PCCN Test Prep

www.100K-Certified-Nurses.com

Presented by:
David W. Woodruff, MSN, RN, CNS

Ed4Nurses.com
We will guarantee your success on the PCCN certification exam!  
-- If you study the right things in the right ways --

Success Checklist:

☐ Attend the entire PCCN: 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.

☐ Use additional study guides for clarification.

☐ Identify areas of weakness that need additional study.

☐ Review the audio CDs (or online videos) 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: pccn
   Password: skill

☐ 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 PCCN: 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
PCCN: Test Prep

Description:
This unique two-day program presents the content of the PCCN exam in a question and answer format. By the conclusion of the program the participant will have answered 125 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 PCCN preparation, or a comprehensive progressive care review.

Objectives:
1. Examine strategies for successful completion of the PCCN exam.
2. Plan care for patients with cardiopulmonary disorders.
3. Compare and contrast septic, hypovolemic, and cardiogenic shock.
4. Describe hemodynamic changes that occur with shock.
5. Explain the benefits of several treatment options for acute respiratory failure.
7. Describe common endocrine dysfunctions in the critical care patient.
8. Compare and contrast diabetic ketoacidosis and hyperosmolar, hyperglycemic syndrome.
9. Describe common hematologic and immunologic dysfunction in the progressive care patient.
10. Describe the process of coagulopathy in DIC.
11. Describe a simple assessment plan for patients with increased intracranial pressure.
12. Evaluate nursing interventions for increased intracranial pressure.
13. Compare and contrast common GI disorders.
15. Describe clinical symptoms of electrolyte disturbances.
17. Identify common etiologies for systemic inflammatory response.
18. Describe the process of initiation, systemic inflammation and multiple organ failure.
20. Define professional and ethical nursing care using AACN definitions.
Day 1

8:00  Introduction and Test Overview
     Professional Caring and Ethical Practice
     Synergy Model
8:30  Cardiovascular (37%)
     Acute Coronary Syndromes
     Acute Heart Failure & Pulmonary Edema
     Acute Peripheral Vascular Insufficiency
     Cardiac Surgery
     Cardiac Inflammatory Disease
     Conduction System Defects
10:00 Break
10:15 Cardiovascular (continued)
     Aortic Aneurysm
     Pericarditis
     Cardiac Trauma
     Hypertensive Crisis
     Shock
11:45 Lunch
12:45 Pulmonary (13%)
     Pulmonary embolism / fat embolism
     ARDS
     Pneumonia
     Aspiration
14:15 Break
14:30 Non-invasive Positive-pressure Ventilation
     Chronic Lung Disease
     Pulmonary Hypertension
     Obstructive Disease (COPD, Asthma)
     Thoracic Surgery
16:00 Adjourn
Day 2

8:00  Endocrine (4%)
      Acute Hypoglycemia
      Diabetes Insipidus
      Diabetes Ketoacidosis & Hyperosmolar Coma
      Adrenal Insufficiency

9:00  Hematologic / Immunologic (5%)
      Immunosuppression-Acquired
      Life-threatening Coagulopathies
      Organ Transplantation
      Sickle Cell Crisis
10:00 Break
10:15 Neurologic (4%)
      Intracranial Hemorrhage
      Seizures
      Stroke (ischemic, hemorrhagic)

11:45 Lunch
12:45 Gastrointestinal (5%)
      GI Bleed / GERD
      Bowel infarction/obstruction/perforation
      GI Surgeries
      Acute Pancreatitis
13:45 Renal (6%)
      Acute & Chronic Renal Failure
      Electrolyte Imbalances

14:15 Break
14:30 Renal (con’t)
      Electrolyte Imbalances
15:00 Multisystem (6%)
      Trauma
      Sepsis / Septic Shock / MODS
      Toxic Ingestions
      Toxic Exposures
16:00 Adjourn
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 “PCCN”?

   a. Registered service mark of AACN.
   b. Credential for certified progressive care nurses.

3. What to Expect from “The Test”

<table>
<thead>
<tr>
<th>AACN – Certification Corporation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fees: $250 non-member</td>
</tr>
<tr>
<td>$170 member of AACN</td>
</tr>
<tr>
<td>Test dates: Year-round</td>
</tr>
<tr>
<td>Requirements: RN license</td>
</tr>
<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 $248 and get member benefits.

Exam is computer-based, 125 questions, with a 2 ½ -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: $215 non-member, $135 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

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

You can do this!
✓ If you are qualified
✓ And you study the right stuff in the right way
✓ You will pass!

I guarantee it!
Professional Caring and Ethical Practice (20%) 25 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. 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

3. 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

4. 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

5. Having a responsibility to a patient describes which essential element of litigation?
   a. Duty
   b. Negligence
   c. Causation
   d. Proximal cause

6. 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
7. 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

8. 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

9. A new research study shows that an intervention would help your 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

10. 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

11. If the progressive-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

12. 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
13. Ms. Regal was involved in a motor-vehicle accident (MVA) and is in fair 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

14. 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

15. 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

16. 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

17. 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

18. 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
19. 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.

20. 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

21. 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

22. 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

23. 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

24. 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
25. 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
healthcare continuum; orchestrates the process that ensures patient's/family's comfort and concerns surrounding issues of death and dying are met

**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
Cardiovascular (37%) 46 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. Which coronary artery supplies the atroventricular (AV) node?
   a. Right coronary artery  
   b. Coronary sinus artery  
   c. Left anterior descending artery  
   d. Nodal artery  

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

17. 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  

18. 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 ECG’s  

19. Your patient, Ms. Smith, is complaining of palpitations. Her telemetry monitor shows the following rhythm. What initial treatment is necessary?

   ![ECG rhythm image]

   a. Administer adenosine  
   b. Immediate defibrillation  
   c. Treat fever, pain, or dehydration  
   d. Perform valsalva maneuver  

20. 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
21. The most common complication of an acute myocardial infarction is:
   a. Dysrhythmia
   b. Congestive heart failure
   c. Cardiogenic shock
   d. Pulmonary embolism

22. The most common cause for the development of a new S₃ heart sound is:
   a. Cardiac tamponade
   b. Papillary muscle rupture
   c. Congestive heart failure
   d. Myocardial infarction

23. 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

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

25. 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

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

27. 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
28. You are caring for a patient recently admitted with and 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 V₁

29. What additional diagnostic test must be done on all patients with an inferior-wall MI?
   a. Pulmonary function tests
   b. Right-sided EKG
   c. Endoscopy
   d. Continuous ST-segment monitoring

30. Oxygen therapy is recommended for all patients with Acute Coronary Syndrome for the first 6 hours after they become stable. The goal of oxygen therapy in ACS is to:
   a. Keep oxygen saturation at 100%
   b. Balance oxygen supply and demand
   c. Reduce workload on the heart
   d. Prevent pulmonary edema

31. Which reperfusion therapy is recommended as the initial treatment for patients with STEMI and a history of intracerebral bleeding?
   a. Thrombolytics (tPA)
   b. Percutaneous coronary interventions (PCI)
   c. Coronary artery bypass grafting (CABG)
   d. Automatic implantable cardiodefibrillator (AICD)

32. A sign of a peripheral arterial occlusion is:
   a. Pallor
   b. Swelling
   c. Redness
   d. Dyspnea

33. 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
34. 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

35. 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

36. 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

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

38. The valve most often affected by infective endocarditis is:
   a. Mitral
   b. Aortic
   c. Tricuspid
   d. Pulmonary

39. 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

40. 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
41. 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

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

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

44. 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

45. Hypertensive crisis is identified by a diastolic blood pressure greater than 120 mmHg. The best medication for initial treatment of hypertensive crisis is:
   a. Nitroprusside
   b. Apresoline
   c. Vasotec
   d. Brevibloc

46. The treatment priority for the rhythm below is:
   a. Immediate electrical conversion
   b. Lidocaine bolus and infusion
   c. Epinephrine infusion
   d. Cardiopulmonary resuscitation
Cardiovascular

The Cardiac Cycle: Flow and Assessment
**Electrocardiogram (ECG)**

1. Arrhythmias  
   a. Normal Sinus Rhythm (NSR)  
      i. Characteristics  
         1. Rate: 60-100  
         2. Regular  
         3. P-wave precedes QRS  
         4. Narrow complex  
         5. All complexes look the same  
      ii. Treatment  
         1. None necessary

b. Sinus Tachycardia (ST)  
   i. Characteristics  
      1. Rate: >100  
      2. Regular  
      3. P-wave precedes QRS  
      4. Narrow complex  
      5. All complexes look the same  
   ii. Treatment  
      1. Treat the underlying condition  
         a. Fever  
         b. Anxiety  
         c. Pain  
         d. Dehydration  
         e. Hypoxemia

c. Sinus Bradycardia (SB)  
   i. Characteristics  
      1. Rate: <60  
      2. Regular  
      3. P-wave precedes QRS  
      4. Narrow complex  
      5. All complexes look the same  
   ii. Treatment  
      1. If symptomatic:  
         a. Atropine, epinephrine  
         b. Pacer
d. Atrial Flutter
   i. Characteristics
      1. Rate: 60-150
      2. Regular
      3. Several F-waves precede QRS
      4. Narrow complex
      5. All complexes look the same
   ii. Treatment
      1. Slow rate: Digoxin, Verapamil, Diltiazem
      2. Convert: Amiodarone, sotalol, flecainide

e. Atrial Fibrillation (A-fib)
   i. Characteristics
      1. Rate: varies
      2. Irregular
      3. Several f-waves precede QRS
      4. Narrow complex
      5. All complexes look the same
   ii. Treatment
      1. Cardioversion
      2. Slow rate: Digoxin, Verapamil, Diltiazem
      3. Convert: Amiodarone, sotalol, flecainide
      4. Anticoagulation

f. Paroxysmal Atrial Tachycardia (PAT)
   i. Characteristics
      1. Rate: 140-250
      2. Regular
      3. P-wave precedes QRS
      4. Narrow complex
      5. All complexes look the same
   ii. Treatment
      1. Vagal maneuvers
      2. Adenosine, beta-blockers, verapamil
      3. Cardioversion
g. Junctional Rhythm
   i. Characteristics
      1. Rate: 40-60 (accelerated 60-100)
      2. Regular
      3. P-wave absent or inverted
      4. Narrow complex
      5. All complexes look the same
   ii. Treatment
      1. If symptomatic:
         a. Atropine
         b. Pacer

h. First-degree AV Block
   i. Characteristics
      1. Rate: 60-100
      2. Regular
      3. P-wave precedes QRS: long PR-interval
      4. Narrow complex
      5. All complexes look the same
   ii. Treatment
      1. None necessary
      2. Watch for progression to greater block

i. Second-degree AV Block
   i. Characteristics
      1. Rate: varies
      2. Irregular
      3. P-wave precedes QRS
      4. Narrow complex, but some dropped
      5. All complexes look the same
   ii. Treatment
      1. D/C digoxin
      2. Pacer

j. Second-degree AV Block (Wenckebach)
   i. Characteristics
      1. Rate: varies
      2. Irregular
      3. P-wave precedes QRS: PR-interval becomes progressively longer, until a QRS is dropped.
      4. Narrow complex
      5. All complexes look the same
   ii. Treatment
      1. D/C digoxin
      2. If symptomatic:
         a. Atropine
b. Pacer

k. Third-degree AV Block
   i. Characteristics
      1. Rate: <60
      2. Regular
      3. No coordination between P-wave and QRS
      4. Narrow complex
      5. All complexes look the same
   ii. Treatment
      1. Atropine
      2. Pacer
      3. ACLS

l. Ventricular Tachycardia (V-tach)
   i. Characteristics
      1. Rate: 100-220
      2. Regular
      3. No P-waves
      4. Wide complex
      5. All complexes look the same
   ii. Treatment
      1. With pulse:
         a. Stable:
            i. Lidocaine
            ii. Amiodarone
            iii. Procainamide
            iv. Sotalol
         b. Unstable:
            i. Cardioversion
            ii. Lidocaine, procainamide
      2. Pulseless:
         a. Defibrillate
         b. CPR
         c. Epinephrine

m. Ventricular Fibrillation (V-fib)
   i. Characteristics
      1. Rate: none detectable
      2. Irregular
      3. No P-wave or QRS
      4. Wide, bizarre, chaotic complexes
      5. Complexes look different
   ii. Treatment
      1. Defibrillation
      2. CPR
3. Epinephrine
4. Lidocaine, amiodarone, procainamide, magnesium

n. Asystole
   i. Characteristics
      1. Rate: none
      2. Flat line
      3. P-waves may be present
      4. QRS complexes absent
      5. No electrical or mechanical activity
   ii. Treatment
      1. CPR
      2. Epinephrine, atropine
      3. Pacer

o. Pulseless Electrical Activity (PEA)
   i. Characteristics
      1. Rate: varies
      2. May be regular or irregular
      3. P-waves may be present
      4. QRS complex present
      5. No detectable pulse or B/P with electrical activity
   ii. Treatment
      1. CPR
      2. Epinephrine
      3. Atropine
      4. Correct mechanical cause

Resources:
Internet:
The EKG Site: www.the-ekg-site.com
EKGs Online: www.ekgonline.com
American Heart Association: www.americanheart.org
Heart Failure Society of America: www.hfsa.org
5 Steps to 12-Lead Interpretation

1. Assess regularity and speed
2. Look for signs of infarction
3. Present in >1 lead, but not all?
4. Assess associated conditions
5. Correlate with clinical condition

1. 12-Lead EKG
   a. Best evidence in first 3 hours after symptom presentation
   b. Normal EKG is associated with ↓ risk
   c. Poor sensitivity for MI (40-50%)

2. Changes in Acute MI
   a. LBBB or left ventricular strain
   b. Hyperacute T-waves
   c. T-wave inversion
   d. ST-segment elevation: (GUSSI-1 criteria)
      i. > 1mm in limb leads
      ii. >2 mm in precordial leads
   e. Q-waves: .04 sec and ½ the height of the R-wave

3. S-T analysis
   a. Measure at 0.04 sec. after the J-point
   b. Compare to baseline
   c. S-T segment elevation and peaked T-waves over ischemic zone
   d. Other leads may have reciprocal S-T segment depression

Reciprocal changes occur when we are looking through the myocardium to see the problem. For example, if a patient has a posterior wall MI, reciprocal changes will be seen in the anterior leads (looking through the front to see the back). Reciprocal changes are mirror-image or opposite changes. Instead of ST-segment elevation, we get ST-segment depression.
Localizing the injury

a. Anterior MI (LCD, LAD) V3, V4
   i. Associated symptoms:
      1. Sinus tachycardia
      2. SA blocks
      3. Pulmonary edema
   ii. Complications:
      1. High risk of sudden death
      2. 2° & 3° AV-block
      3. BBB
      4. Ventricular aneurysm
      5. Outflow obstruction
      6. Heart failure

b. Inferior MI (RCA) II, III, aVF
   i. Associated symptoms:
      7. Bradycardia
      8. Hypotension
   ii. Complications:
      1. 1° AV-block
      2. Papillary muscle rupture
      3. Mitral insufficiency
      4. RV infarct
c. Lateral (LCA, Circumflex) V5, V6, I, aVL
   i. Associated symptoms:
      1. Ventricular dysrhythmias
   ii. Complications:
      1. Transient AV-blocks
      2. Heart failure
      3. Late aneurysm

d. Septal V1, V2
   i. Associated symptoms:
      1. Sinus tachycardia
      2. Atrial fibrillation
   ii. Complications:
      3. Septal rupture

e. Posterior (LCA, Circumflex, RCA)
   i. Pathologic R-waves V1 – V4
   ii. ST depression in V1, V2
   iii. Associated symptoms:
      1. Sinus bradycardia
      2. Junctional rhythms
   iv. Complications:
      3. Mitral insufficiency
      4. Heart failure
f. RV Infarct
   i. Suspect with Inferior MI
   ii. Incidence is about 40% in IWMI
   iii. V4R
   iv. Associated symptoms:
       1. Hypotension
       2. JVD
       3. Clear lungs
   v. Complications:
       1. Right-sided heart failure
       2. Atrial fibrillation
       3. Tricuspid insufficiency

**Pattern of EKG Changes in MI**

<table>
<thead>
<tr>
<th></th>
<th>I</th>
<th>AVR</th>
<th>V1</th>
<th>V4</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>II</td>
<td>AVL</td>
<td>V2</td>
<td>V5</td>
</tr>
<tr>
<td>III</td>
<td>AVF</td>
<td>V3</td>
<td>V6</td>
<td></td>
</tr>
</tbody>
</table>

- Inferior
- Lateral
- Septal
- Anterior

www.Ed4Nurses.com
Preload: I & O, Weight, JVD

Afterload: Diastolic B/P, Skin perfusion

Cardiac Output: Systolic B/P, Organ perfusion, Skin perfusion

70%
Hemodynamic Assessment

<table>
<thead>
<tr>
<th>Component</th>
<th>Factors affecting</th>
<th>Assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preload</td>
<td>Fluid volume</td>
<td>I &amp; O</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Weight</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Jugular veins</td>
</tr>
<tr>
<td>Contractility</td>
<td>Fluid volume</td>
<td>Heart rate</td>
</tr>
<tr>
<td></td>
<td>Oxygenation</td>
<td>Systolic blood pressure</td>
</tr>
<tr>
<td></td>
<td>Resistance</td>
<td>Organ perfusion:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>-Brain</td>
</tr>
<tr>
<td></td>
<td></td>
<td>-Kidneys</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Peripheral perfusion:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>-Capillary refill</td>
</tr>
<tr>
<td></td>
<td></td>
<td>-Pulses</td>
</tr>
<tr>
<td>Afterload</td>
<td>Arterial vascular resistance</td>
<td>Diastolic blood pressure</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Skin temperature and color</td>
</tr>
</tbody>
</table>
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
   ii. Interventional
      1. Pre-procedure
      2. Post-procedure
         a. 6 P’s of peripheral vascular assessment
Acute Myocardial Infarction

1. Clinical Presentation
   a. ECG Changes
      i. 12-lead within 10 minutes of symptom presentation
      ii. Repeat q5-10 minutes, or continuous ST-segment monitoring
   b. Enzymes
      i. CPK-MB
      ii. Troponin
      iii. Myoglobin

2. Manage and Monitor
   a. Reduce size of infarction
   b. Door to diagnosis and treatment
   c. Treatment Paradigm
      i. Oxygen
         1. First 6 hours
         2. Maintain O2 sat of >90%
      ii. Pain management
         1. Nitroglycerine: PO, IV if pain persists / hypertensive
         2. MSO4:
            a. 2-4 mg IV, q 5-15 minutes
      iii. Reperfusion therapies
         1. Thrombolytics (tPA)
         2. Percutaneous coronary intervention (PCI)
         3. CABG
      iv. Increase Myocardial Oxygen Supply
      v. Decrease Myocardial Oxygen Demand

    → Plug in the Pump! ←

   d. ACC/AHA guidelines
      i. Reperfusion Therapy
      ii. ASA:
         1. Initial dose: 162-325mg (chewed)
         2. Maintenance: 75-162mg daily
      iii. ACEi / ACB
      iv. Beta Blocker
      v. Calcium-channel blocker if B-blocker is contraindicated
      vi. Normalize glucose (insulin drip for hyperglycemia)
      vii. Monitor appropriate leads to detect recurrent ischemia
     viii. Anxiolytics
     ix. Statins
     x. Smoking cessation
     xi. Exercise
     xii. Weight reduction / diet
e. 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
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

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
Pulmonary (13%) 16 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. 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

5. 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

6. 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
7. The most common etiology for Acute Respiratory Distress Syndrome (ARDS) is:
   a. Sepsis
   b. Multiple trauma
   c. Pancreatitis
   d. Shock

8. 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

9. 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

10. Appropriate interventions for the patient with hospital-acquired pneumonia include:
    a. Assure adequate fluid intake, endotrachael suction q 1-hour
    b. Antibiotics, percussion and vibration
    c. Turning, positioning, and ambulation
    d. Saline lavage, mechanical ventilation

11. 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

12. 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

13. 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
14. 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

15. 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

16. Evaluate the following ABG: pH 7.32, CO2 55, O2 125, HCO3 22, O2 Sat 94%
   a. Uncompensated metabolic acidosis
   b. Uncompensated respiratory acidosis
   c. Compensated respiratory acidosis
   d. Compensated metabolic alkalosis
Troubleshooting CPAP & BiPAP

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
6-easy steps to ABG analysis

1. Is the pH normal?
2. Is the CO2 normal?
3. Is the HCO3 normal?
4. Match the CO2 or the HCO3 with the pH
5. Does the CO2 or the HCO3 go the opposite direction of the pH?
6. Are the pO2 and the O2 saturation normal?

In order for our analysis to be effective, notes will have to be written next to the results on our lab slip. Alternately, the ABG results can be transcribed onto another paper for analysis (see example one below).
1. The first step in analyzing ABGs is to look at the pH. Normal blood pH is 7.4, plus or minus 0.05, forming the range 7.35 to 7.45. If blood pH falls below 7.35 it is acidotic. If blood pH raises above 7.45, it is alkalotic. If it falls into the normal range, label what side of 7.4 it falls on. Lower than 7.4 is normal/acidotic, higher than 7.4 is normal/alkalotic. Label it.
2. The second step is to examine the pCO2. Normal pCO2 levels are 35-45mmHg. Below 35 is alkalotic, above 45 is acidotic. Label it.
3. The third step is to look at the HCO3 level. A normal HCO3 level is 22-26 mEq/L. If the HCO3 is below 22, the patient is acidotic. If the HCO3 is above 26, the patient is alkalotic. Label it.
4. Next match either the pCO2 or the HCO3 with the pH to determine the acid-base disorder. For example, if the pH is acidotic, and the CO2 is acidotic, then the acid-base disturbance is being caused by the respiratory system. Therefore, we call it a respiratory acidosis. However, if the pH is alkalotic and the HCO3 is alkalotic, the acid-base disturbance is being caused by the metabolic (or renal) system. Therefore, it will be a metabolic alkalosis.
5. Fifth, does either the CO2 or HCO3 go in the opposite direction of the pH? If so, there is compensation by that system. For example, the pH is acidotic, the CO2 is acidotic, and the HCO3 is alkalotic. The CO2 matches the pH making the primary acid-base disorder respiratory acidosis. The HCO3 is opposite of the pH and would be evidence of compensation from the metabolic system.
6. Finally, evaluate the PaO2 and O2 sat. If they are below normal there is evidence of hypoxemia.

Normal Arterial Values (At sea level):

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.35-7.45</td>
</tr>
<tr>
<td>pCO2</td>
<td>35-45 mmHg</td>
</tr>
<tr>
<td>pO2</td>
<td>80-100 mmHg</td>
</tr>
<tr>
<td>O2 Saturation</td>
<td>95-100%</td>
</tr>
<tr>
<td>HCO3-</td>
<td>22-26 mEq/L</td>
</tr>
<tr>
<td>Base Excess</td>
<td>+ or - 2</td>
</tr>
</tbody>
</table>
Compensation

The component of the respiratory system that balances the pH is the dissolved carbon dioxide (CO₂) that is produced by cellular processes and removed by the lungs. The component of the renal system that balances the pH is the dissolved bicarbonate (HCO₃) produced by the kidneys. The kidneys also help control pH by eliminating hydrogen (H⁺) ions. The way the two systems interact is through the formation of carbonic acid (H₂CO₃). Movement through the carbonic acid system is fluid and constant. What this means is that water (H₂O) can combine with CO₂ and form carbonic acid. If necessary, carbonic acid (H₂CO₃) can then break up to form hydrogen ions (H⁺) and bicarbonate (HCO₃). This system works in both directions. By balancing back and forth, a normal pH is achieved. The respiratory system balances the pH by increasing or decreasing the respiratory rate, thereby manipulating the CO₂ level. Fast and deep breathing “blows off” CO₂. Conversely, slow and shallow breathing “retains” CO₂. The renal system balances pH by producing HCO₃ or by eliminating hydrogen ions (H⁺).

The renal system will reflect changes in metabolic activity within the body. For example, a patient in shock will undergo anaerobic metabolism, which produces lactic acid. The production of lactic acid will bind or use up available HCO₃ and will be manifested by a decrease in the HCO₃ level. Therefore, the HCO₃ level is an indicator of metabolic acid-base balance.

\[
\text{H}_2\text{O} + \text{CO}_2 = \text{H}_2\text{CO}_3 = \text{H}^+\text{HCO}_3
\]

Water & carbon dioxide = carbonic acid = hydrogen & bicarbonate

Respiratory side \hspace{1cm} Metabolic side

Balance must always be achieved by the opposing system. If an adult were on one side of a seesaw and a small child on the other, we would expect the child’s side of the seesaw to go up and the adult’s side to go down. We cannot make the child go down by adding another adult to the adult’s side. In the same way, our body regulates pH by using the opposing system to balance pH. So if the pH is out of balance because of a respiratory disorder, it will be the renal system that makes the corrections to balance the pH. Conversely, if the renal system is to blame for the pH disorder, the respiratory system will have to compensate. This process is called compensation. Compensation may not always be complete. Complete compensation returns the pH balance to normal. There are times when the imbalance is too large for compensation to return the pH to normal. This is called partial compensation.
Now let’s try an example:

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.27</td>
<td>Acidotic</td>
</tr>
<tr>
<td>PCO2</td>
<td>53</td>
<td>Acidotic</td>
</tr>
<tr>
<td>PaO2</td>
<td>50</td>
<td>Low</td>
</tr>
<tr>
<td>O2 Saturation</td>
<td>84%</td>
<td>Low</td>
</tr>
<tr>
<td>HCO3-</td>
<td>24</td>
<td>Normal</td>
</tr>
</tbody>
</table>

Step 1. The pH is acidotic
Step 2. The CO2 is acidotic
Step 3. The HCO3 is normal
Step 4. The CO2 matches the pH, therefore the imbalance is respiratory acidosis
Step 5. The HCO3 is normal, therefore there is no compensation
Step 6. The PaO2 and O2 sat are low indicating hypoxemia

Number 1

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.28</td>
<td></td>
</tr>
<tr>
<td>PCO2</td>
<td>59</td>
<td></td>
</tr>
<tr>
<td>PaO2</td>
<td>88</td>
<td></td>
</tr>
<tr>
<td>O2 Saturation</td>
<td>95%</td>
<td></td>
</tr>
<tr>
<td>HCO3-</td>
<td>26</td>
<td></td>
</tr>
</tbody>
</table>

ABG Interpretation: ________________________________________________

Cause: ___________________________________________________________

Treatment: ________________________________________________________

Number 2

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.33</td>
<td></td>
</tr>
<tr>
<td>PCO2</td>
<td>60</td>
<td></td>
</tr>
<tr>
<td>PaO2</td>
<td>75</td>
<td></td>
</tr>
<tr>
<td>O2 Saturation</td>
<td>94%</td>
<td></td>
</tr>
<tr>
<td>HCO3-</td>
<td>31</td>
<td></td>
</tr>
</tbody>
</table>

ABG Interpretation: ________________________________________________

Cause: ___________________________________________________________

Treatment: ________________________________________________________
<table>
<thead>
<tr>
<th>Number 3</th>
<th>Test</th>
<th>Value</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>pH</td>
<td>7.51</td>
<td></td>
</tr>
<tr>
<td></td>
<td>PCO2</td>
<td>32</td>
<td></td>
</tr>
<tr>
<td></td>
<td>PaO2</td>
<td>124</td>
<td></td>
</tr>
<tr>
<td></td>
<td>O2 Saturation</td>
<td>99%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>HCO3-</td>
<td>25</td>
<td></td>
</tr>
</tbody>
</table>

**ABG Interpretation:**

Cause: 

Treatment: 

<table>
<thead>
<tr>
<th>Number 4</th>
<th>Test</th>
<th>Value</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>pH</td>
<td>7.49</td>
<td></td>
</tr>
<tr>
<td></td>
<td>PCO2</td>
<td>40</td>
<td></td>
</tr>
<tr>
<td></td>
<td>PaO2</td>
<td>72</td>
<td></td>
</tr>
<tr>
<td></td>
<td>O2 Saturation</td>
<td>96%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>HCO3-</td>
<td>29</td>
<td></td>
</tr>
</tbody>
</table>

**ABG Interpretation:**

Cause: 

Treatment: 

<table>
<thead>
<tr>
<th>Number 5</th>
<th>Test</th>
<th>Value</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>pH</td>
<td>7.10</td>
<td></td>
</tr>
<tr>
<td></td>
<td>PCO2</td>
<td>25</td>
<td></td>
</tr>
<tr>
<td></td>
<td>PaO2</td>
<td>92</td>
<td></td>
</tr>
<tr>
<td></td>
<td>O2 Saturation</td>
<td>95%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>HCO3-</td>
<td>7</td>
<td></td>
</tr>
</tbody>
</table>

**ABG Interpretation:**

Cause: 

Treatment: 
Number 6

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.42</td>
<td></td>
</tr>
<tr>
<td>PCO2</td>
<td>18</td>
<td></td>
</tr>
<tr>
<td>PaO2</td>
<td>178</td>
<td></td>
</tr>
<tr>
<td>O2 Saturation</td>
<td>99%</td>
<td></td>
</tr>
<tr>
<td>HCO3-</td>
<td>11</td>
<td></td>
</tr>
</tbody>
</table>

ABG Interpretation: ________________________________________________

Cause: ___________________________________________________________

Treatment: ________________________________________________________

Additional Acid-Base Diagnostics

1. Base Excess (BE)
   The base excess evaluates the magnitude of the metabolic component of the pH imbalance. It measures all of the components of the metabolic system that balance the pH. In our blood gas analysis, we used the bicarbonate to evaluate the metabolic acid-base balance. Bicarbonate fluctuates to accommodate fluctuations that cannot be controlled by other body bases such as the hemoglobin and albumin. Base excess weighs all of these bases and determines overall metabolic acid-base balance.

   Normal: +2 to -2  
     Minimal: 3-5    
     Severe: 7-10

   <0 = Base deficit / Acidosis
   >0 = Base excess / Alkalosis

2. Anion Gap (AG)
   The difference between the major positive electrolytes (ions) and the major negative electrolytes (ions) in the body is called the anion gap. The formula is AG = Na⁺ + K⁺ - (Cl⁻ + HCO₃⁻). A venous CO2 level can be substituted for the HCO₃. A patient who has the following lab values would have an AG of 19.

   Na⁺  140  
   K⁺    4.0  
   Cl⁻   100  
   CO₂   25  
   AG = 140 + 4 – (100+25)  
   AG = 19
The value of the AG is to determine the source of a metabolic acidosis. If our patient in the example above has hypotension and has received fluid boluses this morning, and is also suffering from diarrhea, it might be difficult to determine the source of her metabolic acidosis. Hypotension, IV fluid administration, and diarrhea all cause metabolic acidosis. Her AG of 19 is consistent with a wide-ratio anion gap, and is probably the result of hypotension and the resulting lactic acidosis.

A normal-ratio anion gap is 10-15 mEq/L. See table below for causes of metabolic acidosis for normal, high, and low-ratio anion gaps.

<table>
<thead>
<tr>
<th>High Ratio</th>
<th>Normal Ratio</th>
<th>Low Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>2. Lactic acidosis</td>
<td>2. Renal tubular acidosis</td>
<td>2. Hypoalbuminemia</td>
</tr>
<tr>
<td>4. Poisonings</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Resources:
Martin, L. (1999). All you really need to know to interpret arterial blood gases. Lippincott.

On the Internet:
ABG Case Studies:  www.ed4nurses.com/abgs.htm
The ABG Site: www.the-abg-site.com
**Pulmonary embolism**

1. **Brief Pathophysiology**
   a. Sudden obstruction of pulmonary vasculature
      i. Altered ventilation / perfusion
      ii. Increased alveolar dead space
         1. Ventilated but not perfused
      iii. Pneumocostriction
      iv. Bronchoalveolar hypoxemia
      v. Hypoxemia
      vi. Serotonin & histamine
      vii. Thrombin activation
      viii. Increased PVR, ↓ CO, tachycardia
   b. Inflammation
      i. Loss of surfactant
      ii. Atelectasis
      iii. Edema

2. **Diagnostic Tests**
   a. VQ scan (89% sensitivity, 92% specificity)
   b. Pulmonary angiography (the “gold standard”)
      i. Visualize embolus
      ii. Treat locally
         1. Embolectomy
         2. Thrombolysis
   c. Spiral CT
      i. Much faster than V:Q scan
      ii. Similar sensitivity/specificity
   d. Doppler-Ultrasound
      i. Positive U/S, high clinical probability = probable PE
      ii. Negative U/S, low clinical probability = low probability

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

4. **X-rays**
   a. Chest X-ray

---

**Virchow’s Triad**
1. Venous stasis
2. Endothelial injury
3. Coagulopathy
5. EKG
   a. Non-specific findings (can mimic MI)
   b. Classic changes (20%)
      i. **S1** s-wave in lead I
      ii. **Q3** q-wave in lead III
      iii. **T3** t-wave inversion in lead III
   c. Sinus tachycardia
   d. RBBB, right-sided strain
   e. New onset atrial fibrillation

6. Hemodynamics
   a. ↓ preload
   b. ↓ CO
   c. ↑ afterload

7. ABGs
   a. pH
      i. Initially, ↑ from tachypnea
      ii. Later, ↓ from respiratory acidosis
   b. pCO2
      i. Initially ↓
      ii. Later ↑
   c. pO2
      i. ↓ due to V:Q mismatch

8. Symptoms
   a. Many are asymptomatic
   b. Dyspnea
   c. Pleuritic chest pain
   d. Hemoptyisis
   e. Restlessness / apprehension
   f. Calf pain

9. Signs
   a. Tachycardia
   b. Tachypnea
   c. Hypocapnia, hypoxemia
   d. Lung auscultation:
      e. Rales
      f. Wheezing
      g. JVD
   h. Pulsus paradoxus
      i. Hypotension
      j. Diaphoresis
      k. + Homan’s sign

---

**Pulsus paradoxus** occurs when the systolic pressure decreases by 10mmHg or more on inspiration. This finding often results from respiratory disease.

*To measure:* Inflate the B/P cuff and slowly deflate while listening for the first Korotkoff sounds on expiration, then inspiration. The difference between the two should be less than 10mmHg.

---

**From the PIOPED Study:**

*Most common symptoms:*
- Dyspnea 73%
- Pleuritic chest pain 66%
- Cough 37%

*Most common physical exam findings:*
- Tachycardia 70%
- Rales 51%
10. 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

Internet sites:
Preventing Deep Vein Thrombosis and Pulmonary Embolism: www.dvt.org
Canadian Lung Association: www.lung.ca/diseases/pulmonary_embolus.html
The Problem of 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 neonatal 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: cause microclotting
   f. Endothelial cells: cause adherence, release 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. 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
6. Clinical presentation
   a. Latent phase
      i. Beginning changes in the structure of the alveolar-capillary (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
      vi. Consider extracorporeal gas exchange (ECMO, AVCO2R)
      vii. 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
**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
Primary Pulmonary Hypertension

Etiology:
1. Primary:
   a. No known cause
   b. Associated with autoimmune disorders, fibrotic disease
2. Secondary:
   a. Liver disease
   b. Portal hypertension
   c. Diet supplements
3. Mostly affects females in their childbearing years

Brief Pathophysiology

Main Signs and Symptoms:
1. Symptoms:
   a. Dyspnea (60%)
   b. Weakness (19%)
   c. Recurrent syncope (13%)
   d. Women more symptomatic than men
2. Signs:
   a. Tricuspid murmur
   b. JVD, pulsation

Diagnostics:
1. Positive ANA (40%)
2. Often have thyroid disease, HIV
3. EKG
   a. RV hypertrophy
   b. ST-depression in anterior leads
4. Echocardiography

Monitoring:
1. EKG
2. Hemodynamics
   a. PA pressures
   b. PAOP, CVP

Causes of Pulmonary Hypertension:
- Embolic disease
- Pulmonary fibrosis
- Sarcoidosis
- Collagen vascular disease
- Cirrhosis
- Sleep apnea
- HIV
- Cocaine
Treatment:
1. Anticoagulation
2. Digoxin, diuretics
3. Oxygen
4. Calcium channel blockers:
   a. Nifedipine (up to 240 mg/day)
   b. Diltiazem (up to 900 mg/day)
5. Pulmonary vasodilators:
   a. Nitric oxide
   b. Flolan (Epoprostenol)
      i. Prostacycline inhibitor
      ii. Immediate action, 3-5 minute half-life
      iii. Dose: 2-4 ng/kg/min IV
      iv. Continuous infusion
      v. Protect from light
      vi. Needs a special pouch to maintain temperature at 45°F
      vii. Expensive (about $100,000/yr)
   c. Remodulin (Treprostinil)
      i. Similar to Flolan
      ii. Stable at room temperature, longer half-life
      iii. Dose: 1.25 ng/kg/min SC
   d. Tracleer (Bosentan)
      i. Endothelin receptor antagonist
      ii. Oral preparation
         1. <40 kg: 62.5 mg PO bid
         2. >40 kg: 62.5 mg PO bid for 4 weeks, then ↑ to 125 mg PO bid.
6. Lung transplant
   a. Heart-lung transplant
   b. Single, double lung transplants
      i. 70-80% survival at 1 year
      ii. RV will heal itself
   c. 39% of patients develop obliterative bronchiolitis

Internet sites:
Pulmonary Hypertension Association: www.phassociation.org
PHCentral: www.phcentral.org
**Asthma / COPD**

**Asthma**

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. \( \downarrow \) FEV1 may reach 30-35% of personal best
j. \( \downarrow \) FEV1:FVC
k. Pulsus paradoxus > 10 mmHg
l. \( \uparrow \) 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, \( \downarrow \) 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
Endocrine (4%) 5 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 osmolarlity
   b. Serum osmolarlity increased
   c. Serum sodium elevated
   d. All of the above

3. 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

4. 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

5. 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 ↑
   i. Serum Osmolality
   j. Serum ADH level
   k. Water Deprivation Test

3. Diagnostic
   a. Serum Na
   b. BUN
   c. ↑ 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, HNKH)

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>
</tr>
</thead>
<tbody>
<tr>
<td>SIADH</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Dehydration</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Diabetes Insipidus</td>
<td>↑</td>
<td>↑</td>
<td>↓</td>
</tr>
</tbody>
</table>
**Adrenal Insufficiency**

1. Insufficient secretion of adrenal hormones  
   a. Primary (rare)  
      i. Autoimmune  
      ii. Infection  
   b. Secondary  
      i. Corticosteroid cessation / withdrawal

2. Assessment:  
   a. Weakness  
   b. Hyperpigmentation of skin  
   c. Weight loss  
   d. Hypotension  
   e. Bradycardia

3. Diagnostic tests:  
   a. Thyroid function tests  
   b. Adrenocorticotropic hormone stimulation test  
      i. Baseline cortisol and ACTH levels  
      ii. Cosyntropin (synthetic ACTH) 250mcg IV/IM  
      iii. Repeat cortisol levels q30-60 minutes  
      iv. Cortisol levels should rise in response to administration of ACTH

4. Treatment:  
   a. Volume replacement  
   b. Correct electrolytes  
      i. Hyponatremia (most common)  
      ii. Hypoglycemia  
      iii. Hyperkalemia  
      iv. Hypercalcemia (least common)  
   c. Hydrocortisone 100mg IVP q6h.

**Glucocorticoids:**
- ↑ Blood glucose levels  
- Break down ketones  
- ↑ RBC & platelet levels  
- Anti-inflammatory effects

**Resources:**

American Diabetes Association: [www.diabetes.org](http://www.diabetes.org)  
Endocrine Web: [www.endocrineweb.com](http://www.endocrineweb.com)  
Thyroid Today: [www.thyroidtoday.com](http://www.thyroidtoday.com)
Hematologic / Immunologic (5%) 6 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. Common side effects of Cyclosporine include:
   a. Hypertension
   b. Hypokalemia
   c. GI bleeding
   d. Hypomagnesemia

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

4. 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

5. 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

6. Treatment for Sickle Cell Crisis includes:
   a. Oxygen and fluids
   b. Heparin and coumadin
   c. Platelet transfusion
   d. Interferon
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

DIC

Initiation
Platelets
Perfusion

Clotting
Bleeding

(c) 2001-2002 David W. Woodruff
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. PRBC’s
      ii. Platelets
      iii. FFP
      iv. Cryoprecipitate

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

DIC Treatment

Treat Underlying Disorder

Able
Unable
Heparin

Replace Blood Products

(c) 2001-2002 David W. Woodruff
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>
</tr>
</thead>
<tbody>
<tr>
<td>Corticosteroids</td>
<td>↓ Inflammation</td>
<td>↑ Risk of infection</td>
</tr>
<tr>
<td></td>
<td></td>
<td>GI bleed</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hyperglycemia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Adrenal suppression</td>
</tr>
<tr>
<td>Cyclosporine</td>
<td>↓ Immune and inflammatory responses</td>
<td>Potentiates other immunosuppressives</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hepatotoxicity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Nephrotoxicity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hyperkalemia</td>
</tr>
<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>
</tr>
<tr>
<td></td>
<td></td>
<td>Thrombocytopenia</td>
</tr>
<tr>
<td>Imuran (azathioprine)</td>
<td>↓ Immune response</td>
<td>↑ Risk of infection</td>
</tr>
<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>
</tr>
<tr>
<td></td>
<td></td>
<td>Hyperkalemia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hypomagnesemia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Nephrotoxicity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hepatotoxicity</td>
</tr>
<tr>
<td>CellCept (mycophenolate)</td>
<td>↓ Immune response</td>
<td>GI distress</td>
</tr>
<tr>
<td></td>
<td></td>
<td>↓ WBC, platelet levels</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hypertension</td>
</tr>
<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>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Chronic</th>
<th>Incidence</th>
<th>Characteristics</th>
</tr>
</thead>
<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>

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)
Neurologic (4%) 5 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. 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
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

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
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

\[ \text{FiO}_2 \rightarrow \text{Hemoglobin} \rightarrow \text{Cardiac Output} \rightarrow \text{Tissues} \]
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>
Gastrointestinal (5%) 6 Questions

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

2. 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

3. 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

4. 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

5. Pulmonary complications of acute pancreatitis may include:
   a. Adult Respiratory Distress Syndrome (ARDS)
   b. Elevation of the diaphragm and bilateral basilar rales
   c. Atelectasis, especially of the left base
   d. All of the above

6. 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
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%)

<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

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

1. Obstruction of the small bowel
   a. Partial or complete
   b. Simple or strangulated

2. Etiology:
   a. Post-operative adhesions (60%)
   b. Malignancy
   c. Crohn’s disease
   d. Hernia

3. Assessment:
   a. Pain: crampy and intermittent
      i. Short-duration with vomiting: proximal
      ii. Long-duration, progressive in nature: distal
   b. Nausea, vomiting
   c. Fever
   d. Tachycardia

4. Diagnostic tests:
   a. CXR
   b. KUB (sensitivity 75%, specificity 53%)
   c. CT (sensitivity 93%, specificity 100%)
   d. Ultrasound (sensitivity 89%, specificity 100%)

5. Treatment:
   a. Simple / partial
      i. NG drainage
      ii. Antibiotics
      iii. IV fluids
   b. Simple complete / strangulated
      i. NG drainage
      ii. Antibiotics
      iii. IV fluids
      iv. Surgical intervention

6. Complications:
   a. Sepsis
   b. Abscess
   c. Aspiration

**Early:**
- Hyperactive bowel sounds
- Diarrhea

**Late:**
- Hypoactive bowel sounds
- Constipation
Bowel Perforation

1. Puncture or erosion of the GI tract.
   a. Free, with spillage of GI contents
   b. Contained, no spillage

2. Etiology
   a. Abdominal trauma
      i. Penetrating / blunt (more common in children)
   b. Ingestion of aspirin, NSAIDs, or steroids
   c. Appendicitis
   d. Ulcerative colitis
   e. Bowel infarction
   f. Endoscopy
   g. Laparoscopy

3. Assessment:
   a. Peptic ulcer perforation:
      i. Sharp, sudden, severe pain
      ii. Rigid, “board-like” abdomen
      iii. Shoulder pain
      iv. Hiccup
      v. Vomiting is rare
   b. Perforated diverticulitis, ruptured appendicitis
      i. Low abdominal pain
      ii. Pain precedes vomiting by 3-4 hours

4. Diagnostic tests:
   a. ↑ WBC, ↑ HCT (third-spacing)
   b. KUB
   c. Ultrasound
   d. CT

5. Treatment:
   a. IV fluids
   b. NPO
   c. NG suction
   d. Surgery
   e. Perioperative antibiotics

6. Complications:
   a. Abscess
   b. GI bleeding
   c. Obstruction
   d. Sepsis

Bowel sounds are absent in generalized peritonitis.
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:
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, hypertriclyceridemia
   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. **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

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

7. **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
Renal (6%) 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 = \( \uparrow \) forces opposing filtration = \( \downarrow \) 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. \( \uparrow K^+, \downarrow Na^+, \) acidosis
         f. Cardiac arrhythmias
         g. Kussmal’s respirations
         h. Hypervolemia
         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 \( \uparrow \) 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. Hypovolemia
      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
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 \textit{L}K\textit{y}losis is associated with a Low \textit{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. \[ (4 - \text{Alb}) \times 0.8 \] + Calcium = 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)**

2. Intracellular enzymatic reactions and utilization of ATP
3. CNS transmission
4. Cardiovascular tone
5. 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

6. 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)

7. 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
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)

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

d. Osmolality = 2 × Na + Glu / 18 + BUN / 2.8

- Fluid shifts from low osmolality to high!

- 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

### Sodium & Water Imbalances

<table>
<thead>
<tr>
<th>Water Balance:</th>
<th>Sodium Balance:</th>
</tr>
</thead>
<tbody>
<tr>
<td>High</td>
<td>High</td>
</tr>
<tr>
<td></td>
<td>Low</td>
</tr>
<tr>
<td>Normal</td>
<td>High</td>
</tr>
<tr>
<td></td>
<td>Low</td>
</tr>
<tr>
<td>Low</td>
<td>High</td>
</tr>
<tr>
<td></td>
<td>Low</td>
</tr>
</tbody>
</table>
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.

Free Water Deficit = (kg wt. X 0.6) X [(Na/140) – 1]
Multisystem (6%) 8 Questions

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

2. 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

3. 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

4. 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

5. 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

6. Using vasopressors in shock may cause:
   a. Increased splanic perfusion
   b. Decreased cardiac output
   c. Decreased pulmonary perfusion
   d. Increased peripheral perfusion
7. 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

8. 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. ↑ mortality</td>
</tr>
<tr>
<td>Hb substitutes</td>
<td>Varies</td>
<td>Immediate oxygen delivery</td>
<td>Multiple side effects Not proven effective</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
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:

<table>
<thead>
<tr>
<th>Action</th>
<th>Started</th>
<th>Completed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decide which test to take</td>
<td></td>
<td></td>
</tr>
<tr>
<td>When?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Register</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Request time off</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Get study materials</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Emergency planning</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Study guide #1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Study guide #2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Study guide #3</td>
<td></td>
<td></td>
</tr>
</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>
<td></td>
</tr>
<tr>
<td>Who will work the night before the test?</td>
<td></td>
</tr>
<tr>
<td>Who will manage the kids/pets?</td>
<td></td>
</tr>
<tr>
<td>When will you shop for healthy foods?</td>
<td></td>
</tr>
<tr>
<td>Who will you get to care for ill kids, pets, or husbands/wives?</td>
<td></td>
</tr>
<tr>
<td>What will you do if the car doesn’t start?</td>
<td></td>
</tr>
<tr>
<td>What if you get a flat tire?</td>
<td></td>
</tr>
<tr>
<td>What will you do if traffic is bad?</td>
<td></td>
</tr>
<tr>
<td>What alternate routes are available to the testing site?</td>
<td></td>
</tr>
<tr>
<td>When do you need to go to bed the night before?</td>
<td></td>
</tr>
<tr>
<td>What will you eat the morning of the exam?</td>
<td></td>
</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](http://www.ed4nurses.com/certification.htm)
Thanks for attending “PCCN: Test Prep”!

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

**The Progressive 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 Progressive Care Mastery Package**

Progressive 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.

**The Progressive Care Full Curriculum Package**

Get both the Essentials and Mastery Packages together and really supercharge your career!