

Course Objectives

- Prepare you to take the CCRN Examination
 - Not what's new in critical care
 - Not an update
- A REVIEW of the essentials of critical care nursing

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Requirements

- Current unrestricted RN license in the USA
- Clinical practice in critical care
 - 1750 hours in 2 year period
 - Current clinical practice
- BSN is <u>not</u> a requirement

Application

• Obtain application:

American Association of Critical-Care Nurses

• (800) 899-2226 or www.aacn.org

Application

1. Receive notice of processed application

- AACN will send you an email confirming that you have successfully applied to take the CCRN exam.

2. Receive approval-to-test email

- AACN's testing service (aMP) will send an email and mail a postcard to eligible candidates within 5 to 10 days after the confirmation email that will include:

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Application

- 1. A toll-free number and online instructions to schedule your testing appointment
- 2. The 90-day period during which you must schedule and take the exam
- 3. Schedule the exam.
- 4. Sit for the exam
- 5. You need 87 correct out of 125 scored questions

Blueprint			
Clinical	80%	Professional	20%
 Cardiovascular 	17	 Advocacy/Moral 	3
 Pulmonary 	15	 Caring Practice 	4
 Multisystem 	14	 Collaboration 	4
 Gastrointestinal 	4	 System Thinking 	2
• Renal	4	 Diversity 	2
 Endocrine 	4	Clinical Inquiry	2
 Hematology 	4	• Learning	3
Immunology	4		
 Muscul/Neuro/ 	Psych		
14			
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Blueprint

Professional: Synergy Model

- Patient-Centered Care
- Needs of the patient matched with the nurse's ability

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CCRN Test:

- Exam:
- -150 questions
- -3 hours to complete
- READ ALL INSTRUCTIONS!
 - Will not need pencil, calipers or calculator
- Passing: 71% overall

Certification CCRN • Maintaining CCRN: - Certification 3 years • Recertification: - Retaking exam - Continuing Education Units (CEU)

Renewal

- Completion of 432 hours of direct bedside care of acutely/critically ill patients as an RN or APRN within the 3-year certification period, with 144 of those hours in the 12month period preceding the scheduled renewal.
- Competition of CERPS or take and pass the CCRN exam.

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Recertification CCRN Category A • Clinical • Min 60 Max 80 Category B • Leadership, Team Building, Caring • Min 10 Max 30 Category C • Collaboration, Precepting • Min 10 Max 30

Certification PCCN Requirements

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Application

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- Apply
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- Schedule Test: H&R Block
- 90 day window to take exam

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Blueprint Clinical 80% Cardiovascular 27 Pulmonary 17 • Endocrine, Renal, Gastrointestinal, Hematology 18 Neurology, Multisystem, Behavioral **Professional** 20% Advocacy/Moral Caring Practice Collaboration System Thinking Diversity 2 Clinical Inquiry Learning

Blueprint

Professional: Synergy Model

- Patient-Centered Care
- Needs of the patient matched with the nurse's ability
- Concept: Whole patient and resources that patient needs for successful outcome

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PCCN: The Test

Exam:

125 questions, 100 questions scored2.5 hours to complete

READ ALL INSTRUCTIONS!

Will not need pencil, calipers or calculator

Passing: 68% overall

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Recertification/Renewal

- Maintaining PCCN:
 - Certification 3 years
- Recertification:
 - Retaking exam
 - Continuing Education Units (CEU)

Category B • Safety, Mental Illness, Caring, • End of Life, Diversity, HIPAA • Min 10 Max 30 • Collaboration, Precepting,	Category A	Recertification/Renewa • Clinical • Min 60 Max 80
• End of Life, Diversity, HIPAA • Min 10 Max 30 • Collaboration, Precepting,		
	Category B	End of Life, Diversity, HIPAA
• Min 10 Max 30	Category C	Communication, Team Work

A patient's family expresses anxiety regarding the meaning of numbers on the patient's monitor and asks the nurse for clarification. The nurse's most appropriate

- a. The numbers indicate the patient is having problems.
- b. The numbers help us to determine the best treatment.
- c. Which numbers on the monitor concern you?

response would be:

d. What don't you understand about the monitor?



When teaching a family member to perform an aspect of patient care, the nurse realizes that family members:

- a. Are affected by timing of teaching.
- b. Learn best if they perceive a need to learn.
- c. Learn best if shown a complex procedure all at once.
- d. Learn unrelated tasks first.



A patient with cerebral edema after a subarachnoid hemorrhage has been ordered Nifedipine 10 mg by mouth every 4 hours.

The patient's blood pressure is 150/85 mmHg.

How should the nurse respond to this order?

- a. Ask the pharmacist to clarify the order.
- b. Discuss the purpose of the order with the physician.
- c. Research the indications and safety of Nifedipine.
- d. Administer the medication to control the blood pressure.

Hematology - Immunology

Hematological:

- Provides medium for transportation of O₂, CO₂, and nutrients
- Maintains Hemostasis
- Maintains internal environment: temperature/acid/base

Immunological:

- Protects from invading foreign material

Stress Response -Immunosuppression

Stress Response:

Acute Stress vs. Chronic Stress: total body response Sympathetic Nervous System Stimulation – vital signs

- Impaired gag, cough or swallow
 Changed gastric pH, colonization, volume aspiration, pneumonitis
 Malnutrition
 Acute Phase Stress Reactions=catabolism, decreased healing, inhibit immune
- response
 Sequential infections

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Hematology • Hemostasis: - Termination of bleeding - Vascular response - Vasospasm - Thromboxane A₂ - Platelet response

Platelets • ETOH • Aspirin/Plavix/Effient • GP IIb IIIa Inhibitors • NSAIDS	• Thrombocytopenia - HITT – Heparin-Induced Thrombocytopenia - ♥ production - ↑ destruction - Dilutional

Disseminated Intravascular Coagulopathy: Definition

- A syndrome characterized by thrombus formation and hemorrhage secondary to over-stimulation of the normal coagulation process, with resultant decrease in clotting factors and platelets.
- DIC may be chronic or acute
- Thrombosis; then hemorrhage
- Always secondary

DIC: Etiology

- Sepsis
- Hematological
 - Anaphylaxis
 - Hemolytic blood transfusion reaction
 - Massive blood transfusion
 - Prolonged cardiopulmonary bypass
 - Sickle cell crisis
 - Transplant reaction

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DIC Other Causes

- Trauma
- Cancers
- Burns
- OB complications
- Acute anoxia
- Embolism
- Heat stroke
- GI complications
- Crush injury
- Pulmonary complications
- Head injury
- Toxins
- Surgery
- Dissecting aneurysm

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DIC: Clinical Presentation

- Abnormal bleeding
- Signs of thrombosis
- Change in level of consciousness
- Chest pain, ST-T wave changes, **Ψ** systolic BP
- Dyspnea, hypoxemia
- ullet urine output, proteinuria, electrolyte imbalance
- Abdominal pain, diarrhea

Clinical Indications of Platelet Dysfunction

- Petechiae (first indication of DIC)
- Purpura
- Ecchymosis

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Labs: DIC

- ◆ Platelets: (<100,000/mm²)
 </p>
- ↑ PTT (>60-90 seconds)
- ↑ PT (normal: 11-15 seconds, abnormal >15)
- ◆ Fibrinogen (<200mg/100ml)
 </p>
- ↑ FDP/FSP (>10g/ml but < 100)
- ↑ D-dimer (<2mg/L: abn >2mg/L)
- ♣ Antithrombin III (normal 80 to 120%, abnormal <70%)</p>

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Complications Of DIC

- Mortality 40-60%
- Hypovolemic shock
- Acute kidney injury
- Infection
- Acute respiratory distress syndrome
- Stroke
- GI dysfunction

Medical Management

- Maintain ABC's
- Treat stimuli
- Stop bleeding
- Careful of oral and mucosal bleeding
- Correct hypovolemia, hypotension, hypoxia, and acidosis
- Stop microclotting to maintain perfusion

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Blood Products

Risks of transfusion

- Non-infectious: wrong blood
- Infectious: HIV, hepatitis
- Immunologic: Cytomegalovirus (CMV)
- Aged blood :Hgb problems

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Blood Products

RBC's

- Action: increase O₂ carrying capacity
- Indications:
 - Significant ullet H&H with normal volume
 - -Slow blood loss
- Avoid fluid and circulatory overload
- Administration: blood filter, 2-4 hours
- Complications: transfusion reaction, hepatitis

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Blood Replacement

- Coagulation components: Platelets
 - Action: ↑ platelet count, aides clotting

 - Administration: component filter, rapid infusion
 - Complications: transfusion reaction, mismatching, hepatitis, allergic reactions, febrile reaction

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Blood Replacement

- Fresh Frozen Plasma
 - Action: ↑ clotting factors, water and electrolytes
 - No platelets
 - Indications: coagulant deficiencies, viable
 Factor V and VIII
 - Administration: filter, rapidly, thaw
 - Complications: same as platelets

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Blood Replacement

- Cryoprecipitate
 - Action: raises Factors VIII + XIII prevents and controls bleeding,

fibrinogen and anti III

- **Indications:** Hemophilia A, von Willebrands, DIC
- Administration: filter, rapidly
- Complications: hepatitis

Blood Replacement

- Adverse Reactions
 - Hyperkalemia, Hypocalcemia
 - Decreased 2,3Diphosphoglycerate, ammonia intoxication
 - Hypothermia, Hepatitis, HIV
 - Cytomegalovirus, Acute Respiratory Distress Syndrome (ARDS) – Transfusion Related Acute Lung Injury (TRALI)

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Anemia's

- Deficiency of hemoglobin
- Most common causes
 - Excessive blood loss
 - Excessive blood cell destruction (hemolysis)
 - Deficient red blood cell production (ineffective hematopoiesis)

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Classifications

- Production vs. destruction of loss
- Red Blood Cell Size
 - Microcytic anemia: iron deficiency, thalassemia
 - Macrocytic anemia: megaloblastic—deficiency of B12, folate, hypothyroidism, ETOH abuse, drugs
 - Normocytic anemia: acute blood loss, aplastic
 - Dimorphic anemia: 2 causes act together
 - Heinz Body anemia: cytoplasm of RBC's have dark spots--dietary

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Complications	
Decreased Exercise Tolerance/fatigue	
Hypoxemia	
Cold intolerance	
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Treatments

- Depends on cause
 - Iron deficiency, vitamin supplementation, epoetin alfa
 - Blood transfusion

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Cancer

TO REMEMBER

- Immunosuppression
 - Due to drugs, cancer itself
 - Nutritional concerns
 - Organ dysfunction
- Hypercoagulable: increased risk of DVT
 - Cancer cells
 - -Stress hormones
 - Bed rest

Wounds

- Pressure Ulcers
 - A pressure ulcer is an area of skin that breaks down when an irritant has occurred
 - -Risk Factors
 - Wheelchair, cannot move, malnutrition
 - Decreased blood flow
 - Alzheimer's disease, fragile skin
 - Urinary incontinence or bowel incontinence

Symptoms of Pressure Ulcer

- Red skin that gets worse over time, blister then open
- Commonly occur
 - Buttocks, elbow, hips, heels, ankles,
 - Shoulders, back, back of head

Pressure Ulcer Stages

• Stage I: reddened area, when pressed does

not turn white

• Stage II: skin blisters or forms open sore

• Stage III: skin now develops an open, sunken

crater

• Stage IV: pressure ulcer becomes so deep that

there is damage to the muscle, bone,

tendons and joints

Peripheral IV Review

Complications: phlebitis, extravasation of IV fluids and/or medications, hematoma formation

Thrombophlebitis 15%

Minimize catheter movement, upper extremity only, smallest suitable catheter, less than 3 days

Extravasation

- 1. Stop injection, leave catheter in place
- 2. Slowly aspirate, apply pressure
- 3. Remove IV access
- 4. Inform MD and obtain orders
- 5. Elevate area 48 hours, warm/cold compress
- 6. Initiate substance-specific antidote Phentolamine (regitine), Sodium Thiosulfate, Hyaluronidase (Hylenex, Vitrase)

Extravasation

- Observe the region for pain, induration or necrosis
- Continue warm/cold therapy for 48-72 H
- Advise patient to resume activity with affected limb as tolerated
- Consider surgical evaluation for persistent or worsening symptoms

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Know!

- <u>Cellulitis</u>:common and sometimes painful bacterial skin infection. It may first appear as a red, swollen area that feels hot and tender to the touch. The redness and swelling can spread quickly. It most often affects the skin of the lower legs, although the infection can occur anywhere on a person's body or face.
- <u>Necrotizing fasciitis</u>:an infection that results in the death of parts of the body's soft tissue. A severe disease of sudden onset that spreads rapidly. Symptoms include red or purple skin in the affected area, severe pain, fever, and vomiting.

Hematology Pearls

- <u>DIC=high PT/PTT, low fibrinogen, low platelets, high FSP (FDP), high D-dimer</u>
- Give heparin in DIC → accelerates formation of antithrombin III, inactivates thrombin and prevents conversion of fibrinogen into fibrin

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A primary chemical mediator in anaphylactic reaction is:



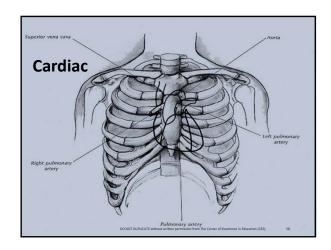
- A. Myocardial depressant factor
- B. Histamine
- C. Complement
- D. Interferon

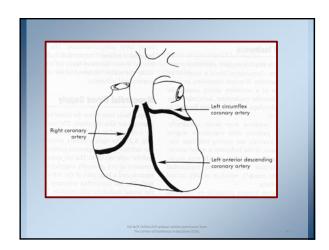
(The pathophysiology of anaphylaxis includes: Bronchospasm, hemolysis and rapid DIC, increased vascular permeability and third spacing)

Which of the following lab diagnostic findings will most likely be seen in DIC?
A. PT & PTT prolonged
B. Fibrinogen increased
C. Platelet count increased
D. D-dimer normal
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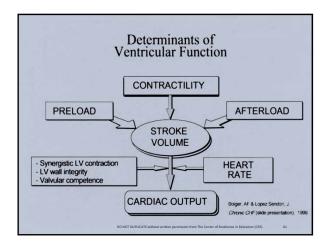
A. Signs of thrombus formation B. Excessive bleeding C. Decrease in platelet count D. All the above

Essentials of Care: Vital Signs Heart rate, respiratory rate, temperature Blood Pressure Systolic – determines SV Diastolic – Arterial tone Tissue Oxygenation Supply and Demand GOAL: Enhance O₂ Delivery and Decrease O₂ Demand

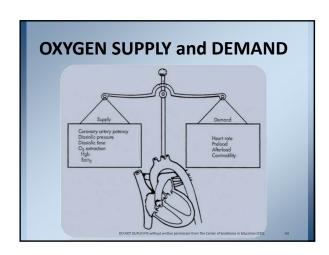


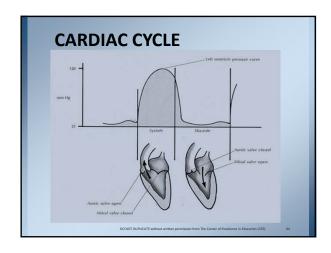


Coronary Perfusion Cardiac cycle Coronary arteries are perfused during diastole Coronary Artery Perfusion Pressure (CAPP) CAPP= Diastolic BP - pulmonary artery wedge pressure Normal is 60-80 mmHg



Afterload Afterload Decrease: A: ACEI, ARB, Alpha Antagonists (Prazosin, Tamsulosin, Terazosin, Silodosin) B: Beta Blockers C: Calcium Channel Blockers Afterload Increase: Levophed Vasopressin Neosynephrine



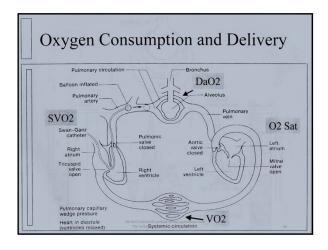


Heart Sounds

- S₁: beginning of ventricular systole
- S₂: beginning of ventricular diastole
- S₃: always pathologic, heard in early diastole.

 Think fluid overload
- S₄: always pathologic, late sound.

 Think decreased compliance



Hemodynamics

Cardiac output 4-8 liters/min
Cardiac index 2.5-4 liters/min/m²

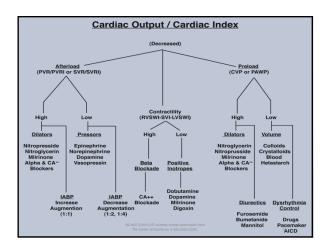
Central venous pressure 2-6

Pulmonary artery pressure 20-25/10-15
Pulmonary artery wedge pressure 4-12

Pulmonary vascular resistance < 250dynes/sec/cm²

Systemic vascular resistance 800-1200dynes/sec/cm²

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Electrolytes and the Heart • Hypokalemia: ventricular irritability • T with prominent U wave • T-wave + U-wave same amplitude • Prolongation of QT interval (K+ < 2.0)

Electrolytes and the Heart Hyperkalemia; asystole - > 5.5 tall, narrow, peaked T waves - P-wave widens - QRS widens - > 8.0 Sine wave

Electrolytes and the Heart

Hyperkalemia

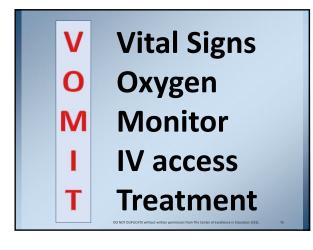
- -Treatment:
 - Remove Potassium: kayexalate or dialysis, lasix
 - Shift Potassium: insulin and dextrose, sodium bicarb and calcium
 - Albuterol breathing treatment

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Electrolytes and the Heart Hypocalcemia: Torsades de Pointes Prolonged QT Prolonged ST segment Hypercalcemia: agonal or asystole Shortened QT Shortened ST segment **Torsades de Pointes** **

Electrolytes and the Heart Hypomagnesemia: Torsades de Pointes - Prolonged QT - Broad, flattened T-wave - Torsade's de Pointes Hypermagnesemia: agonal to asystole - PR, QT prolonged - Prolonged QRS

Coronary Artery Disease Definition Pathophysiology Etiology Risk Factors Clinical Manifestations Heart failure, angina, unstable angina, STEMI, NSTEMI, sudden death



• Clinical Presentation • ECG Presentation • Rest, NTG, ASA - Supply and Demand

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Angina Management

- Antiplatelet Therapy
 - Aspirin (ASA)
 - Phosphodiesterase (PDE) inhibitors (Persantine)
 - ADP inhibitors (Plavix/Effient)
- Anticoagulants
 - Heparin/low molecular weight
 - $\\ Coumadin$

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Angina Management

- Vasodilator
 - Nitroglycerin: patch, sublingual, longer acting--Imdur
- Beta Blocker
 - Decreases MVO₂
 - Regulates blood pressure, heart rate & rhythm
- ACE Inhibitor
 - Blood pressure control, reduces remodeling

Unstable Angina (UA) • Clinical Presentation, ECG, enzymes • Pathophysiology: Blood Clot • ACS-Acute Coronary Syndrome

UA/NSTEMI

(Unstable angina/Non-ST elevation myocardial infarction)

- Biochemical Markers
 - Troponin CPK -
 - Diagnosis: unstable angina
 - Prognosis: high risk

Biochemical Markers

- Troponin + CPK +
- Diagnosis: NSTEMI
- Prognosis: high risk

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UA/NSTEMI

- Treatment management
- Cath lab
- ♠ MVO₂supply
- **₩** MVO₂ demand
- -ASA
- Beta Blockers
- Heparin
- NTG
- Morphine
- GP IIb-IIIa Inhibitor drugs

UA/NSTEMI

- Interventional
 - Percutaneous Transluminal Coronary Angioplasty (PTCA)
 - -Stent placement
 - DCA (Directional Coronary Atherectomy)

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Nursing Care of Interventional Cardiology Patient

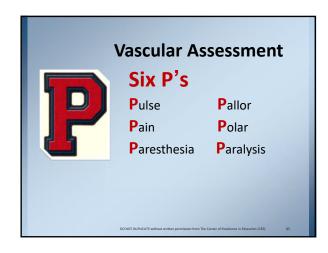
Pre-procedure

- NPO, consent
- Labs, ECG, insulin orders, oral medications for patient with diabetes, pre-hydrate, Mucomyst for renal insufficiency patients.
- Vascular exam, allergies

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Post Procedure

- Monitor ECG
- Vascular assessment
- Labs, heparin protocol, IIb IIIa infusion
- Activity restrictions, progression
- Sheath removal
- Medications



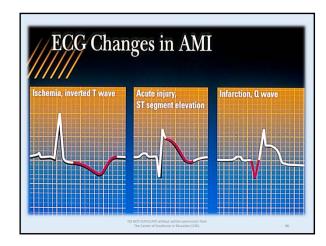
Peripheral Vascular Insufficiency

- Arterial/Venous
- Carotid artery stenosis—endarterectomy
- Fem-Pop Bypass—stent
- Peripheral Stents
- Improving flow—perfusion assessments

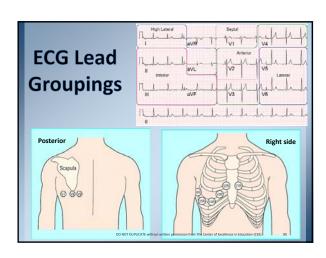
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STEMI

- Etiology: occlusive clot causing full thickness death
- Clinical Presentation
- Labs: Troponin, LDH, CPK, MB Band all +
- ECG, ECHO, Chest x-ray



ECG Lead Groupings			
LOCATION	LEADS	CORONARY ARTERY AFFECTED	
Anterior	V ₃ , V ₄	Left Anterior Descending	
		Left Anterior Descending	
Anteroseptal		Left Anterior Descending	
	I, aVL (high lateral), V ₅ , V ₆ (low lateral)		
Anterolateral	V ₃ , V ₄ , V ₅ , V ₆ (I, aVL)	Left Coronary Artery	
Right Ventricle	V _{4R} , V _{5R} , (V _{5R} may be transient)	Right Coronary Artery	
	V_7,V_8,V_9 or reciprocal in V_1,V_2,V_3	RCA and/or LCA	



STEMI: Acute Management

- ABC, VOMIT, MONA
 - ECG, vital signs, BLS, ACLS
 - Hemodynamic parameters
- Reduce size of infarct
 - Door to diagnosis and treatment
 - Door to drug = 30 min
 - Door to cath lab = 90 min

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STEMI: Management

- Diagnose: Clinical Presentation, ECG, Enzymes
- Treatment paradigm: open artery
 - Reperfusion therapies
 - Cath lab Percutaneous Coronary Intervention (PCI)
 - Fibrinolysis
 - CABG

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AMI: Management

Decrease myocardial oxygen consumption

- -Oxygen
- Pain control
- Beta Blockers
- ACE Inhibitors
- Rhythm control

STEMI: Right Ventricle Infarction

Assess for clinical indications of right ventricle myocardial infarction

- ECG changes V_{4R} , V_{5R} , V_{6R}
- ↓RAP, ↓PAWP
- ↓CO, CI, MAP, ↑ SVR
- Clinical indications of right ventricle failure
- Minimal to absent pulmonary congestion

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Intra-Aortic Balloon Pump

- Two functions
 - Decrease afterload
 - Increase coronary perfusion
- Absolute contraindication: aortic insufficiency
- Monitor for:
 - Vascular exam
 - -Timing

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Right Ventricle Infarction: Management

• Maintain adequate filling pressures:

Administer volume

Avoid diuretics and/or venodilators

Maintain contractility

Hemodynamics				
✓ C\ ✓ PA ✓ PA	VP AP AOP /CO	ricle Infarction ↑ normal or ↓ normal or ↓ normal or ↓	Left Ventr ✓ CVP ✓ PAP ✓ PAOP ✓ CI/CO ✓ SVR	ricle Infarction normal or ↑ ↑ ↓ ↑
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Complications: AMI			
Dysrhythmias Heart failure Cardiogenic shock Papillary muscle dysfunction/or rupture Ventricular septal defect (VSD)	 Cardiac rupture Ventricular Aneurysm Pericarditis Dressler's Syndrome Sudden death 		
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Coronary Artery Disease Treatment • Medical: two arms - Medical management: medications only - Interventional: stents with meds • Meds: antiplatelet, vasodilator, Beta Blockers, ACE inhibitors, Statin • Surgical: CABG: continue med treatment • Pre-op, post-op care

Aneurysm
Dissecting
Repair
Rupture
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Aortic Dissection

Type I, II, III

- Goal: Control the BP
- **Treatment:** surgery for repair or stent
- Post Op watch: BP, pulmonary concerns, renal
- Nursing: Ambulate, incentive spirometer
- Dissecting
- Repair

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Cardiac Surgery (PCCN Only) Cardiac surgery (e.g., open chest surgery) more than 48 hours postoperative Care of patient: Splinting with cough, DVT prevention

Valve Surgery (PCCN Only) Mitral/Aortic valve repair/replacement - Median sternotomy - Right/left thoracotomy - Post operative care - Valve types - Anticoagulation

Etiology of Heart Failure Left Ventricular Failure Right Ventricular Failure • CAD/RV infarct • CAD/LV infarct Dysrhythmias Dysrhythmias Volume overload Volume overload Valvular disease Valvular disease • VSD • VSD • CMP • CMP • Coarctation of Aorta Myocardial contusion Tamponade • Pulmonary hypertension

Clinical Presentation: LVF • Tachycardia • Weakness, fatigue • Tachypnea, dyspnea, Mental confusion orthopnea, PND Murmur mitral regurg • Left sided S₃ • ABG's Displaced point max Chest x-ray intensity ECG: atrial arrhythmia, • Cough, pulsus alternans LAE, LVH Oliguria

Clinical Present	ation: RVF
JVD Hepto-jugular Reflux (HJR) Dependent edema Heave at sternum Hepatomegaly Anorexia, nausea, vomiting, abdominal pain Ascites	 Nocturia Weakness, fatigue Weight gain Murmur tricuspid regurg Right sided S₃ CVP Abnormal liver functions ECG: RAE, RVH, atrial arrhythmia

Heart Fail	ure: Ma	anagement
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- Treat the cause, improve oxygenation
- Decrease MVO₂: Decrease preload
 - Monitor volume status: low sodium diet
 - Diuretics, Natrecor
 - NTG, ACE Inhibitors, pulmonary vasodilators, IABP
- Decrease Afterload
- Beta Blockers, Carvedilol (Coreg)
- Control dysrhythmias

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Heart Failure: Management Increase contractility: 1 Mechanical: Intra-aortic balloon pump 2. Pharmacological Dobutamine - Phosphodiesterase inhibitors: Milrinone - Dopamine - Digoxin

Cardiomyopathies PCCN			
Dilated			
• Hypertrophic			
• Stress induced (Takotsubo)			
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Cardiomyopathies CCRN			
Cardiomyopathies CCRN			
• Dilated			
Dilated Hypertrophic			
• Dilated			
DilatedHypertrophicIdiopathic	nucleon (KTE) 110		

Cardiovascular Issues 1. Acute Inflammatory Disease: - Myocarditis - Endocarditis - Pericarditis - Pericarditis 2. Hypertensive crisis 3. Cardiac tamponade

Transcatheter aortic valve replacement (TAVR) Minimally invasive heart procedure to replace a narrowed aortic valve Pts who are intermediate or high risk of complications from surgical aortic valve replacement Carried Friedh arch of the acrts Transagenal saccess #vrough 8p of heart) Transferent (BCC698 Byrough femoral artery)

TAVR Risks

- Blood vessel complications
- Problems with the replacement valve, such as the valve slipping out of place or leaking
- Heart rhythm problems (arrhythmias) and the need for pacemaker implantation
- Kidney disease
- Heart attack
- Infection
- Death

Know Your Dysrhythmias!

- Bradycardias
- Heart blocks
- Ventricular rhythms
- Tachycardias
- Know how to treat above!
- Devices (ICD's pacemakers)

Know Your Lines and Monitoring Lines Arterial lines PA catheters CVP Monitoring Grinterval monitoring To segment monitoring: ST alarm parameter 1 mm or less above and below the patient's baseline ST segment. Set the isoelectric point and the ST-segment measurement point (60 milliseconds beyond the J point) before the start of ST-segment monitoring

Cardiac Pearls

- ABC's
- Cardiac output/index--preservation of PERFUSION
- Maintaining HR X SV
 - PRELOAD
 - AFTERLOAD
 - CONTRACTILITY

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Cardiac Pearls

- ST segment depression = ischemia
- ST segment elevation = current injury
- IABP=increase coronary perfusion, decrease afterload: so it increases myocardial oxygen supply and decreased demand

You are caring for a patient recently admitted with an inferior wall myocardial infarction. Which of the following 12 lead ECG findings would you anticipate?

A. T wave inversion I, and AVL
B. Q wave formation and ST segment elevation in II, III, and AVF
C. QRS duration > 0.01 in all 12 leads
D. R wave taller in V₆

Your patient with an inferior wall myocardial infarction also has a right ventricular infarction.

He soon develops right ventricular failure. Which of the following data obtained would correlate this?

A. PAP 23/8 PCWP 19 CVP 20
B. PAP 54/28 PCWP 14 CVP 14
C. PAP 28/10 PCWP 10 CVP 20
D. PAP 12/4 PCWP 24 CVP 18

The most common complication of a myocardial infarction is: A. Arrhythmias B. Heart failure C. Cardiogenic shock D. Pulmonary edema

A normal wedge pressure, increased pulmonary artery pressures, and evidence of right ventricular failure would most likely indicate:

- A. Cardiac tamponade
- B. Left ventricular failure
- C. Myocardial infarction
- D. Pulmonary embolism



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Medical management of valvular disease includes:

- A. Prevention of infection
- B. Treatment of heart failure
- C. Treatment of dysrhythmias
- D. All of the above



Symptoms to evaluate for the diagnosis of heart failure may include: A. Dyspnea at rest B. Orthopnea C. Nocturnal cough D. All the above

Endocrine

Functions: endocrine system regulates secretion of hormones that alter metabolic functions



- Chemical reactions, transport of chemicals
- Growth and development, metabolism
- Fluid and electrolyte, acid-base balance
- Adaptation, reproduction

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Diabetes Insipidus

- Definition: clinical condition characterized by impaired renal conservation of water, resulting in polyuria, low urine specific gravity, dehydration, ↑ serum Na⁺: caused by <u>deficiency of Antidiuretic</u> <u>Hormone</u> (ADH) from the pituitary or decreased renal responsiveness to ADH
- Etiology: neurogenic, nephrogenic (lithium), psychogenic (polydipsia)

71)

Pathophysiology

- Deficiency of ADH or inadequate renal tubule response to ADH
- Diuresis of large volumes of hypotonic urine
- Dehydration and hypernatremia
- Shock and/or neuro effects
- Permanent vs temporary

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Clinical Presentation

- Polyuria: 5 to 15 liter/day (>200ml/h X 2h)
- Thirst, fatigue
- Dehydration: weight loss, poor skin turgor, postural hypotension, ↓CVP, ↓PAP, ↓PWCP, ↓CO/CI
- Neurologic: restlessness, confusion, irritability, seizures, lethargy, coma

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Clinical Presentation

- Urine specific gravity: <1.005
- Serum sodium >145 mEq/liter
- Elevated BUN
- Increased serum osmolality
- Elevated H&H

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- Management: detect clinical indication of diabetes insipidus
 - Monitor urine output, weight, serum labs, hypovolemia
 - Correct fluid deficit
- Administer exogenous ADH, aqueous vasopressin IV or sq, Desmopressin (DDAVP), Diapid (intranasal)

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Diabetes insipidus:

Dehydration and high serum Na⁺

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Syndrome of Inappropriate Antidiuretic Hormone (SIADH)

 Definition: clinical condition characterized by impaired renal excretion of water, resulting in oliguria, high urine specific gravity, water intoxication and hyponatremia

SIADH: Neurogenic

- Neurogenic: Pituitary tumor, CNS trauma, stroke, ICH, CNS infection, Guillain-Barre syndrome, CVA, nonmalignant pulmonary disease
- Ectopic SIADH: production of a substance indistinguishable from ADH by tissue
 - -Oat-cell CA
- **Nephrogenic** SIADH: general anesthetics, narcotics, tricyclics, Tylenol, anticonvulsants
- Hypoxia, stress, multifactorial in ICU patient

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SIADH

- Oliguria: urine output less than 0.5ml/kg/hour actual body weight
- Urine specific gravity: >1.030
- Clinical indications of over-hydration
- ↑CVP, ↑PAOP
- Anorexia, nausea/vomiting, diarrhea
- Dyspnea and pulmonary edema

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SIADH

- Headache, personality changes, altered level of consciousness
- Seizures
- Muscle weakness or cramps
- Serum sodium <120mEq/liter
- Serum ADH level ↑ if neurogenic
- ↓ BUN
- ↓Serum osmolality
- ↓ H&H

SIADH Treatment

- Detect SIADH in high risk patients:
 - Monitor urine output, specific gravity
- Treat cause
 - Decrease water intake
 - Surgery to remove malignancy
 - Discontinue causative drugs

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SIADH Treatment

- Correct fluid volume excess
 - Fluid restriction
 - Diuretics
- Correct electrolyte imbalance
 - Increase dietary sodium
 - Hypertonic saline for Na⁺ <125 or if experiencing seizures
- Institute seizure precautions

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SIADH

- Swimming in water
 - LOW Serum NA⁺

Diabetic Ketoacidosis

- <u>Diabetes mellitus (DM)</u>: a group of metabolic diseases characterized by hyperglycemia that results from defects in insulin secretion, insulin action or both
- <u>Diabetic ketoacidosis (DKA)</u>: hyperglycemic crisis associated with metabolic acidosis and elevated serum ketones, the most serious metabolic disturbance of type I DM
- Hyperglycemic hyperosmolar nonketotic condition (HHNK): hyperglycemic crisis associated with the absence of ketone formation, most serious metabolic disturbance type 2 DM

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DKA

- Insufficient insulin=hyperglycemia=osmotic diuresis=glycosuria, dehydration, and electrolyte imbalance
- Breakdown of glycogen is activated and its synthesis inhibited=impaired glucose uptake by adipose tissue causes impaired triglyceride synthesis and liberation of free fatty acids into the blood
- Excessive free fatty acids enter the liver=ketoacidosis

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DKA

- Undiagnosed type I DM
- Known type I DM
 - Illness, infection, omission of insulin, trauma, surgery, non-compliance
- Non-diabetic: Cushing's syndrome, hyperthyroidism, pancreatitis, drugs (steroids, thiazide diuretics, dilantin), pregnancy

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DKA Presentation

- Serum Glucose > 300-800
- Na normal
- K elevated due to ketosis and then decreases with insulin
- Ketones ↑, BUN/creatinine ↑
- Serum osmolality > 295-330 (dehydrated)
- ABG's = metabolic acidosis (due to ketones, corrects with fluid)
- WBC may be increased

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DKA Presentation

- Nausea/vomiting, abdominal pain, polyphagia, polydipsia, polyuria
- Weakness, fatigue, weight loss
- Clinical indications of dehydration
- Tachycardia, orthostatic hypotension
- Kussmaul's breathing
- Lethargy progressing to coma

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DKA Treatment

- ABC's, monitor
- Identify and treat cause: infection: cultures
- Correct fluid volume deficit
- Correct blood sugar

DKA: Treatment Normalize serum glucose Regular insulin 0.1-0.15 units/kg followed by infusion Serum glucose should drop no more than 75-100 mg/dl per hour to avoid hypoglycemia hypokalemia cerebral edema

Normalize serum glucose Infusion decreased when blood glucose < 250 mg/dl Subcutaneous insulin by sliding scale started before IV infusion discontinued Replace potassium, phosphate, magnesium Correct acid-base imbalance: fluids Maintain safety Manage until the gap closes!

Complications	
<u>Cardiovascular</u>	Neurologic
Hypovolemic shock	Seizures, cerebral edema, coma
Dysrhythmias	Renal
Embolism	Acute kidney injury
MI	Electrolyte
Pulmonary edema	imbalance
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Metabolic Acidosis

- Causes of metabolic acidosis
 - Diabetic or ETOH ketoacidosis
 - Renal failure
 - Lactic acidosis
 - Poisoning: ASA overdose

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Hyperosmolar Hyperglycemic Nonketotic Condition (HHNK)

- **Definition:** hyperglycemic crisis
- Etiology: usually seen in patients over 50 years of age, with glucose intolerance: may follow:
 - Pancreatitis, burns, hepatitis, trauma, ETOH, hypertonic nutrition, drugs (beta blockers, Thiazide, Dilantin, steroids)

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HHNK Pathophysiology

- Relative insulin deficiency
- DEHYDRATION

HHNK Presentation

- Glucose >600-2000
- Low serum Na⁺ (appears to be low to > BS, don't fix it!)
- Low K+
- BUN/creatinine ↑
- Serum osmolality ≈ 330-450 high
- ABG's: normal pH. If acid consider lactic acidosis

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Treatment

- ABC's, monitor, identify cause: infection
- Correct fluid volume deficit
- Normalize serum glucose level
- Correct electrolyte imbalance
- Safety
- Monitor for complications:
 - Hypovolemic shock, dysrhythmias, acute renal failure, thromboembolism, myocardial infarction, pulmonary embolism, cerebral edema

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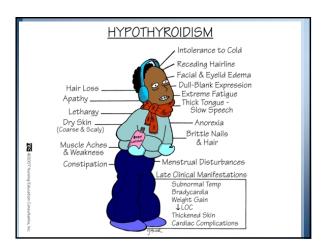
Hypoglycemia

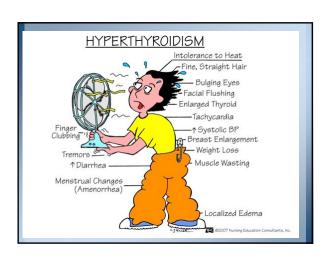
- Females > Males
- More common in the elderly
- ETOH, infection
- Signs and symptoms of acute hypoglycemia
- Treatment: replace

More Endocrine Disorders: A Little of This and Too Much of That

- **Hyperthyroid:** Thyrotoxicosis. Thyroid storm. Too much thyroid.
- Hypothyroid: Myxedema. Deficiency of thyroid







Endocrine Pearls SIADH = low sodium levels Fluid restriction, 3% NS DI = neurological injury High serum sodium Dehydration Vasopressin = ADH = Pitressin HHNK = Severe dehydration Normal serum osmolarity= 275-295 Acidosis causes shift of cellular K* to serum

SIADH is clinically manifested by:

- A. Hyperosmolar state
- B. Low output state
- C. Myxedema state
- D. Water intoxication state

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The "cardinal sign" of SIADH is: A. Hyponatremia B. Urinary output of 10 liters/day C. Hypotension D. Systemic edema

Which of the following is characteristic of diabetes insipidus?

- A. Low urine osmolarity
- B. Serum osmolarity increased
- C. Serum sodium elevated
- D. All of the above



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True or False



- In DKA the patient is initially hyponatremic?
- In HHNK the patient is initially hyponatremic?
- Do you treat the low Na⁺?

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GI Hemorrhage

Introduction

- Loss of new or old blood from GI tract
- Emesis or stool
- 85% of all GI hemorrhages are upper GI tract
- Death can result from circulatory failure and shock



Pathophysiology

- Ulceration when mucosa is injured, allowing acid to diffuse through broken barrier
- Hemorrhage, perforation or scarring with obstruction
 - Gastric ulcers: hematemesis or perforation
 - Duodenal ulcers: melena, perforation, scarring with obstruction

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Pathophysiology

- Most common causes of upper GI hemorrhage
 - Peptic ulcers
 - Esophageal or gastric varices
 - Gastritis
 - Mallory-Weiss tear
- Lower GI hemorrhage
 - Diverticular disease, tumors, ulcerative colitis

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Diagnostics

- Chemistry: amylase, lipase, proteins, albumin, transferrin, hemoglobin and hematocrit
- GI scope

Management

- Ensure airway, oxygenation, ventilation
- Restore circulating blood volume and control bleeding:
 - IV, isotonic crystalloids, colloids
 - Blood transfusion
 - Vasopressin: WATCH FOR????
- NG tube
- Prepare for procedures and/or OR or IR, H₂blockers or PPI - IV

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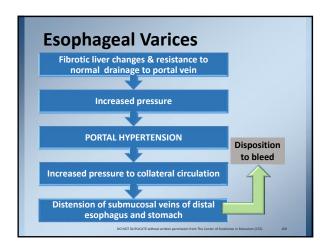
Treatment: ABC's

- Therapy for H. pylori
- Maintain fluid and electrolytes
- Nutritional concerns
- Complications:
 - Aspiration pneumonitis, recurrent bleeding, perforation, acute pancreatitis, MI, DIC, sepsis, shock
 - Ammonia levels

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Esophageal Varices

- **Definition:** dilation of the submucosal esophageal veins
- **Etiology:** cirrhosis, portal vein thrombosis, hepatic venous outflow obstruction, congenital hepatic fibrosis



Esophageal Varices Management: - ABC's, restore circulating blood volume - Endoscopy: sclerotherapy, ligation or banding - Vasopressin, administer sandostatin (Octreotide) - TIPS: transjugular intrahepatic portosystemic shunt - All other complications: electrolyte, coagulation, liver fracture, ETOH withdrawal, renal failure, pneumonia

Treatment Decrease gastric production Local vasoconstriction Esophageal balloon tamponade Octreotide

Hepatic Failure

- **Definition hepatic failure:** inability of liver to perform organ functions
- **Hepatic encephalopathy:** neurologic failure as a result of hepatic failure

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Hepatic Failure: Acute

- Viruses
- Fulminant viral hepatitis
- Herpes simplex
- CMV
- Hepatotoxic drugs
- Ischemia
- Trauma
- Reye's syndrome
- Acute fatty liver of pregnancy
- Acute hepatic vein occlusion

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Hepatic Failure: Chronic

Chronic liver failure

- Cirrhosis
- Wilson's disease
- Primary or metastatic tumor of the liver

Cirrhosis

- Liver parenchymal cells are progressively destroyed and replaced with fibrotic tissue, results impaired hepatic function: 3/4 of liver can be destroyed before symptoms appear
- Distortion, twisting, and constriction of central sections cause impedance of portal blood flow and portal hypertension

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Portal Hypertension

- Esophageal varices
- Splenomegaly --thrombocytopenia, vitamin K deficiency
- Inability to produce adequate bile
- Impaired carbohydrate, fat, protein metabolism (hypoglycemia)
- Inability to store vitamins and manufacture clotting factors

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Portal Hypertension

- Inability to detoxify toxins and drugs and remove bacteria
 - Drug or toxin intoxication
 - Hepatic encephalopathy
 - Ammonia: protein metabolism
 - Convert ammonia to urea

Clinical Presentation

Fulminant hepatic failure

 Jaundice, tachycardia, hypotension, fluid retention, ascites, ↓ urine output, spider nevi, palmar erythema, bleeding, electrolyte imbalance, asterixis, hyperventilation, increased ICP, sepsis, portal hypertension

Cirrhosis

 Azotemia, bruising, bleeding, nutritional abnormalities, fatigue, weight loss, impaired bilirubin metabolism, respiratory alkalosis

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Management

- Identify and treat cause of liver failure
 - Avoid hepatotoxic drugs
 - Avoid ETOH
 - Monitor liver function tests
- Airway
- Aspiration: Safety

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Management

- Ascites, fluid overload, pleural effusion, LeVeen or Denver shunt: fluid status
- Renal insufficiency (hepatorenal), fluid restrictions, diuresis (aldosterone antagonists: Aldactone)
- Immunocompromised
- Electrolyte imbalance: <u>↓K+, ↓Ca+</u>

Management • Empty bowel of nitrogen-containing materials - Neomycin orally or NGT - Lactulose - Blood in bowel

Treatment

Decrease portal HTN, Beta Blockers

- Nutritional support
- Prevent increased ICP—positioning, fluid status
- Prevent and monitor bleeding
- Monitor for infection
- Skin breakdown
- ETOH withdrawal

• Malnutrition - Immunosuppression - Poor wound healing - Edema, ascites • Hemorrhage - Esophageal varices - Coagulopathy, DIC • Hypoglycemia • Electrolyte imbalance	• ARDS • Peritonitis • Sepsis • Hepatorenal syndrome • Gradual loss of function. • Associated with cirrhosis • Oliguria and ↑ urine Na* • ATN • Cerebral edema
Electrolyte imbalance	Cerebral edema

Pancreatitis

- Definition: acute inflammation of the pancreas forms include:
 - Interstitial: edematous pancreas, hypovolemia
 - Hemorrhagic: extensive necrosis of pancreas and peripancreatic tissue and fat, erosion into blood vessels, hemorrhage, SIRS often occurs
 - Acute vs. Severe Acute Pancreatitis (SAP)

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Etiology

- Alcoholism
- Obstruction of common bile duct
 - Cholelithiasis
 - Post ERCP
 - Hypertriglyceridemia
 Thiazide
 - Lasix, estrogen
- Peptic ulcer w/ perforated
- Cancer

- Trauma, surgical
- Radiation
- Pregnancy
- Ovarian cyst
- Hypercalcemia
- Lupus
- Infections
- Ischemia, Post CPB
- Idiopathic (20%)

Pathophysiology

- Etiologic factor triggers activation of pancreatic enzymes and pancreatic cell injury = autodigestion of pancreas = damage to acinar cells = erosion into vessels = inflammatory process = necrosis of fat and exudates with high albumin content = hypoalbuminemia and ascites
- Hypocalcemia
- Release of necrotic toxins (cascade) may cause sepsis and SIRS

Clinically

- Acutely ill, hyperthermic
- PAIN
- Nausea & vomiting, dyspepsia, flatulence, weight loss, weakness
- Look like AMI
 - Tachycardia, fever, hypotension, jaundice, Grey Turner's sign, abdominal distention, ascites, ↓BS, steatorrhea, respiratory findings
- Shock

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Clinically

- ↓Ca^{++,} ↓K⁺, hyperglycemia
- Elevated serum amylase and lipase
- Elevated urine amylase
- Elevated liver function tests
- CT, MRI = pancreatic swelling, edema or necrosis

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Treatment: ABC'S

Decrease release of and destruction by pancreatic enzymes

- Pain management
- Nutritional care, TPN, lipids, electrolytes
- Prevent infection, ETOH withdrawal

Prevent Complications Hypoglycemia Hypocalcemia Pseudocysts Pancreatic abscess Pancreatic fistula Hypovolemic shock ARDS Prevent Complication Perforation Bleeding ETOH withdrawal Immobilization SIRS

Intestinal Infarction Definition: necrosis of intestinal wall resulting from ischemia Etiology arteriosclerosis, vasculitis, mural thrombus, emboli (atrial fibrillation), hypercoagulability, surgical procedures (aorta clamp), vasopressors, strangulated intestinal obstruction, intra-abdominal infection, cirrhosis

Pathophysiology
INFARCTION: Decrease in blood flow to major mesenteric vessels causes vasoconstriction, vasospasm, prolonged ischemia
Edema of intestinal wall, full thickness necrosis
peritonitis
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Infarction

- Clinical: anorexia, pallor, abdominal pain, severe cramping or nonspecific diffuse
 - Abdominal tenderness, urgent bowel movements
- Objective: tachycardia, hypotension, tachypnea, fever, dehydration, vomiting (persistent and/or bloody), abdominal guarding and rigidity

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Infarction

- Management: ABC's
- Maintain adequate circulating volume
- D/C vasopressors with bowel ischemia
- Prevent and treat pain (morphine)
- Prevent perforation (bowel rest)
 - NG tube, elevate HOB
- Prepare for surgical intervention

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Intra-Abdominal Hypertension

- Renal dysfunction
- Respiratory compromise
- Intra-abdominal pressure greater than 18 mmHg

Abdominal Compartment Syndrome Intra-abdominal Hypertension in adults is >12 mmHg ACS is present when intra-abdominal pressure rises and is sustained at > 20 mmHg and there is new organ dysfunction or failure. What Happens to the Body's Organs? A Vicious Cycle Total body fluid third space due to howel defense or the compression entitle difference due to howel defense organization for compression of the compartment of the c

Complications

- Fluid and electrolyte imbalance
- Hemorrhage, sepsis, shock
- Peritonitis
- ARDS
- Abscess
- Perforation

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GI Surgery

Obstruction: fluid balance, most common cause is adhesions, hernias, tumors, ulcers, infections, post-op patient

- Most frequent indications for GI surgery
- Diagnosis: X-ray, CT
- Treatment: fluids, electrolytes, NG tube, prepare OR

GI Surgery

Perforation: sudden onset abdominal pain, very ill

- Most common appendicitis, ruptured tic
- Presentation: hypovolemia, abdominal signs
- Diagnosis: WBC's, electrolytes, X-ray, CT
- Treatment: surgical repair

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Remember: The Gut

- Nutritional Support
- Electrolytes



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GI Pearls

- Arterial perfusion of small intestine
 - Superior mesenteric artery
- Complications of pancreatitis=bilateral rales, atelectasis of left base, pleural effusion and ARDS, HHNK, low Ca⁺
- Cullen's Sign: ecchymosis around umbilicus in hemorrhagic pancreatitis
- Kehr's Sign: splenic rupture=left shoulder pain due to diaphragmatic irritation.
- GI assessment: inspection, auscultation, palpation, labs

GI Pearls

- Retention of phosphorus causes a reciprocal drop in serum calcium – pancreatitis
- Elevated PO₄⁺=renal fracture, excess vitamin D, hypoparathyroidism, chemotherapy agents
- Decreased PO₄⁺ =ETOH, TPN, chronically acutely ill

Question



The administration of vasopressin should be most carefully monitored in patients who have:

- A. Diabetes insipidus
- B. Coronary artery disease
- C. Hypotension secondary to GI bleed
- D. Diabetes

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Question



The inability of the liver to conjugate what substance is a primary contributor to hepatic coma?

- A. Ammonia
- B. Urea
- C. Fatty acids
- D. Bilirubin

Question



Cimetidine or rantidine, act to reduce stress ulcers by inhibiting the production of which substance?

- A. Histamine
- B. Gastrin
- C. Acetylcholine
- D. Calcium

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Question



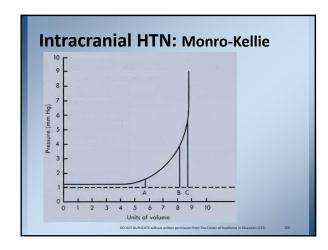
Which of the following laboratory findings is **MOST** specific for pancreatitis?

- A. Leukocytosis
- B. Elevated serum and urine amylase
- C. Hyperglycemia and hypokalemia
- D. Decreased serum albumin and total protein

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Cerebral Metabolism

- Oxygen requirements
 - 2% of body weight, 20% of cardiac output
 - Cerebral cortex most sensitive to O₂ delivery
 - Anoxia caused cerebral edema + neuron death
- Nutrient
 - High metabolic rate, glucose (ATP)
 - Brain does not require insulin to use glucose



Cerebral Blood Flow Cerebral perfusion pressure (CPP)= mean arterial pressure (MAP) - mean intracranial pressure (ICP) Changes in MAP or ICP affect CPP Normal MAP 70-105 mmHg Normal ICP 5 - 15 mmHg Normal CPP 60 - 100 mmHg

Cerebral Perfusion Pressure (CPP) Increase Cerebral Decrease Cerebral Blood Flow Blood Flow Hypercapnia Hypocapnia ◆ Hypoxemia • 🛧 Hyperoxemia Blood viscosity Blood viscosity Hyperthermia Hypothermia Drugs: vasodilators • Drugs: anesthetics, barbiturates

Assessment NEURO EXAM - Mental status Motor function - Sensory function - Cranial nerves – Deep tendon reflexes

Additional Assessment

- Inspect and palpate: face, head, orbits, raccoon eyes, nose, CSF leaks, (test + for glucose) halo, ears
- Clinical indications of meningeal irritation

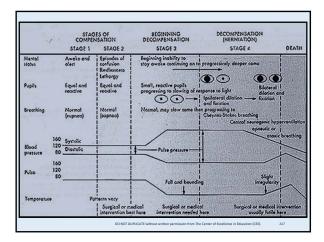
Additional Assessment

- Blood pressure and heart rate
- Respiratory rate and rhythm
 - Bradypnea: CNS depression
 - Cheyne-Stokes: cerebral hemisphere
 - Hyperventilation: lower midbrain or upper pons

 - Apneustic: mid to lower pons
 - Ataxic: medulla

Additional Ass	essment			
Temperature				
• Central fever: injury	to hypothalamus			
– Does <u>not</u> respond t	o antipyretics			
 Peripheral Fever 				
– Caused by infection				
– Does respond to ar	itipyretics			
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Nursing Care of	Neuro Patient			
• ABC's	Neuro Patient • Infection			
ABC's Aspiration	InfectionComplications of			
ABC'sAspirationICP/CPP/MAP	InfectionComplications of bed rest			
ABC'sAspirationICP/CPP/MAPVolume status	InfectionComplications of bed restDVT/PE			
ABC'sAspirationICP/CPP/MAPVolume statusNutrition	InfectionComplications of bed restDVT/PERehab potential			
ABC'sAspirationICP/CPP/MAPVolume status	InfectionComplications of bed restDVT/PE			
ABC'sAspirationICP/CPP/MAPVolume statusNutrition	InfectionComplications of bed restDVT/PERehab potential			

Clinical Picture • Change in LOC: early signs, late signs • Central nerve changes: change in pupils, vision, corneal reflex, swallow, contralateral motor, vomiting, head ache, seizures • LATE: vital signs = Cushing's Triad: — HTN with widened PP — Bradycardia — Change in respirations



Complications of Increased ICP

- Ischemia and edema
- Seizures
- Diabetes insipidus, SIADH
- Hydrocephalus
- DVT, stress ulcer
- Respiratory insufficiency , pneumonia

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Primary Types of Brain Injury

- Trauma: concussion, contusion, sheer injury
- Ischemia: global, regional
- Inflammatory/infection: meningitis--viral vs. bacterial
 - Glucose levels of CSF
- Compression tumor, edema, hematoma
- Metabolic: encephalopathies--anoxic, hypoxicischemic, metabolic, infectious

Closed Head Injury

- Etiology: blunt trauma, cell injury
- Pathophysiology:
 - -Focal injury: contusion
 - Partial or complete dysfunction for less than 24 hours, bruising, petechial hemorrhages, laceration may occur, areas of infarction and necrosis may occur = edema, intracranial hypertension



Closed Head Injury

- Concussion: transient state of partial or complete paralysis of cerebral functioning with complete recovery within 12 hours; headache
- Mild: no loss of consciousness or memory loss
- Classic: loss of consciousness or memory loss

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Closed Head Injury

- **Diffuse injury:** loss of consciousness > 24 hours, axonal disruption
 - Amnesia, residual deficits in memory
- Diffuse axonal injury: severe mechanical disruption of axons and neuronal pathways in both cerebral hemispheres, diencephalon, and brainstem

Closed Head Injury

Hypoxic Brain Damage

- Occurs most frequently in the arterial distribution between anterior cerebral artery and the MCA
- Occurs as a result of ↓CPP associated with a period of hypotension after the initial injury

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Space-Occupying Lesion

Think about:

- -Acute trauma
- -Chronic tumor
- -Growing in size
- -Signs and symptoms of increased intracranial pressure

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Management

- ABC's
- Prepare for OR
- Maintain CPP > 70
- Institute seizure precautions
- Assess for additional injuries
- Prevent/detect intracranial HTN and secondary brain injury

Brain Death

Cardinal finding in brain death

- Coma or unresponsiveness
- Absence of cerebral motor responses to pain in all extremities
- Absence of brain stem reflexes
- Apnea

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Brain Death

- Cerebral angiography: no Intracerebral filling at level of carotid bifurcation
- EEG: no electrical activity during a period of at least 30 minutes
- Transcranial doppler: no diastolic or reverberating flow
- Somatosensory and brain stem auditory evoked potentials
- Technetium Tc 99m brain scan: no uptake

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Intracranial Hematomas

Subdural hematoma (SDH):

- Spontaneously, older, ETOH
- Usually venous bleed, accumulated below dura mater, classification
 - Acute SDH: clinical indications occur within 24 hours
 - Subacute: within 2 weeks
 - Chronic: weeks to months

Surgical Interventions

- Burr holes: remove clots or blood
- <u>Cranial window</u>: allow expansion of brain tissue to < ICP
- <u>Craniotomy:</u> remove foreign objects, repair aneurysm

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Intracranial Hematomas

Epidural Hematoma (EDH):

- Linear skull fracture, usually arterial bleeding associated with tearing of arteries, accumulates above the dura mater
- History of precipitating event, history of short period of unconsciousness followed by lucid interval and then rapid deterioration, headache, increasing irritability

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Stroke: Ischemic

 Sudden, severe disruption of cerebral circulation with a subsequent loss of neurologic function caused by thrombus or embolus



Stroke: Ischemic • Etiology: atrial fibrillation, atherosclerosis, HTN, hypercoagulability • Clinical: sudden onset • Diagnosis: CT, MRI, cerebral angiogram

Risk Factors	
• Family history	Substance abuse
 Hypertension 	Oral contraceptives
• Smoking	• Dysrhythmias
 Hyperlipidema 	Hypercoagulability
Obesity	Sedentary lifestyle

Management ABC's VOMIT Time last know normal Identify type: ischemic or hemorrhagic CT Scan or MRI

Strokes: Ischemic

Management: ABC's

- Oxygenation, ventilation, prevent aspiration
- Decrease metabolic requirements
- Maintain cerebral perfusion
- Platelet aggregation inhibitors
- Anticoagulants
- Thrombolytic TPA
- Prevent complications

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Stroke: Hemorrhagic



- Definition: neurologic deficit caused by interruption of blood flow to the brain caused by vessel rupture
- ICH: trauma, HTN, tumor, thrombolytic, anticoagulants, bleeding disorders
- **Subarachnoid hemorrhage:** hemorrhage into the subarachnoid space
 - Aneurysms, AV malformations

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Clinical Presentation

- 90% of cases of ruptured aneurysm = HTN
- Warning: headache, generalized weakness, fatigue, ptosis, diplopia, blurred vision
- Sudden severe headache
- Nausea and vomiting

Clinical Presentation

- Restless, change in level of consciousness
- Meningeal irritation signs
- Seizures
- Site and size determine specific clinical presentation
- Diagnosis: CT scan, lumbar puncture, MRI, cerebral angiogram

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Treatment

- Maintain airway, ventilation, oxygenation
- Prevent/monitor clinical indications of intracranial hypertension
- Prevent/monitor for delayed ischemia following SAH
- <u>Identify vasospasm</u> by worsening of neurologic status: occurs anytime from the 3-21 days

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Treatment: Vasospasm

- Administer calcium channel blockers
- Triple H therapy: hypertension, hypervolemia, hemodilution
 - -SBP=120-150, Hct=30-33%
- Minimize potential for re-bleed and promote stability
- Stent

Procedures

- Aneurysm:
 - -Surgical: clipping, wrapping, ligation
 - Endovascular: coiling (embolization coils), intravascular balloon placement
- Arteriovenous malformation:
 - Surgical excision, embolization
- Intracranial hemorrhage:
 - -Surgical removal of clot

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Postoperative

Monitor

- Vasospasm
- Re-bleeding
- Cerebral edema and intracranial hypertension
- Hydrocephalus
- -SIADH
- Seizures

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Overall Stroke Care: Goals of Treatment

- Minimize damage and maximize recovery
- Essential initial care
- Stabilize patient airway and breathing
 - Monitor breathing patterns, swallowing, aspiration, intubate
- Optimization of cardiovascular function:
 - BP management: < 185/110, labetalol or calcium channel blocker

Overall Stroke Care: Goals of Treatment

- Cardiovascular function
 - Dysrhythmias, acute MI, 20% of stroke patients have change in CPK - MB
 - Hypotension and ↓cardiac output
- Elevated ICP
- Pulmonary hygiene
- Seizures, hyperglycemia, nutrition, bowel function, DVT, pressure sores, fever (33%), depression

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Spinal Cord Injury

- Etiology: trauma, disease process, tumor, abscess, hematoma
- · Pathophysiology: mechanism of injury
- Hyperextension-flexion, rotational, compression, penetrating

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Neurologic Infectious Disease

- Viral
- Bacterial
- Fungal

Neuromuscular Disorders



- Guillian Barre: condition in which the immune system attacks the nerves. May be triggered by an acute bacterial or viral infection.
- Symptoms start as weakness and tingling in the feet and legs that spread to the upper body. Paralysis can occur.
- Special blood treatments (plasma exchange and immunoglobulin therapy) can relieve symptoms. Physical therapy is needed.
- Myasthenia: rare chronic autoimmune disease marked by muscular weakness without atrophy, and caused by a defect in the action of acetylcholine at neuromuscular junctions

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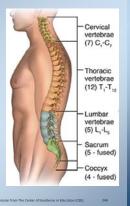
Spinal Cord Injury

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Spinal Cord Injury

- Know spinal shock
- Know Brown-Sequard Injury
- Know Autonomic Dysreflexia



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Spinal Cord Injury Assessment: clinical presentation - Overall assessment: associated injuries - Respiratory function - Motor/sensory function - Physical exam: - Palpation - (spine, skin temperature, sensory and motor) - Deep tendon reflexes

Spinal Cord Injury

Spinal Shock: occurs within minutes; lasts several days to months: T6 or higher

- Results: inhibition of descending tracts
- Loss of all motor, sensation, reflexes
- Bradycardia and hypotension
- Loss of autonomic control
- Flaccid
- Poikilothermy (loss of temp regulation from hypothalamus)

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Spinal Cord Injury

Diagnostics

- Spinal X-rays
- Must clear C1-T1 to rule out cervical spine injury
- -CT scans

Spinal Cord Injury

- Brown-Sequard
 - Knife or bullet hemisection
 - Ipsilateral paralysis
 - Ipsilateral loss of light vibration
 - Contralateral loss of pain sensation, temperature

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Management

- ABC's
- Prevent further damage to spinal cord
- Immediate immobilization
- Prevent further edema
- Maintain airway, ventilation, and oxygenation
- Monitor and treat spinal shock
- Prevent/treat complications

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Autonomic Dysreflexia

- Does not occur until spinal shock is over
- Etiology: massive sympathetic discharge that cannot traverse the spinal cord to communicate with the brain: common noxious stimuli
 - Full bladder, full sigmoid colon, skin pain
- Treatment: eliminate cause, antihypertensives

Status Epilepticus

- Definition: sudden episode of exaggerated activity
- Etiology: withdrawal, toxic levels of drugs, CNS infection, stroke, brain tumors, cerebral edema, metabolic disorders

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Treatment

- Airway and ventilation
- Assess causes or contributing factors
- Protect patient from injury
- Stop seizure activity
- Monitor and prevent complications
- Monitor and document duration of seizure activity
- Fluid and electrolytes

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Seizures

- Prevent seizure activity
- Administer anti-seizure medications as ordered
- Obtain and monitor drug levels
- Avoid exposing the patient to precipitating events
- Monitor lab values carefully

Behavioral Antisocial behavior Agitation (PAD; pain, agitation, delirium) Scales to assess agitation: Richmond Agitation Sedation Scale (RASS) Sedation Agitation Scale (SAS) Dementia

Delirium

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Delirium

What is delirium?

-Acute onset of mental status changes

And

-Inattention

And/or

-Disorganized thinking/altered LOC

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Delirium

- Imbalance of neurotransmitters
- Who is at risk? Anyone
- Facts:
 - -66-90% of ICU patients,
 - onset ICU day 2,
 - how long--4 days,
 - 10% remain delirious at the time of discharge
- Overall 7 out of 10 patients will have delirium

Delirium

- Associated with:
- Increased length of stay
- Increased time of ventilator
- Higher costs
- Increased mortality
- 3-fold increase risk of death at 6 months

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Treatment

- Identify the etiology: assessments
- Modify risk factors
- Haldol 2-10 mg IV every 20-30 minutes, then 25% of loading dose every 6 hours
- Effects on heart: prolonged QT interval

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Treatment

- Development of a protocol
- Assessment and rapid treatment
- Look at current medications
- Do not over sedate
- Aspiration precautions

Dementia

- Loss of mental functions: such as thinking, memory, and/or reasoning
- Not a disease--group of symptoms
 - Substance abuse, severe depression, medications, stroke, vitamin B₁₂ deficiency, AIDS-associated dementia
- Alzheimer's most common cause of dementia

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Depression: A Complex Matter

- Depressive disorder: a syndrome that reflects a sad and/or irritable mood
- Negative thoughts, moods, and behaviors

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Treatment of Depression

- SSRI's: medications that increase amount of neurochemical serotonin in brain
- Fewer side effects than tricyclic antidepressant and MAOI's
- First line drug of treatment
- Paxil, Zoloft, Celexa, Luvox and Lexapro

Treatme	ent of	Depre	ssion
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- Dual-acting antidepressants
- More severe depression
- Act on both the serotonin and norepinephrine systems
- Effexor, Cymbalta

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Substance Abuse

- ETOH
- Drugs: Opioids
- Withdrawal: stages of withdrawal
- Benzodiazepines

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Suicide

- Are you thinking of killing yourself?
- Do you have a plan?
- Do you have a gun?
- When are you going to do this?
- Needs help....

Failure to Thrive (PCCN Only) • Think • Nutrition • Endocrine: AI, Thyroid dysfunction

CCRN

- Post traumatic stress disorder
- Medical non-adhere
- Agitation
- Risk taking behavior

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Other CCRN

- Post traumatic stress disorder
- Medical non-adhere
- Agitation
- Risk taking behavior
- Guillian Barre: A condition in which the immune system attacks the nerves.
- The condition may be triggered by an acute bacterial or viral infection.
- Symptoms start as weakness and tingling in the feet and legs that spread to the upper body. Paralysis can occur.
- Special blood treatments (plasma exchange and immunoglobulin therapy) can relieve symptoms. Physical therapy is needed.

PTSD Prevention Support systems Ability to discuss the event(s) Do Not Self Medicate Hospital post ICU conferences, round table discussions, support group

Neuro Pearls

Multisystem effects of ICP

- 1. Airway Issues--pulmonary compromise
- 2. ECG abnormalities--hemodynamic
- 3. GI bleeding
- 4. Effects of bed rest

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Neuro Pearls

- Temperature=hypothalamus
- No hypotonic solutions in patient with ↑ICP
- Amicar prevents a re-bleed, acts as an antifibrinolytic agent

Question



The patient suddenly becomes unresponsive as you are speaking to him, and he develops trembling of all extremities. Your priority is:

- A. Notify MD
- B. Administer diazepam IV
- C. Establish an airway
- D. Perform a rapid neuro check

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Question



The most common cause of subarachnoid hemorrhage is:

- A. Aneurysms
- B. Coagulopathies
- C. Trauma from falls
- D. Ischemia

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Question



In a patient with increased intracranial pressure, cerebral perfusion pressure should be maintained at:

- A. 40 mmHg
- B. 50 mmHg
- C. 60 mmHg
- D. 70 mmHg

Question



The single most important index of the neurologic state is the:

- A. Level of consciousness
- B. Pupillary reaction
- C. Extremity movement
- D. Vital signs

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Question



A patient is admitted to the ICU after sustaining a knife wound to the back. Assessment findings include loss of pain and temperature on the right side and loss of motor function on the left. Vital signs are stable and he is alert and oriented. No other injuries are noted. Based on the preceding information, which type of neurologic syndrome is likely to be developing?

- A. Central cord
- B. Brown-Sequard
- C. Anterior cord
- D. Horner

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Question



Which of the following is a necessary immediate assessment for an injury of C3-C4?

- A. Motor Ability
- B. Heart Rate
- C. Temperature
- D. Ventilation

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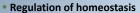


Which vital sign changes (due to loss of sympathetic nervous stimulation) would occur after a spinal cord lesion about T5?

- A. Bradycardia and hypotension
- B. Bradycardia and hypertension
- C. Tachycardia and hypotension
- D. Hypertension and bradycardia

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Renal



- -Extracellular volume and osmolality
- -Electrolytes
- -Excretion of metabolic wastes
- Regulation of acid-base balance
- Production and release of hormones
 - -Aldosterone and ADH
 - -Erythropoietin
 - -Bone mineralization

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Renal Blood Flow

- Kidneys receive 20-25% of cardiac output
- Autoregulation: maintains constant in GFR
- MAP 80-180 mmHg prevents changes in GFR
 Afferent arteriole's ability to dilate or constrict
- Filtration ceases if MAP 40 to 60 mmHg

Renal Assessment

- Weight and fluid changes
- Serum osmolality: 275-295mOm/liter
- BUN:creatinine ratio: 10:1
 - If BUN is elevated disproportionate to creatinine
 - Dehydration (prerenal)
 - Catabolism
 - Blood in gut

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Pathophysiology

Hypovolemia:

- Tachycardia, orthostatic hypotension, ↓CVP, PCWP, CO/CI, ↑ SVR, flat jugular veins, weakness, lethargy, anorexia, poor skin turgor, thirst, lowgrade fever, syncope, oliguria, ↑ BUN with normal creatinine, ↑ H+H, ↑ serum osmolality

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Management

- Monitor intake + output, weight
- Replace fluid: with similar fluids
- Provide frequent oral and skin care

Pathophysiology

Hypervolemia:

- Excessive fluid intake
- Retention of Na⁺ and water
 - Steroid therapy, heart failure, liver failure, stress response, nephrotic syndrome, acute or chronic renal failure
- Clinical presentation: tachycardia, ↑BP, ↑CVP, ↑PWCP, weight gain, JVD, tachypnea, dyspnea, lethargy, apathy, disorientation, indications of pulmonary or cerebral edema, ↓Hct, ↓BUN

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Treatment

- Monitor I+O, daily weight, labs
- Decrease excess volume
 - Restrict fluid intake and Na⁺ intake
 - Administer diuretics
 - Hemodialysis
- Prevent complications: skin and mouth care

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Acute Renal Failure: ARF

- Definition: any sudden severe impairment or cessation of kidney function: characterized by accumulation of nitrogenous wastes and fluid and electrolyte imbalances
- Prerenal: disrupted blood flow to the kidney
- Low intravascular volume, ↓CO, vasodilation, renovascular disease: most common in floor patient

Acute Renal Failure

Cortical: intra-renal damage to renal tissue

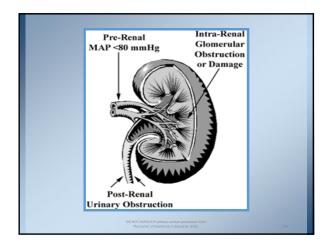
 Glomerulonephritis, vasculitis, interstitial nephritis (renal capillary swelling)



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Acute Renal Failure

- Medullary: Acute Tubular Necrosis
 - Nephrotoxic drugs, prolonged ischemic injury, any causes of prerenal failure that is prolonged: prolonged ischemia destroys tubular basement membrane: most common in ICU patient
- Postrenal: disrupted urine flow
 - Mechanical obstruction, functional obstruction: neurogenic bladder, diabetic neuropathy



Stages of Acute Renal Failure

- **Onset:** period of time from the precipitating event to beginning of oliguria or anuria
- Duration: hours to days
- BUN/Creatinine: normal or slightly decreased
- Mortality: 5%

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Stages of Acute Renal Failure

- Oliguric-Anuric: when urine output is less than 400 mL in 24 hours
- Duration: 1-2 weeks
- BUN/creatinine: increases
- Mortality: 50-60%
- Other: metabolic acidosis, water gain with dilutional hyponatremia, hyperkalemia, hyperphosphatemia, hypocalcemia, hypermagnesemia, azotemia

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Stages of Acute Renal Failure

- Diuretic: urine output is > 400 mL/24h until lab values stabilize
- Duration: 1-2 weeks
- Urine output: may > 3L/24h
- Mortality: 25%
- Other: metabolic acidosis, Na⁺ may be normal or low, high K⁺ continues

Stages of Acute Renal Failure

- Recovery: period of time between when the lab values stabilize until they are normal
- Duration: 3-12 months
- BUN/creatinine: back to 100% normal
- Mortality: 10-15%
- Other: uremia, acid-base imbalances, and electrolyte imbalances gradually resolve

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Treatment

- Support renal perfusion and improve GFR
 - Volume
 - Inotropes (dopamine), vasopressors
 - Administer diuretic challenge
- Maintain fluid, electrolyte, and acid-base balanced
- Na+, K+, phosphorus, magnesium
- Diminish accumulation of nitrogenous wastes
 - Protein restriction, dialysis

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Treatment

- Prevent further damage to kidney
 - Eliminate nephrotoxic agents
 - Monitor peak and trough levels of drugs
 - Nutrition
 - Prevent infection
 - Monitor and treat anemia (Epogen)

Management: Complications

Renal failure increases mortality overall

- Renal: chronic renal failure in 25-30% of acute renal failure
- Cardiovascular: dysrhythmias, hypertension, pericarditis, pulmonary edema, heart failure
- · Neurologic: coma, seizures
- Metabolic: electrolyte imbalance
- GI: peptic ulcer disease, hemorrhage, anorexia, nausea & vomiting, abdominal distention, pancreatitis, ileus

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Management: Complications

- Infection: pneumonia, immunosuppressed
- Pulmonary: pulmonary edema, hyperventilation, acid-base imbalance
- Nutrition

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Renal Replacement Therapies

- Dialysis
- Semipermeable membrane; blood/dialysate
- Principles:
- Types:
 - Peritoneal
 - Hemodialysis
 - Continuous renal replacement therapy

Acute Rei	nal Fail	ure	
	Pre-renal	Acute tubular necre	<u>osis</u>
Urine Na ⁺	< 20	> 40-100	
BUN: Creatinine	<u>></u> 20:1	10:1	
Lasix or fluids	+ urine	No urine	
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Chronic Renal Failure (PCCN Only)

- CRF is a slowly progressive disease that causes gradual loss of kidney function. It can range from mild dysfunction to severe renal failure
- Over number of years
- Asymptomatic
- Progression may be so slow: symptoms occur when renal failure is 1/10th of normal

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Incidence (PCCN Only)

- 2 out of 1000 people in US
- Diabetes and hypertension are the two most common causes and account for most cases
- Other Causes
 - Heart failure, hypotension, glomerulonephritis, kidney stones, obstructive uropathy, polycystic disease

Chronic Renal Failure (PCCN Only)

- Categorized as diminished renal reserve, renal insufficiency, or renal failure.
- Decreased renal function interferes with the kidney's ability to maintain fluid and electrolyte homeostasis. Changes precede predictability



Chronic Renal Failure

- First: ability to concentrate urine declines early
- Followed by decreases in ability to excrete phosphate, acid and potassium
- Renal failure advanced: (GFR < 10mL/min/1.73m²)
- ullet Ability to dilute urine is lost so volume ullet

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Chronic Renal Failure

- As renal failure progresses;
 - Abnormalities of Ca⁺, phosphate, parathyroid hormone, vitamin D metabolism, renal osteodystrophy occur
 - Decreased renal excretion of Calcitriol leads to hypocalcemia
 - Secondary hyperparathyroidism is common
 - Monitoring parathyroid hormone is recommended

Symptoms

- Fatigue: anemia
- Frequent hiccups
- General ill feeling
- Generalized itching (pruritus)
- Headache
- Nausea and vomiting
- Unintentional weight loss

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Late Symptoms

- Hyper vomiting, uremic frost
- Confusion, change in behavior and level of consciousness
- Decreased sensation in the hands, feet
- Easy bruising or bleeding
- Increased or decreased urine output
- Muscle twitching or cramps
- Seizures

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Diagnosis

- Urinalysis
- Creatinine
- Creatinine clearance
- Potassium electrolyte disturbances
- Metabolic acidosis
- CT scan, abdominal MRI, ultrasound
- Renal biopsy

Classification

- Stage 1: normal GFR (>90mL/min/1.73m²)
 - Plus persistent albuminuria
- Stage 2: GFR 60 to 89
- Stage 3: GFR 30 to 59
- Stage 4: GFR 15 to 39
- Stage 5: GFR < 15

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End Stage Renal Disease

- 90% nephrons damaged
- Renal function has deteriorated so that chronic and persistent abnormalities exist
- Patient requires artificial support to sustain life
- Uremic syndrome

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Treatment

- Goal: control symptoms, reduce complications, and slow the progression of the disease (treat underlying problem)
- Fluid restriction, diet control, BP monitoring and control, diabetes control, vitamin D supplements, electrolyte control
- Doses of all drugs adjusted
- Dialysis ??

More Nutrition

- Vitamin D supplements
 - Calcitriol: as indicated by levels
 - Stage of renal failure = and phosphate
 - Target Ca⁺ = 8.4 to 9.5
- Starting dose 0.25 μg by mouth daily
- Statin if cholesterol is elevated

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Renal Pearls

- Signs and symptoms of hypophosphatemia=reciprocal hypercalcemia, weakness, apathy and confusion, TPN, ETOH
- Seizures are seen with hyperphosphatemia
- Creatinine best indicator of renal function:
 - Inversely proportional to GFR
- Low sodium causes aldosterone release

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Question



Mr. J., age 24, boxes on the weekends. He has sustained blunt trauma to the left kidney during a boxing match. Which of the following indicates renal trauma?

- A. Severe flank pain and diaphoresis
- B. Hematuria and flank tenderness
- C. Urethral bleeding
- D. Side pain and hemoptysis

Question



A patient with chronic renal failure asks the nurse why he is anemic. The nurse explains that anemia accompanies chronic renal failure due to:

- A. Blood loss via the urine
- B. Renal insensitivity to vitamin A
- C. Inadequate production of Erythropoietin
- D. Inadequate retention of serum iron

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Question



The primary etiology of hyperphosphatemia is:

- A. Over-replacement
- B. Hypercalcemia
- C. Renal failure
- D. Hypoalbuminemia

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Question

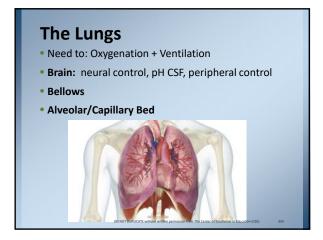


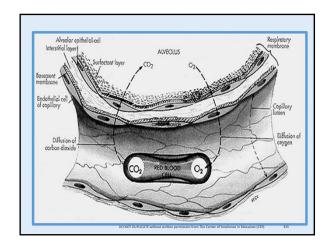
Bradycardia, tremors and twitching muscles are associated with which electrolyte disorder?

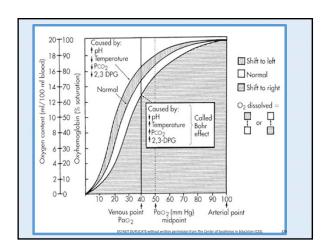
- A. Hypokalemia
- B. Hyperkalemia
- C. Hypophosphatemia
- D. Hyperphosphatemia

Question	
Hyponatremia is usually associated with:	
A. Fluid overload	
B. Dehydration	
C. Diuresis	
D. Over-administration of normal saline	
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Renal Pearls
• Know the electrolytes!!!
• Know the electrolytes for all systems!!!
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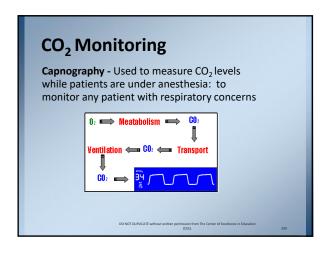




Pulmonary Assessment: Respiratory rate and rhythm Oxygen saturations Breath sounds ETCO₂

Assessment: Pulmonary Exam ABG Chest x-ray

Arterial Blood Gas pH 7.55 7.21 7.30 CO₂ 28 28 38 Bicarb 24 14 18 O₂ 88 97 68



Pulmonary Hypertension

- Primary: rare lung disorder, pressure in the lung circulation is high for no apparent reason.
- Mean PAP greater than 25 mmHg at rest and 30 mmHg during exercise
- Causes: Raynaud's, appetite suppressants, cocaine and HIV

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Primary Pulmonary Arterial Hypertension

- Symptoms
 - Fatigue or tiredness, dizziness, swelling of ankles, advanced to severe pulmonary failure
- Treatment: cath, response to oxygen
 - Calcium channel blockers
 - IV prostacyclin, endothelin receptor antagonists: Bosenten or Tracleer
 - Transplantation

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Secondary Pulmonary Arterial Hypertension

- Other reasons for pulmonary pressure increases
- Pulmonary emboli, heart failure, obstructive sleep apnea, any condition that causes hypoxemia, lung disease, valve disease
- Treatment: underlying disease

Lung Abnormalities				
<u>RESTRICTIVE</u>		<u>OBSTRUCTIVE</u>		
Atelectasis		Asthma		
Pneumonia		Chronic		
 Pneumothorax 		bronchitis		
 Pulmonary edema 		Emphysema		
 Pulmonary fibrosis 				
• ARDS				
Obesity				
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Acute Respiratory Failure

- Pulmonary system is no longer able to meet the metabolic demands of the body.
 - Hypoxemic: $PaO_2 < 50 \text{ torr}$ - Hypercapnic: $PaCO_2 > 50 \text{ torr}$

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Hypoxemia

- V/Q mismatch primary cause
- Shunt Effect
 - Blood is not oxygenated as it travels through the lungs
 - Treatment: removing the obstruction, reopening (recruiting) atelectatic zones, preventing closure (derecruitment) of affected lung units

Assessment

Clinical indications of hypoxemia/hypoxia

- Tachycardia = dysrhythmias
- Tachypnea/Dyspnea
- Accessory muscle use
- Cyanosis
- Restlessness-confusion-lethargy-coma

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Acute Respiratory Failure

HYPERCAPNIA

- Abnormality of alveolar minute ventilation
- Tidal volume (VT)
- Dead space (DS)
- Frequency (f)

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Acute Respiratory Failure

Hypercapnic respiratory failure, patient has:

- Central (depressed respiratory drive)
- Neuromuscular
- Abnormalities of the chest wall (restrictive)
- Abnormalities of gas flow in airways (obstructive)
- Increased dead space (air sees no blood)
- Increased CO₂ production

Assessment

Clinical indications of hypercapnia

- Tachycardia = dysrhythmias
- Bradypnea
- Irritability, confusion
- Inability to concentrate somnolence -coma
- Hypotension
- Facial rubor, headache

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Assessment of Acute Respiratory Failure

- Altered mental status: agitation-somnolence
- Increased work of breathing: nasal flaring, tachypnea, dys-synchronous breathing
- Cyanosis
- Diaphoresis, tachycardia, hypertension, signs of stress-catecholamine release

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Management

- Oxygen supplementation
- Tracheal intubation and mechanical ventilation
- Pharmacologic Adjuncts
 - Beta₂ agonists, anticholinergic agents
 - Corticosteroids, antibiotics





Review O ₂	
Airways	
1. Cannula <40%	
2. Simple mask 40-60%	
3. Partial re-breather mask 60-80%	
4. Nonrebreather mask 80-100%	
5. Noninvasive positive-pressure ventilation	
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Mechanical Ventilation (MV)	
• Indications	
Acute ventilatory failure with acidosis	
– Hypoxemia despite adequate O ₂ therapy	
-CO ₂ retention	
– Apnea	
• Parameters: VC< 10ml/kg, NIF < -20cmH ₂ O	
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Machanias Newtiletie	
Mechanical Ventilation	
Prove beneficial	
Decrease systemic or MVO₂	
– Permit sedation	
– Reduce intracranial pressure	
– Prevent atelectasis	
– Secure airway	

Mechanical Ventilation

Types of ventilators

- Positive pressure
 - Inspiration created by positive pressure
 - Expiration passive
 - Classifications: pressure-cycled, volume cycled

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Goal

Most important goals of mechanical ventilation are:

- Reduction in work of breathing
- Assurance of patient comfort
- Synchrony with ventilator
- Adequacy of ventilation and oxygenation
- Airway protection

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Strategies of mechanical ventilation: lung-protective ventilation

Lung-protective ventilation

- Protect the lung: low VT
- Recruit the lung: PEEP
- Perfuse the lung: right ventricle is filled and contracting

Improve Oxygenation

PEEP: Physiologic 3 - 5 cm

- Actions: improves the PaO_2 without increasing FiO_2 , ψ surface tension, ψ intrapulmonary shunt
- Uses: ARDS, acute respiratory failure
- <u>Adverse effects</u>: hemodynamic changes, barotrauma, ↑intracranial pressure
- <u>Contraindications</u>: untreated hypovolemia, hypovolemic-neurogenic-anaphylactic or septic shock

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Removal CO₂

- Alveolar Ventilation
- Frequency
- Tidal volume
- Dead space

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Guidelines

- 1. Ventilator mode: Goals
- 2. Initial FiO₂= 100%, then wean to keep sats > preset %
- 3. Initial VT 5-10ml/kg: acute respiratory failure may require more to satisfy air hunger, <u>ARDS</u> less
- 4. Respiratory rate = target pH and PaCO₂
- 5. Add PEEP: diffuse lung injury and reduce FiO₂

Assessment of Patient with Mechanical Ventilation

Pulmonary

- Airway: type, size, position, cuff pressure
- Chest excursion, breath sounds bilaterally
- Ventilatory mechanics: VC, VT, RR
- Ventilator parameters: mode, VT, RR, FiO₂, PEEP, PIP, alarms
- Pulse Oximetry, ABG's, chest x-ray

Assessment of Patient With Mechanical Ventilation

- Cardiovascular
 - Heart rate, rhythm, heart sounds, blood pressure, hemodynamic parameters
- Neurologic, renal, metabolic, GI (bowel sounds, abdominal distention), nutritional, immunologic, psychological

Complications

- ↓Cardiac output
- Fluid retention
- Baro-Biotrauma
- Atelectasis
- Hypercapnia-hypo
- Oxygen toxicity
- Aspiration
- GI effects
- Infection
- Asynchrony
- Anxiety
- Inability to wean

Weaning

- Gradual withdrawal of ventilatory support
- Indications: NIF, VC, VT, RSBI
 - Resolution or improvement
 - Patient's strength, nutritional, neurologic status
 - Hemodynamics stable
 - $-PEEP \le 5$ cm, $FiO_2 < 50\%$
 - -RR < 25

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Post-extubation

COMPLICATIONS

- Hoarseness
- Difficulty in swallowing and risk of aspiration
- Severe glottic edema leading to post-extubation stridor and obstruction
- Failure to wean: What do you do?

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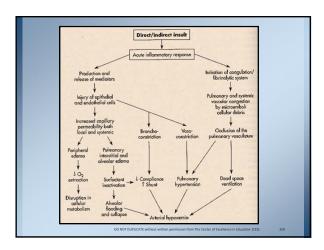
Summary Treatment: Acute Respiratory Failure

- Treat cause
- Maintain airway, oxygenation + ventilation
 - $\\ Positioning$
 - Hydration
 - Bronchial hygiene
 - Bronchoscopy
 - Intubation

Acute Respiratory Distress Syndrome

 Definition: syndrome of acute respiratory failure characterized by noncardiac pulmonary edema and manifested by refractory hypoxemia caused by intrapulmonary shunt

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Clinical Presentation

- Severe oxygenation defect
- Chest x-ray: diffuse bilateral infiltrates: ground glass appearance, white out
- Static compliance: stiff lung
- PCWP: < 18 mmHg
- ↑PAP
- ABG's: refractory hypoxemia
- Lung volumes are ↓VT, FVC

Treatment: Restore Oxygenation

- Improve delivery and reduce consumption
- With mechanical ventilation:
 - -Low VT
 - High PEEP
- Decrease intra-alveolar fluid:
 - CPAP/PEEP, diuretics
 - Avoid overhydration

Treatment

- Hemodynamic monitoring
- Inotropes as indicated by cardiac index
- Decrease oxygen consumption and increase supply
- Decrease pulmonary hypertension
- General support

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Pneumonia

- **Definition**: acute infection of the lung parenchyma, including alveolar spaces and interstitial tissue
- Etiology
- Predisposing factors

Pneumonia Diagnostic - Fever	
– Fever	
-WBC's	
-Sputum	
– Chest X-ray	
 Increased respiratory rate 	

Treatment PREVENTION Maintain airway and ventilation Positioning Organism-specific antibiotics Hydration Bronchial hygiene Bronchoscopy Intubation

Complications
Monitor
 Acute respiratory failure
– Pleural effusion
– Empyema
- Lung abscess
– Septic shock
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Aspiration Pneumonia

- Definition: lung injury related to the inhalation of stomach contents, saliva, food, or other foreign material into the tracheobronchial tree
- Pathophysiology: oropharyngeal secretions are most common

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Risk Factors

Altered consciousness and/or gag reflex

- Anesthesia, CNS disorder, altered anatomy,
 GI conditions (hiatal hernia, vomiting).
 Prolonged intubation, aortic surgery
- Enteral nutritional support
- NG tube
- Position
- Residual content

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Status Asthmaticus

- Definition: a recurrent, reversible airway disease characterized by increased airway responsiveness to a variety of stimuli that produce airway narrowing
- Status asthmaticus: exacerbation of acute asthma not relieved after 24 hours of maximal therapy

Management

- ABC's
- Maintain airway and ventilation
- · Bronchodilators-short acting
- Anticholinergics
- Mechanical ventilation
- STEROIDS

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Pulmonary Embolism

- Definition: obstruction of blood flow to one or more arteries of the lung by a thrombus lodged in a pulmonary vessel: fat, air, amniotic fluid, tumor, foreign body
- Etiology: hypercoagulability, alteration in vessel wall, venous stasis
 - Fat emboli: osteomyelitis, sickle cell anemia, multiple long bone fractures, burns

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Clinical Presentation

Most common symptoms

DyspneaPleuritic painCough73%66%37%

Most common signs

Tachycardia 70%Crackles 51%

Hemodynamics • ↑CVP • ↑ PAP with normal PCWP • ↑PVR • ↓CO/CI in massive PE • Hypoxemia

ECG

- Dysrhythmias: tachycardia, atrial fibrillation
- Tall, peaked P-waves (P-pulmonale)
- New right bundle branch block
- Right axis deviation
- Right ventricle strain pattern
- McGinn White: S1 Q3 T3

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Diagnostics

- ABG's
- Chest X-ray
- ECG
- Echo
- V/Q scan
- CT scan with PE protocol
- Pulmonary angiography

Treatment

- PREVENTION
- Maintain airway and ventilation
- Arrest thrombus: baseline clotting profile, fibrinolytic therapy
- Heparin therapy
- Oral anticoagulants: Coumadin 3-6 months
- Pulmonary embolectomy
- Surgical interruption of inferior vena cava: filter

Complications

- Pulmonary infarction
- Cerebral infarction
- Myocardial infarction
- Right ventricle failure
- Hepatic congestion
- Pneumonia
- Empyema

- Pulmonary abscess
- Acute respiratory failure
- DIC
- Shock
- Bleeding secondary

to therapy

Pulmonary Fibrosis

- Causes: medications, idiopathic
- Treatment: oxygen, dilators, supportive care



New ABCDEF Bundle

- A = Assess, prevent, and manage pain
- B = Both SAT and SBT
- C = Choice of analgesia and sedation
- D = Delerium: Assess, prevent and manage
- E = Early mobility and exercise
- F = Family engagement and empowerment

Pulmonary Pearls

- Asthma: ominous signs = absence of wheezing, ↑CO₂
- Oxygenate and ventilate

Pulmonary Pearls

Ventilatory Adjuncts

- Aerosol treatments: bronchodilators and mucolytics
- Inhaled nitrous oxide
- Helium
- Prone position
- Rotational beds--vibration and percussion

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Pearls

- IV Magnesium- 2 grams IV
 - Acts as bronchodilator
 - Decrease inflammation
 - Effective with respiratory failure

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Question



The hallmark of acute respiratory distress syndrome is:

- A. Refractory hypercapnia
- B. Refractory hypoxemia
- C. Low functional residual capacity
- D. Increased compliance

Question



The most common ECG changes that occur during pulmonary embolus are:

- A. Q-waves in AVR and Lead I
- B. Tachycardia and atrial fibrillation
- C. Bradycardia and ST-segment depression
- D. High-degree AV blocks

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Question



The principal contributing factors to venous thrombosis include all of the following except:

- A. Atrial fibrillation
- B. Stasis of blood flow
- C. Endothelial injury or vessel wall abnormality
- D. Hypercoagulability

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Question



Which of the following features of pleural drainage systems indicates an active pleural leak?

- A. Bubbling in the water-seal chamber
- B Bubbling in the suction control chamber
- C. Fluctuation of water level in the water-seal chamber with respiration
- D. No fluctuation of water level in the waterseal chamber with respiration

Question



Which type of condition can lead to a tension pneumothorax?

- A. Closed pneumothorax
- B. Open pneumothorax
- C. Subcutaneous emphysema
- D. Pneumomediastinum

Question



Pressure-support ventilation (PSV) differs from synchronized intermittent mandatory ventilation (SIMV) and AMV in which of the following ways?

- A. PSV includes a level of PEEP with each breath
- B. PSV is negative pressure regulated
- C. SIMV and AMV are volume-limited, PSV is pressure limited.
- D. SIMV and AMV do not reduce the work of breathing, whereas PSV reduces the work of breathing and is therefore a better weaning tool.

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Question



You are helping another nurse to move a patient up in bed when the low-pressure alarm on the ventilator goes off.

- It also indicates a low tidal volume.
- \bullet The patient is becoming short of breath and his ${\rm SpO}_2$ has dropped from 0.95 to 0.84.
- The PETCO₂ waveform is absent.
- The endotracheal tube appears to be in place and there is no obvious disconnection from the ventilator.
- The other nurse goes to call the respiratory therapist.

What should you do?

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- A. Increase the VT on the ventilator while instructing the patient to remain calm
- B. Increase the FiO₂ on the ventilator while instructing the patient to remain calm
- C. Remove the ventilator and begin manual respiration (ambu)
- D. Increase the ventilator respiratory rate and peak flow

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Question



The major signs and symptoms of acute respiratory failure include:

- failure include:

 A. Increased respiratory rate, tachycardia, change in mental status
- B. No change in respiratory rate, tachycardia
- C. The major sign is the complaint of shortness of breath
- D. There are no early signs of respiratory failure

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Multisystem Organ Failure (MSOF)

- SHOCK
- **Definition:** condition of insufficient perfusion of cells and vital organs, causing tissue hypoxia, perfusion is inadequate to sustain life: results in cellular, metabolic, and hemodynamic derangements
- Malperfusion

Classification

- <u>Hypovolemic</u>: caused by inadequate intravascular volume: external losses, internal losses
- <u>Cardiogenic</u>: caused by impaired ability of heart to pump blood: contractility, filling, emptying
- <u>Distributive or vasogenic</u>: caused by massive vasodilation caused by release of mediators of inflammatory process in response to overwhelming infection: septic, anaphylactic, neurogenic

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Shock

Important concepts to remember:

- Preload
- Contractility
- Afterload
- Heart Rate

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SIRS

- Systemic inflammatory response syndrome (SIRS): the systemic response to a variety of insults that begin as local inflammation (collection of immune-mediated responses to infections, foreign materials, tissue ischemia and reperfusion injuries)
 - The Cascade

Clinical Presentation

- Criteria (2 or more of the following):
 - Tachycardia (>90/min)
 - Hyperpnea (RR >20/min, PaCO₂ <32mmHg)
 - Hyperthermia (temp >38°C) or hypothermia (<36°C)
 - WBC >12,000 or below 4,000

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Definitions

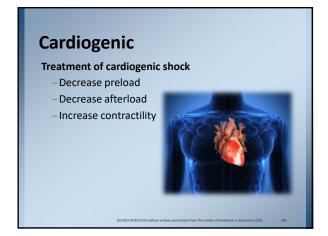
- Fever + leukocytosis = SIRS
- SIRS + infection = sepsis
- Sepsis + MODS = severe sepsis
- Severe sepsis + refractory hypotension= septic shock

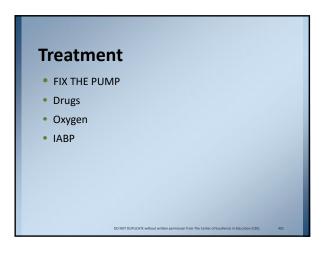
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Specific To Septic

- Avoid NPO status
- Antibiotic therapy: treat infection and neutralize toxin
- Control hyperthermia
- Volume: 30ml/kg body weight
- Support cardiovascular function
- Pharmacotherapy

Management: Must Identify Infection! Maximize O₂ Delivery - Fluids - Vasopressors - Maintain Hct > 21% - Inotropes Minimize O₂ Consumption - Mechanical ventilation - Monitor SVO₂ - Control temperature and electrolytes - Nutritional status - Control pain and anxiety - Prevent complications





Specific To Hypovolemic

- · ABC's
- Volume resuscitation
- Treat the cause: stop source of fluid loss, restore intravascular volume
- Inotropes: after volume restored

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Specific To Anaphylactic

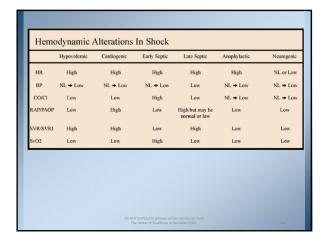
- Remove the offending agent, antigen
- Maintain a patent airway (ABC's)
- Volume resuscitation
- Modify or block the effects of biochemical mediators
 - Administer sympathomimetics
 - Epinephrine, antihistamines, bronchodilators,
 - IV Steroids

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Specific To Neurogenic

- ABC's
- Spinal cord immobilization
- Warming measures
- Maintain MAP, prevent venous stasis
- Volume replacement
- Monitor for complications of shock, or other reason for shock
- Steroids

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Shock Pearls

- Know hypovolemic shock
- Cardiogenic shock

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Question



One on the most effective therapies in the treatment of sepsis is:

- A. Early antibiotic therapy
- B. Early treatment with multiple cardiac inotropes
- C. Early treatment with mechanical ventricular assistance
- D. No treatment has been shown to be successful

Question In the treatment of shock, the team should: A. Promote oxygenation and ventilation B. Enhance oxygen delivery C. Decrease oxygen consumption D. All the above

Infectious Diseases MRSA VRE CRE Influenza: pandemic or epidemic

Secure catheter with leg strap or tube holder Strict hand washing Perform per-care daily and after each BM Sterile technique Always scan bladder prior to catheterization to determine urine volume and necessity

VAP

- HOB elevated 30 degrees
- Oral care every 2 hours
- Turn patient every 2 hours
- Sedation vacation
- PUD prophylaxis
- DVT prophylaxis

Bariatric Considerations

- Abdominal Pain: Post Operative
 - Anastomosis leak: considerable pain, acute abd
 - Gastric bleeding
 - Persistent vomiting and abdominal pain
- Pulmonary Embolis always in the differential
- Long Term Post Operative Complications
 - Nutritional concerns
 - 1/3 of patients develop gall stones
 - 20% of all patients require follow-up surgeries to correct complications

Maternal/Fetal Complications

- Eclampsis: Seizures that occur during a woman's pregnancy or shortly after giving birth.
- Can follow a condition of high BP and excess protein in the urine during pregnancy (preeclampsia).
- Symptoms include upper right abdominal pain, severe headache, and vision and mental status changes.
- Magnesium sulfate is a loading dose of 4 to 6 g given over 15 to 20 minutes, followed by a maintenance dose of 2 g/h as a continuous IV solution to prevent seizures and reduce high blood pressure. The baby may need to be delivered early.
- HELLP syndrome: A serious complication of elevated BPduring pregnancy.
- Hemolysis, elevated liver enzymes, low platelet count (HELLP) syndrome usually develops before the 37th week of pregnancy but can occur shortly after delivery. Many women are diagnosed with preclampsia beforehance.
- . Symptoms include nausea, headache, belly pain, and swelling.
- Treatment usually requires delivery of the baby, even if the baby is premature.

Maternal/Fetal Complications

- Post partum hemorrhage: Causes of postpartum bleeding include loss of tone in the uterine muscles, a bleeding disorder, or the placenta failing to come out completely or tearing.
- Symptoms include vaginal bleeding that doesn't slow or stop. This can lead to a drop in BP
- Treatment often includes uterine massage and medication. In rare cases, blood transfusion, removal of residual placenta, or a hysterectomy may be needed.

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Maternal/Fetal Complications

- Amniotic Fluid Embolism: Amniotic fluid embolism is most likely to occur during delivery or in the immediate postpartum period.
- Sudden SOB
- · Pulmonary edema
- Sudden decrease in BP, tachycardia
- Disseminated intravascular coagulopathy
- Altered mental status, such as anxiety or a sense of doom
- Fetal distress, seizures
- Tx: O₂, PRBC, FFP, cyroprecip for fibrinogen < 100, platelets for < 20,000

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Health-Care Associated Infections (HAI)

- Central Line-associated bloodstream infections (CLABSI)
- Catheter-associated urinary tract infection(CAUTI
- Ventilator-associated event (VAE) (VAP)

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