MICHIGAN STATE UNIVERSITY
COLLEGE OF NURSING

NUR 430

Nursing Care of the
Critically Ill Client

COURSE SYLLABUS
REQUIRED ON CAMPUS
Section 1-2

COURSE FACULTY:
Mary Kisting, RN, MS

Fall, 2002
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MICHIGAN STATE UNIVERSITY
COLLEGE OF NURSING
OUTREACH AND PROFESSIONAL DEVELOPMENT

NUR 430/491

Nursing Care of the Critically Ill Client

Number of Credits: 3 (2-1)

Placement: Fall Term, 2001

Course Faculty: Mary Kisting, RN, MS, CCRN, CNS

NUR 430/491 - Room - A117-123 Life Sciences Building
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I. Course Description
The theory portion of this course provides fundamental information about the nursing process of clients in critical care settings. The clinical piece gives students opportunities to explore critical care nursing and to work in peer relationships with critical care staff. Both require application and synthesis of pathophysiological, pharmacological, and therapeutic concepts. Utilization of family theory, stress-adaptation, psychosocial concepts and legal/ethical issues are incorporated throughout.

Fourteen weeks will be spent in the classroom theory, and 45 hours will be spent in clinical in critical care settings. While maintaining professional standards for critical care nursing, the student will continue to develop and practice the professional role. For clinical, students will work on an individual basis with a professional nurse preceptor who practices in a critical care setting.

II. Course Objectives
At the completion of Nursing Care of the Critically Ill Client the student will be able to:

1. Define and participate in the nursing care of clients in critical care settings.

2. Apply psychosocial concepts to individuals and families in actual and potentially life-threatening situations.

3. Discuss the legal and ethical issues that commonly occur in the critical care setting.

4. Describe needs of families of the critically ill.
5. Interpret basic dysrhythmias and describe the appropriate interventions.

6. Describe the physiologic basis, indications and implications for hemodynamic monitoring.

7. Explain the concept of oxygen delivery and consumption, and how it can be altered in the critically ill.

8. Identify medications commonly used in the critical care setting and their indications and side effects.

9. Describe the pathophysiology and management of clients with acute nervous system alterations.

10. Contrast the pathological cause and the effect of cardiac disturbances, identifying the treatment modalities for each.

11. Examine the etiology, pathophysiology, assessment and intervention for acute respiratory, hematologic, renal, endocrine and GI dysfunction.

12. Correlate the different types of shock to their pathophysiology and treatment.

13. Discuss the unique considerations for the neonatal, pediatric, geriatric and burn client in the critical care setting.

14. Demonstrate behavior consistent with ANA Standards of Nursing Practice and the ANA Code for Nurses when interacting with clients who are in actual and potentially life-threatening situations.

III. Learning Experiences - Teaching Methods

The course will consist of a variety of instructional methods including lecture, discussion, seminars and clinical application in critical care settings.

IV. Instructional Model (14 weeks of didactic and 45 hours of clinical)

Credits: 3(2-1)
Course lecture content is scheduled to precede and overlap with the clinical experience. The completion of assigned readings prior to class is necessary to facilitate understanding and integration of content presented. Attendance at class is strongly recommended.

For clinical, students will work in a one-to-one relationship with a clinical nurse preceptor in a critical care setting. Active participation on the part of the student in client nursing care is expected. While the student will not be totally responsible for the patient
care, each student will be accountable for the portions of care which he/she has performed. Clinical hours will be negotiated with the clinical nurse preceptor.

**Attendance at Clinical is Required**

One UNEXCUSED absence will result in failure of the clinical portion of the course.

V. **Clinical Agencies:**
   - Ingham Medical Center
   - Sparrow Hospital
   - Others as arranged

VI. Student-preceptor informal clinical conferences may be conducted at selected times during the term on a pre-arranged basis with students, and a formal final conference will be conducted during finals week. Faculty and students are expected to engage in discussion as needed for the purposes of feedback, clarification, evaluation of student completion of course objectives, and concerns relative to the course. Students who have concerns regarding lecture material may request consultation from the faculty member. Students may meet by appointment with faculty members.

VII. **Evaluation**
   Evaluation is viewed as an essential skill for the professional nurse practitioner. Skill in self-evaluation is significant. Students can increase their self-evaluation skills through discussion and validation with other students, faculty, and their clinical nurse preceptors.

   By the end of the first week of the clinical experience, students will prepare three to five personal and professional goals for the term. A written summary of progress in goal attainment will be reviewed with the clinical instructor at the final evaluation conference.

   Safety, honesty, and integrity are expected of all students. Any student who does not meet minimal safety standards with clients or who willfully falsifies client records or reports will fail the clinical portion of the course.

VIII. **Grading**
   A. The theory grade will be based upon three quizzes and analysis and presentation of a published research study. Students are responsible for all reading assignments and lecture material.

   | Quiz 1   | 20% |
   | Quiz 2   | 20% |
   | Quiz 3   | 20% |
   | Research presentation | 5% |
   | Final Exam | 35% |
B.  The clinical grade is pass/fail, based on the clinical objectives. Students not passing the clinical portion of the course will receive 0.0 for the course.

C.  The following scale will be used for grade determination.

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<thead>
<tr>
<th>%</th>
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<tr>
<td>94-100</td>
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IX.  Academic Dishonesty Policy
Academic dishonesty in ANY FORM will not be tolerated. If cheating in any form is observed and documented by a faculty member, the student will be reported to the Student Affairs Committee and a grade of 0.0 will be issued for the course.

X.  Exam Policy
All students are expected to take examinations on the designated date unless prior arrangements are made with the course faculty. Alternative exams may be used for make-up. Alternative exams may include essay and/or short answer questions.

XI.  Attendance
Attendance at class lecture is STRONGLY recommended. Attendance at clinical is REQUIRED. Due to the limited amount of clinical experience, one UNEXCUSED absence will result in failure of the course.

In case of illness, each situation will be considered individually. A health provider's statement may be requested.

Students must notify their clinical instructor and the critical care unit PRIOR to the clinical experience in the event of illness or other reasons for absence. Failure to do so may result in an unexcused absence.

Students MUST schedule a final conference with the clinical instructor in order to receive a grade for the course. If a final evaluation is not done, NO GRADE REPORTED will be given for the course.
XII. **CPR Certification**
All students in the course must demonstrate CURRENT certification in Basic Life Support. This can be achieved from either the American Red Cross or the American Heart Association for a minimal fee. The student must show evidence of current certification to the clinical instructor during clinical orientation.

XIII. **Required Textbook:**

XIV. **Required Equipment**
In addition to textbooks, students are required to have:

1. Uniform - see Student Handbook for guidelines. No pullover sweaters, sweatshirts, tennis or jogging shoes, or dangling jewelry are permitted. Name tag is required. Additional guidelines will be provided during orientation with clinical preceptor.

2. Watch with a second hand or second function.

3. EKG calipers.

4. Stethoscope with both a bell and diaphragm.

5. Penlight

6. Black ballpoint pen (NO MARKERS).

XV. **Taping Policy:** Lectures may be audio taped with instructor permission.

XVI. **CON & MSU Policy:** Students should refer to the College of Nursing and MSU Student Handbooks for any areas not specifically covered in the syllabus.

Weather Policy: Each clinical faculty will establish an attendance policy related to severe weather. In general, students will arrive at clinical as soon as safely possible and notify the clinical site if they will be late by more than 2 hours of usual starting time. If faculty are unable to attend clinical, they will establish contact with the student.

**Recommended Media:**

"SVO₂: The delicate balance" (11 minutes) in the Media Lab.
**Supplemental Reading:**

Dubin, D. *Rapid Interpretation of EKG's*. Cover Publishing Co.


## Michigan State University
### College of Nursing
#### Nursing 430/491
Nursing Care of the Critically Ill Client
**Course Outline**

<table>
<thead>
<tr>
<th>Class</th>
<th>Date</th>
<th>Content</th>
<th>Assignments</th>
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| 1     | 8-29-02 | **Introduction**  
Critical Care Nursing – Individual and family response  
Psychosocial & legal-ethical issues  
ECG Interpretation | Pp. 9-23  
Pp. 25-37  
Pp. 41-54 |
| 2     | 9-5-02  | **Dysrhythmias**                                          | Pp. 54-80                                       |
| 3     | 9-12-02 | **Hemodynamic monitoring**                                | Pp. 83-114                                      |
| 4     | 9-19-02 | **Oxygen delivery/consumption and monitoring**  
Ventilators  
ABGs | Pp. 117-163 |
| 5     | 9-26-02 | Quiz #1  
**Medications**                                           | Pp. 180-193                                      |
| 6     | 10-3-02 | **Nervous system alterations and treatment**  
Increased ICP  
Head/spinal cord injury  
Acute cerebrovascular disease  
Seizures  
Meningitis | Pp. 283-343 |
| 7     | 10-10-02| **Cardiovascular dysfunction and treatment**  
Coronary artery disease  
Congestive heart failure  
Pericardial/endocardial disease  
Aortic aneurysm | Pp. 239-281 |
| 8     | 10-17-02| **Respiratory dysfunction and treatments**  
Respiratory failure  
Acute respiratory distress syndrome  
Chronic obstructive pulmonary disease  
Asthma  
Pneumonia  
Pulmonary embolism  
Pneumothorax | Pp. 345-368 |
| 9     | 10-24-02| Quiz #2  
**Acute renal failure and treatment**  
Pre, intra, and post renal failure  
Renal replacement therapies | Pp. 371-401 |
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<td>10-31-02</td>
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<td>Anemias</td>
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<td>Malignant white blood cell disorders</td>
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<td>Pancreatic endocrine emergencies</td>
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<td>Acute adrenal crisis</td>
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<td>Thyroid crises</td>
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<td>Antidiuretic hormone disorders</td>
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<td>Pancreatitis</td>
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<td>11-14-02</td>
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<td>Mechanisms of trauma injury</td>
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<td>Initial phases of care</td>
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<td>Specific organ injuries</td>
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<td>11-28-02</td>
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<td>Special and unique considerations</td>
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<td>Pediatric critical care considerations</td>
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<td>Geriatric considerations</td>
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<td>12-12-02</td>
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Course Notes and Reference Information
NUR 430
Nursing Care of the Critically Ill Client

Class 1 and 2

Individual & Family Responses to the Critical Care Experience

1) AACN’s Vision
   a) A healthcare system driven by patient’s and family’s needs where critical care nurses
      make their optimum contributions

2) Many Stressors
   a) Physical
      i) Pain, restraints
   b) Psychological
      i) Powerlessness, fear, anxiety
   c) Environmental
      i) Alarms, lights
   d) What are other examples?

3) Ain’t got no rhythm??

4) Circadian Cycles 24 hours
   a) Sleep
   b) Temperature
   c) Blood Pressure
   d) Urine production

5) Ultradian Cycles < 24 hours
   a) REM/NREM sleep
   b) SA node firing
   c) Nerve action potentials

6) Infradian Cycles > 24 hours
   a) Menstrual cycle

7) Powerlessness
   a) Lack of control
   b) Who is more apt to experience powerlessness?
   c) What can nurses do to prevent it?

8) Anger
   a) Protects the patient’s integrity when dealing with a perceived threat
   b) What signs do patients exhibit?
   c) How should the nurse respond to angry behavior?

9) ICU Syndrome
   a) Often related to environment
      i) Past studies have found this in patients after cardiac surgery
   b) Sleep deprivation
c) Sensory overload

10) Nursing Strategies to Assist the Family
   a) Information is the top priority
   b) Identify role of the nurse
   c) Consistent nurses
   d) Education about the environment and equipment
   e) Facilitate the family’s participation in care
   f) Explain the patient’s behavior (e.g., confusion, agitation)
   g) Individualize visiting
   h) Recommend Support groups

11) Nursing Strategies to Assist the Family
   a) Explain crisis situations in the unit
   b) Provide support

12) Who Should Visit the Patient?

13) Visitation that Works
   a) Individualized or contracted
   b) People and times mutually determined by patient, family, and nurse
   c) Avoid change of shift, rounds
   d) Evaluate response of patient and family

14) Career Development: Important!
   a) Critical care course
   b) Good preceptor and comfort with him/her
   c) Continuing
   d) Education
   e) Professional
   f) Development
   g) Ongoing continuing education in the field
   h) ASK QUESTIONS!!

Ethical Issues

1) Advocacy is Essential
   a) ANA Code for Nurses
      i) Practice ethically on behalf of patients
      ii) Patients and families are vulnerable
      iii) Nurses establish a unique relationship with them

2) Autonomy
   a) Patient’s right of self = determination concerning medical care
   b) Opposite of paternalism: health care workers (especially MDs) know best

3) Beneficence
   a) Duty to prevent harm, remove harm, and promote the good of another person
   b) Related to futility

4) Nonmaleficence
   a) Not to intentionally inflict harm
   b) “Above all, do no harm”

5) Veracity
a) To tell the truth, the whole truth, and nothing but the truth

6) **Fidelity**
   a) Be faithful to commitments

7) **JCAHO**
   a) Formal mechanisms for dealing with ethical issues required
   b) Committees versus case-by-case consultation
   c) Investigate what is in place at clinical settings in the area to meet JCAHO requirements

8) **Solving Ethical Dilemmas**
   a) Diversity of approaches
   b) Goal is to determine most ethically justifiable course of action and the one that minimizes violation of ethical principles

9) **Considerations**
   a) What is the dilemma?
   b) Who are the parties involved?
   c) Known and unknown facts
   d) Ethical principles
   e) Options and consequences
   f) Promotes the best interest of the patient
   g) Not required to practice if situation violates moral or religious beliefs

10) **Selected Ethical Issues**

11) **Informed Consent**
    a) Decisions based on information and understanding
    b) Decisions are voluntary

12) **Life-Sustaining Treatment**
    a) Vegetative state versus brain death
       i) Brain death is death; life support can be terminated based on state laws
       ii) Vegetative state involves many more issues

13) **CPR Issues**
    a) Should a patient be “coded?”
    b) Specific orders need to be written and documented on chart
    c) Orders should be written in collaboration with patient and/or family members
       i) Advance directives useful in guiding decision making

14) **Withholding or Withdrawal of Life Support**
    a) Ordinary versus extraordinary care
    b) Quality of life
    c) What are some issues?

15) **Helping Families Make Decisions About Life Support**
    a) Communicate frequently
    b) Consistent, honest communication
    c) Base on PATIENT’S wishes
    d) Provide psychological support to the family

16) **Advance Directive**
    a) Communication about treatment should patient become incapacitated
       i) Living will
(a) Treatment desired and what should be withheld
   ii) Durable power of attorney for health care
(a) Determines who makes decisions

17) Organ/Tissue Transplantation
   a) Patients who are brain dead are often candidates for organ donation
   b) Everyone has right to donate organs
   c) “Designated requester” is a new strategy to facilitate consent for organ donation

18) Brain Death
   a) How is brain death determined?

Introduction to Electrocardiography

1) Electrocardiography
   a) Electrical activity
   b) Graphic representation

2) Basis for Electrical Activity
   a) AUTOMATICITY
   b) Cardiac muscle can generate its own electrical activity

3) Two Types of Cardiac Cells
   a) Electrical initiates
   b) Mechanical contractions

4) Principles
   a) Electrical precedes mechanical
   b) Electrical activity can occur without mechanical
      i) Pulseless electrical activity (PEA)
      ii) Lack of pulse or blood pressure
   c) Electrical + Mechanical = Cardiac contraction

5) Cardiac Cycle
   a) Depolarization = systole = contraction
   b) Repolarization = diastole = resting/filling

6) Electrical Activity
   a) As the atria repolarize the ventricles depolarize
   b) The ECG cannot show both repolarization and depolarization

7) Polarization
   a) Electrical charges ready for discharge
   b) K intracellular and Na extracellular

8) Depolarization
   a) Discharge of energy that accompanies the transfer of electrical charges across the cell
   membrane
   b) Na moves into cell and K moves out of the cell
   c) Associated with the MECHANICAL act of systole

9) Repolarization
   a) Return of electrical charges to original state
   b) Associated with the MECHANICAL act of diastole

10) Conduction System
   a) SA node
b) Intra-atrial and internodal pathways to AV node
c) Bundle of His
d) Left and right bundle branches
e) Purkinje fibers

11) Inherent Rates (Automaticity)
   a) SA node = 80 to 100 beats/min
   b) AV node = 40 to 60 beats/min
   c) Ventricles (Purkinje fibers) = 15 to 40 beats/min

12) Which Predominates?
   a) Site with fastest rate = pacemaker
      i) SA node is normal pacemaker
   b) Irritability—another site speeds up and takes over
   c) Escape—normal site slows down and lower site takes over

13) Autonomic Influences
   a) Sympathetic nervous system effects
   b) Parasympathetic nervous system effect

14) Sympathetic Stimulation
   a) Increased heart rate
   b) Increased AV conduction
   c) Increased irritability

15) Parasympathetic Stimulation
   a) Decreased heart rate
   b) Decreased AV conduction
   c) Decreased irritability

16) Waves and Measurement
   a) P wave – positive deflection (atria contracting)
   b) QRS – large deflection (ventricles depolarize)
   c) T wave – upright, rounded (ventricles repolarize)

17) Electrodes
   a) Transmit electrical activity
   b) Lead is SINGLE view of activity

18) Methods
   a) 12-lead EKG
   b) Bedside monitoring
   c) Holter

19) 12-Lead EKG
   a) Limb leads (I, II, III, AVR, AVL, AVF)
      i) Einthoven’s triangle
   b) Precordial leads (V1 - V6)
   c) Additional right precordial leads
   d) Additional posterior leads

20) Lead Placement
    Determines Configuration
    a) Impulses toward electrode = positive
    b) Impulses away from electrode = negative
21) Bedside Monitoring
   a) 3-lead versus 5-lead
   b) Electrode placement
   c) Choosing which lead to monitor
      i) Choose leads to monitor for ischemia
      ii) Newer monitors have the capacity for monitoring more than one lead

22) Monitoring Systems
   a) Hard wire
      i) Cable connected from patient directly to bedside monitor
      ii) Rhythm viewed on bedside and central station monitors
   b) Telemetry
      i) Cable connected to battery pack
      ii) Signal transmitted to a central station for viewing

23) Waves and Measurement
   a) Wave analysis done routinely
   b) Documented by shift and with change

24) Graph Paper
   a) Used to standardize tracings
   b) Vertical lines = time
   c) Horizontal lines = voltage

25) Squares
   a) Small = .04 seconds / 0.1 mv
   b) Large = .20 seconds / 0.5 mv
      i) 10 large blocks = 2 seconds
      ii) 15 large blocks = 3 seconds
   c) Hash marks used to designate seconds at top of paper

26) Waves
   a) P-wave = atrial depolarization
      i) normally indicates firing of the sinoatrial node
   b) QRS = ventricular depolarization
      i) Various configurations
   c) T wave = repolarization

27) PR Interval
   a) Atrial depolarization/ delay in AV node
   b) Beginning of P-wave to beginning of QRS complex
   c) .12 to .20 seconds
   d) Shorter interval = impulse from AV junction
   e) Longer interval = 1st degree AV block

28) QRS Duration
   a) Ventricular depolarization
   b) 0.06 to .10 seconds
   c) Various configurations
   d) Wide: slowed conduction
      i) Bundle branch block (BBB)
      ii) Ventricular rhythm
29) **ST Segment**
   a) Look for depression or elevation
      i) ST elevation: myocardial injury
      ii) ST depression: reciprocal changes, digoxin, ischemia

30) **QT Interval**
   a) Beginning of QRS complex to end of T wave
   b) .32 to .50 seconds
   c) Varies with heart rate

31) **U wave**
   a) Sometimes seen after T wave
   b) May indicate hypokalemia

32) **Can Get Artifact**
   a) Movement
   b) 60-cycle interference
   c) ALWAYS CHECK THE PATIENT!

33) **Analyzing Rhythm Strips**
   a) Know rules
   b) Follow a pattern or formula

34) **Look at:**
   a) Rhythm = regularity
   b) Rate
   c) P wave
   d) PR interval
   e) QRS complex
   f) QT interval

35) **Rhythm**
   a) P - P intervals (atrial)
   b) R - R intervals (ventricles)

36) **Determining heart rate**
   a) Regular
      i) Small blocks into 1500
      ii) Large blocks into 300
   b) Irregular = 6-second strip

37) **Heart Rate**
   a) Calculate both ATRIAL and VENTRICULAR rates
   b) Atrial rate = P - P
   c) Ventricular rate = R – R

38) **Sinus Rhythms**
   a) The gold standard against which other rhythms are measured

39) **Normal Sinus rhythm**
   a) NSR or SR
   b) P, QRS, T
   c) Normal; intervals
   d) Rate 60 to 100 beats/min

40) **Sinus Bradycardia**
a) Sinus rhythm with rate less than 60
b) Causes; vagal, drugs, ischemia, suctioning, ICP, athletes
c) Treat according to symptoms—transcutaneous pacing/drugs

41) Sinus Tachycardia
a) Sinus rhythm with a rate of 100 to 150 beats/min
b) Causes; stimulants, exercise, fever, volume overload, shock
c) Treat cause
d) Assess for symptoms of low cardiac output

42) Sinus Dysrhythmia
a) Sinus rhythm
b) Rate varies with respirations
   i) Inspire = increase
   ii) Expire = decrease

43) Sinus Arrest/Exit Block
a) Sinus node fails to initiate an impulse
b) Causes; vagal, CAD, drugs that slow heart rate
c) Treat; monitor symptoms

44) Atrial Rhythms
a) Increased automaticity in the atrium
b) Generally have P-wave changes

45) Causes of Atrial Dysrhythmias
a) Stress
b) Electrolyte imbalances
c) Hypoxia
d) Atrial injury
e) Digoxin toxicity
f) Hypothermia
g) Hyperthyroidism
h) Alcohol
   i) Pericarditis

46) Premature Atrial Contractions
a) Premature Atrial Contractions
b) Early beats initiated by atrium
c) P waves and PR interval may vary
d) Noncompensatory pause

47) Wandering Atrial Pacemaker
a) Varying configurations of P-waves
b) Common in COPD

48) Atrial Tachycardia
a) Multifocal ectopic foci
b) Heart rate exceeds 100 beats/min

49) Supraventricular Tachycardia
a) Fast rate (> 150 beats/min) from atrial foci

50) Atrial Flutter
a) Ectopic foci in atria
b) Classic “sawtooth” pattern
c) Atrial rate fast and regular (250 to 350 beats/min)
d) Ventricular rate slower
e) Degree of conduction varies may be 1:3, 1:4
f) May need drugs or cardioversion

51) Atrial Fibrillation
   a) Erratic impulse formation in atria
   b) No discernable P wave
   c) Irregular ventricular rate
   d) Aberrant (abnormal) ventricular conduction can occur
   e) Results in loss of atrial kick
   f) High risk for pulmonary or systemic emboli

52) Functional (Nodal) Rhythms
   a) Dysrhythmias from the AV junction
   b) P-wave changes
      i) Shorter PR intervals
      ii) No P-wave
      iii) Retrograde P-waves

53) Junctional Rhythm
   a) Rhythm initiated from AV junction
   b) P-wave changes
   c) Rate usually 40 to 60 beats/min
   d) Often an escape rhythm
   e) Assess for symptoms of decreased cardiac output

54) Premature Junctional Contractions
   a) Early beats initiated by AV junction
   b) P-wave changes
   c) Usually involve a noncompensatory pause

55) Junctional Tachycardia
   a) Rhythm initiated from the AV junction
   b) Rate is higher than 60 beats/min
   c) Rate is faster than the AV junction usually fires

56) Ventricular Dysrhythmias
   a) Impulses initiated from the Purkinje fibers
   b) Common causes
      i) Myocardial infarction
      ii) Low potassium or magnesium
      iii) Hypoxia
      iv) Acid-base imbalances

57) Idioventricular Rhythm
   a) Rhythm initiated by the Purkinje fibers
   b) Slow rate (15-40 beats/min)
   c) Widened QRS
   d) Usually an escape rhythm
   e) Assess for low cardiac output
6) NO lidocaine
7) Accelerated: rate > 40 beats/min

58) **Premature Ventricular Contractions**
   a) Wide, bizarre, and ugly
   b) Compensatory pause
   c) Patterns
      i) Bigeminy and trigeminy
      ii) Couplets and triplets
   d) Unifocal versus multifocal

59) **Ventricular Tachycardia**
   a) Three or more PVCs in a row
   b) Fast rate (> 100 beats/min)
   c) Initiated by ventricles
   d) Wide QRS complex
   e) Usually regular
   f) Patient may or may not have a pulse

60) **Ventricular Fibrillation**
   a) Chaotic pattern
   b) Coarse versus fine
   c) No cardiac output
   d) Emergent defibrillation

61) **Absence of Rhythm**
   a) Asystole
   b) Assess in two leads

62) **AV blocks**
   a) Coronary artery disease
   b) Myocardial infarction (e.g., inferior wall)
   c) Infections
   d) Enhanced vagal tone
   e) Drug effects (e.g., digoxin toxicity)
   f) Always assess for decreased cardiac output and treat the cause

63) **Bundle Branch Blocks**
   a) Delayed conduction through the bundle branches
   b) LBBB and RBBB
   c) P wave, P-R interval, widened QRS complex

64) **First Degree Block**
   a) Delayed conduction from sinus node to AV node
   b) Prolonged (> .20 seconds) PR interval

65) **Second Degree Block**
   a) Mobitz I—Wenckebach’s phenomena
   b) Mobitz II

66) **Wenckebach’s phenomena**
   a) Progressive lengthening of PR interval
   b) Followed by P wave without a QRS

67) **Mobitz Type II**
a) Occasional P waves without a QRS complex
b) P to P interval is regular
c) Observe for progression to 3rd degree block

68) Third Degree Block
   a) Complete heart block
   b) Atria and ventricles beat independently of each other
   c) P waves not associated with QRS complex
      i) P-P intervals regular
      ii) R-R intervals regular
d) Junctional or ventricular escape rhythms
e) May need pacemaker

69) Treatments Based on Symptoms and Cause
   a) ABC is the top priority
   b) Treatment based on arrhythmia

70) Tachydyrsrhythmias
   a) Mediate effect of sympathetic nervous system
   b) Vagal maneuvers
c) Drugs
d) Cardioversion/defibrillation
e) Overdrive pacemakers
f) Ablation

71) Bradydyrsrhythmias
   a) Mediate affect of parasympathetic nervous system
   b) Drugs
c) Pacemakers

72) Pacemakers
   a) Temporary versus permanent
   b) Method of pacing
      i) Transcutaneous-emergency
      ii) Transvenous
      iii) Epicardial

73) Pacemakers
   a) Pulse generators
      i) External versus internal
   b) Demand versus asynchronous modes

74) Types
   a) Atrial
   b) Ventricular
c) Dual chamber

75) Pacemaker Terms
   a) Output; milliamperes (mA)
b) Sensitivity
c) Spike
d) Capture

76) Complications
a) Failure to pace
b) Failure to capture
c) Failure to sense

**77) Interventions for Complications**

a) Positioning patient (e.g., on left side)
b) Check/replace batteries
c) Adjust settings

*Practice rhythm strips weekly.*

**Class 3**

1) *Hemodynamic Monitoring*

2) *Hemodynamics*

a) Interrelationships of physical forces that affect circulation
b) Pressure, flow and resistance
c) Garden hose analogy

3) *Why are thermodynamics important in critical care?*

a) Monitoring used extensively in patient assessment and decision making
b) Nurses need to understand types and meaning of data

4) *Common Types of Monitoring*

a) Arterial pressure monitoring
   i) A-line
b) Pulmonary artery pressure monitoring
   i) PA catheter; “Swan Ganz”
c) Right atrial pressure monitoring
   i) RAP; CVP

5) *Principles*

a) Pressure = flow \times resistance
b) Greater flow = higher pressure
c) Greater resistance = higher pressure

6) *Principles*

a) Blood Pressure = flow (cardiac output) \times resistance (lumen size)
b) Circulatory system is a continuous circuit
c) Liquids flow from areas of higher pressure to areas of lower pressure
   i) Heart pressures

7) *Cardiac Output*

8) *Physiology*

a) Blood volume is constant
   i) Increased demands = increased circulation
   ii) Decreased demands = storage
      (1) Veins are capacitance vessels
      (2) No tone, out of shape

9) *Physiology*

a) Ability of blood vessels to change lumen size (especially veins)
i) Neuroendocrine
   (1) sympathetic system
ii) Renin-angiotensin system
   (1) retains volume

### 10) Cardiac Cycle

a) Review how blood flows through the heart
   i) What part of cardiac cycle do ventricles receive blood from atria?
   ii) What part of the cycle do the ventricles contract?
   iii) Tri = right; might (mit) is not right

### 11) Cardiac Output

a) CO = HR X SV
b) SV = Preload, afterload, and contractility
   i) the amount of blood in the LV after the mitral valve closes makes up the SV
   ii) heart never blows its wad - only a portion or fraction (ejection fraction)
   iii) norm is 60-70%

### 12) Preload

a) Muscle fibers stretch before systole
b) Determined by LVEDV/LVEDP
c) Starling’s law
   i) Increased stretch = increased volume
   ii) Stretch is within physiological limits
d) Example = balloon

### 13) Afterload

a) Pressure or resistance against flow
b) R/t lumen size and viscosity
c) SVR = common measure
   i) (MAP-RAP)/CO x 80
d) Example = opening door against wind/opening of the balloon

### 14) Contractility

a) Force of ventricular contraction
b) How well is the heart pumping
c) The quality/elasticity of the balloon

### 15) Measurement

a) Direct: Preload and afterload
b) Indirect: Contractility

### 16) Ejection Fraction

a) Percentage of blood ejected with systole
b) 60% to 70% is normal

### 17) Components of Hemodynamic Monitoring

a) Transducer
   i) Converts physiological events into electrical signals (e.g. pressure, temperature, light)
b) Amplifier
   i) Picks up electrical signal and transmits to display through cable

### 18) Components of Hemodynamic Monitoring

a) Monitor Display
i) Display readings (waveforms, pressure values)
b) Catheter tubing / flush system
   i) Keep catheter patent
   ii) Heparinized solution (✓ protocol)
   iii) Pressure bag
19) Reliable Measurements
   a) Level = phlebostatic axis (4th intercostal space, midaxillary line)
   b) Balance = zero reference (negates atmosphere pressure)
   c) Calibration = numerical accuracy
   d) Square wave test
20) Intraarterial Monitoring
   a) Indications
      i) Continuous blood pressure monitoring
      ii) Blood drawing
21) Sites
   a) Radial
   b) Brachial
   c) Femoral
22) Equipment
   a) Pressurized flush solution with transducer
   b) A-line catheter (angiocath)
23) Nursing interventions
   a) Monitor waveform
   b) Compare values with cuff
      i) A-line should be more accurate reading
   c) Check connections in system
   d) Check site and circulation to extremities
   e) Set alarms
24) Arterial line tracing
25) Complications
   a) AIR Embolus: major complication
   b) Hemorrhage: keep connections tight
   c) Thrombosis
26) Right Atrial Pressure
   a) RV preload/RVEDP
   b) Right pressure changes usually occur late
27) Catheters that Measure RAP
   a) Pulmonary artery catheter (proximal port)
   b) Central line (e.g. triple lumen catheter)
   c) Peripherally inserted central catheter (PICC) line
28) Measurement
   a) Measured via pressure system
   b) Normal value is 0 to 8 mm Hg
   c) Recorded as MEAN value
29) Interpretation of Values
a) **Low CVP**
   i) Hypovolemia
   ii) Vasodilation
      (1) relative hypovolemia
b) **High CVP**
   i) Hypervolemia
   ii) Vasoconstriction
   iii) Right CHF
   iv) Pulmonary hypertension

30) **Complications of Insertion**
   a) Pneumo/hemothorax
   b) Heart perforation
   c) Dysrhythmias
   d) What assessments should be done during insertion and immediately after insertion?

31) **Nursing Implications**
   a) Zero/balance
   b) Waveform analysis
   c) Respiratory variation - end-exp.
   d) Monitor complications - Infection
   e) Correlate values obtained with nursing assessment

32) **Left Atrial Pressure**
   a) Catheter into left atrium
   b) Mean value—normal range is 1 to 10 mm/hg
   c) When to use
      i) For accurate measurements
      ii) Cardiac surgery; children

33) **Nursing implications**
   a) Zero/level/balance
      i) Positioning patient
      ii) Respiratory cycle

34) **Complications of LAP**
   a) Air
   b) Clots
   c) Infection

35) **Pulmonary Artery Catheter**
   a) Balloon-tipped
   b) Measures PA systolic, diastolic, mean, and wedge (PCWP; PAWP; PAOP)
   c) Multi-lumen
      i) Proximal (RA)
      ii) Proximal injectate
      iii) Distal (PA)
      iv) Balloon

36) **Swan-ganz catheter**

37) **Types**
   a) Four / five (VIP) lumen
b) Variations
   i) SvO₂ catheter
   ii) RefOx right ejection fraction
   iii) Continuous cardiac output (CCO)
   iv) SvO₂/CCO

38) Insertion
   a) Provide explanation and obtain informed consent
   b) Site: usually subclavian or internal jugular
   c) Incision: guide wire, introducer, catheter, sheath
   d) Flush lumens

39) Insertion
   a) Inserted with balloon down, selected inflation to get into PA
   b) Waveform changes as catheter progresses
   c) Check for proper wedging
   d) Secure and dress site

40) Pulmonary Artery catheter insertion

41) During insertion
   a) Monitor pressures in each chamber
   b) Record values
   c) Assess for complications
      i) Dysrhythmias
      ii) Pneumo/hemothorax

42) PA Values
   a) Normal is 25 to 10 with mean is 15
      i) Systolic 15 to 30 mm Hg
      ii) Diastolic 4 to 12 mm Hg
      iii) PCWP 6 to 12 mm Hg
   b) Diastolic reflects PCWP unless pulmonary hypertension present (mitral valve open
during end diastole; therefore, open circuit)

43) Interpretation
   a) Increased PA pressures = volume overload, CHF
   b) Decreased PA pressures = volume depletion

44) Complications
   a) Infection
   b) Dysrhythmias
   c) Air embolus
   d) Thromboembolism
   e) PA rupture
   f) Pulmonary infarction

45) Cardiac Output Measurements
   a) Thermodilution method
   b) Newer methods for CCO
   c) Bioimpedence methods (noninvasive)

46) Equipment
   a) Closed system or syringes
b) NS or D5W

c) Cool versus room temperature injectate

d) CO computer

47) Measurement

a) Inject solution within 4 seconds
b) Increased accuracy at end expiration
c) Check waveform obtained
d) Repeat at least three measurements
e) Average values within 10% of each other
f) Calculate CO and cardiac index (computer does this)

48) Cardiac Output vs. Index

a) Index based on body size
b) Uses body surface area

49) SvO2 monitoring

a) Measurement of oxygen saturation in PA
b) Fiberoptic technique
c) Reflects CO, SaO2, Hgb, and VO2
d) Reflects overall tissue oxygenation
   i) Rate of greater than 60% is desirable
e) Can calculate oxygen delivery and consumption profiles

Class 4

1) Oxygen delivery/consumption and monitoring

2) Physiology of breathing

a) Rest:
   i) intrapleural pressure < atmospheric
   ii) intraalveolar = atmospheric
b) Inspiration:
   i) Intrapleural pressure more negative
   ii) Intraalveolar negative = airflow
c) Expiration:
   i) Passive when intrapulmonary pressure exceeds atmospheric

3) Gas Exchange

a) Ventilation = movement of O2 and CO2 in and out of the alveoli
b) Diffusion of O2 and CO2 occurs at pulmonary capillary
c) Oxygenated blood is perfused or transported to the tissues

4) Gas Exchange

a) Diffusion of O2 and CO2 occurs at the cellular level
b) Transport of CO2 to the right side of the heart

5) Regulation of Breathing

a) Respiration stimulated by elevated CO2
b) Not true for COPD; stimulus is hypoxia
   i) Rationale for low oxygen in patients with COPD
6) **Terminology**
   a) Work of breathing = effort

7) **Compliance**
   a) Distensibility or stretch
   b) Determined by elasticity, “recoil”
   c) Elastic recoil and compliance are inversely related

8) **Resistance**
   a) Opposition to gas flow in the airways

9) **LUNG VOLUMES and CAPACITIES**

10) **Tidal Volume (VT)**
    a) Normal breath
    b) Average 500 ml or 5 to 10 ml/kg.

11) **Inspiratory Reserve Volume (IRV)**
    a) Maximum amount of gas inspired at the end of a normal breath (over and above the VT)
    b) Average: 3000 ml

12) **Expiratory Reserve Volume (ERV)**
    a) Maximum amount of gas forcefully expired at the end of a normal breath
    b) Average 1100 mL

13) **Residual Volume (RV)**
    a) Amount of air remaining in the lungs after maximum expiration
    b) Average: 1200 mL

14) **Inspiratory Capacity (IC)**
    a) Maximum volume of gas inspired at normal resting expiration
    b) Average: 3500 ml
    c) IC=VT+IRV

15) **Functional Residual Capacity (FRC)**
    a) Volume of gas remaining in the lungs at normal resting expiration
    b) Average: 2300 mL
    c) FRC=ERV+RV

16) **Vital Capacity (VC)**
    a) Maximum volume of gas forcefully expired after maximum inspiration
    b) Average: 4600 mL
    c) VC=VT+IRV+ERV

17) **Total Lung Capacity (TLC)**
    a) Volume of gas in the lungs at the end of maximum inspiration
    b) Average: 5800 mL
    c) TLC = VT + IRV + ERV + RV

18) **Abnormal Breathing Patterns**
    a) Tachypnea: rate > 20
    b) Bradypnea: rate < 10
    c) Cheyne-stokes: cyclical with apneic periods
    d) Kussmaul’s: deep, regular, and rapid
    e) Biot’s: cluster breathing
    f) Apneustic: gasping inspirations

19) **Arterial Blood Gases**
a) Adequacy of oxygenation and ventilation  
b) Acid-base status  
c) Interpret in conjunction with  
   i) Clinical history  
   ii) Physical assessment  

20) Oxygenation  
a) \( \text{PaO}_2 \) Partial pressure of oxygen dissolved in arterial blood  
   i) Normal value 80 to 100 mm/Hg  
   ii) Decreases in elderly  
b) \( \text{SaO}_2 \) Amount of oxygen bound to hemoglobin  
   i) Normal value 93 to 99%  
   ii) Frequently measured via pulse oximetry  

21) Terms  
a) Hypoxemia—decreased oxygenation of arterial blood  
b) Hypoxia: decreased oxygenation at tissue level  

22) Oxyhemoglobin Dissociation Curve  

23) \( \text{PaO}_2 \) & \( \text{SaO}_2 \) Relationship  
a) Oxyhemoglobin dissociation curve  
b) Critical zone: \( \text{PaO}_2 < 60 \text{ mm/Hg} \)  
c) Shifts of oxyhemoglobin dissociation curve  
   i) Acidosis: Release of oxygen to tissues  
   ii) Alkalosis: hemoglobin holds on to oxygen  

24) Ventilation/Acid-Base Status  

25) \( \text{pH} \)—Concentration of Hydrogen Ions (H+)  
a) Normal range is 7.35 to 7.45  
   i) \( \text{pH} < 7.35 \) = acidosis  
   ii) \( \text{pH} > 7.45 \) = alkalosis  

26) \( \text{PaCO}_2 \)  
a) Partial pressure of carbon dioxide in arterial blood  
b) Normal value 35 to 45 mm/Hg  
   i) \( \text{PaCO}_2 > 45 \text{ mm/Hg} \) = Acidosis  
   ii) \( \text{PaCO}_2 < 35 \text{ mm/Hg} \) = Alkalosis  
c) Hint: Think of CO2 as an acid  

27) \( \text{HCO}_3^- \)—Bicarbonate  
a) Concentration of sodium bicarbonate in the blood  
b) Normal value is 22 to 26 meq / l.  
   i) \( \text{HCO}_3^- < 22 \) = Acidosis  
   ii) \( \text{HCO}_3^- > 26 \) = Alkalosis  
c) Hint: Think of HCO3 as a buffer or neutralizer  

28) Regulation of Acid-Base Balance  

29) Blood Buffer System  
a) Increased H+ ions combined with \( \text{HCO}_3^- \) to form Carbonic Acid (\( \text{H}_2\text{CO}_3 \))  
b) Carbonic Acid breaks down into \( \text{H}_2\text{O} \) and \( \text{CO}_2 \)  

30) Respiratory Buffer System  
a) Excretes excess \( \text{CO}_2 \) from system when metabolic disorder occurs
b) Immediate action

31) Renal Buffer System
   a) Excretes excess H+ ions and retains HCO₃
   b) Slow activation (up to 2 days)

32) Interpretation of ABGs
   a) STEP 1: Evaluate oxygenation
   b) STEP 2: Evaluate the pH
   c) STEP 3: Evaluate the acid-base status
   d) STEP 4: Determine the primary cause of the acid-base status
   e) STEP 5: Determine compensation

33) Compensation
   a) None
   b) Partial: mechanisms occurring; pH abnormal
   c) Complete: mechanisms occurring; pH normal range

34) Example—Resp Arrest
   a) pH
   b) PO₂
   c) PCO₂
   d) Bicarbonate
   e) 7.15
   f) 55 mm Hg
   g) 85 mm Hg
   h) 24 mEq/L

35) Assessment Of Oxygenation
   a) Pulse oximetry (SPO₂)
   b) Continuous ABG monitoring
   c) Periodic ABGs to assess PaO₂

36) Assessment of Ventilation
   a) End-tidal CO₂ (ETCO₂)
   b) Must compare with ABGs and use for trending

37) Airway Management
   a) Positioning
   b) Oral airways
   c) Nasal airways
   d) Oxygen delivery devices

38) Laryngoscopes & Blades

39) Endotracheal Tube

40) Endotracheal Intubation
   a) Ventilate during preparation
   b) Sniffing position
   c) Topical anesthetic/paralytic medication
   d) Eudotrachel tube (sizes 7.5 to 9.0 mm), lubricant, stylet (check balloon)
   e) Laryngoscope and blade

41) Airway Management of the Mechanically Ventilated Patient

42) Suctioning
a) How often? How should it be done to prevent hypoxemia?
b) Conventional
   i) Hyperinflation
   ii) Hyperoxygenation
   iii) Hyerventilation
c) Closed suction devices
d) Adapters
43) Suctioning: Research Issues
   a) Saline or no saline?
   b) Use of mucolytics
44) Manually Ventilate: Ambu
45) Reasons for Mechanical Ventilation
   a) Acute respiratory failure
   b) Impairment of:
      i) Alveolar ventilation
      ii) Pulmonary vascular perfusion
   c) Causes severe hypoxia
   d) Life-threatening
46) Indicators for Ventilation
   a) Hypoxemia: PO₂ < 60
   b) Hypercapnea: PCO₂ > 50
   c) Acidemia: pH < 7.25
   d) PCO₂ = PO₂
   e) RR > 35/min
47) Indicators for Ventilation
   a) VT < 3.5 ml/kg
   b) VC < 10 to 15 mL/kg
   c) NIF (NIP) < -20
      i) e.g., −5 or −10
      ii) Not able to generate enough negative pressure
   d) Worsening chest x-ray
   e) Signs and symptoms of hypoxia
48) Noninvasive Methods of Ventilation
49) Negative Pressure Ventilation
   a) Iron lung
   b) Cuirass
   c) Poncho
50) Noninvasive Positive Pressure Ventilation (NIPPV)
   a) Examples
      i) Nasal CPAP
      ii) Bi PAP
   b) Used in conjunction with portable ventilator
   c) Requirements
      i) Tight seal of mask or mouthpiece
      ii) Intact respiratory drive
iii) Able to protect airway

51) Volume-Targeted Ventilation
a) Ventilator set to allow air to flow until preset tidal volume is achieved
b) Commonly used in critical care settings

52) Pressure-Targeted Ventilation
a) Ventilator allows air to flow into lungs until a preset pressure is achieved
b) Tidal volumes vary
c) Risk of hypoventilation

53) Volume-Targeted Modes

54) Controlled Mechanical Ventilation (CMV)
a) Preset tidal volume at a preset respiratory rate
b) Risk of competition
c) For which patients would you consider using CMV?

55) Assist/Control Ventilation (A/C)
a) Preset tidal volume whenever the patient exerts a negative inspiratory effort
b) Helps to preserve respiratory muscle tone
c) Risk of hyperventilation and respiratory alkalosis

56) Synchronized Intermittent Mandatory Ventilation (SIMV)
a) Preset tidal volume at a preset respiratory rate
b) Permits patient to breathe spontaneously at own rate and depth in between the ventilator breaths
c) What are the risks of SIMV?

57) Pressure-Targeted Mode
a) Pressure support ventilation (PSV)
b) Provides pressure during inspiration
c) Decreases work of breathing
d) Used alone or with SIMV

58) Ventilation Adjuncts

59) Positive End Expiratory Pressure (PEEP)
a) Positive airway pressure to mechanically assisted breaths
b) Keeps airways open at end of expiration and increases FRC
c) Use to decrease amount of FiO₂ needed
d) Risk for barotrauma and decreased cardiac output

60) Continuous Positive Airway Pressure (CPAP)
a) Augments FRC in spontaneous breaths
b) Can be given via ventilator or by face mask in spontaneously breathing patients

61) Advanced Modes
a) Review table in book

62) Ventilator Settings
a) Tidal Volume (Vt): 10 to 15 ml/kg
   i) This amount currently being debated and lower values ordered
   ii) Allowing PaCO₂ to rise—permissive hypercapnea
b) Respiratory rate (RR): breaths/min by ventilator
c) Fraction of inspired oxygen (FiO₂): amount of oxygen delivered
d) PEEP/pressure support

63) Ventilator Settings
   a) Sigh: deeper breath; not routinely used
   b) Sensitivity: amount of effort needed to trigger the ventilator
   c) Inspiratory: expiratory ratio (I:E): 1:2
   d) Peak inspiratory pressure (PIP): highest amount of pressure needed to ventilate the patient; i.e., deliver the tidal volume
   e) Pressure limits: settings for the alarms

64) Barotrauma
   a) Pneumothorax and tension pneumothorax
   b) Signs and symptoms
   c) Nursing interventions

65) Intubation of the Right Main Stem

66) ETT Malposition / Extubation
   a) How do you assess for the presence of this condition?
   b) What interventions are appropriate if it is suspected?

67) Tracheal Damage
   a) Monitor cuff pressure

68) Damage to the Mouth and Nose
   a) How is this assessed?
   b) What interventions protect the mucosa?
   c) What are strategies for mouth care in intubated patients?

69) Associated with \(O_2\)
   a) Chronic lung disease
   b) Try to get FiO\(_2\) to lowest level possible

70) Acid-Base Disturbances
   a) Hypocapnia or hypercapnia
   b) How can these conditions be prevented?

71) Aspiration
   a) How can you prevent aspiration?
   b) What assessments indicate aspiration?
   c) Can a patient with a tracheostomy eat and drink? (swallowing studies recommended)

72) Infection
   a) Normal protective mechanisms bypassed by ETT tube
   b) What strategies can be used to prevent infection?

73) Ventilator Dependence/Inability to Wean

74) Low cardiac output
   a) Related to increased intrathoracic pressure
   b) Positive end expiratory pressure

75) Gastrointestinal
   a) Stress ulcer
   b) Nutrition

76) Endocrine
   a) Baroreceptor stimulation
   b) Release of ADH
c) Fluid retention

77) **Does My Patient Need to be Medicated?**

a) Analgesics: morphine
b) Sedatives: benzodiazepines, neuroleptics, and propofol
c) Neuromuscular blocking agents (NMBAs): paralytics
   i) Pancuronium, atracurium, and vecuronium
   ii) Many newer agents

78) **Nutritional Support**

a) Enteral feedings preferred
b) Protect from aspiration
c) Consider specialty formulas
d) May need formulas with lower carbohydrates content to reduce CO₂ production

79) **Troubleshooting**

a) Never shut alarms off; silence only
b) Manually ventilate if uncertain of problem

80) **Low -Volume Alarm**

a) Patient does not receive preset tidal volume

b) Causes
   i) Disconnection
   ii) Leak in cuff
   iii) Tube out of position

c) Fix the cause

81) **High -Pressure Alarm**

a) High pressure alarm sound if pre-set pressure limit is exceeded

b) Causes
   i) Secretions and mucus plugs
   ii) Kinks in ETT tube or tubing
   iii) Talking
   iv) Dysynchrony
   v) Barotrauma

82) **Weaning Patients from Ventilator**

a) Individualized decisions
b) Collaborative team effort

83) **Readiness to Wean**

a) Underlying cause for mechanical ventilation resolved
b) Hemodynamic stability; adequate cardiac output
c) Assessment tools available such as Burns Wean Assessment Program (BWAP)

84) **Readiness to Wean**

a) Adequate respiratory muscle strength
   i) Respiratory rate < 25 breaths/min
   ii) Negative inspiratory force > -20 cm H₂O
   iii) Spontaneous tidal volume 4 to 5 ml/kg
   iv) Vital capacity is 10 to 15 ml/kg
   v) Minute ventilation 5 to 10 l/min

85) **Readiness to Wean**
a) Adequate ABGs without a high FiO\textsubscript{2} and/or high PEEP
   i) PaO\textsubscript{2} > 60 mm Hg with FiO\textsubscript{2} < .5
   ii) PaCO\textsubscript{2} < 45 mm Hg
   iii) PEEP < 5 cm H\textsubscript{2}O
b) Adequate level of consciousness

86) **Readiness To Wean**
   a) Good nutritional status and hydration
   b) Absence of factors that impair weaning e.g., infection, anemia, fever
   c) Mental readiness
   d) Minimal need for medicines that cause respiratory depression

87) **Short-Term Ventilation Weaning**
   a) T-piece trials or CPAP
      i) Strengthens respiratory muscles
   b) Synchronized intermittent mechanical ventilation
   c) Can be extubated if able to sustain 1 hour of spontaneous breathing
   d) Observe for complications of extubation
      i) What are the complication?

88) **Long-Term Ventilation Weaning**
   a) Protocol-driven is more successful
   b) High-pressure, low-volume
      i) T-Piece or CPAP trials
      ii) Low IMV rates
      iii) Strengthen respiratory muscles
   c) Low pressure, high volume
      i) Pressure support ventilation
      ii) Strengthens endurance

89) **Stop the weaning process?**
   a) Respiratory rate > 25 to 30 or < 8 breaths/min
   b) Blood pressure increases to > 30 mm Hg or decreases to > 20 mm Hg
   c) Heart rate increases > 30 beats/min
   d) Dysrhythmias (e.g., PVCs)
   e) ST-segment elevation

90) **Stop the weaning process?**
   a) Significant decrease in spontaneous, V\textsubscript{T}
   b) Use of accessory muscles
   c) Labored respirations
   d) Diaphoresis
   e) Decreased level of consciousness
   f) Restlessness
   g) Anxiety

91) **Weaning Difficulties**
   a) ABGs
   b) Back on ventilator
   c) Identify cause
92) Terminal Weaning
   a) Indications for terminal weaning
   b) Strategies to assist the family with coping
   c) Interventions promote patient comfort

Class 5

1) Medications
   a) Emergency and Critical Care Drugs

2) Oxygen
   a) 100% oxygen
   b) Via Bag, mask, ETT

3) IV Access
   a) Large bore IVs
   b) Biggest veins
   c) May insert central line

4) Drugs & Alternative Administration
   a) Use of ET tube if needed: ALE
      i) Atropine
      ii) Lidocaine
      iii) Epinephrine

5) Fluids
   a) Normal saline
   b) First line defense
   c) Replace what is needed

6) Sympathetic Nervous System
   a) Fight or flight response
      i) Response comes from sympathetic fiber stimulation
      ii) Some effect from catecholamines

7) Sympathetic Nervous System
   a) Sympathetic receptors
      i) Alpha
         1) Vasoconstriction
         2) Increased contractility
      ii) Beta-1
         1) Increased HR
         2) Increased contractility

8) Sympathetic Nervous System
   a) Sympathetic receptors (cont’d)
      i) Beta-2
         1) Vasodilatation
         2) Relaxation of bronchial and GI smooth muscle
      ii) Dopaminergic
         1) Vasodilatation of renal, mesenteric, and cerebral vessels

9) Chemicals of the SNS
a) Catecholamines
   i) Epinephrine (adrenaline—from adrenal medulla)
   ii) Norepinephrine

10) Terms
    a) Sympathomimetic
    b) Catecholamines
    c) Adrenergic
    d) Combine with alpha and beta terms

11) Calculations
    a) Mg/min
    b) Mcg/kg/min

12) Epinephrine
    a) Sympathomimetic
    b) Ventricular fibrillation, pulseless ventricular tachycardia, asystole, PEA
    c) 1 mg. IV push every 3 to 5 minutes
    d) Can be given via ET tube
    e) Infusion if needed

13) Lidocaine
    a) Ventricular ectopy
    b) Bolus 1 to 1.5 mg/kg; additional bolus 0.5 to 0.75 mg/kg every 5 to 10 min up to 3 mg/kg
    c) Follow with infusion at 2 to 4 mg/min (250 mL 5% dextrose in water with 1 Gm)
       i) Concentration—1 mg/min = 15 ml/hour
    d) Assess for lidocaine toxicity

14) Atropine
    a) Asystole
       i) Start with 1 mg. IV push
       ii) Repeat every 3 to 5 minutes to maximum dose of 0.04 mg/kg
    b) Can be given via ET tube; 2-3 mg in 10 ml normal saline
    c) External pacer on standby

15) Atropine
    a) Anticholinergic
    b) Symptomatic bradycardia
       i) 0.5 mg every 3 to 5 min IV push
       ii) Maximum of 0.03 to 0.04 mg/kg

16) Amiodarone
    a) Unique drug; prolongs action potential and slows sinus rate
    b) Used for ventricular fibrillation and ventricular tachycardia refractory to treatment
    c) May also be given for atrial fibrillation or flutter
    d) IV push; follow with infusion if needed

17) Adenosine
    a) Miscellaneous antidysrhythmic agent
    b) Slows conduction through AV node
    c) Primary use for paroxysmal supraventricular tachycardia
    d) IV push; rapid; through port nearest insertion site of IV
    e) Half-life 10 seconds; duration 1 to 2 minutes
18) **Verapamil**
   a) Calcium-channel blocker
   b) Decreases fast rates
   c) Treat PSVT
   d) Contraindicated in PSVT if patient has Wolff Parkinson White syndrome
   e) IV bolus

19) **Diltiazem (Cardizem)**
   a) Calcium channel blocker
   b) Useful in PSVT, especially associated with atrial fibrillator or flutter
   c) IV bolus followed by infusion

20) **Magnesium**
   a) Refractory ventricular fibrillation
   b) Torsades de pointes (type of ventricular tachycardia)
   c) Known deficiency
   d) IV bolus followed by infusion titrated by magnesium levels

21) **Dopamine**
   a) Vasoactive (vasoconstrictor) to **increase** blood pressure
   b) Continuous drip
   c) 1 to 20 mcg/kg/min (learn calculations)

22) **Dopamine**
   a) Effects are dose-related
      i) Lower doses may increase renal perfusion (not supported by recent studies)
      ii) Moderate doses = cardiac doses
      iii) Higher doses = vasopressor doses
   b) Consider need for fluids versus dopamine

23) **Norepinephrine**
   a) Vasopressor
   b) Continuous infusion of 0.5 to 30 mcg/min
   c) Very potent

24) **Calcium Chloride**
   a) Underlying problem
      i) Hypocalcemia
      ii) Hyperkalemia
      iii) Calcium blocker toxicity
   b) IV push

25) **Sodium Bicarbonate**
   a) According to ABGs
   b) Rarely given

26) **Morphine Sulfate**
   a) Ischemic chest pain
   b) Pulmonary edema
      i) Increases venous capacitance

27) **Ventricular Fibrillation/ Pulseless VTach**
   a) ABCD
   b) Shock, shock, shock
c) 200, 300, 360 joules
d) Intubate
e) Epinephrine or vasopressin
f) Defibrillate

28) Ventricular Fibrillation / Pulseless V-Tachycardia
a) Drug-Shock Continues
   i) Epinephrine repeated as needed
   ii) Consider other drugs
       (1) Amiodarone
       (2) Lidocaine
       (3) Mg sulfate
       (4) Procainamide
       (5) Sodium bicarbonate

29) Symptomatic Tachycardia (Including VT)
   a) ABCD
   b) Airway, oxygen, IV access
   c) Sedation
   d) Cardioversion

30) Asystole
   a) ABCD
   b) Airway, oxygen, intubate, IV access
   c) Confirm in two leads
   d) Treat cause: hypoxia, hypo/hyperkalemia, acidosis, overdose, hypothermia
   e) Transcutaneous pacemaker
   f) Epinephrine
   g) Atropine

31) Pulseless Electrical Activity
   a) Rhythm without pulse ABCD
   b) Airway, oxygen, intubate, IV access
   c) Treat cause
   d) Epinephrine and atropine

32) Causes of PEA
   a) Hypovolemia
   b) Hypoxia
   c) Hydrogen ion (acidosis)
   d) Hyper/ hypokalemia
   e) Hypothermia
   f) Tablets (overdose)
   g) Tamponade
   h) Tension pneumothorax
   i) Thrombosis (AMI)
   j) Thrombosis (pulmonary Embolism)

33) Ventricular Ectopy
   a) Can also give:
      i) Procainamide
ii) Magnesium (if levels are low)

Class 6

1) Neurological Alterations
2) Central Nervous System
   a) Peripheral Nervous System
      i) 31 pairs of spinal nerves
      ii) 12 pairs of cranial serves
         (1) On old Olympus towering tops a Fin and German viewed some hops
   b) Autonomic Nervous System
      i) Motor nerves to viscera
      ii) Sympathetic and parasympathetic
3) Monro-Kellie Hypothesis
   a) Intracranial volume
      i) Volume of circulating blood
      ii) Volume of cerebrospinal fluid (CSF)
      iii) Volume of brain
4) Intracranial Pressure (ICP) Increases
   a) Alterations in intracranial contents
   b) Goal is to prevent ICP from rising
5) Examples of Alterations
   a) Increase in Circulating Blood
      i) Hypercarbia
      ii) Hypoxia
      iii) Aneurysm
      iv) Arteriovenous malformation
   b) Increase in CSF
      i) Hydrocephalus
6) Examples of Alterations
   a) Increase in brain volume
      i) Cerebral edema
   b) Extra “contents”
      i) Hematoma
      ii) Tumor
7) Intracranial Compliance
   a) Volume-pressure curve
   b) Compensatory mechanisms present = compliance
   c) Compensatory mechanisms fail = low compliance
      i) Small increase in volume
      ii) Causes large increase in pressure
8) Pressure volume curve
9) Normal ICP
   a) 4 to 15 mm Hg
10) **Cerebral Perfusion Pressure (CPP)**
   a) Mean arterial pressure (MAP) - ICP
   b) Average = 85 mm Hg
   c) Must be > 65 mm Hg to prevent cerebral anoxia

11) **Concern if ICP is High and/or MAP is Low**

12) **Compensatory Mechanisms**
   a) Accommodation
   b) Autoregulation

13) **Accommodation**
   a) Contents of intracranium change
      i) Decreased cerebral blood flow secondary to venous compression
      ii) Displacement of brain tissue
      iii) Decreased CSF secondary to:
         1. Increased reabsorption
         2. Translocation of fluid to spinal subarachnoid space

14) **Autoregulation**
   a) Automatic alteration in size of cerebral arterioles
   b) Temporary
   c) Sensitive to
      i) Changes in blood pressure
      ii) PaCO₂—high levels vasodilate
      iii) PaO₂—low levels vasodilate

15) **Etiology of Increased ICP (IICP)**
   a) Loss of compliance
   b) Altered cerebral blood flow
   c) Loss of autoregulation
   d) Obstructed venous outflow
   e) Cerebral edema

16) **Cerebral Edema**
   a) Swelling
   b) Cytotoxic—intracellular swelling; r/t hypoxia and low osmolality
   c) Vasogenic—breakdown of blood-brain barrier; trauma and tumor are common causes

17) **Herniation**
   a) Why is this a dangerous phenomenon?

18) **Cingulate**
   a) Lateral herniation of cerebral hemisphere across the midline
   b) Signs and symptoms vary
   c) Change in level of consciousness (LOC)

19) **Central**
   a) Downward displacement of cerebral hemisphere onto brainstem
   b) Change in LOC and respiration
   c) Small pupils
   d) Positive Babinski reflex
   e) Posturing and Cheyne-Stokes respirations are late signs

20) **Uncal**
a) *(Also known as transtentorial or supratentorial)*
b) Herniation of uncal portion of temporal lobe through the tentorial notch; compresses midbrain
c) Cranial nerve III: ipsilateral dilated pupil
d) Contralateral hemiplegia
e) Worsening: fixed midposition pupils, altered respirations, posturing
f) Quick progression to coma

21) *Cerebellar Tonsil*
a) Compression of brainstem by cerebellar tonsils
b) Cardiorespiratory compromise

22) **Assessment**
a) Glasgow Coma Scale (GCS)
b) Mental status
   i) Consciousness
   ii) Language
   iii) Memory

23) **Assessment**
a) Cranial nerves
   i) What can you assess in critically ill patients?
   ii) Pupillary, corneal, cough, gag, and swallow are routinely assessed

24) **Assessment**
a) Motor status
   i) Posturing
   ii) Babinski reflex

25) **Assessment**
a) Vital signs—LATE SIGNS

26) **Symptoms of IICP**
a) **CHANGE IN LEVEL OF CONSCIOUSNESS**
b) Headache
c) Pupillary changes
d) Nausea/vomiting
e) Papilledema

27) **Symptoms of IICP**
a) Seizures
b) Changes in vital signs *(late)*
   i) Slow, bounding pulse
   ii) Irregular respirations—depends on level of lesion
   iii) Increased systolic blood pressure with widening pulse pressure

28) **ICP Monitoring**
a) Various techniques
b) Used to monitor continuous ICP and CPP
c) Record on flow sheet and note trends
d) Fluid-filled (similar to hemodynamic set-up)
e) Newer fiberoptic and filament sensor systems available

29) **Methods of ICP Monitoring**
a) Probes, catheters, and bolts

30) **Intraparenchymal Sensor**
   a) Fiberoptics
   b) Minimal trauma to the brain
   c) No need to level the device
   d) Safe but expensive
   e) Fiberoptics can break
   f) Cannot withdraw fluid

31) **Epidural**
   a) Probes into epidural space
   b) Reduces risk of infection
   c) Questionable accuracy at high ICP

32) **Subarachnoid/Subdural**
   a) Bolt placed in subarachnoid space via drill
   b) Advantages
      i) Fairly accurate, easily placed
      ii) Small amounts of CSF can be sampled
   c) Disadvantages
      i) Clogs easily
      ii) Risk of infection

33) **Intraventricular**
   a) Catheter placed into lateral ventricle on nondominant side
   b) Level with foramen of Monro
   c) Advantages
      i) Most accurate
      ii) Can evaluate compliance
      iii) Can withdraw CSF in large amounts: ventriculostomy

34) **Intraventricular**
   a) Disadvantages
      i) Increased risk of infection
      ii) Increased risk of neurological deficit
      iii) Catheter may be difficult to place if ventricles are small
      iv) Frequent calibration needed to ensure accuracy

35) **Ventriculostomy**
   a) Drainage
      i) Continuous versus prn
   b) Level is very important

36) **Nursing Care: ICP Monitoring**
   a) Maintain closed system
   b) Monitor for infection
   c) Change dry sterile gauze according to protocol
   d) Level with ventricles
   e) Zero and calibrate frequently
   f) Record ICP and CPP (e.g. every 1 hour)

37) **Monitor Waveforms**
a) Normal
   i) Looks similar to PA
   ii) Mean value; 4 to 15 mm Hg is normal

b) Others (need *trending* to view)
   i) A waves
   ii) B waves
   iii) C waves

38) A Waves
   a) Elevations for 15 to 20 minutes
   b) Dangerous

39) B Waves
   a) Change with respirations
   b) Potentially dangerous

40) C Waves
   a) Small change with blood pressure or respirations
   b) Not significant

41) Other Neurological Monitoring
   a) Jugular bulb oxygen saturation gives global picture of cerebral oxygenation
   b) Fiberoptic system placed into jugular bulb
   c) Measures oxygen saturation
   d) < 50% suggests cerebral ischemia

42) Other Neurological Monitoring
   a) Continuous EEG monitoring
   b) Transcranial Doppler—cerebral blood flow
   c) Evoked potential

43) Neurological Diagnostics
   a) Computerized tomography of the head
   b) Skull x-rays
   c) Magnetic resonance imaging
   d) EEG
   e) CBF studies
   f) Cerebral arteriogram
   g) Culture of CSF

44) Nursing Management of IICP
   a) Avoid precipitating factors
   b) Schedule rest periods between activities
   c) Assess response to visitors
   d) Ongoing assessment
      i) Document
      ii) Report changes

45) Factors Which May Precipitate IICP
   a) Supply and demand or flow vs drainage

46) Increased Cerebral Blood Flow
   a) Hypercapnia
   b) Hypoxia (e.g. suctioning)
c) Fluid overload

47) Jugular Drainage Obstruct
   a) Neck flexion, hyperextension
   b) Neck swelling
   c) Tight tracheostomy ties

48) Increased Intrathoracic Pressure
   a) Positive pressure ventilation
   b) Positive end-expiratory pressure
   c) Valsalva maneuver

49) Increased Oxygen Demand — Increased CBF
   a) Seizures
   b) Hyperthermia
   c) Shivering
   d) Hyperactivity
   e) Pain

50) Medical Management of ICP
   a) Goal ICP: < 20 mm Hg
   b) CPP: 65 to 70 mm Hg

51) Airway/Hyperventilation
   a) Intubate and ventilate as needed
   b) Goals
      i) PaO₂: 100 mm Hg
      ii) PaCO₂: 35-45 mm Hg—current controversy as to how much to lower

52) Diuretics
   a) Osmotic diuretics — mannitol
      i) Withdraw fluid from normal tissue
      ii) Work in 20 minutes
      iii) May cause rebound cerebral edema
   b) Loop diuretics — furosemide
      i) Reduce rate of CSF production

53) Corticosteroids
   a) Dexamethasone (Decadron)
   b) Actions and effectiveness questionable
      i) Stabilize cell membranes
   c) Only documented effectiveness is with brain tumors

54) Oxygenation
   a) Short term
   b) Long term

55) Blood Pressure Control
   a) Avoid both hypotension and hypertension
   b) Keep systolic blood pressure < 160 mm Hg
      i) Except in management of vasospasms associated with subarachnoid hemorrhage—
         higher blood pressure may be required

56) Fluid Restriction
   a) 75 to 100 mL/hr
b) Except in management of vasospasms associated with subarachnoid hemorrhage—these patients often need higher rates to prevent vasospasms

57) Control Cerebral Metabolism
   a) Treat fever
   b) Seizure prophylaxis
   c) Sedation
      i) Propofol is newer agent; short-acting
   d) Neuromuscular blockade
      i) Always sedate as well

58) Control Cerebral Metabolism
   a) Barbiturate coma—pentobarbital
   b) Actions
      i) Decreases metabolism
      ii) Decreases cerebral edema
      iii) Better CBF
   c) Best when used early to manage ICP refractory to standard treatment
   d) Related nursing care—TOTAL CARE

59) Medical Management
   a) Surgical interventions may be needed as determined by diagnosis

60) Head Injury
   a) The leading cause of death in 1-44yr olds

61) Etiology
   a) Motor vehicle accidents
   b) Falls
   c) Assaults
   d) Injury + alcohol

62) Mechanisms of Injury
   a) Closed head—scalp intact
   b) Acceleration/deceleration
   c) Coup-contrecoup

63) Scalp Lacerations
   a) Break in scalp; risk for infections
   b) Scalp is very vascular

64) Skull Fractures
   a) Oh my head!

65) Linear
   a) Simple crack on X-Ray
   b) Symptoms may include swollen, ecchymotic areas over scalp
   c) May lead to epidural hematoma if in temporoparietal area; signs would include increased ICP

66) Depressed
   a) Broken bones may penetrate meninges or brain tissue
   b) Types include:
      i) Closed with scalp intact
      ii) Compound; scalp open but dura intact
iii) Complex; dura lacerated by bone fragments
c) Symptoms vary
d) Skull depressed
e) Dura may be intact, bruised, or torn
f) Complications may include:
   i) Laceration of brain
   ii) Intracranial hemorrhage
   iii) Infection
g) Surgical treatment needed

67) Basilar
   a) Fracture at base of skull into either anterior, middle, or posterior fossa
   b) Usually not seen on X-Ray

68) Brain Injury
   a) Primary and Secondary

69) Primary Injury
   a) Direct injury to the brain
   b) Coup-contrecoup injury
   c) Concussion, contusion, diffuse axonal, and penetrating

70) Concussion
   a) Transient loss of consciousness
   b) Reversible

71) Cerebral Contusion
   a) Bruising of the brain
   b) Signs, symptoms, and severity vary, depending on location of injury

72) Diffuse Axonal Injury
   a) Shearing
   b) Prolonged coma
   c) Intense rehabilitation

73) Penetrating Injury
   a) Knife, gunshot, ice picks, etc.
   b) Signs and symptoms vary according to location and extent of injury
   c) What are the problems associated with penetrating injury?

74) Hematomas
   a) Collections of blood where it doesn’t belong

75) Epidural Hematoma
   a) Blood clot between dura and skull; frequently in temporal area
   b) Laceration of middle meningeal artery or veins
   c) Develops rapidly, in minutes to hours (usually within first 24 to 48 hours)

76) Epidural Signs and Symptoms
   a) Lucid interval often precedes neurologic decline
   b) Early signs—headache, irritability, restlessness
   c) Typical later signs—from brainstem compression
      i) Ipsilateral dilated pupil
      ii) Contralateral hemiparesis
      iii) Decerebration
iv) Progressive unconsciousness

77) **Management of Epidural Hematoma**
   a) Surgical evacuation through burr hole
   b) SURGERY MUST BE PERFORMED EARLY

78) **Subdural Hematoma**
   a) Clot within subdural space; may be bilateral
   b) Usually from torn cortical vein
   c) May be multiple and associated with contusions

79) **Onset of Subdural Hematoma**
   a) Acute—Develops rapidly within minutes to 48 hours
   b) Subacute—Occurs 2 days to 2 weeks after injury
   c) Chronic—Occurs weeks to months after injury

80) **Signs of Subdural Hematoma**
   a) Acute—signs and symptoms are often the same as epidural hematoma
   b) Lucid interval often precedes neurologic decline
   c) Early signs—headache, irritability, restlessness
   d) Progressive unconsciousness

81) **Management**
   a) Surgical evacuation through burr hole
   b) Acute—SURGERY MUST BE DONE EARLY; includes excision of hematoma, resection of contused brain, and draining

82) **Miscellaneous Information**
   a) Acute subdural
   b) Postoperative cerebral edema common; may be refractory to treatment
   c) Chronic subdural—frequently attributed to other causes such as senility or alcohol; may have problems with gait

83) **Intracerebral Hematoma**
   a) Clot deep within brain tissue
   b) May occur from trauma, ruptured aneurysm, AVM, vascular tumor, or ruptured vessels from hypertension
   c) Varied signs and symptoms

84) **Secondary Brain Injury**
   a) Occurs secondary to another problem
   b) Examples
      i) Hypoxia
      ii) Fluid and electrolyte imbalance
      iii) Hypotension

85) **Nursing Interventions**
   a) Maintain patent airway
   b) May need intubation
   c) Maintain ventilation and oxygenation
   d) Monitor for shock
   e) Monitor neurological status—obtain good baseline
   f) Provide support to patient and family

86) **Nursing Interventions**
a) Continue care plan for increased ICP; monitor and control ICP  
b) Control pain and agitation with extreme caution  
c) Monitor fluid and electrolyte status  
d) Maintain normal temperature  
e) Prevent and/or treat seizure activity  
f) Prevent infection  
g) Provide cognitive retraining  

87) Spinal Cord Injury (SCI)  
a) Etiology  
   i) Hypoxia  
   ii) Edema  
   iii) Biochemical changes/vasoactive substances  
   iv) Hemorrhage  
   v) Ischemia  

88) Priority Assessments  
a) Airway and ventilation  
b) Paralysis of diaphragm and intercostal muscles will result in ineffective breathing patterns  
   i) C1-C3: ventilator dependent  
   ii) C4-C5: may or may not need ventilator  
   iii) Below C-5: have intact diaphragmatic breathing  

89) Assessment of SCI  
a) Entire body—motor/sensory  
b) Reflexes  
c) Spinal shock  
d) Sensory/motor loss  
e) Autonomic dysfunction  
f) Bowel/bladder dysfunction  
g) Spinal shock  

90) Spinal Shock  
a) Common with complete lesions  
b) Decreased venous return  
c) Venous stasis  
d) Hypotension  
e) Bradycardia  

91) SCI Lesions  
a) Complete  
b) Incomplete  
   i) Anterior cord syndrome  
   ii) Central cord syndrome  
   iii) Brown-Sequard’s syndrome  

92) Functional Ability r/t Level of Lesion  
a) Higher level - greater deficit  

93) Emergency Management of SCI  
a) Ventilation/perfusion  
b) Stabilization
i) Tongs  
ii) Surgery  
iii) Halo  
c) Corticosteroid protocol  
d) Kinetic bed—Why?

94) **Nursing Care: SCI**  
   a) Oxygenation  
   b) Potential for ineffective airway clearance  
   c) Potential for decreased cardiac output r/t spinal shock  
   d) Fluids  
   e) Drugs  
   f) Improve venous return  
   g) Long-term rehabilitation needs  

95) **Autonomic Dysreflexia**  
   a) Occurs T6 or above after resolution of spinal shock  
   b) Intense sympathetic response to stimuli  
      i) Kinked catheter  
      ii) Impaction  
   c) Severe hypertension, headache, and bradycardia  
   d) Assess and remove the cause

96) **Cerebrovascular Disease**  
   a) Stroke  
      i) Occlusive/ischemic  
      ii) Hemorrhagic  
   b) Brain Attack

97) **Occlusive Stroke**  
   a) Caused by thrombus  
   b) Early thrombolitics have reduced mortality and morbidity

98) **Hemorrhagic Stroke**  
   a) Most comonly caused by ruptured aneurysm—hypertensive crisis  
   b) May also be associated with AVM  
   c) Must *not* be treated with thrombolitics  
   d) Result in SAH

99) **Classic Stroke Symptoms**  
   a) Hemiparesis or weakness  
   b) Slurred speech  
   c) Confusion  
   d) Visual disturbances

100) **Assessment is Essential**  
   a) Must differentiate occlusive from hemorrhagic types  
   b) Emergent CT  
   c) *Onset* of symptoms must be established

101) **Thrombolitics**  
   i) Only effective if given within 3 hours of *Onset*  
   ii) t-PA is used (similar to AMI)
102) **Intracranial Aneurysms**
   a) Commonly occur at bifurcations in the circle of Willis
   b) Many are congenital; often precipitated by hypertension
   c) Patient usually well until rupture occurs

103) **Signs and Symptoms**
   a) Headache
   b) Seizures
   c) Change in sensorium
   d) Nuchal rigidity

104) **Preventing Vasospasms**
   a) Fluids
   b) Adequate blood pressure
   c) Calcium channel blockers—nimodipine
   d) Vasopressors as needed

105) **Nursing Care Aneurysms**
   a) Assess ICP
   b) Complete bedrest
   c) Elevate the head of the bed
   d) Quiet, dark environment
   e) Avoid straining

106) **Management of Aneurysms**
   a) Keep blood pressure at prerupture levels
   b) Medication for headache
   c) Prepare for surgical repair

107) **Transient Cerebral Ischemia**
   a) Patients have early symptoms that can progress to stroke
   b) May need carotid endarterectomy

108) **Seizures / Status Epilepticus**
   a) Seizures lasting more than 30 minutes
   b) Common with nonadherence to medical treatment for seizures
   c) Prolonged seizures deplete energy stores and lead to hypoglycemia, lactic acidosis, and cerebral edema

109) **Interventions**
   a) Maintain airway and ventilation
   b) STOP SEIZURE ACTIVITY
   c) First line
      i) Diazepam (Valium)
      ii) Lorazepam (Ativan)
   d) Second line
      i) Phenobarbital
      ii) Phenytoin (Dilantin)
      iii) Fosphenytoin

110) **Interventions**
   a) Maintain therapeutic levels of drugs

iii) Increased risk of bleeding
b) Prevent injury  
c) Assess and treat causes  
d) Prevent complications (e.g., aspiration, hyperthermia)  

**Class 7**

1) **Cardiac Alterations**
2) **Overview of Coronary Artery Disease (CAD)**  
   a) Progressive narrowing of coronary arteries by atherosclerosis  
3) **Pathophysiology**  
   a) Injury to Endothelium  
4) **Fatty Streak**  
   a) Lipoproteins enter intima  
   b) Yellowish, flat, lipid-rich  
   c) Monocytes develop into macrophages  
   d) Lipid-rich cells are called "foam cells"  
5) **Fibrous Plaque (Atheroma)**  
   a) White, pearly elevations of intimal lining  
   b) Damage to intima liberates platelet derived growth factor  
   c) Proliferation of smooth muscle cells  
   d) Stimulates movement of smooth muscle cells from media to intimal layer  
6) **Fibrous Plaque (Atheroma)**  
   a) Fibrous cap forms from connective tissue and low-density lipoprotein (LDL)  
   b) Necrotic core forms from inadequate blood flow  
7) **Advanced Lesion**  
   a) Rupture of the fibrous cap  
   b) Thrombus formation with gradual increase in size  
   c) Rupture of plaque starts clotting cascade  
8) **Platelet Changes at Site**  
   a) Adhesion—platelets bind to receptors  
   b) Activation—platelets change shape and activate receptors  
      i) Release: Thromboxane A2 and serotonin  
      ii) Activate glycoprotein IIb/IIIa receptors  
   c) Aggregation—platelets clump together  
   d) Changes cause growing thrombus—drugs such as ASA and Gp IIb/IIIa inhibitors used to stop the process  
9) **Platelet Changes**  
   a) Cause thrombus to grow  
   b) Drugs such as acetylsalicylic acid (ASA) and glycoprotein IIb/IIIa inhibitors used to stop the process  
10) **Risk Factors for CAD**  
   a) Age (men > 45; women > 55)  
   b) Family history  
   c) Cholesterol (high LDL or low high-density lipoproteins [HDL])
d) Smoking
e) Hypertension
f) Diabetes
g) Overweight
h) Inactivity

11) **Cholesterol in CAD**
   a) Categories
      i) HDL — good
      ii) LDL — bad
      iii) Very low-density lipoproteins (VLDL) — ???

12) **Recommendations**
   a) Total cholesterol < 200 mg/dL is desirable
   b) Need *fasting* levels for accuracy

13) **LDL Target Levels (NCEP)**
   a) No CHD and < 2 risks, 160 mg/dL
   b) No CHD and ≥ 2 risks, 130 mg/dL
   c) CHD, 100 mg/dL
   d) **What are the implications of these target levels?**

14) **Treatment**
   a) Diet: low cholesterol, low salt
   b) Exercise: aerobic
   c) Medications: lipid-lowering agents

15) **Medications for Hyperlipidemia**
   a) HMG CoA reductase inhibitors; slow production—most effective
      i) Statins
   b) Bile acid resins; bind and excreted via bowel
      i) Cholestyramine
      ii) Colestipol

16) **Medications for Hyperlipidemia**
   a) Nicotinic acid; inhibits LDL synthesis and increases HDL; many side effects
   b) Fibric acid derivatives; increase VLDL clearance
      i) Gemfibrozil

17) **Medications for Hyperlipidemia**
   a) Patients carefully monitored
   b) Combination therapy may be needed to reach target goals

18) **Medications Affecting Platelets**
   a) ASA (inhibits Thromboxane A2)
   b) Others: dipyridamole, ticlopidine, clopidogrel

19) **Diagnostic Tests**
   a) Symptoms (typical versus atypical)
   b) 12-lead ECG
   c) Holter monitoring
   d) ET tube (stress — with or without medications)
   e) Heart scans/imaging
   f) Serum enzymes/troponin
g) Cardiac arteriography

20) Cardiac Enzymes
   a) Enzymes released with tissue death
   b) CK (total)
      i) 2 to 6 hrs; peak 18 to 36 hrs
   c) CK-MB (cardiac specific)
      i) 4 to 8 hrs; peak 18 to 24 hrs
   d) Troponin I and T
      i) As early as 1 hour after injury
   e) Myoglobin
      i) 30 to 60 minutes after injury

21) Cardiac Catheterization / Arteriography
   a) Catheter (right or left)
      i) Heart pressures (similar to PA catheter)
      ii) Cardiac output
   b) Arteriography
      i) Visualize blood vessels

22) Post-Catheterization Management
   a) Bedrest; head of bed no higher than 30 degrees
   b) Monitor bleeding; newer collagen agents for hemostasis may be used
   c) Monitor pulses
   d) Antiplatelet drugs after the procedure (usually after interventions such as PCI)
   e) May be discharged in 6 to 8 hours; depends on diagnosis and procedures done in catheterization laboratory

23) Outcomes of Atherosclerosis
   a) Asymptomatic
   b) Angina
      i) Stable
   c) Acute coronary syndromes
      i) Unstable angina
      ii) Acute myocardial infarction (AMI)
         (1) Q-wave
         (2) Non Q-wave

24) Supply and Demand
   a) Delicate balance
   b) Degree of CAD lesions
   c) Collateral circulation
   d) Energy expenditure

25) Chest Pain
   a) Classic symptom
   b) Often midsternal
   c) May radiate to arms, jaw, or back

26) Angina Pectoris
   a) Myocardial ischemia: demand higher than supply
   b) Types
i) Stable (chronic exertional) = effort, classic
ii) Unstable (crescendo) = more often and severe, less relief
iii) Variant = Prinzmetal’s; associated with vasospasm; marked ST elevation during attack

27) Angina Pectoris
   a) Stable
      i) T-wave inversion on ECG
      ii) Rx: rest and nitroglycerin
   b) Unstable
      i) May see ST elevation on ECG
      ii) Rx: rest and nitroglycerin; drugs affecting platelets; revascularization
   c) Variant = Prinzmetal’s
      i) ST elevation during pain episodes
      ii) Rx: Calcium-channel blockers

28) Nursing Management: Angina
   a) Maintain cardiac output
   b) Pain relief
   c) Self-care; risk-factor modification

29) Myocardial Infarction
   a) Causes
      i) Platelet aggregation/thrombus
      ii) Spasm
   b) Types
      i) Q-wave: total occlusion of coronary artery with thrombus
      ii) Non Q-wave: partial occlusion of coronary artery

30) Symptoms of AMI
   a) Midsternal chest pain
      i) Severe, crushing, squeezing, pressure
      ii) May radiate
      iii) Unrelieved with nitrates
   b) Pale and diaphoretic
   c) Dyspnea
   d) Syncope
   e) Nausea and vomiting
   f) Dysrhythmias

31) Diagnosis of AMI
   a) Signs and symptoms; be alert for atypical
   b) 12-lead: ST elevation followed by Q-wave (Q-wave MI); ST depression (non Q-wave MI)
   c) Elevated cardiac enzymes: CPK-MB
   d) Elevated serum troponin I/T, myoglobin

32) Critical Thinking Challenge
   a) Describe people who may have atypical signs and symptoms of AMI

33) Nursing Goals: AMI
a) Maintain cardiac output
b) Treat pain
c) Assess for complications
d) Increase activity tolerance
e) Relieve anxiety
f) Ongoing and discharge teaching

34) **Complications of AMI**
a) Dysrhythmias
b) Sudden death
c) Congestive heart failure; cardiogenic shock
d) Ventricular aneurysm/rupture
e) Papillary muscle dysfunction
f) Pericarditis
g) Dressler’s syndrome

35) **Medical Management: AMI**
a) Emergent revascularization
   i) Thrombolysis
   ii) Primary angioplasty

36) **Medical Management: AMI**
a) Pain relief—MS, nitroglycerin
b) Oxygen
c) Drugs affecting platelets—ASA, glycoprotein IIb/IIIa inhibitors
d) Beta blockers
e) Nitrates
f) ACE inhibitors

37) **Thrombolytic Therapy**
a) Time is muscle; 6-hour window
b) Drugs
   i) Tissue plasminogen activator (t-PA)
   ii) Streptokinase
c) Heparin and glycoprotein IIb/IIIa inhibitors used in conjunction with thrombolytics

38) **Thrombolytic Therapy**
a) Drugs must be initiated as soon as possible after diagnosis
b) Newer drugs approved that can be given in 2 bolus doses

39) **Percutaneous Interventions**
a) Angioplasty (PCA)
b) Stent placement
c) Atherectomy
d) Many procedures combine PCA and stent

40) **Percutaneous Transluminal Coronary Angioplasty (PTCA) Procedure**
a) Guiding catheter, dilating catheter
b) Balloon inflated with pressure for 15 to 30 seconds
c) During procedure: IV nitroglycerin, IV heparin, possible newer agents
d) After procedure: sheaths in 4 to 6 hours, antiplatelet drugs, glycoprotein IIb/IIIa inhibitors
41) Stents
   a) Tubes placed in conjunction with angioplasty to keep vessel patent
   b) Help prevent the restenosis associated with angioplasty
42) Atherectomy/Rotoblater
   a) Complex lesions
43) Surgical Revascularization
   a) Coronary artery bypass graft (CABG) surgery
   b) Minimally invasive cardiac (Mid-CAB) surgery
   c) Transmyocardial laser revascularization (TMR)
44) CABG Surgery
   a) Provides additional conduits for blood flow
      i) Saphenous vein
      ii) Internal mammary artery
      iii) Radial artery
   b) Why are arterial conduits being used?
45) Goals of CABG Surgery
   a) Increase blood flow to myocardium
   b) Relieve symptoms
   c) Prolong survival
   d) Improve quality of life
      i) Issues related to older adult patients
46) Indications
   a) Unstable angina
   b) Acute MI
   c) Failure of percutaneous interventions
47) Risks
   a) Increased mortality associated with:
      i) Left ventricle dysfunction
      ii) Emergency surgery
      iii) Age
      iv) Sex (female)
      v) # diseased vessels
      vi) Decreased ejection fraction with congestive heart failure
48) Traditional CABG
   a) Median sternotomy / sternum split
   b) Excision of pericardium
   c) Cardiopulmonary bypass
   d) Myocardial preservation/ cardioplegia
   e) Grafts
   f) Wean bypass; defibrillate if needed
   g) Mediastinal and chest tubes
   h) Epicardial pacing wires
   i) Wire sternum
49) Complications of CABG
   a) Dysrhythmias
b) Impaired contractility; low cardiac output

c) Intra-operative MI

d) Pericardial tamponade

e) Respiratory insufficiency

f) Pain

g) Emboli; stroke

50) **Complications of Cardiac Surgery**

a) Renal impairment

b) Gastrointestinal dysfunction

c) Impaired peripheral circulation

d) Infection—very serious if sternal

e) Post-cardiotomy delirium

f) Poor nutrition status

51) **Mid-CAB**

a) Minimally invasive CABG

b) “Peripheral” bypass with balloon cross-clamping of aorta

c) Fewer complications and quicker recovery

52) **TMR**

a) Laser channels into ventricle created

b) Goal is to increase perfusion of heart muscle from inside out

c) Relief of symptoms occurs over time

d) Mixed results from clinical trials

53) **Critical Thinking Challenge**

a) Patients are transferred to step-down units soon after CABG; what interventions are important to prevent pulmonary complications?

54) **Dysrhythmias**

a) Additional treatments

   i) Medications

   ii) Pacemakers

   iii) Implantable cardioverter-defibrillators (ICD)

   iv) Ablation

55) **Permanent Pacemakers**

a) Modified generic code

b) Can pace atria, ventricles, or dual chamber

56) **Cardioverter/Defibrillator (ICD)**

a) To treat survivors of sudden death or those at high risk

b) Some also have pacemakers built in

57) **Ablation**

a) Used to treat patients with dysrhythmias related to extra accessory pathways

b) Radiofrequency used to eliminate the pathways

58) **Critical Thinking Challenge**

a) Patients need to have electrophysiological studies (EPS) before implantation of ICDs and ablation

59) **Heart Failure**

a) Inability to generate adequate flow
b) Systolic (impaired contractility)
c) Diastolic (impaired filling)
d) Left versus right
   i) Left usually occurs first

60) **Etiology**
   a) Acute myocardial infarction
   b) Hypertension
   c) Idiopathic; cardiomyopathy
   d) Valvular disease

61) **Symptoms: Left**
   a) **Pulmonary**
   b) Crackles; gurgles
   c) Cyanosis
   d) Dyspnea
   e) Hypoxemia
   f) S3

62) **Symptoms: Right**
   a) **Systemic**
   b) Edema
   c) Jugular venous distention
   d) Liver engorgement

63) **Heart Failure Treatment**
   a) Diuretics
   b) ACE inhibitors/angiotensin-receptor blockers
   c) Digoxin
   d) Beta blockers (carvedilol; Coreg)
   e) Calcium-channel blockers (diastolic failure)

64) **Management of Congestive Heart Failure**
   a) Diet: low salt
   b) Exercise
   c) Daily weight
   d) Diuretic regulation

65) **Pulmonary Edema**
   a) *Acute, life-threatening* congestive heart failure
   b) Dyspnea
   c) Cyanosis
   d) Gurgles
   e) Pink, frothy sputum
   f) Hypoxemia

66) **Pulmonary Edema**
   a) Oxygen
   b) Possible intubation
   c) Diuretics
   d) Morphine
   e) Assess/rx cause
67) **Inflammatory Heart Disease**
   a) Endocarditis—inflammation of the endocardium
   b) Pericarditis—inflammation of the pericardium

68) **Infective Endocarditis**
   a) Endocardium continuous with valves
   b) Vegetation
   c) Embolization
   d) Valvular dysfunction

69) **Infective Endocarditis**
   a) DX—echocardiogram, transesophageal echocardiography
   b) RX—antibiotics, rest
   c) Prevention—Antibiotic prescription before treatments (e.g., dentist)
      i) Heart valve
      ii) History of endocarditis
      iii) Microvascular pressure with regurgitation

70) **Pericarditis**
   a) Acute versus chronic
   b) Etiology
      i) After a myocardial infarction
      ii) Uremia
      iii) Cancers
   c) Can lead to infusion, tamponade, and scarring

71) **Hallmarks of Pericarditis**
   a) Friction rub
   b) Pulsus paradoxus
   c) Initial ST elevation

72) **Summary: Nursing Measures for the Cardiac Patient**
   a) Education
   b) Diet
   c) Balance of exercise and rest
   d) Psychosocial
   e) Sex

**Class 8**

1) **Acute Respiratory Failure (ARF)**
   a) Altered gas exchange (room air)
      i) \( \text{PaO}_2 < 60 \text{ mm Hg} \)
      ii) \( \text{PaO}_2 > 50 \text{ mm Hg} \)
      iii) \( \text{pH} \leq 7.30 \)

2) **Etiology of ARF**
   a) Failure of oxygenation
   b) Failure of ventilation
   c) Both of the above
   d) Develops RAPIDLY
3) Hypoventilation
   a) Reduced alveolar ventilation
      i) Drug overdose
      ii) Central nervous system (CNS) disorders
      iii) Thoracic or abdominal surgery
   b) Also causes increase in PaCO₂

4) Intrapulmonary Shunting
   a) Blood shunted from right to left side of heart without oxygenation
   b) Qs/Qt disturbance
   c) Causes: atrial or ventricular septal defect, atelectasis, pneumonia, pulmonary edema

5) V/Q Mismatch
   a) Most common cause of low O₂
   b) Normal ventilation (V) is 4 L/min
   c) Normal perfusion (Q) is 5 L/min
   d) Normal V/Q ratio is 4/5 or .8
   e) A mismatch occurs if either
      i) V is decreased
      ii) Q is decreased

6) Diffusion Defects
   a) Diffusion of O₂ and CO₂ does not occur
      i) Fluid in alveoli
      ii) Pulmonary fibrosis

7) Other Problems of Low O₂
   a) Low cardiac output
   b) Low hemoglobin level
   c) Low barometric pressure (altitude)
   d) Tissue hypoxia and acidosis

8) Failure of Ventilation
   a) Hypercapnia
   b) Related to:
      i) Alveolar hypoventilation—decrease in ventilation and hypoxemia
      ii) V/Q mismatch

9) Assessment of ARF
   a) Neurologic—CNS shows earliest signs of hypoxemia and hypercapnia
   b) Respiratory
   c) Cardiovascular
   d) Nutrition
   e) Psychosocial
   f) Chest x-ray
   g) Pulmonary function tests
   h) ABGs
      i) Pulse oximetry and end tidal CO₂

10) Interventions for ARF
    a) Maintain airway—ET tube or tracheostomy may be needed
    b) Optimize oxygen delivery
c) Reduce oxygen demands
d) Treat the cause
e) Prevent complications

11) Medical Mgt for ARF
   a) Oxygen
   b) Bronchodilators
   c) Corticosteroids
   d) Sedation
   e) Transfusions
   f) Therapeutic paralysis
   g) Nutritional support
   h) Hemodynamic monitoring

12) ARDS
   a) Non-cardiogenic pulmonary edema
   b) Diagnostic criteria (Bernard, 1994)
      i) \( \text{PaO}_2/\text{FiO}_2 \) ratio of less than 200
      ii) Bilateral infiltrates
      iii) Pulmonary capillary wedge pressure < 18 mm Hg
   c) Acute lung injury scoring can be helpful

13) ARDS Pathophysiology
   a) Insult—systemic inflammatory response (SIRS)
   b) Release of inflammatory mediators
   c) Damage to alveolar-capillary membrane
   d) Increased capillary permeability
   e) Pulmonary edema (noncardiogenic)

14) ARDS Pathophysiology
   a) Microatelectasis
   b) Decreased compliance (stiff lungs)
   c) Decreased surfactant (damage to Type II pneumocytes)
   d) Impaired gas exchange
   e) V/Q mismatch

15) Symptoms of ARDS
   a) Insidious
   b) Symptoms of hypoxemia
   c) Respiratory alkalosis
   d) Worsening chest x-rays that progress to “white out”
   e) Increased PIP on ventilation
   f) Eventual severe hypoxemia

16) Treatment of ARDS
   a) Treat the cause
   b) Oxygenation and ventilation
      i) Positive end-expiratory pressure (PEEP)
      ii) Possible non-traditional modes of ventilation: high-frequency, pressure-control, inverse-ratio
17) **Treatment of ARDS**
   a) Comfort
      i) Sedation
      ii) Pain relief
      iii) Neuromuscular blockade
   b) Decrease O₂ consumption
   c) Positioning
      i) Prone positioning shows good results
      ii) Continuous lateral rotation therapy

18) **Treatment of ARDS**
   a) Fluid and electrolyte balance
   b) Adequate nutrition
   c) Psychosocial support

19) **ARDS—Experimental Therapy**
   a) Inhaled nitric oxide
   b) Liquid ventilation
   c) Extracorporeal lung assist
   d) Surfactant (works well in premature infants)
   e) Inflammatory mediators

20) **ARDS**
   a) **Be alert for complications**
      i) Multiple-organ dysfunction syndrome
      ii) Renal failure
      iii) Disseminated intravascular coagulation
      iv) Long-term pulmonary effects associated with high oxygen and other therapies

21) **ARF in Chronic Obstructive Pulmonary Disease (COPD)**
   a) Worsening V/Q mismatch (e.g., secretions, and bronchoconstriction can lead to ARF)
   b) Causes: acute exacerbations, CHF/ pulmonary edema, dysrhythmias, pneumonia, dehydration, electrolyte imbalances
   c) **Asthma discussed later**

22) **Medical Management of ARF in COPD**
   a) Correct hypoxemia
      i) Cautious administration of O₂
      ii) Noninvasive positive pressure ventilation
      iii) Ventilate if needed
   b) Medications
      i) Beta₂ agonists
      ii) Theophylline
      iii) Antibiotics (depends on cause)
      iv) Cautious administration of sedatives

23) **ARF in Asthma**

24) **Exacerbation of Asthma**
   a) Wheezing
   b) Dyspnea
   c) Chest tightness
d) Cough  
e) Hyperventilation initially  

25) **Exacerbation of Asthma**  
a) Causes  
   i) Bronchodilators no longer working  
   ii) NONCOMPLIANCE with medications  
b) Effects  
   i) Hyperventilation with air trapping results in respiratory acidosis  
   ii) Severe hypoxemia  

26) **Medical Management of Exacerbation**  
a) Oxygen; ventilation in severe cases  
b) IV corticosteroids  
c) Inhaled bronchodilators  
d) Helium  

27) **Ventilator-Associated Pneumonia (VAP)**  
a) Aspiration of bacteria from oropharynx or gastrointestinal tract  
b) Controversies about best way to diagnose  

28) **Prevention of VAP**  
a) Handwashing and standard precautions  
b) Aseptic suctioning of ET tube  
c) Oral and nasal care  
d) Maintain cuff pressure of ET tube  
e) Head of bed elevated for enteral feedings  
f) Assess gastric residual volumes  
g) Turn and reposition frequently  

29) **Treatment of VAP**  
a) Bacteria-specific antibiotic therapy  

30) **ARF: Pulmonary Embolus (PE)**  
a) Virchow’s triad  
   i) Venous stasis  
   ii) Altered coagulability  
   iii) Damage to vessel wall  
b) Embolus results in a lack of perfusion to ventilated alveoli (V/Q mismatch)  

31) **PE Assessment**  
a) Symptoms of deep venous thrombosis  
b) Chest pain (worse on inspiration)  
c) Dyspnea  
d) Tachycardia  
e) Cough; hemoptysis  
f) Crackles, wheezes  
g) Hypoxemia  

32) **Diagnosis of PE**  
a) Clinical signs and symptoms  
b) Chest x-ray (nonspecific)  
c) V/Q scan with high probability of PE
d) Pulmonary angiogram—DEFINITIVE

33) Complications of PE
   a) Heart failure
   b) Obstructive shock
   c) Death

34) Prevention of PE
   a) Antiembolic hose
   b) Sequential compression devices
   c) Position changes
   d) Treatment of atrial dysrhythmias
   e) Prophylactic anticoagulant therapy

35) Treatment for PE
   a) ABCs; oxygen
   b) Thrombolytics (dissolve the clots)
   c) Heparin
   d) Surgical procedures
      i) Embolectomy
      ii) Vena cava umbrella (prevention)

Class 9

1) Acute Renal Failure
   a) The Ins and Outs

2) Fluid and Electrolytes; Wastes
   a) Filtration
      i) Glomerular filtration rate (GFR)
      ii) 85 to 125 mL/min
   b) Reabsorption
   c) Secretion

3) Acid-Base Balance
   a) Bicarbonate
   b) H+ ions

4) Blood Pressure Regulation
   a) Juxtaglomerulus apparatus
   b) Renin-angiotensin-aldosertone

5) Recognizing Renal Failure
   a) Low urine output
   b) Accumulation of nitrogenous wastes: azotemia
   c) Acid-base disturbances

6) Acute Renal Failure Causes
   a) Prerenal
   b) Renal: intrinsic; parenchymal
   c) Postrenal

7) Prerenal Etiology
a) Diminished blood flow; hypoperfusion of the kidney
   i) Volume depletion
   ii) Vasodilation
   iii) Decreased cardiac output
b) Can progress to intrarenal damage

8) Postrenal Etiology
   a) Obstruction of flow

9) Intrarenal Etiology
   a) Glomerular, vascular, and hematologic problem

10) Intrarenal Etiology
   a) Acute tubular necrosis
      i) Ischemia
      ii) Nephrotoxic agents
         (a) Antibiotics
         (b) NSAID
         (c) Dyes
      iii) Rhabdomyolysis

11) Pathophysiology Summary
   a) Prerenal: decreased blood supply
   b) Renal: failure of nephrons
   c) Postrenal: obstruction of outflow

12) Phases of Acute Renal Failure (ARF): Initiation
   a) Time from event to signs of decreased renal perfusion
   b) Few hours to 2 days
   c) Potentially reversible

13) Phases of ARF: Maintenance
   a) BUN and creatinine increase daily
   b) Oliguric—output < 400 ml/day
   c) Fluid overload, electrolyte imbalances, and acidosis
   d) Dialysis required

14) Phases of ARF: Recovery
   a) Return of tubular function
   b) 4 to 6 months for blood urea nitrogen (BUN) and creatinine to return to normal
   c) Residual impairment of GFR
   d) Early dialysis may prevent the traditional “diuretic” phase of ARF

15) Importance of History
   a) Predisposing factors
      i) Exposure to nephrotoxic agents
      ii) Cardiac failure
      iii) Shock

16) Clinical Presentation of ARF
   a) Vital signs may be altered
      i) Kussmaul’s respirations to compensate for metabolic acidosis
   b) Assess for volume depletion and volume overload

17) Specific Test for ARF
a) Creatinine clearance  
   i) 24-hour urine  
   ii) Most specific  
   iii) Normal: 80 to 125 mL/min  
   iv) Can calculate an estimated value

18) Serum Tests for ARF  
   a) Creatinine  
   b) BUN  
      i) Affected by catabolism, bleeding, and dehydration  
   c) BUN:creatinine ratio  
      i) 20:1—suspect nonrenal causes of laboratory abnormalities  
   d) Serum osmolality

19) Urine Tests for ARF  
   a) Urine osmolality  
   b) Urine specific gravity  
   c) Urine electrolytes

20) Diagnostic Studies for ARF  
   a) X-ray of kidneys, ureter, and bladder—structures  
   b) IVP—structures  
   c) Computed tomography—structures, accumulation of fluid  
   d) Renal scan—renal uptake of isotopes  
   e) Renal angiography—abnormalities in blood flow; infarction, masses  
   f) Renal biopsy—histological changes

21) Physical Findings of ARF  
   a) Neurological—confusion, lethargy, decreased level of consciousness, stupor  
   b) Gastrointestinal—nausea, vomiting, anorexia, distention, constipation, or diarrhea  
   c) Respiratory—Kussmaul’s respirations, crackles, pulmonary edema

22) Physical Findings of ARF  
   a) Cardiovascular—tachycardia, dysrhythmias, rub, pericarditis, increased blood pressure  
   b) Integumentary—dry skin, pruritus, edema, bruising, pallor, uremic frost

23) Fluid/Electrolyte Imbalances  
   a) Hyperkalemia: low excretion  
   b) Hyponatremia: fluid retention  
   c) Hypocalcemia: low excretion of phosphorus; decreased level of vitamin D  
   d) Hyperphosphatemia: low excretion  
   e) Hypermagnesium: low excretion

24) Nursing Diagnoses  
   a) Alterations in fluid volume (excess)  
   b) Fluid restriction  
   c) Daily weights  
      i) 1 lb = 500 ml of fluid  
   d) Intake and output  
   e) Assessment of overload/deficit  
   f) Risk for infection  
      i) Strict aseptic care with IV lines
ii) Not all patients need Foley catheters; avoid use if not required

g) Alterations in nutrition
   i) Small feedings
   ii) Oral hygiene
   iii) Increase carbohydrate intake
   iv) Limit protein, sodium, and potassium

h) Potential for injury (mentation changes from uremia, electrolyte changes, acidosis)
   i) Restrict potassium intake
   ii) Monitor electrolytes
   iii) Monitor ECG changes
   iv) Neurological assessment
   v) Preparation for dialysis

25) Prerenal
   a) Fluid/volume replacement
   b) Caution in patients with underlying cardiac disease

26) Intrarenal
   a) Hydration
   b) Renal perfusion
      i) Drugs: dopamine
      ii) Volume
      iii) Diuretics: furosemide
   c) Antibiotic regulation

27) Postrenal
   a) Alleviate obstruction

28) Treatment of Hyperkalemia
   a) Reduce content
      i) Kayexalate
      ii) Mineralocorticoids
      iii) Diuretics

29) Treatment of Hyperkalemia
   a) Shift intracellularly
      i) Glucose and insulin
      ii) Alkali (sodium bicarbonate)
   b) Antagonize cellular membrane effect
      i) Calcium gluconate

30) Dietary Management
   a) Adequate nutrition while monitoring fluid/electrolytes
   b) Vitamin supplements

31) Diuretic therapy
   a) Osmotic versus loop
   b) EARLY administration
   c) Mannitol
   d) Furosemide
   e) Bumetanide
   f) Ethacrynic acid
32) **Dialysis**
   a) LIFE-SAVING TREATMENT
   b) Hemodialysis
   c) Continuous renal replacement therapy (CRRT)
   d) Peritoneal dialysis

33) **Dialysis Indications**
   a) Fluid overload
   b) Electrolyte imbalances
   c) Acid-base disturbances
   d) **BEGIN EARLY!**

34) **Hemodialysis Access**
   a) Percutaneous catheters—most common in ICU
   b) AV fistulas
   c) Grafts
   d) External shunts

35) **Hemodialysis**
   a) Usually done at the bedside in the ICU
   b) Pre- and post-dialysis labs and weights
   c) Monitor for complications
      i) Volume depletion
      ii) Dysrhythmias
      iii) Hypoxemia
      iv) Disequilibrium syndrome

36) **Continuous renal replacement therapy (CRRT)**
   a) Used with patients too unstable for hemodialysis
   b) Advantages
      i) More gradual solute removal
      ii) Flexible fluid administration
      iii) Minimal heparin
      iv) Can be done by staff nurses at the bedside
   c) Disadvantages
      i) Bedrest
      ii) 1:1 nursing care

37) **CRRT—Types**
   a) Continuous arteriovenous hemofiltration (CAVH)
   b) Continuous venovenous hemofiltration (CVVH)
   c) Continuous arteriovenous hemodialysis (CAVHD)
   d) Continuous venovenous hemodialysis (CVVHD)

38) **CRRT**
   a) Automated devices now being marketed

39) **Peritoneal Dialysis**
   a) Removal of solutes and fluids using the peritoneal membrane as a filter
   b) Rarely used in the critical care setting, because it is less efficient
   c) High risk of peritonitis

40) **Chronic Renal Failure**
a) May result from acute renal failure
b) May be a concurrent disease in patients admitted to the ICU for other reasons

Class 10

1) Hematological and Immune Disorders
   a) That bloody (white and red) lecture

2) Terms
   a) Many terms are important in the understanding of these disorders
   b) Review Table 13-1

3) Hematopoiesis
   a) Blood cell formation
   b) Stem cells differentiate

4) Blood
   a) Plasma
   b) Solutes (e.g., ions)
   c) Serum proteins
      i) Coagulation
      ii) Healing
      iii) Transport
      iv) Osmotic pressure
   d) Blood cells

5) Cells
   a) Erythrocytes: red blood cells (RBCs)
      i) Erythropoietin stimulates production
      ii) O\textsubscript{2} transport
   b) Thrombocytes: platelets
      i) Thrombopoietin stimulates production
      ii) Clotting
   c) Leukocytes: white blood cells (WBCs)
      i) Fight infection and antigens
      ii) Granular and agranular

6) Granular Leukocytes
   a) Neutrophils
      i) Bacterial infections
   b) Eosinophils
      i) Parasites
   c) Basophils
      i) Allergic response

7) Agranulocytes
   a) Monocytes
      i) Mature into macrophages
   b) Lymphocytes
      i) Microorganisms; tumor immunity
(a) Humoral—B lymphocytes  
(b) Cell-mediated—T lymphocytes

8) **Immune System**
   a) Antigen-antibody responses  
   b) Autoimmune: body sees self as “nonself” and activates immune response

9) **Nonspecific Defenses**
   a) Intact epithelium  
   b) Inflammation and phagocytosis  
      i) involves neutrophils and mediators  
   c) Cytokines  
      i) involves interleukins, TNF, interferons, colony-stimulating factor

10) **Humoral Immunity**
   a) B-lymphocytes  
   b) Formation of antibodies  
   c) Immunoglobulins  
      i) IgG  
      ii) IgM  
      iii) IgA  
      iv) IgE  
      v) IgD

11) **Cell-Mediated Immunity**
   a) T-lymphocytes  
   b) Initiated by macrophages  
   c) Cells  
      i) Helper/inducer  
      ii) Suppressor  
      iii) Killer

12) **Hemostasis**
   a) Local vascular reaction  
   b) Platelet aggregation at site  
   c) Activation of clotting factors—become an insoluble fibrin clot  
      i) Each factor activates another factor  
      ii) Vitamin-K—dependent factors synthesized by liver  
      iii) Calcium required for almost all process

13) **Clotting Cascade**
   a) Initiating event  
      i) Intrinsic pathway: injury to blood  
      ii) Extrinsic pathway: tissue injury  
   b) Final common pathway (factor X)  
      i) Prothrombin to thrombin  
      ii) Fibrinogen to fibrin  
      iii) Clot

14) **Fibrinolysis**
   a) Stimulated by clot formation  
      i) Stimulates conversion of plasminogen to plasmin
b) Breakdown yields fibrin degradation product (FDP) or fibrinogen split products (FSP)

15) Assessment
   a) Cues to hematologic or immunologic problems:
      i) Altered oxygenation
      ii) Bleeding
      iii) Infection

16) Erythrocyte Disorders
   a) Too much or too little

17) Anemia
   a) Poor tissue oxygenation
   b) Blood flow shunted to vital organs
   c) Enhanced release of oxygen by Hgb

18) Causes of Anemia
   a) Blood loss (common in critical care)
   b) Deficient production
   c) Abnormal function
   d) Premature lysis

19) Types of Anemia
   a) Hemorrhagic
   b) Marrow failure
   c) Aplastic
   d) Hemolytic
   e) Sickle cell
   f) B₁₂ deficiency
   g) Folic acid deficiency
   h) Iron deficiency

20) Assessment of Anemia
   a) Decreased circulating volume
      i) Signs of hypovolemia
   b) Decreased oxygenation
      i) Signs of hypoxemia
   c) Compensatory mechanisms
      i) Increased heart rate
      ii) Congestive heart failure may result

21) Diagnosis of Anemia
   a) CBC
   b) Bone marrow biopsy
   c) Reticulocyte count (hemolytic)
   d) Transferrin (hemolytic)
   e) Haptoglobin (hemolytic)
   f) Electrophoresis (sickle cell)
   g) Schilling (B₁₂)
   h) Folate (Folic acid deficiency)
   i) Iron and ferritin (Iron deficiency)

22) Treatment of Anemia
a) Depends on the cause and type
b) Erythropoietin (Epogen) used more commonly to stimulate RBC production

23) Nursing Management of Anemia
   a) Describe common nursing diagnoses
   b) List general strategies for care of the patient with anemia

24) White Blood Cell Disorders
   a) Immune Disorders

25) Immunocompromise
   a) Defects in WBCs or immune physiology
   b) Lack of normal defenses
   c) May be asymptomatic
   d) Infection is leading cause of death

26) Immunocompromise
   a) Symptoms of infection often absent
   b) Fever may be only sign of infection
   c) Pain without signs of inflammation is also a cue
   d) Anergy may be noted with the use of skin tests

27) Laboratory Analysis: Immune
   a) Leukopenia
   b) Low CD4 counts
   c) Decreased immunoglobulins

28) Immune: Medical Management
   a) Reverse the cause
      i) Cell replacement
      ii) Bone marrow transplant
      iii) Immunoglobulins
   b) Treat infections
   c) Nutrition

29) Immune:
    Nursing Management
   a) Controversy about isolation needs
   b) Hygiene
   c) Aseptic technique for procedures
   d) Good assessment for cues of infection
   e) Maintain skin integrity
   f) Optimum nutritional support

30) Neutropenia
   a) Absolute neutrophil count < 1000
   b) Causes
      i) Inadequate production
      ii) Excessive destruction
   c) High risk of infection

31) Neutropenia Risks
   a) Overwhelming infection
   b) Radiation therapy
c) Chemicals and drugs
   i) Antibiotics
   ii) Chemotherapy

d) Disease states

32) Assessment of Neutropenia
    a) Classic symptoms of infection often absent
    b) Areas of heavy bacteria at risk for infection (mouth, perineum, IV sites, catheter)

33) Neutropenia: Management
    a) Prevent and treat infection
    b) Reverse cause
    c) Colony-stimulating factors
    d) Prophylactic antibiotics

34) Neutropenia: Nursing Management
    a) Similar to that used for immunocompromised patient

35) Malignant WBC Disorders
    a) Leukemia
    b) Lymphoma
    c) Multiple myeloma

36) Malignant WBC Disorders
    a) Decreased number of functional WBCs
    b) Risk for infection
    c) Alteration in immune response
    d) Fever difficult to interpret—Why?

37) Malignant WBC Disorders
    a) Leukemia
       i) Large number of immature cells
    b) Lymphoma
       i) Cancer affects lymphocytes
    c) Multiple myeloma
       i) Abnormal immunoglobulins

38) Malignant WBC Disorders
    a) Symptoms are often non-specific
       i) Fatigue, malaise
       ii) Myalgias
       iii) Activity intolerance
       iv) Night sweats
       v) Fever
    b) Other symptoms
       i) Bruising and bleeding
       ii) Enlarged lymph nodes
       iii) Thrombosis

39) Malignant WBC Disorders
    a) Staging of disorders determines treatment
       i) Chemotherapy
ii) Biotherapy
iii) Bone marrow transplant
b) Nursing care is based on treating and preventing infections

40) Immunodeficiency: AIDS
a) Infection with HIV
b) Depletes helper-T cells and macrophages
c) Increased risk for opportunistic infections

41) Diagnosis of AIDS
a) CD4 count < 200/μl
b) Indicator diseases (see Figure 13-6)

42) Medical Interventions: HIV
a) Antiretroviral medications
b) Drugs continuously being developed
c) Multidrug therapy has been more effective than monotherapy
d) Supportive therapy: prevent infection, nutrition, analgesia, etc.

43) Nursing Interventions: HIV
a) Prevent infection
b) Observe for adverse reactions to medications
c) Psychosocial support
d) Prevent transmission

44) Bleeding Disorders: Causes
a) Abnormality in stages of clotting
   i) Vasoconstriction
   ii) Creation of platelet plug
   iii) Development of clot
   iv) Fibrinolysis
b) Inherited or acquired
c) Common in renal, hepatic, and gastrointestinal disorders; malnutrition

45) Diagnosis of Bleeding
a) CBC
b) Fibrinogen
c) PT, aPTT

46) Bleeding Risks
a) Hypovolemic shock
b) Altered tissue perfusion
c) Decreased platelets
d) Risk for bleeding
e) Treated with platelet transfusions

47) Disseminated Intravascular Coagulation (DIC)
   a) Accelerated activation of clotting cascade
   b) Depletion of clotting factors
   c) Bleeding
   d) Secondary problem

48) Pathophysiology of DIC
a) Initiating event: procoagulants
b) Stimulation of intrinsic or extrinsic pathway
c) Clots in microvasculature
d) Consumptions of clotting factors
e) Fibrinolysis
f) FDPs: potent anticoagulants

49) **Etiology of DIC**
a) Infection
b) Trauma (e.g. burns, crush)
c) Obstetric conditions (e.g., abruptio, amniotic fluid embolus, retained dead fetus)
d) Hematologic disorders
e) Oncologic disorders
f) Other: shock/sepsis, acute respiratory distress syndrome

50) **Assessment of DIC**
a) Evidence of occult bleeding
b) Signs of platelet deficiency
   i) Petechiae
   ii) Ecchymosis

51) **Assessment of DIC**
a) Overt bleeding/oozing
b) Decreased perfusion to organs
   i) Changes in mental status
   ii) Acral cyanosis
   iii) Infarction of tissue in digits and nose

52) **Laboratory Diagnosis: DIC**
a) Decreased platelets
b) Decreased fibrinogen
c) Prolonged PT, aPTT, thrombin time
d) Elevated FDP/FSP
e) Increased d-Dimer
f) Decrease in coagulating factors
g) Decrease in hemoglobin and hematocrit

53) **Treatment of DIC**
a) Correct underlying cause
b) Administer blood and components
   i) Platelets
   ii) Fresh frozen plasma
   iii) Cryoprecipitate
   iv) Packed RBCs
c) Stop abnormal coagulation
   i) Heparin: CONTROVERSIAL

54) **Other Treatments of DIC**
a) Antithrombin III (inhibits thrombin)
b) Epsilon-amino caproic acid (prevents fibrinolysis)

55) **Nursing Management of DIC**
a) Assess and prevent
b) Frequent laboratory analysis
c) Administration of blood products
d) Assess circulation
e) Relieve pain
f) Assess for complications: shock, multisystem organ failure, impaired circulation

**Class 11**

1) **Endocrine Alterations**
2) **Hormones**
a) Regulate physiologic processes
b) Hypothalamus and pituitary play big roles in regulation
c) Controlled by feedback loops
   i) Hormone low—stimulus to release more
   ii) Hormone high—stimulus to limit production
3) **Review**
a) Figures 15-1 through 15-3 show feedback mechanisms
4) **Pancreatic Imbalances**
a) Diabetic Ketoacidosis (DKA)
b) Hyperglycemic, hyperosmolar nonketotic diabetic coma (HHNC)
c) Hypoglycemia
5) **Diabetic Ketoacidosis (DKA)**
a) Pathophysiology
   i) Relative or absolute insulin deficiency
   ii) Increase in antagonistic hormones
   iii) Hyperglycemia
6) **DKA**
i) Increase in lipolysis, release of free fatty acids
   (a) Increase in Ketone production; impaired ketone metabolism
   (b) Respiratory compensation (Kussmaul's respirations)
   ii) Osmotic fluid shifts
   iii) Altered potassium balance
   iv) Excess acids result in increased anion gap
   v) Altered consciousness r/t acidosis and dehydration
7) **DKA: Etiology**
a) Initial presentation of type 1 diabetes
b) Insufficient insulin
   i) Stress/infection
   ii) Growth spurts
   iii) Pregnancy
8) **DKA: Etiology**
a) Missed or reduced insulin
   i) Nonadherence to insulin regimen
ii) Insulin pump failure
b) Medications

9) Hyperglycemic, Hyperosmolar, Nonketotic Coma
   i) HHNC

10) HHNC: Pathophysiology
   a) Decreased use of glucose and/or increased production
   b) Hyperglycemia; increased extracellular osmolality
   c) Osmotic diuresis
   d) Profound dehydration
   e) No ketoacidosis—hyperglycemia with hyperosmolarity blocks lipolysis

11) HHNC: Etiology
   a) Inadequate insulin secretion; usually type 2 diabetics
   b) Often in elderly with decreased compensatory mechanisms
   c) Stress response
   d) Medications

12) Medications that cause an Increase in Blood Sugar
   a) Thiazides
   b) Phenytoin
   c) Glucocorticoids
   d) Beta blockers

13) Symptoms of DKA and HHNC
   a) Dehydration/hypovolemia
   b) Nausea and vomiting
   c) Classic polyuria, polyphagia, and polydipsia
   d) Decreased level of consciousness

14) Differences
   a) DKA
      i) Blood sugar > 300 mg/dL; average = 675 mg/dL
      ii) Metabolic acidosis; bicarbonate < 15 mEq/L
      iii) Kussmaul’s respirations
      iv) High anion gap
      v) Ketones in urine and blood

15) Differences
   a) HHNC
      i) Blood sugar > DKA; average > 1000 mg/dL
      ii) More “normal” ABGs
      iii) More electrolyte imbalances and renal dysfunction
      iv) Higher serum osmolarity than DKA

16) Common Interventions
   a) Rapid infusions of 0.9% normal saline until stabilized (1 to 2 liters/hr initially)
   b) Solution then changed to 0.45 normal saline
   c) Monitor for overload, especially in older patients
   d) Electrolyte replacement
      i) Potassium, phosphorus (K-Phos replacement), magnesium
   e) Regular Insulin
i) Loading dose
ii) Continuous drip at 0.1 to 0.2 U/kg/hr
iii) Hourly glucose monitoring
iv) Decrease when glucose gets to 300 mg/dL
v) Change IV to 5% D / 0.45 normal saline at same time

17) Interventions: DKA
   a) Treatment of acidosis
      i) Assess respiratory compensation and level of consciousness
      ii) Usually corrected by fluids and insulin
      iii) Bicarbonate if pH < 7.10

18) Hypoglycemia
   a) Pathophysiology
      i) Glucose production below utilization
      ii) Glucose level 45 to 60 mg/dL

19) Hypoglycemia: Etiology
   a) Excess insulin/oral agents
   b) Alcohol potentiates hypoglycemic effects
   c) Missed meals or delayed enteral feedings
   d) Drug interactions—ASA, haldol
   e) Excess exercise

20) Hypoglycemia: Clinical Signs
   a) (Brain and stimulation of epinephrine)
   b) Sweating
   c) Blurred vision
   d) Change in level of consciousness or behavior
   e) Circumoral paresthesia, anxiety, chilling, lack of coordination
   f) Slurred speech, palpitations, nausea, headache
   g) Stupor and coma

21) Hypoglycemia: Diagnosis
   a) Blood glucose
   b) Signs and symptoms
      i) Some patients with chronically high blood sugar levels will be symptomatic at higher levels than expected

22) Hypoglycemia: Interventions
   a) Treat hypoglycemia with 10 to 20 gm of carbohydrate
      i) Glucose (50% dextrose common in emergency department and ICU settings)
      ii) Glucagon
      iii) Oral glucose
   b) Assess response—should improve rapidly
   c) Prevention and teaching

23) Acute Adrenal Crisis
   a) Pathophysiology
      i) Acute deficiency of glucocorticoids (cortisol) and mineralocorticoids (aldosterone)
      ii) Chronic dysfunction
iii) Acute exacerbation

24) Lack of Cortisol
   a) Decreased production of glucose
   b) Decreased metabolism of protein and fat
   c) Gastrointestinal symptoms
   d) Decreased vascular tone
   e) Decreased effect of catecholamines
   f) UNABLE TO RESPOND TO STRESS

25) Lack of Aldosterone
   a) Decreased sodium and water retention
   b) Decreased circulating volume
   c) Hyperkalemia

26) Adrenal Crisis: Primary
   a) Hypofunction
      i) Autoimmune—Addison's disease
      ii) Diseases—tuberculosis, hemorrhage, sepsis, HIV
      iii) Drugs—ketoconazole and others

27) Adrenal Crisis: Secondary
   a) Interference with adrenocorticotropic hormone (ACTH) secretion or suppressed production of steroids
      i) Tissue destruction
      ii) Pituitary necrosis
      iii) Acute withdrawal of chronic steroids
      iv) Inadequate steroids in highly stressed patient who has received chronic steroid therapy

28) Adrenal Crisis: Clinical Signs
   a) Good history essential to look for cues
   b) Symptoms of Hypovolemia
   c) Fluid and electrolyte imbalances
      i) Postural hypotension
      ii) Change level of consciousness
      iii) Hyperkalemia
   d) Fatigue, weakness
   e) Gastrointestinal complaints
   f) Decreased renal perfusion and decreased urine output
   g) Skin changes (hyperpigmentation)
   h) Decreased libido, amenorrhea, scanty pubic hair

29) Adrenal Crisis: Diagnosis
   a) Laboratory values (hyponatremia, hyperkalemia, azotemia)
   b) Fasting hypoglycemia
   c) Low plasma cortisol levels and urinary metabolites
   d) Cosyntropin test

30) Adrenal Crisis: Interventions
   a) Correct fluid and electrolyte imbalances
   b) Hormonal replacement
i) Hydrocortisone
ii) Fludrocortisone (mineralocorticoid)
c) Patient/family education

31) Thyroid Storm
a) Pathophysiology
   i) Superacceleration of metabolic processes
   ii) Women more common
   iii) High mortality (10-20%)

32) Thyroid Storm: Etiology
a) Goiter—most common cause
b) Inadequately controlled hyperthyroidism
c) Ingestion of too much thyroid hormone
d) Cancer of thyroid

33) Thyroid Storm: Clinical Signs
a) Increased cardiac workload—possible congestive heart failure, dysrhythmias (atrial fibrillation and flutter common)
b) Increased oxygen demands and alterations in respirations
c) Restless, agitation, manic
d) Fever
e) Nausea, vomiting, diarrhea, and cramps
f) Splenomegaly
g) Jaundice
h) Classic symptoms
   i) Change in vision; exophthalmus
   ii) Fine, soft hair; hair loss
   iii) Dermopathy
   iv) Enlarged thyroid
   v) Thyroid bruit

34) Thyroid Storm: Diagnosis
a) Elevated T3 and T4
b) Elevated T3 resin intake
c) Low plasma thyroid stimulating hormone (TSH)
d) Electrolyte imbalances

35) Thyroid Storm: Interventions
a) Monitor cardiovascular status; assess for CHF and dysrhythmias
b) Administer meds
   i) Propylthiouracil (PTU) and methimazole (Tapazole) inhibit thyroid synthesis
   ii) Satured solution of potassium iodine (SSKI) retards release of hormones
   iii) Medication to block effects—beta blockers, steroids

36) Myxedema Coma
a) Pathophysiology
   i) Hypofunction of thyroid
   ii) Hypometabolism and decreased functioning
   iii) Occurs mainly in women
   iv) Mortality up to 50%
37) **Myxedema Coma: Etiology**
   a) Primary disease
   b) Insufficient thyroid stimulation due to hypothalamus or pituitary disease
   c) Exacerbation of hypothyroid state

38) **Myxedema Coma: Signs**
   a) **Insidious**
   b) Cognitive changes
   c) Activity intolerance
   d) Cardiovascular
      i) Bradycardia, hypotension
      ii) Cardiomegaly
      iii) Decreased cardiac output
      iv) ECG changes
   e) Hypoventilation
   f) Decreased peristalsis; constipation
   g) Decreased reflexes and slow movements
   h) Hypothermia
   i) Skin changes
      i) Edema
      ii) Skin is cool, dry, scaly, thin, yellowish
      iii) Dry, coarse hair
   j) Decreased libido; irregular menses

39) **Myxedema Coma: Diagnosis**
   a) Decreased T3 and T4; decreased T3 resin intake; elevated TSH (primary)
      i) TSH normal or low if problem in hypothalamus or pituitary
   b) Hypoxemia, hypercapnia
   c) Hyponatremia (from water retention)
   d) Elevated liver enzymes
   e) Increased cholesterol and lipids

40) **Myxedema Interventions**
   a) Treat with replacement drugs
   b) Fluid and electrolyte replacement; thyroid replacement usually corrects sodium
   c) Monitor gas exchange/respiratory status
   d) Monitor cardiovascular status
   e) Manage hypothermia
   f) Protect from injury and infection

41) **Diabetes Insipidus (DI)**
   a) Pathophysiology
      i) Deficiency in synthesis or release of antidiuretic hormone (ADH)
      ii) Excess water losses
      iii) Neurogenic versus nephrogenic
         (a) Neurogenic—ADH deficiency
         (b) Nephrogenic—kidneys insensitive to ADH
      iv) Secondary causes

42) **DI: Etiology Neurogenic**
a) Familial
b) Trauma and diseases
   i) Head trauma, hypophysectomy
   ii) Infections
   iii) Tuberculosis

43) **DI: Etiology Nephrogenic**
a) Familial
b) Renal disease
c) Metabolic disease
d) Drugs

44) **DI: Clinical Signs**
a) High urine output; low specific gravity
b) Thirst
c) Dehydration
d) Central nervous system signs of volume depletion

45) **DI: Diagnosis**
a) Increased serum osmolarity
b) Dilute urine with low specific gravity
c) Water deprivation test
d) Vasopressin test

46) **DI: Interventions**
a) Fluids; assess for hypovolemia
b) ADH replacement (neurogenic)—vasopressin, DDAVP

47) **DI: Interventions**
a) Nephrogenic
   i) Thiazide diuretics
   ii) Sodium restriction

48) **Syndrome of Inappropriate Secretion of Antidiuretic Hormone (SIADH)**
a) Pathophysiology
   i) Excess ADH
   ii) Plasma hypotonicity

49) **SIADH Etiology**
a) Malignancy
b) Pulmonary disorders
c) Central nervous system trauma, tumors
d) Stress, pain
e) Drugs

50) **SIADH Clinical Signs**
a) Central nervous system changes r/t low serum sodium and cerebral edema—confusion, headache, agitation, weakness
b) Pulmonary edema from volume overload
c) Anorexia, nausea, vomiting

51) **SIADH Diagnosis**
a) Hyponatremia
b) Decreases osmolarity
c) High urine sodium
d) Concentrated urine

52) SIADH Interventions
a) Fluid restriction (800 to 1000 mL/day)
b) If needed, hypertonic saline and diuretics
c) Severe cases—demeclocycline increases renal response to ADH
d) Input and output, specific gravity, weights
e) Mouth care

Gastrointestinal Alterations

1) Caloric Needs
a) Nitrogen balance
b) Harris-Benedict equation
c) Determines basal caloric needs for men and women

2) Principles
a) If gut works, use it
b) Early feeding/intervention

3) Enteral Feedings
a) Gastric versus small intestine
b) Various formulas
   i) 1.5 cal/mL HN common in critical care
   ii) Specialty formulas (e.g., renal, respiratory)

4) Enteral Feeding Care
a) Prevent aspiration
   i) Ensure placement
   ii) Positioning
   iii) Check residuals
b) Rinse tube well
c) Care of nose
d) Check blood sugars until stable

5) Feeding Controversies
a) Continuous versus bolus feedings
b) No need to dilute
c) Dye or not
d) Best ways to unclog tube (PREVENT CLOGGING)

6) Parenteral Nutrition
a) Use if unable to tolerate enteral nutrition or NPO
b) Given via a central line: subclavian, internal jugular, or peripherally inserted central catheter line

7) Parenteral Nutrition
a) Total parenteral nutrition
   i) Protein (amino acids), carbohydrates (glucose), vitamins, minerals, and trace elements
   ii) May need insulin in formula
iii) Histamine blockers sometimes added
b) Fats (lipids)
i) Added to formula or given separately

8) Parenteral Nutrition Care
a) Observe fluid status
b) Routine blood sugar levels
c) Infection control—sepsis is a high risk

9) Upper Gastrointestinal Bleeding
a) GIB

10) Upper Gastrointestinal Bleeding
a) Etiology
   i) Ulcer
   ii) Mallory-Weiss tear
   iii) “Itis”
   iv) Varices
b) Potential for Hypovolemic shock

11) Peptic Ulcer Disease
a) Main risks
   i) h. pylori bacteria
   ii) NSAIDs

12) Contributing Factors Ulcers
a) Alcohol
b) Drugs—NSAIDs, ASA, steroids
c) Smoking
d) Stress

13) Stress Ulcer
a) Trauma
b) Sepsis
c) Burns

14) Mallory-Weiss tear
a) Forceful retching
b) Drugs—NSAIDs, alcohol

15) “itis”
a) Gastritis
b) Esophagitis
c) What are contributing factors?

16) Esophageal Varices
a) Portal hypertension
b) Elevated ammonia and change level of consciousness
c) Bleeding can be profuse

17) Assessment
a) Blood loss
   i) Color, amount, and consistency of emesis and stool
b) Symptoms of Hypovolemia
c) Pain/discomfort
18) Laboratory Studies
   a) Hemoglobin and hematocrit
   b) White blood cell count
   c) Electrolytes and blood urea nitrogen level often reflect dehydration
   d) Gastric pH
   e) Liver function and ammonia if liver disease suspected

19) Diagnostic Tests
   a) Endoscopy
   b) Barium studies

20) Priority Treatment
   a) Fluid/blood resuscitation
   b) Nasogastric tube; gastric lavage

21) Pharmacologic Therapy
   a) Antacids
   b) Histamine blockers (cimetidine and others)
   c) Mucosal barrier enhancers (sucralfate)
   d) Antibiotics (H. pylori)

22) Endoscopic Treatment
   a) Electrocautery
   b) Sclerotherapy
   c) Band ligation (varices)

23) Surgical Treatment
   a) Types
      i) Gastric resection
      ii) Vagotomy
      iii) Pyloroplasty

24) Specialized Therapy for Varices
   a) Vasoconstrictive drugs
      i) Vasopressin drip
         (a) Risk of cardiac compromise
         (b) Central nervous system abnormalities secondary to low sodium
      ii) Ocreotide (somatostatin)
         (a) Fewer risks
   b) Balloon tamponade
      i) Blakemore tube
      ii) Minnesota tube: how is it different?
   c) Sclerotherapy
   d) Transjugular intrahepatic portosystemic (TIP) shunt
   e) Surgery (portocaval shunts)
   f) Both surgical and transjugular shunts have higher risks of encephalopathy after procedures

25) Acute Pancreatitis
   a) Inflammation of pancreas
      i) Mild to severe
      ii) Severe can lead to necrosis and hemorrhage
iii) Severe has high mortality rate

26) Acute Pancreatitis
   a) Etiology
      i) Metabolic--alcoholism
      ii) Mechanical--biliary tract disease
      iii) Trauma
      iv) Idiopathic

27) Pathophysiology
   a) Proteolytic enzymes \( \rightarrow \) autodigestion
      i) Trypsinogen \( \rightarrow \) trypsin \( \rightarrow \) edema; necrosis; activation of other enzymes
      ii) Lipase \( \rightarrow \) fat necrosis
      iii) Phospholipase A \( \rightarrow \) acinar membrane damage
      iv) Kallikrein \( \rightarrow \) edema; increased vascular permeability
   b) Autodigestion \( \rightarrow \) edema; interstitial hemorrhage; necrosis
   c) Release of histamine and bradykinin \( \rightarrow \) increased vascular permeability; edema
   d) Inflammation may lead to obstruction of duct; ischemia

28) Signs and Symptoms
   a) Severe epigastric pain; radiates to back; worse supine
      i) Patient may curl up with both arms over abdomen to relieve pain
   b) Nausea/vomiting
   c) Abdominal distention/rigidity
   d) Low-grade fever
   e) Hypotension/shock

29) Signs and Symptoms
   a) Jaundice
   b) Signs of low calcium
      i) Chvostek's sign
      ii) Trousseau's sign
   c) Turner's sign (discoloration of flanks)
   d) Cullen's sign (discoloration around umbilicus)

30) Laboratory Data
   a) Elevated
      i) Serum and urine amylase
      ii) Serum lipase
      iii) White blood cell count
      iv) Glucose
      v) Liver function studies; bilirubin
   b) Decreased
      i) Calcium
      ii) Albumin
      iii) Potassium

31) Diagnostic Tests
   a) Computed tomography (CT) of abdomen
   b) Magnetic resonance imaging (MRI)
   c) Abdominal and chest X-rays
d) Upper gastrointestinal (UGI)  
  e) IV cholangiogram  
  f) Endoscopic retrograde cholangiopancreatography (ERCP)

32) Fluids and Electrolytes  
   a) Volume replacement with colloids, FFP, blood  
   b) Pulmonary artery catheter to aid in monitoring  
   c) Electrolyte replacements  
      i) Calcium and potassium  
   d) Treatment of hyperglycemia

33) Rest the Pancreas  
   a) Decreasing secretions  
      i) NPO  
      ii) Gastric suction  
   b) Parenteral nutrition  
   c) Newer trend evaluating intestinal feedings

34) Pain Relief  
   a) Demerol (prevents spasm of sphincter of Oddi)  
   b) Morphine may be acceptable as long as pancreatitis is not caused by gallbladder disease

35) Other Medical Therapy  
   a) Peritoneal lavage  
   b) Drainage of pseudocysts  
   c) Pancreatic resection

36) Complications  
   a) Hypovolemic shock  
   b) Anemia, diffuse intravascular coagulation (DIC)  
   c) Atelectasis, acute respiratory distress syndrome (ARDS), effusion  
   d) Pseudocysts and abscess  
   e) Renal Failure  
   f) High glucose and triglycerides, low calcium

37) Hepatic Failure  
   a) Hepatitis  
   b) Hepatotoxicity (drugs, toxins)  
   c) Decreased perfusion  
   d) Cirrhosis

38) Hepatitis  
   a) Acute inflammation of liver cells  
      i) A—common; fecal contamination  
         (a) Vaccine available  
      ii) B—blood-borne  
         (a) Vaccine; begin in infancy  
      iii) C—blood-borne, sexual contact  
         (a) More common, including among health care workers  
         (b) Risk of chronic hepatitis and liver damage  
      iv) D—combined with hepatitis B  
      v) E—similar to A; found in developing countries
vi) G—newer; percutaneous transmission

39) Symptoms of Hepatitis
   a) Gastrointestinal pain
   b) Fever, chills
   c) Jaundice
   d) Brown urine
   e) Right upper quadrant pain
   f) Clay-colored stools

40) Management of hepatitis
   a) Rest
   b) Nutritional support
   c) Prevention of spread of the virus

41) Fulminant Failure
   a) Can occur secondary to hepatitis
   b) Patients are acutely ill
   c) Often die within 10 days of onset

42) Cirrhosis
   a) Destruction of liver parenchyma and replacement by scar tissue
   b) Types
      i) Laënnec’s (alcoholic; portal)
      ii) Biliary
      iii) Cardiac
      iv) Postnecrotic

43) Stages
   a) Liver enlarges due to increased fat accumulation
   b) Inflammation and necrosis of cells
   c) Yellow, orange, fatty, scarred liver
      i) Liver shrinks; hobnail
      ii) Flow of blood is obstructed
      iii) Portal hypertension

44) Complications
   a) Portal hypertension
      i) Varices
   b) Impaired metabolism
   c) Impaired clotting
   d) Impaired bile flow
   e) Inability to detoxify drugs and toxins, including ammonia

45) Signs and Symptoms
   a) Early: anorexia, dyspepsia, flatulence, nausea, vomiting, changes in bowel patterns
   b) Related to altered metabolism

46) Signs and Symptoms
   a) Late
      i) Jaundice
      ii) Skin lesions
      iii) Hematologic
iv) Endocrine
v) Encephalopathy
vi) Ascites

47) Diagnostic Tests
   a) Elevated enzymes
   b) Elevated bilirubin
   c) Elevated ammonia
   d) Coagulation studies
   e) Plasma proteins
   f) CT of abdomen

48) Supportive Therapy
   a) Fluids
   b) Prevents injury and bleeding
   c) Treats hypoglycemia

49) Aggressive Therapy
   a) Liver transplant
      i) Acute hepatic failure
      ii) Transport to center that does transplants
   b) Extracorporeal liver assist
      i) Experimental
      ii) Bridge to transplant or healing

50) Ascites
   a) Pathophysiology
      i) Low albumin + obstruction to flow
      ii) Fluid in peritoneal cavity
      iii) Increased aldosterone
         (a) Sodium and water retention
      iv) Peripheral edema and further ascites

51) Medical Management: Ascites
   a) Bedrest
   b) Sodium and fluid restriction
   c) Albumin (questionable effectiveness)
   d) Diuretics
   e) Potassium
   f) Paracentesis
   g) LeVeen “peritoneovenous” shunt
   h) Nutritional support

52) Encephalopathy
   a) Cerebral toxicity from elevated ammonia levels
   b) Pathophysiology
      i) Increased ammonia levels
      ii) Neural disturbances: asterixis, euphoria, anxiety, impaired speech, progressive decrease in level of consciousness

53) Medical Management: Encephalopathy
   a) Limit protein intake
b) Neomycin  
c) Lactulose  
d) Restrict toxic medications  
e) Prevent gastrointestinal bleeding  
f) Dialysis

54) Hepatorenal Syndrome  
a) Acute renal failure  
b) R/t decreased renal circulation secondary to cirrhosis

55) Hemorrhage  
a) Pathophysiology  
   i) Increased portal pressure (portal hypertension)  
   ii) Esophageal varices and hemorrhoids  
   iii) See previous discussion  
   iv) Gastrointestinal bleeding

Class 12

1) Trauma  
a) Major problem  
b) Fourth leading cause of death  
c) Affects the young  
d) Often associated with drugs and alcohol  
e) Financial implications  
   i) Treatment  
   ii) Rehabilitation  
   iii) Disability

2) Trauma Team: Essential  
a) Similar to code team  
b) Includes trauma surgeons, emergency department physicians, and specialists  
c) Nurses  
d) Ancillary services: radiology, laboratory, and social work

3) Critical Thinking Challenge  
a) What is the role of pastoral services on a trauma team?  
b) Why is it important to have security officers as part of the trauma team?

4) Levels of Trauma Care  
a) Level I—regional resource, state-of-the-science care, education, outreach, research  
b) Level II—provides care for trauma patients and transfer to Level I if needed, outreach  
c) Level III—rural hospital with protocols for managing patients and transferring as needed  
d) Level IV—rural clinic with protocols

5) Trauma Triage  
a) Essential for determining if patients need to be transferred to a Level I trauma center  
b) If in doubt, transfer  
c) Revised Trauma Score (Table 16-1) used to determine severity of injury

6) Mechanisms of Injury
a) Knowledge helps to identify potential problems
b) Blunt trauma versus penetrating trauma

7) **Blunt Trauma**
   a) Common vehicular trauma, assault with blunt objects, falls
   b) Severity depends on duration of impact and body part injured

8) **Penetrating Trauma**
   a) Stab wounds and gunshot wounds
   b) Injuries depend of body part(s) involved and on the trajectory of the impaled (or sharp) object or bullet

9) **Prehospital Care**
   a) Emergency stabilization and quick transport
   b) ABCs (with cervical stabilization)
   c) IV access and fluid administration
   d) Control hemorrhaging
   e) Stabilize fractures

10) **Primary Survey**
    a) Done in 1 to 2 minutes
       i) Airway patency (with C-spine immobile)
       ii) Breathing effectiveness
       iii) Circulation, including hemorrhage and pulses
       iv) Disability (overview of neurological status)
    b) Baseline vital signs
    c) Insertion of Foley catheter and nasogastric tube
    d) Continuous ECG monitoring

11) **Secondary Survey**
    a) Performed after life-threatening injuries are identified and treated
    b) Examination of all body systems: head-to-toe and front-to-back
    c) Maintain C-spine immobilization until cleared by x-ray
    d) X-ray studies (as determined by injury)
    e) Laboratory studies

12) **Airway Management in Trauma**
    a) Many factors affect the airway (e.g., facial fractures, bleeding, vomiting, decreased sensorium)
    b) Use oral or nasopharyngeal airways if needed in spontaneously breathing patients
    c) Endotracheal intubation often needed
    d) If unable to intubate, emergency cricothyrotomy is performed

13) **Ineffective Breathing**
    a) Breathing patterns are affected by a variety of traumatic injuries; ongoing assessment is essential
    b) Depending on the injury, the nurse needs to be prepared to assist with:
       i) Mechanical ventilation
       ii) Needle thoracostomy and chest tube insertion
       iii) The administration of fluids and blood products
       iv) The administration of sedation and analgesics

14) **Impaired Gas Exchange**
a) Injuries often affect gas exchange
b) Patients may need supplemental oxygen or mechanical ventilation
c) Ongoing assessment of oxygen saturation, end-tidal CO₂, and ABGs needed, along with physical assessment

15) Decreased Cardiac Output / Hypovolemia
   a) Hypovolemic shock occurs often in patients with trauma
   b) Ongoing assessment of vital signs, urine output, mental status, and hemodynamic parameters

16) Treatment of Hypovolemia
   a) Stop bleeding
      i) Pressure
      ii) Pneumatic antishock garment (PASG)
   b) Venous access—2 large-bore IVs; central line may be needed

17) Treatment of Hypovolemia
   a) Administration of crystalloids and blood products
      i) Ringer’s lactate is fluid of choice
      ii) Blood administration based on response to initial fluid resuscitation and laboratory values
      iii) Autotransfusion may be an option

18) Response to Treatment
   a) Rapid responders
   b) Transient responders
      i) Patient is still bleeding; surgery needed
   c) Minimal or no responders
      i) Emergent surgical intervention needed to stop bleeding

19) Signs of Deterioration
   a) Falling hematocrit
   b) Falling PaO₂
   c) Decreasing urine output
   d) Increased serum lactate levels

20) Assessment of Internal Abdominal Bleeding
   a) Varies by institutional protocol
   b) Diagnostic peritoneal lavage (DPL)
   c) Abdominal ultrasound
   d) CT of abdomen

21) Complications of Massive Fluid Resuscitation
   a) Acid-base abnormalities; metabolic acidosis
   b) Fluid-electrolyte imbalances
   c) Hypothermia
   d) Coagulopathies
   e) Organ dysfunction
   f) Volume overload

22) Specific Organ Injuries
   a) Chest
   b) Spinal cord
c) Head
d) Abdomen

23) Tension Pneumothorax
   a) Life-threatening as increased intrapleural and intrathoracic pressures cause compression of heart and great vessels
   b) Cardiovascular collapse
   c) Emergent treatment with needle thoracostomy
   d) Chest tube inserted after needle decompression

24) Hemotorax and Open Pneumothorax
   a) Blood (hemo) or air (pneumo) in pleural space
   b) Chest tube insertion needed
   c) Cautious application of dressing to open (sucking) chest wound; tension pneumothorax can result
      i) Allow small amount of air to escape from occlusive dressing

25) Cardiac Tamponade
   a) Bleeding into pericardial space
   b) Impairs pumping ability of heart
   c) May be difficult to diagnose; suspect in patient with symptoms of decreased cardiac output who does not respond to treatment
   d) Treated by pericardiocentesis

26) Pulmonary Contusion
   a) Bruising of lung tissue
   b) Often results in pneumonia and ARDS
   c) May require long-term ventilatory support

27) Rib Fractures
   a) Seriousness varies; treatment also varies
   b) May result in flail chest
      i) Paradoxical respirations result
      ii) Treated with intubation, ventilation, pain management

28) Aortic Disruption
   a) Life-threatening injury requiring emergency surgical intervention
   b) Symptoms include weak pulses, pain, and hoarseness
   c) Chest x-ray shows widened mediastinum
   d) Confirmed by aortogram

29) Spinal Cord Injury
   a) Immobilization
   b) X-rays and possible CT studies
   c) Reduction with cervical or halo traction
   d) Emergency administration of methylprednisolone
   e) Assess for distributive (spinal) shock
      i) May need vasopressors

30) Head Injury
   a) Primary injury associated with trauma
   b) Secondary injury associated with hypoxemia, hypotension, increased intracranial pressure, cerebral edema, hypercapnia, hypothermia, and infection
c) Monitor and treat intracranial pressure
d) Ongoing neurological assessment

31) Musculoskeletal Injuries
   a) Many types of fractures; multiple fractures often present in trauma patient
   b) Unstable pelvic fractures and femur fractures can result in a large amount of blood loss

32) Musculoskeletal Injuries
   a) Assess:
      i) Pain
      ii) Pallor
      iii) Pulses
      iv) Paresthesia
      v) Paralysis

33) Musculoskeletal Treatment
   a) Closed or open reduction; may need traction
   b) Treatment of hypovolemia and blood loss
   c) Wound care
   d) Tetanus prophylaxis
   e) Possible antibiotics
   f) Assess for neurological and/or vascular injury

34) Musculoskeletal Complications
   a) Ischemia of affected muscle tissue
   b) Rhabdomyolysis—increased myoglobin secondary to crush injury; can result in renal failure

35) Compartment Syndrome
   a) Severe pain is first symptom
   b) May need compartmental pressure monitoring
   c) Treated with fasciotomy

36) Fat Embolism
   a) Suspect with long-bone, pelvic, and multiple fractures
   b) Symptoms include fever, tachycardia, and new onset of respiratory distress
   c) Treated with oxygen, intubation, ventilation, and positive end-expiratory pressure

37) Abdominal Injuries
   a) Trauma can result in damage to any of the organs; liver damage most common
   b) Pain is a classic sign
   c) Diagnosis facilitated by diagnostic peritoneal lavage, ultrasound, and CT of abdomen
   d) Laboratory tests can also help in identifying organ damage (e.g., liver function studies, renal studies)
   e) Ongoing assessment is essential

38) Critical Care Phase
   a) During this phase, the nurse provides ongoing monitoring and assessment for complications discussed previously

39) Acute Respiratory Distress Syndrome
   a) Identify patients at risk: flail chest, pulmonary contusion, prolonged hypovolemia, massive fluid resuscitation, aspiration, sepsis
   b) Observe serial chest x-ray studies for infiltrates
c) Patient will require mechanical ventilation
d) Fluid therapy often guided by hemodynamic monitoring

40) Deep Venous Thrombosis and Pulmonary Embolism
   a) Ongoing assessment of extremities, pulmonary status
   b) Interventions such as range of motion exercises, early ambulation, sequential compression devices, and heparin prophylaxis are important
   c) Patient may require a vena cava filter

41) Infection
   a) Trauma predisposes patients to a wide variety of infections
      i) Nosocomial pneumonia
      ii) Catheter sepsis
      iii) Sinusitis
      iv) Wounds

42) Acute Renal Failure
   a) All classifications of renal failure possible
      i) Prerenal—decreased blood flow
      ii) Intrarenal—hypovolemia, nephrotoxicity, and rhabdomyolysis
      iii) Postrenal—injury or obstruction
   b) Careful monitoring of urine output, daily weight, and laboratory values
   c) Anticipate in high-risk patients (e.g., hypovolemic shock, crush injuries)

43) Altered Nutrition
   a) Nutritional support is essential; high metabolic demands are associated with traumatic injury
   b) Early enteral nutrition preferred
   c) Parenteral nutrition given if needed

44) Multisystem Organ Failure
   a) Inflammatory response predisposes patient to this condition
   b) Risks include sepsis, extensive trauma and tissue damage, hypotension and hemorrhagic shock, inadequate fluid resuscitation, and multiple transfusions
   c) Prevention includes treating infections, maintaining oxygenation, and nutritional support

Class 13

1) Shock
   a) Life-threatening response to alterations in circulation
   b) Results in:
      i) impaired tissue oxygenation
      ii) impaired cellular metabolism

2) Shock is a SYNDROME, it is not low blood pressure.
   a) Shock is a pathophysiologic abnormality involving inadequate blood flow to vital organs and/or the inability of the tissues or cells to metabolize nutrients normally

3) Clinical Indicators
   a) Blood pressure may be misleading
b) Hemodynamics may be more useful
   i) CO/CI, SVR, SvO₂

   c) Need good history to help detect cause

4) Alteration in Circulation
   a) Blood volume
   b) Myocardial contractility
   c) Blood flow
   d) Vascular resistance

5) Systemic Responses
   a) If inadequate perfusion persists, organ systems begin to malfunction
   b) Signs and symptoms appear
   c) At this point hypoxemia, hypotension, and acidosis activate compensatory mechanisms

6) Stages
   a) Early compensatory
   b) Progressive
   c) Refractory

7) Stage 1: Compensatory
   a) Neural response
   b) Endocrine response
   c) Chemical response

8) Neural
   a) Baroreceptors
      i) Epinephrine and norepinephrine

9) Epinephrine
   a) Increases heart rate and contractility
   b) Dilates coronary arteries
   c) Bronchodilates
   d) Increases sweating to dissipate heat
   e) Breaks down glycogen to glucose

10) Norepinephrine
    a) Potent vasoconstriction
       i) Improves blood pressure
       ii) Improves venous return

11) Combined Action
    a) Preserves cardiac output and perfusion to heart and brain

12) Endocrine Compensation
    a) Renin-angiotensin-aldosterone mechanisms
       i) Decreased renal perfusion
       ii) Stimulates release of renin
       iii) Stimulates release of angiotensin I and II
       iv) Stimulates release of aldosterone, which conserves water

13) Chemical Compensation
    a) Chemoreceptors
    b) Respirations increase to increase oxygenation
    c) Metabolic alkalosis occurs
14) **Stage II: Progressive Shock**
   a) Continued vasoconstriction to preserve heart and brain
      i) Tissue hypoxia
      ii) Lactic acidosis
      iii) Impaired cellular function (swelling)
15) **Stage II: Progressive Shock**
   a) Microcirculation dilates
      i) Sphincters relax and blood flows back into nonvital organs and sludge
      ii) This further leads to the development of METABOLIC ACIDOSIS as waste products, WBCs, and clots accumulate
      iii) Impaired coronary perfusion and contractility develop
16) **Stage III: Refractory Shock**
   a) Pooling of blood in capillary beds
   b) Inadequate perfusion of vital organs
   c) Multisystem organ failure
17) **Classification**
   a) Shock
18) **Hypovolemic Shock**
   a) Inadequate blood volume to fill intravascular space
19) **Critical Thinking Challenge**
   a) What are some of the common etiologies of hypovolemia?
20) **Direct Losses—Quantified**
   a) Hemorrhage
   b) Diarrhea
   c) Vomiting
   d) Massive diuresis
   e) Loss of plasma (burns/wounds)
21) **Indirect Losses**
   a) Ascites
   b) Intestinal obstruction
22) **Severity**
   a) Volume lost
   b) Rate of loss
   c) Age
   d) Comorbid conditions
23) **Cardiogenic Shock**
   a) Inability of heart to pump; cardiac output cannot meet tissue demands
   b) Causes include myocardial infarction, post-cardiac surgery, dysrhythmias, and cardiomyopathy
   c) Goal: Restore adequate pumping
24) **Cardiogenic Shock**
   a) *Compensatory mechanisms cause further detriment to myocardium*
      i) Increased sympathetic discharge; increased SVR
      ii) Increased left ventricular end diastolic pressure
      iii) Pulmonary congestion
25) **Obstructive**  
   a) Compression and obstruction to flow  
   b) Tamponade, pulmonary embolism, and dissecting aneurysm

26) **Distributive Shock**  
   a) Vasodilation and decreased SVR  
      i) Abnormal distribution of intravascular volume  
      ii) Relative hypovolemia  
   b) Septic, anaphylactic, and neurogenic

27) **Neurogenic**  
   a) Loss of vasomotor tone  
   b) Secondary to deep general anesthesia, spinal anesthesia, and spinal cord injury

28) **Anaphylactic**  
   a) Antigen-antibody reaction  
   b) Direct damage to vascular walls

29) **Septic Shock**  
   a) Secondary to vasodilation from endotoxin release  
   b) Activates clotting mechanisms  
   c) Alters capillary permeability

30) **Stages of Septic Shock**  
   a) Infection  
   b) SIRS: mediators; vasodilate  
   c) Sepsis: hypoperfusion; high cardiac output; low SVR  
   d) Septic shock: hypotension; hypoperfusion; acidosis  
   e) Multiple organ dysfunction syndrome: renal failure; acute respiratory distress syndrome, disseminated intravascular coagulation

31) **Assessment**  
   a) History  
   b) Clinical picture  
   c) Laboratory studies

32) **Objective Data**  
   a) Skin color and temperature  
   b) Level of consciousness  
   c) Blood pressure  
   d) Peripheral pulses  
   e) Respiratory status and ABGs  
   f) Urine output  
   g) Heart sounds; jugular venous distention  
   h) Gastrointestinal

33) **Objective Data**  
   a) Hemodynamic parameters  
      i) Cardiac output; CI  
      ii) PA pressures  
      iii) SVR  
      iv) SVO2  
   b) Lactate
c) Hematocrit
d) WBC
e) Clotting profile

34) **Objective Data**
   a) Urine tests
   b) Chest x-ray
   c) Echocardiogram
d) DPL
e) CT
   f) Gastric Tonometry

35) **Hypovolemic Shock**
   a) Classic symptoms
   b) Low PA pressures
c) Increased SVR
d) Treat with *volume*

36) **Cardiogenic Shock**
   a) Symptoms of heart failure
   b) High PA pressures
c) Low cardiac output
d) Increased SVR
e) Treatments aimed at *pump*
   i) Inotropic drugs
   ii) Intra aortic balloon pump
   iii) Venous access device

37) **Obstructive Shock**
   a) Symptoms vary depending on cause
   b) Decreased cardiac output/CI
c) Treat the *etiology*

38) **Neurogenic Shock**
   a) Hypotension
   b) Decreased PA pressures, SVR, and cardiac output/CI
c) Treatment
   i) Fluids
   ii) Vasopressors
   iii) Atropine

39) **Anaphylactic Shock**
   a) Hypotension
   b) Urticaria
c) Wheezing / airway obstruction
d) Edema

40) **Anaphylactic Shock**
   a) Treatment
   i) Intubate and ventilate
   ii) Bronchodilators
   iii) Steroids
iv) Antihistamines
v) Sympathomimetics

41) Septic Shock
a) Early—warm shock
b) Warm, flushed skin
c) High cardiac output/CI
d) High SvO₂
e) Low SVR
f) Treatment
   i) Fluids
   ii) Vasopressors
   iii) Antibiotics

42) Septic Shock
a) Late phase - cool phase
   i) Cool, clammy skin, edema
   ii) CO decreasing/SVR increasing
   iii) Lactic acidosis
   iv) Oliguria
   v) Depressed LOC, unresponsive
   vi) Multiple organ dysfunction (MODS)

43) Fluid Challenge
a) Obtain baseline measurements-RAP, pulmonary capillary wedge pressure
b) Administer fluids (250 ml to 2000 ml)
c) Monitor patient response and duration of response
   i) PA pressures
   ii) Vital signs
   iii) Urine output

44) Responses
a) Determine course of action
b) Rapid—fluids
   c) Transient—fluids, blood, possible surgery
d) No response—massive resuscitation with fluids and blood; emergent surgery

45) Crystalloids
a) Ringer’s lactate (or normal saline) for hypovolemia
b) Replace with 3 mL for every mL of blood loss

46) Colloids
a) Increase colloidal osmotic pressure; keeps fluid in vascular space
b) Albumin: 5% and 25% (SPA)
   i) ml 5% ↑ intravascular volume by 1 ml
   ii) 25 gm 25% (100 ml) ↑ volume by 400 ml
c) Plasmanate (P-Nate)
d) Hetastarch (Hespan)
e) Dextran

47) Blood and Blood Products
a) Whole blood
b) Packed cells
c) Fresh frozen plasma
d) Blood components
e) Blood substitutes

48) Improve Contractility
   a) Positive inotropes
      i) Dobutamine
      ii) Amrinone
      iii) Digoxin

49) Improve Preload
   a) Volume resuscitation
   b) Vasopressors (if not hypovolemic)

50) Reduce Preload
   a) Diuretics

51) Reduce Afterload
   a) Nitroprusside

52) Increase Afterload
   a) Dopamine
   b) Norepinephrine

53) Treat Dysrhythmias
   a) Alter rate
   b) Manage dysrhythmias

54) Positioning
   a) Avoid trendelenburg’s position
      i) Tricks the baroreceptors!
   b) Head of bed should be at a 20 to 30 degree angle and the patient should be turned frequently

55) Maintenance
   a) Body temperature
   b) Skin integrity
   c) Psychological support

56) Mechanical Interventions
   a) External management

57) Intra-Aortic Balloon Pump
   a) Cardiogenic shock
   b) Inflated diastole; deflated systole
      i) Increases coronary artery perfusion
      ii) Decreases afterload
   c) Close monitoring for complications
      i) Thromboembolism
      ii) Decreased perfusion to extremities

58) Ventricular Assist Device
   a) Cardiogenic shock
   b) Often a bridge to transplant
   c) Newer models becoming more portable
59) **Pneumatic Antishock Garment (PASG)**
   a) PASG; MAST trousers
   b) Hypovolemic shock
   c) Contraindicated in pregnancy and head injuries and pulmonary edema
   d) Must be deflated slowly with constant assessment

60) **Outcomes/complications**
   a) CNS
   b) CV
   c) Respiratory
   d) Renal
   e) Hepatic
   f) GI
   g) MODS

**Class 14** (You made it)

**Burns**

1) **Improvement in Management**
   a) If burned area less than 81% of body surface area, good chance of survival
   b) Highest morbidity
      i) Burns cover > 50% of body surface area
      ii) Patient is older
      iii) Patient is younger

2) **Phases of Burn Care**
   a) Resuscitative (emergent)
      i) First 48 hours
      ii) Fluid management to prevent shock
   b) Acute phase
      i) Onset of diuresis to wound closure
   c) Rehabilitative phase
      i) Restore to functional status

3) **Functions of Skin**
   a) Protect from infection
   b) Prevention of loss of body fluids
   c) Thermoregulation
   d) Production of vitamin D
   e) Excretion
   f) Determination of identity
   g) Sensation reception

4) **Severity of Burns**
   a) Duration of contact
   b) Temperature of agent
   c) Amount of tissue exposed
   d) Ability of agent to dissipate energy

5) **Classification**
6) **Partial Thickness Burns**
   a) Superficial
      i) Epidermis only or small depth of dermis
      ii) Manifestations:
          (a) Red (e.g. sunburn)
          (b) Pink, mottled red, blisters
   b) Deep
      i) Epidermis and most of dermis
      ii) Pale, mottled, pearly, dry
      iii) Often painless
      iv) Usually need skin grafts

7) **Full-Thickness Burns**
   a) Destruction of all layers down to or past fat, fascia, muscle, or bone
   b) Thick, dry, leathery appearance
   c) Insensate (no pain)

8) **Zones of Injury**
   a) Zone of coagulation: greatest injury
   b) Zone of stasis: potentially reversible damage
   c) Zone of hyperemia: minimal injury

9) **Local Response to Burns**
   a) Acute inflammation
   b) Cellular enzymes and vasoactive substances
   c) Activation of complement
   d) Altered vascular permeability

10) **Local Response: Fluid Shifts**
    a) Fluid shifts to extravascular space
    b) Burns > 25% total body surface area; edema in burned and unburned areas
    c) Maximum edema 18 to 24 hours postburn

11) **Systemic Response to Burns**
    a) Cardiovascular: varies; decreased cardiac output common
    b) Altered defenses: increased risk of infection
    c) Pulmonary: possible pulmonary hypertension or direct injury

12) **Systemic Response to Burns**
    a) Renal: initial decrease in urine output related to decreased glomerular filtration rate; followed by diuresis as fluids shift
    b) Gastrointestinal: ileus, ulcers
    c) Metabolic: Hyper state; peaks 6 to 10 days postburn

13) **Thermal Injury**
    a) Skin damaged by contact with heat

14) **Chemical Injuries**
    a) Systemic and local effects
b) Must be completely removed or neutralized or damage continues

c) Types
   i) Alkalis
   ii) Acids
   iii) Organic compounds

15) Electrical Injuries
   a) High versus low voltage
   b) Injury affected by type and path of current, duration of contact, and resistance of tissues

16) Nonburn “Burns”
   a) Some syndromes mimic with burn-like injury
      i) Toxic epidermal necrolysis
      ii) Staphylococcal scalded skin
   b) May need to be treated in burn unit
   c) No fluid shifts with these syndromes

17) Assessment in Resuscitative Phase
   a) Respiratory: Assess for hypoxemia, carbon monoxide poisoning
   b) Cardiovascular: Assess for fluid volume status, vital signs, hemodynamics, and carbon monoxide
   c) Neurological: Assess for changes in level of consciousness
   d) Renal: Urine output essential
   e) Gastrointestinal: Assess for occult bleeding, pH, bowel sounds (ileus)
   f) Integumentary: Document extent of injury; edema may be very severe
   g) Psychosocial: Assess response to injury; pain assessment essential

18) Acute Phase Assessment
   a) Continues with assessments performed during resuscitative phase
   b) Complications assessed (e.g., pneumonia, hypoxemia, decreased cardiac output, infection/sepsis, ulcer, ileus)
   c) Pain assessment and management is ongoing

19) Prehospital Interventions
   a) Remove the source of thermal damage
   b) ABCs and cervical spine
   c) Oxygen at 100%; intubation if needed
   d) Assess circulation
   e) Remove clothing and jewelry
   f) Assess for additional trauma

20) Prehospital Interventions
   a) Cover and prevent hypothermia
   b) Large-bore IV fluids
   c) Pain management with narcotics
   d) Vital signs and baseline assessment

21) Emergency Department Interventions
   a) ABCs; C-spine evaluation
   b) Circulation checks—assess need for escharotomy or fasciotomy
   c) Calculation of fluid requirements
   d) Administration of fluids—Ringer’s lactate
e) Chest x-ray
f) Nasogastric tube
g) Pain management

22) Fluid Resuscitation
a) Rule of 9s
b) Lund and Browder chart
c) Are only Guidelines—must assess patient’s response

23) Fluid Guidelines for Adults
a) to 4 mL / kg / % of body surface area burned
b) Maintain urine output of 30 to 50 mL/hr
c) Administer half of total fluids during first 8 hours postburn
d) Administer a quarter of total fluids during second 8 hours postburn
e) Administer a quarter of total fluids during third 8 hours postburn

24) Fluid Guidelines: Electrical
a) Patients with electrical injury need higher volumes of fluid
b) Keep urine output at 75 to 100 mL/hr
c) Rationale: electrical injury results in the release of myoglobin, which can cause acute renal failure

25) Transfer
a) Transfer patient to burn center early, according to criteria

26) Acute Care Interventions
a) Primary and secondary survey
b) Ongoing assessment: vital signs, pulses, urine output, pulse oximetry, hemodynamics, gastric pH, occult blood
c) Daily weights
d) Fluid administration and monitoring
e) Maintain joint function and mobility

27) Acute Care Interventions
a) Ongoing monitoring:
   i) Respiratory
   ii) Cardiovascular
   iii) Renal
   iv) Gastrointestinal
   v) Fluid and electrolytes
b) Prevent hypothermia
c) Labs and x-rays: electrolytes, blood urea nitrogen, creatinine, ABGs, lactate, chest x-ray

28) Pain Control
a) Adequate, accurate assessment tools
b) Opiates; IV route
c) PCA may be useful
d) Nonpharmacologic strategies

29) Wound Care
a) Remove nonviable tissue
b) Promote coverage of the burned area; graft as needed
30) **Wound Care**
   a) Clean and debride wounds
      i) Hubbard tank is best
   b) Application of antimicrobial agents
   c) Open versus closed dressings
   d) Various types of biological and biosynthetic dressings (Table 17-8)
   e) Grafts for deep partial-thickness and full-thickness burns

31) **Skin Grafts**
   a) Autograft is the only permanent method of grafting
   b) Meshed versus sheet grafts—What are the advantages and disadvantages of each?
   c) Care of donor site—various dressings
   d) Cultured epithelium autograft is newer method for growing skin

32) **Special Concerns**
   a) Facial burns—risk of respiratory injury; oral hygiene; promote healing
   b) Ears—prevent breakdown
   c) Eyes—ophthalmology consult as needed, keep moist
   d) Hands and feet—adequate circulation; maintain function
   e) Genitalia—meticulous wound care to prevent infection

33) **Nutritional Support**
   a) Hypermetabolic-catabolic state
   b) Consult to determine caloric needs
   c) Early enteral nutrition warranted

34) **Psychosocial Issues**
   a) What are the many psychosocial outcomes of burn injury?
   b) What are strategies for addressing the psychosocial needs of the patient with burns?

**Neonatal considerations**
   a) Temperature
   b) Access
   c) Fluids
   d) Airway

**Pediatric considerations**
   a) Family considerations
   b) Weight dependent dosing
   c) Fluids
   d) Developmental issues
   e) Airway

**Geriatric considerations**
   a) Adverse drug reactions
   b) Functional decline
   c) Physical changes