat susceptible; somewhat protected
- Level 5 - Minimally vulnerable - Safe; out of the woods; protected, not fragile

Stability--the ability to maintain a steady-state equilibrium.
- Level 1 - Minimally stable - Labile; unstable; unresponsive to therapies; high risk of death
- Level 3 - Moderately stable - Able to maintain steady state for limited period of time; some responsiveness to therapies
- Level 5 - Highly stable - Constant; responsive to therapies; low risk of death

Complexity--the intricate entanglement of two or more systems (e.g., body, family, therapies).
- Level 1 - Highly complex - Intricate; complex patient/family dynamics; ambiguous/vague;
atypical presentation
Level 3 - Moderately complex - Moderately involved patient/family dynamics
Level 5 - Minimally complex - Straightforward; routine patient/family dynamics; simple/clear cut; typical presentation

**Resource availability**—extent of resources (e.g., technical, fiscal, personal, psychological, social) the patient, family and community bring to the situation.
Level 1 - Few resources - Necessary knowledge and skills not available; necessary financial support not available; minimal personal/psychological supportive resources; few social systems resources
Level 3 - Moderate resources - Limited knowledge and skills available; limited financial support available; limited personal/psychological supportive resources; limited social systems resources
Level 5 - Many resources - Extensive knowledge and skills available and accessible; financial resources readily available; strong personal/psychological supportive resources; strong social systems resources

**Participation in care**—extent to which the patient and family engage in aspects of care.
Level 1 - No participation - Patient and family unable or unwilling to participate in care
Level 3 - Moderate level of participation - Patient and family need assistance in care
Level 5 - Full participation - Patient and family fully able to participate in care

**Participation in decision-making**—extent to which the patient and family engage in decision-making.
Level 1 - No participation - Patient and family have no capacity for decision-making; requires surrogacy
Level 3 - Moderate level of participation - Patient and family have limited capacity; seeks input/advice from others in decision-making
Level 5 - Full participation - Patient and family have capacity, and makes decision for self

**Predictability**—a summative characteristic that allows one to expect a certain trajectory of illness.
Level 1 - Not predictable - Uncertain; uncommon patient population/illness; unusual or unexpected course; does not follow critical pathway, or no critical pathway developed
Level 3 - Moderately predictable - Wavering; occasionally-noted patient population/illness
Level 5 - Highly predictable - Certain; common patient population/illness; usual and expected course; follows critical pathway

For example:

A healthy, uninsured, 40-year-old woman undergoing a pre-employment physical could be described as an individual who is (a) stable (b) not complex (c) very predictable (d) resilient (e) not vulnerable (f) able to participate in decision-making and care, but (g) has inadequate resource availability.

On the other hand: a critically ill infant with multisystem organ failure can be described as an individual who is (a) unstable (b) highly complex (c) unpredictable (d) highly resilient (e) vulnerable (f) unable to become involved in decision-making and care, but (g) has adequate resource availability

**Nurse Characteristics**

Nursing care reflects an integration of knowledge, skills, experience, and attitudes needed to
meet the needs of patients and families. Thus, continuums of nurse characteristics are derived from patient needs. The following are levels of expertise ranging from competent (1) to expert (5):

**Clinical judgment**—clinical reasoning, which includes clinical decision-making, critical thinking, and a global grasp of the situation, coupled with nursing skills acquired through a process of integrating formal and experiential knowledge.

**Level 1** - Collects basic-level data; follows algorithms, decision trees, and protocols with all populations and is uncomfortable deviating from them; matches formal knowledge with clinical events to make decisions; questions the limits of one's ability to make clinical decisions and delegates the decision-making to other clinicians; includes extraneous detail

**Level 3** - Collects and interprets complex patient data; makes clinical judgments based on an immediate grasp of the whole picture for common or routine patient populations; recognizes patterns and trends that may predict the direction of illness; recognizes limits and seeks appropriate help; focuses on key elements of case, while shorting out extraneous details

**Level 5** - Synthesizes and interprets multiple, sometimes conflicting, sources of data; makes judgment based on an immediate grasp of the whole picture, unless working with new patient populations; uses past experiences to anticipate problems; helps patient and family see the "big picture;" recognizes the limits of clinical judgment and seeks multi-disciplinary collaboration and consultation with comfort; recognizes and responds to the dynamic situation

**Advocacy/moral agency**—working on another's behalf and representing the concerns of the patient, family, and community; serving as a moral agent in identifying and helping to resolve ethical and clinical concerns within the clinical setting.

**Level 1** - Works on behalf of patient; self assesses personal values; aware of ethical conflicts/issues that may surface in clinical setting; makes ethical/moral decisions based on rules; represents patient when patient cannot represent self; aware of patients' rights

**Level 3** - Works on behalf of patient and family; considers patient values and incorporates in care, even when differing from personal values; supports colleagues in ethical and clinical issues; moral decision-making can deviate from rules; demonstrates give and take with patient's family, allowing them to speak/represent themselves when possible; aware of patient and family rights

**Level 5** - Works on behalf of patient, family, and community; advocates from patient/family perspective, whether similar to or different from personal values; advocates ethical conflict and issues from patient/family perspective; suspends rules - patient and family drive moral decision-making; empowers the patient and family to speak for/represent themselves; achieves mutuality within patient/professional relationships

**Caring practices**—the constellation of nursing activities that are responsive to the uniqueness of the patient and family and that create a compassionate and therapeutic environment, with the aim of promoting comfort and preventing suffering. These caring behaviors include, but are not limited to, vigilance, engagement, and responsiveness.

**Level 1** - Focuses on the usual and customary needs of the patient; no anticipation of future needs; bases care on standards and protocols; maintains a safe physical environment; acknowledges death as a potential outcome

**Level 3** - Responds to subtle patient and family changes; engages with the patient as a unique patient in a compassionate manner; recognizes and tailors caring practices to the individuality of patient and family; domesticates the patient's and family's environment; recognizes that death may be an acceptable outcome

**Level 5** - Has astute awareness and anticipates patient and family changes and needs; fully engaged with and sensing how to stand alongside the patient, family, and community; caring practices follow the patient and family lead; anticipates hazards and avoids them, and promotes safety throughout patient's and family's transitions along the
Collaboration--working with others (e.g., patients, families, healthcare providers) in a way that promotes and encourages each person's contributions toward achieving optimal and realistic patient goals. Collaboration involves intra- and interdisciplinary work with all colleagues.

**Level 1** - Willing to be taught, coached and/or mentored; participates in team meetings and discussions regarding patient care and/or practice issues; open to various team members' contributions

**Level 3** - Seeks opportunities to be taught, coached, and/or mentored; elicits others’ advice and perspectives; initiates and participates in team meetings and discussions regarding patient care and/or practice issues; recognizes and suggests various team members' participation

**Level 5** - Seeks opportunities to teach, coach, and mentor and to be taught, coached and mentored; facilitates active involvement and complementary contributions of others in team meetings and discussions regarding patient care and/or practice issues; involves/recruits diverse resources when appropriate to optimize patient outcomes

**Systems thinking**--the body of knowledge and tools that allow the nurse to appreciate the care environment from a perspective that recognizes the holistic interrelationship that exists within and across healthcare systems.

**Level 1** - Uses a limited array of strategies; limited outlook - sees the pieces or components; does not recognize negotiation as an alternative; sees patient and family within the isolated environment of the unit; sees self as key resource

**Level 3** - Develops strategies based on needs and strengths of patient/family; able to make connections within components; sees opportunity to negotiate but may not have strategies; developing a view of the patient/family transition process; recognizes how to obtain resources beyond self

**Level 5** - Develops, integrates, and applies a variety of strategies that are driven by the needs and strengths of the patient/family; global or holistic outlook - sees the whole rather than the pieces; knows when and how to negotiate and navigate through the system on behalf of patients and families; anticipates needs of patients and families as they move through the healthcare system; utilizes untapped and alternative resources as necessary

**Response to diversity**--the sensitivity to recognize, appreciate, and incorporate differences into the provision of care. Differences may include, but are not limited to, individuality, cultural differences (e.g., in child rearing, family relations), spiritual beliefs, gender, race, ethnicity, disability, family configuration, lifestyle, socioeconomic status, age values, and alternative medicine involving patients and their families and members of the healthcare team.

**Level 1** - Assesses cultural diversity; provides care based on own belief system; learns the culture of the healthcare environment

**Level 3** - Inquires about cultural differences and considers their impact on care; accommodates personal and professional differences in the plan of care; helps patient/family understand the culture of the healthcare system

**Level 5** - Responds to, anticipates, and integrates cultural differences into patient/family care; appreciates and incorporates differences, including alternative therapies, into care; tailors healthcare culture, to the extent possible, to meet the diverse needs and strengths of the patient/family

**Clinical inquiry or Innovator/Evaluator**--the ongoing process of questioning and evaluating practice, providing informed practice, and innovating through research and
experiential learning. The nurse engages in clinical knowledge development to promote the best patient outcomes.

**Level 1 -** Follows standards and guidelines; implements clinical changes and research-based practices developed by others; recognizes the need for further learning to improve patient care; recognizes obvious changing patient situation (e.g., deterioration, crisis); needs and seeks help to identify patient problem

**Level 3 -** Questions appropriateness of policies and guidelines; questions current practice; seeks advice, resources, or information to improve patient care; begins to compare and contrast possible alternatives

**Level 5 -** Improves, deviates from, or individualizes standards and guidelines for particular patient situations or populations; questions and/or evaluates current practice based on patients’ responses, review of the literature, research and education/learning; acquires knowledge and skills needed to address questions arising in practice and improve patient care; (The domains of clinical judgment and clinical inquiry converge at the expert level; they cannot be separated)

**Facilitator of learning of patient/family educator**—the ability to facilitate patient and family learning.

**Level 1 -** Follows planned educational programs; sees patient/family education as a separate task from delivery of care; provides data without seeking to assess patient's readiness or understanding; has limited knowledge of the totality of the educational needs; focuses on a nurse's perspective; sees the patient as a passive recipient

**Level 3 -** Adapts planned educational programs; begins to recognize and integrate different ways of teaching into delivery of care; incorporates patient's understanding into practice; sees the overlapping of educational plans from different healthcare providers' perspectives; begins to see the patient as having input into goals; begins to see individualism

**Level 5 -** Creatively modifies or develops patient/family education programs; integrates patient/family education throughout delivery of care; evaluates patient's understanding by observing behavior changes related to learning; is able to collaborate and incorporate all healthcare providers' and educational plans into the patient/family educational program; sets patient-driven goals for education; sees patient/family as having choices and consequences that are negotiated in relation to education

From the AACN Cert Corp website: [www.certcorp.org](http://www.certcorp.org) © 2004 AACN
References:


Resources:


Certification Exam Planner

- Read the question carefully
- If the most logical answer is readily apparent, choose it
- If not, re-read the question and start eliminating obviously wrong answers
- Then narrow the remainder down to what makes the most sense

You will have 1 minute, and 12 seconds for each question, use that time wisely.

Your action plan:

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<tr>
<th>Action</th>
<th>Started</th>
<th>Completed</th>
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</thead>
<tbody>
<tr>
<td>Decide which test to take</td>
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<td></td>
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<tr>
<td>When?</td>
<td></td>
<td></td>
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<tr>
<td>Register</td>
<td></td>
<td></td>
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<tr>
<td>Request time off</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Get study materials</td>
<td></td>
<td></td>
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<tr>
<td>Emergency planning</td>
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<tr>
<td>Study guide #1</td>
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<td>Study guide #2</td>
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<tr>
<td>Study guide #3</td>
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</tbody>
</table>

Areas to study:

Where will you study?

When will you study?

What study aids do you plan to get?

Where will you get them?

How will you test your progress?
Planning:

<table>
<thead>
<tr>
<th>Question</th>
<th>Plan</th>
</tr>
</thead>
<tbody>
<tr>
<td>Who will cover on-call/emergencies?</td>
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<tr>
<td>Who will work the night before the test?</td>
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<tr>
<td>Who will manage the kids/pets?</td>
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<tr>
<td>When will you shop for healthy foods?</td>
<td></td>
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<tr>
<td>Who will you get to care for ill kids, pets, or husbands/wives?</td>
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<tr>
<td>What will you do if the car doesn’t start?</td>
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<tr>
<td>What if you get a flat tire?</td>
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<tr>
<td>What will you do if traffic is bad?</td>
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<tr>
<td>What alternate routes are available to the testing site?</td>
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<tr>
<td>When do you need to go to bed the night before?</td>
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<tr>
<td>What will you eat the morning of the exam?</td>
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</tr>
<tr>
<td>What content will you study the night before the exam?</td>
<td></td>
</tr>
<tr>
<td>Will you need a hotel room the night before the exam?</td>
<td></td>
</tr>
<tr>
<td>How will you pace yourself during the exam?</td>
<td></td>
</tr>
<tr>
<td>How will you reward yourself for preparing and taking the exam?</td>
<td></td>
</tr>
</tbody>
</table>

Cramming:
The night before the exam it is OK to study subjects that need memorization, or to briefly review your notes. Don’t start a new topic or study difficult content. It is generally not a good idea to study the day of the exam.

Relaxation Tips the Day of the Exam:
- Slow, deep breathing is relaxing and restores oxygen to the brain.
- Gentle stretching or walking stimulates circulation and increases oxygen delivery to the brain.
- Listen to music that you like
- Avoid ingesting alcohol, cold medications, or unusual amounts of caffeine.
- Proper preparation will clear your mind of unnecessary details the day of the exam!

Find more certification resources at:
www.ed4nurses.com/certification.htm
Thanks for attending “CCRN: Test Prep”!

Additional resources are available from Ed4Nurses, Inc. that will help you prepare for the exam:

**The Critical Care Skills Package**

High-acuity patients often bring along critical care equipment like ventilators, central lines, pacemakers and chest tubes. Proper management of these patients is vital to prevent complications.

**The Critical Care Essentials Package**

Critically ill patients are everywhere these days – on the med-surg floor, in the ICU, the PACU, the ED, even long-term care! A good working knowledge of these essential concepts is indispensable.

**The Critical Care Mastery Package**

Critical Care Mastery will give you a strong foundation, while integrating tips, timesavers, and stories about real nurses who make a difference in their patient’s lives.