The Rescue Company 1

Critical Care Study Guide

FP-C Examination Content

The Certified Flight Paramedic Examination consists of 125 Questions and the candidate is provided 2.5 hours to complete the examination. The certification process is focused on the knowledge level of accomplished, experienced paramedics currently associated with a Flight and/or Critical Care Transport Teams. The Questions on the examination are based in sound paramedicine. The candidate is expected to maintain a significant knowledge of current ACLS, PALS, NALS, and PHTLS/BTLS standards. This examination is not meant to test entry-level knowledge, but rather to test the experienced paramedic's skills and knowledge of critical care transport.

As you prepare for the examination, please consider there are a variety of mission profiles throughout the spectrum of transport medicine. Please remember this examination tests the candidates overall knowledge of the transport environment, not the specifics of one individual program. We have included a brief outline below of the topics and skills included in the exam. Though some outline topics are within the paramedic's scope of practice, the exam questions will be related to critical care and are of a much higher level of difficulty.

1. Trauma Management (9)

- Perform patient triage
- Differentiate injury patterns associated with specific mechanisms of injury
- Identify patients who meet trauma center criteria
- Perform a comprehensive assessment of the trauma patient
- Initiate the critical interventions for the management of the trauma patient
- Provide care for the patient with life-threatening thoracic injuries (e.g., pneumothorax, flail chest, tamponade, and myocardial rupture)
- Provide care for the patient with abdominal injuries (e.g., diaphragm, liver and spleen)
- Provide care for the patient with orthopedic injuries (e.g., pelvic, femur, spinal)
- Administer appropriate pharmacology for trauma management

2. Aircraft Fundamentals, Safety and Survival (12)

- Assess the safety of the scene
- Conduct preflight checks to ensure aircraft integrity
- Conduct preflight checks to ensure equipment is present, functional, and stowed
- Observe for hazards during aircraft operation
- Utilize proper safety equipment while in flight
- Maintain a sterile cockpit during critical phases of flight
- Approach and depart the aircraft in a safe manner
- Ensure safety around the aircraft
- Secure the patient for flight
- Participate in crew resource management (CRM)
- Participate in flight mission safety decisions (e.g., Go – No Go, abort)
- Respond to in-flight emergencies
- Fire
- Emergency egress
- Emergent landing
- Adverse weather conditions
- Perform immediate post-accident duties at a crash site
- Build survival shelters
- Initiate emergency survival procedures
- Ensure the safety of all passengers (e.g., specialty teams, family, law enforcement, observer)

- Estimate weather conditions that are below weather minimums

3. Flight Physiology (10)

- Identify causes of hypoxia
- Take corrective measures to prevent altitude related hypoxia
- Identify signs of barometric trauma
- Identify stressors related to transport (e.g., thermal, humidity, noise, vibration, or fatigue related conditions)
- Take corrective action for patient stressors related to transport
- Relate the relevant gas laws to patient condition and treatment
- Relate the stages of hypoxia to patient condition and treatment
- Identify immediate causes of altitude related conditions in patients
- Identify immediate causes of altitude related conditions as they affect the air medical crew
- Provide appropriate interventions to prevent the adverse effects of altitude changes during patient transport

4. Advanced Airway Management Techniques (5)

- Identify the indications for basic and advanced airway management
- Identify the indications and contraindications for specific airway interventions
- Perform advanced airway management techniques
- Administer appropriate pharmacology for airway management
- Implement a failed airway algorithm
- Identify esophageal intubation
- React to intubation complications
- Perform alternative airway management techniques (e.g., needle cricothyrotomy, surgical cricothyrotomy, Seldinger technique, retrograde intubation, LMA)
- Monitor airway management and ventilation during transport
- Use mechanical ventilation

5. Neurological Emergencies (10)

- Conduct differential diagnosis of coma patients
- Manage patients with seizures
- Manage patients with cerebral ischemia
- Initiate the critical interventions for the management of a patient with a neurologic emergency
- Provide care for a patient with a specific neurologic emergency
- Perform a baseline neurologic assessment of a trauma patient
- Perform an ongoing serial evaluation of a neurologic patient
- Assess changes in intracranial pressure using patient level of consciousness
- Perform a focused neurological assessment
- Assess a patient using the Glasgow coma scale
- Manage patients with head injuries
- Manage patients with spinal cord injuries
- Evaluate muscle strength and motor function
- Administer appropriate pharmacology for neurological management

6. Critical Cardiac Patient (20)

- Perform a detailed cardiovascular assessment
- Identify patients experiencing an acute cardiac event (e.g., acute myocardial infarction, heart failure, cardiogenic shock, primary arrhythmias, and hemodynamic instability)
- Use invasive monitoring during transport, as indicated, for the purpose of clinical management

- Provide treatment for patients with acute cardiac events and hemodynamic abnormalities
- Control cardiopulmonary support devices to patient condition as part of patient management (e.g., ventricular assist devices, transvenous pacer, intra-aortic balloon pump)
- Assist in the control of cardiopulmonary support devices to patient condition as part of patient management (e.g., ventricular assist devices, transvenous pacer, and intra-aortic balloon pumps)
- Conduct defibrillation during transport
- Administer appropriate pharmacology for cardiac management

7. Respiratory Patient (10)

- Perform a detailed respiratory assessment
- Identify patients experiencing respiratory compromise
- (e.g., acute respiratory distress syndrome, spontaneous
- pneumothorax, pneumonia)
- Monitor patient's respiratory status using laboratory
- values and diagnostic equipment (e.g., pulse oximetry, capnography, blood gas values, chest radiography)
- Provide treatment for patients with acute respiratory events
- Administer appropriate pharmacology for respiratory management

8. Toxic Exposures (6)

- Conduct a physical examination of a toxicological patient
- Decontaminate toxicological patients when indicated
- Administer poison antidotes when indicated
- Provide emergency care for victims of envenomation (e.g., snake bite, scorpion sting, and spider bite)
- Administer appropriate pharmacology for toxic exposures
- Provide treatment for toxicological patients (e.g., medication overdose, chemical/biological/radiological exposure)

9. Obstetrical Emergencies (4)

- Perform an assessment of the obstetrical patient
- Perform fetal assessment
- React to special transport considerations of the obstetrical patient
- Provide treatment for high-risk obstetrical patients
- Assess uterine contractions
- Conduct interventions for obstetrical emergencies (e.g., pregnancy induced hypertension, hypertonic or titanic contractions, cord prolapsed, placental abruption)
- Assess whether transport can safely be attempted or whether delivery should be accomplished at the referring facility
- Administer appropriate pharmacology for obstetrical patients
- Manage emergent delivery

10. Neonates (4)

- Perform an assessment of the neonatal patient
- Reevaluate the clinical assessment and management of the neonate when initial emergency measures fail
- Administer appropriate pharmacology for neonatal patients
- Implement neonatal resuscitation according to established practice
- Manage the isolette transport
- Provide treatment of neonatal emergencies

11. Pediatric (10)

- Perform an assessment of the pediatric patient
- Identify the pediatric patient experiencing an acute
- respiratory event (e.g., epiglottitis, bronchiolitis, asthma)
- Identify the pediatric patient experiencing an acute
- medical event (e.g., meningitis, overdose, seizures)
- Identify the pediatric patient experiencing an acute
- cardiovascular event (e.g., shock, cardiac anomaly,
- dysrhythmias)
- Identify the pediatric patient experiencing an acute
- traumatic event (e.g., auto v. pedestrian, falls, child abuse)
- Administer appropriate pharmacology for pediatric
- patients
- Provide treatment of pediatric emergencies

12. Burn Patients (5)

- Perform an assessment of the burn patient
- Calculate the percentage of total body surface area
- burned
- Calculate appropriate fluid replacement amounts based on the patient's burn injury and physiologic condition
- Diagnose inhalation injuries in burn injury patients
- Administer appropriate pharmacology for burn patients
- Provide treatment of burn emergencies

13. General Medical Patient (16)

- Perform a focused medical assessment
- Identify patients experiencing a medical emergency (e.g., AAA, GI bleed, bowel obstruction, HHNC)
- Use invasive monitoring during transport, as indicated, for the purpose of clinical management
- Provide treatment for patients with medical emergencies
- Manage patient condition utilizing available laboratory values (e.g., blood glucose, CBC, H/H) Administer appropriate pharmacology for the medical patient
- Prevent transmissions of infectious disease
- Provide appropriate pain management
- Evaluate and record patient pain levels

14. Environmental (4)

- Perform an assessment of the patient suffering from an environmental emergency
- Identify the patient experiencing a cold related emergency (e.g., frostbite, hypothermia, cold water submersion)
- Identify the patient experiencing a heat related emergency (e.g., heat stroke, heat exhaustion, heat cramps)
- Identify the patient experiencing a diving related emergency (e.g., decompression sickness, arterial gas emboli, near drowning)
- Identify the patient experiencing a altitude related emergency (e.g., HAPE, cerebral edema)
- Administer appropriate pharmacology for environmental emergency patients
- Provide treatment of environmental emergencies

CFRN Examination Content

The CFRN exam consists of 180 items of which 150 items are scored. The remaining 30 items are pretest items for use in future versions of the exam. Pretest items are distributed throughout the exam, the pretest items are not identified, and your answers to these items will not affect your test score in any way.

This Exam is based on flight nursing practice in the United States. The applicant can assume that an item on the exam is referring to an adult patient unless otherwise noted.

CFRN Content List – The CFRN exam contains, but is not limited to the following items.

Single System Emergencies

Cardiopulmonary (27 items)

- Obtain 12-lead ECG
- Evaluate Characteristics of chest pain
- Initiate and mange noninvasive transcutaneous cardiac pacemaker
- Interpret 12-lead ECG
- Determine need for and perform cardiopulmonary resuscitation
- Recognize pericardial Tamponade
- Manage transvenous pacemaker
- Initiate fibrinolytic infusion
- Perform cardioversion/defibrillation
- Auscultate heart sounds
- Initiate ACLS protocols
- Identify patients who are candidates for fibrinolytic agents
- Assist with pericardiocentesis
- Assess for neck vein distention
- Assess peripheral pulses and capillary refill
- Initiate PALS protocols
- Manage IABP during transport
- Perform pericardiocentesis

GI/GU and OB Emergencies (11 items)

- Insert and irrigate NG/OG tubes
- Inspect, auscultate, percuss, and palpate the abdomen
- Assess for flank tenderness and ecchymosis
- Perform neonatal resuscitation
- Assess fetal heart tones (Doppler)
- Perform emergency delivery
- Position a patient who has a prolapsed cord during labor
- Position a pregnant patient for uterine displacement
- Determine risk for delivery during transport
- Manage OB hypertensive disorders
- Manage neonates during transport
- Massage uterine fundus postpartum
- Manage vaginal bleeding/drainage
- Palpate for uterine contractions and fetal movement
- Interpret fetal monitoring strips
- Initiate and manage tocolytic therapy

Maxillofacial and Orthopedic Emergencies (8items)

- Assess for upper respiratory distress/difficulty
- Assess for maxillofacial trauma
- Inspect oropharynx
- Manage ocular emergencies
- Reduce orthopedic injuries
- Apply a traction splint
- Preserve amputated body part for reimplantation
- Manage pelvic instability
- Immobilize injured extremities
- Assess neurovascular status of injured or immobilized extremity (e.g. compartment syndrome)

Neurological Emergencies (17 items)

- Perform cranial nerve assessment
- Assess drainage from ears and nose for blood and/or CSF
- Perform or maintain full spinal immobilization
- Assess level of consciousness/mental status
- Assess seizure activity and initiate seizure precautions
- Evaluate reflexes (DTRs and clonus)
- Manage increased intracranial pressure
- Identify patients at risk for increased intracranial pressure
- Assess for nuchal rigidity
- Manage ventilation for a patient with suspected head injury
- Assess sensation and motor function
- Monitor intracranial pressure monitor devices

Multi-System Emergencies

Environmental Emergencies (9 items)

- Manage fluid resuscitation for burn injury
- Measure/restore core body temperature
- Determine degree and extent of burn injury
- Evaluate decontamination of patient after exposure to hazardous materials
- Manage heat/cold related emergencies
- Plan measures for treating hazardous materials exposure
- Perform escharotomy
- Manage decompression sickness

General/Medical Emergencies (9 items)

- Assess/manage hematological disorders
- Initiate isolation precautions
- Identify and manage symptoms of hypoglycemia or hyperglycemia
- Assess risk of communicable disease transmission/exposure
- Assess patient for signs of envenomation
- Identify and manage fever
- Assess patient's fluid status
- Maintain patients who are potential organ/tissue donors

Patient Management

Patient Care (12 items)

- Interpret laboratory results
- Perform bedside laboratory testing (glucose,Hgb/Hct)
- Manage Pain
- Evaluate a chest x-ray
- Apply/manage patient restraints
- Evaluate cervical spine x-rays
- Perform primary and secondary surveys
- Plan priorities of patient care management
- Evaluate patient response to interventions (medications, procedures)
- Initiate use of vasoactive drugs
- Calculate and titrate intravenous medication infusions
- Determine care based on age/weight parameters
- Monitor arterial line pressure
- Assess pulmonary artery (Swanz Ganz) wave forms
- Monitor central venous pressure readings
- Calculate appropriate fluid resuscitation for the pediatric patient
- Plan care based on cultural considerations
- Plan care based on developmental stages and age appropriate considerations
- Manage patient with delayed consequences of trauma (DIC, SIRES, MODS, ARDS)
- Develop transport diagnosis/impression based on assessment
- Determine death

Advanced Airway Care (9 items)

- Determine treatment plans based on colormetric end-tidal CO2 results
- Manage patients based on continuous capnography (wave form)
- Select appropriate measures to provide an airway (basic and advanced)
- Determine treatment plans based on pulse oximetry values
- Manage mechanical ventilation
- Perform a needle cricothyrotomy
- Perform oral intubation
- Choose device for oxygen delivery (nasal cannula, mechanical ventilator, mask)
- Perform nasal intubation
- Perform rapid sequence induction intubation
- Manage patients with surgical airway (Cricothyrotomy and tracheostomy)
- Suction Airway
- Insert nasal and/or oral airway
- Ventilate patient using a bag-valve-mask device
- Maintain neuromuscular blockade with sedation
- Facilitate airway management using alternative airway devices (LMAs, Combitube, bougie)
- Manage BiPAP
- Perform a surgical cricothyrotomy

Respiratory (7 items)

- Evaluate the patient's response to oxygen therapy
- Interpret results of arterial blood gas studies
- Maintain chest tube and drainage systems
- Assess for abdominal breathing patterns
- Assess for inhalation injury
- Perform needle Thoracotomy
- Administer nebulizer treatment
- Assess for pulsus paradoxus
- Inspect, palpate, percuss, and Auscultate chest
- Manage chest wall injury
- Develop plan of care based on mechanism of injury
- Perform tube Thoracotomy

Shock/Trauma (7 items)

- Assess and manage shock
- Administer blood products
- Monitor urinary output
- Perform rapid volume resuscitation
- Initiate ATLS/TNATC protocols
- Perform Trauma Score assessment
- Control visible hemorrhage
- Apply dressings to wounds
- Assess for soft tissue injury
- Manage patient based on type of shock
- Place a central intravenous line

Substance Abuse/Toxicological (3 items)

- Determine need for medication antagonist in an overdose situation
- Perform gastric emptying for toxic substance ingestion
- Plan measures for treating complications of toxic ingestion/inhalation/exposure
- Consult information resources about management of poisoning
- Recognize patients with drug overdose
- Identify substance abuse patients at risk for acute withdrawal symptoms

Transport Considerations (6 items)

- Assess and manage violent behavior
- Manage patient transport stressors
- Determine necessity for diversion
- Determine appropriate mode of transport (rotor, ground, fixed wing)
- Determine emergent versus non-emergent transport status (lights and sirens, code 3)
- Plan for treatment of effects due to illness

Safety Issues

Safety (9 items)

- Assist in safe transport vehicle operation
- Perform vehicle decontamination and disposal of biohazardous waste
- Secure patient in the transport vehicle
- Secure equipment in the transport vehicle
- Manage crew transport stressors
- Communicate with EMS personnel
- Manage radio communication operations
- Report patient's condition to receiving staff
- Teach pre-hospital care workers to prepare a landing zone for helicopter
- Ensure security of aircraft at landing areas
- Assist with extrication
- Assist with flight following
- Assist with navigation

Disaster Management (3 items)

- Triage patient care during a disaster
- Implement steps of disaster plan
- Participate in a plan for mass casualties
- Participate in a plan for effects of Weapons of mass destruction

Survival (4 items)

- Train for transport vehicle emergencies
- Ensure safety of team and patient(s) in post-crash situation
- Participate in emergency operations plan
- Operate emergency locator transmitter (ELT)

Professional Issues

Legal (3 items)

- Notify authorities of reportable situations
- Evaluate patient's capacity to refuse treatment
- Ensure preservation evidence for legal/forensic situations
- Assess for signs of abuse
- Recognize legal requirements for transport (EMTALA, HIPPA)
- Provide documentation of transport care

Organizational Issues (3 items)

- Participate in conflict resolution
- Participate in critical incident stress management
- Participate in quality assurance/improvement activities
- Evaluate applicability of published evidenced-based research to practice

Patient/Community Education (3 items)

- Inform patients/families regarding procedures/patient condition
- Provide public education
- Provide transport safety briefing to patient/family member prior to transport
- Provide support to family after patient death
- Participate in continuing education/in-service
- Orient new staff to work responsibilities

FLIGHT PHYSIOLOGY

Boyle's Law -The volume of a gas is inversely proportional to the pressure of a gas at a constant temperature.

(P1)(V1)= (P2)(V2)

Ascent=Expansion of gas

Descent=Contraction of gas

Effects on Patients

-Barotitis media

-Barodontalgia

-Barosinusitis

-Barobariatrauma

-GI Complications

Effects on Equipment

-ETT cuffs

-MAST pants

-IV drip rates

Charles' Law - At a constant pressure, the volume of gas is directly proportional to the absolute temperature of the gas. It is directly proportional relationship between temperature and volume.

V1/T1=V2/T2

Climb 100m = 1degree Celsius drop

Effects on Patients/Equipment with Charles' Law:

-Gas volume expands as temperature increases.

-Gas volume shrinks as temperature decreases.

Gay-Lussac's Law - Directly proportional relationship between temperature and pressure

P1/T1=P2/T2

Similar to Charles' Law

Henry's Law - Deals with the solubility of gases in liquid. The quantity of gas dissolved in 1cc of a liquid is proportional to the partial pressure of the gas in contact with the liquid.

Graham's Law - The diffusion rate of a gas through a liquid medium is directly related to the solubility of the gas & inversely proportional to the square root of its density.

Means gases diffuse from a higher concentration to an area of lower concentration.

Dalton's Law - The total pressure of a gas mixture is the sum of the partial pressures of all the gases in the mixture.

Pt=P1+P2+P3+P4+…

Stressors of Flight

1. Decreased partial pressure of oxygen
2. Barometric pressure
3. Thermal changes
4. Decreased humidity
5. Noise
6. Vibration
7. Fatigue
8. G-forces

DEATH-factors effecting flight stressors

D – Drugs

E – Exhaustion

A - Alcohol

T – Tobacco

H – Hypoglycemia

The Principle of Atmospheric pressure

At sea level, the weight of a one square inch column of air extending to the edge of space is called "one atmosphere". This refers to "atmospheric pressure". (ATM) 1 ATM weighs 14.7 lbs (760 mmHg [torr])

As you ascend the pressure becomes less (0.5 ATM or 380 mmHg at 18,000ft)

As you dive in water you increase the forces (or weight) on your body by 1 ATM for every 33 feet you are submerged, hence the term diving "1 atmosphere".

Altitude Zones

Physiologic zone – sea level to 10,000 ft

Physiologically deficient zone – 10,000 to 50,000 feet

Space-equivalent zone – 50,000 to 250,000 feet

Space – 250,000 feet

Hypoxic Hypoxia-deficiency in alveolar O2 exchange- AKA altitude Hypoxia

Hypemic Hypoxia-reduction in the O2 carrying capacity in the blood, hemorrhage, anemia, and drugs (sulfanilamide nitrates)

Histotoxic Hypoxia-a result of poisoning or metabolic disorders; cyanide, ETOH, CO poisoning

Stagnant Hypoxia-reduced cardiac output or pooling of blood; heart failure, PE, Shock

Stages of Hypoxia

Indifferent stage – slightly increased HR and RR, decreased night vision

Compensatory stage – increased BP occurs, impairment of task performance occurs

Disturbance stage- dizziness, sleepiness, tunnel vision, and cyanosis

Critical stage – marked mental confusion and incapacitation

Changes with altitude

PaO2 decreases 5 mmHg per every 1000 ft increase in altitude

Oxygen Adjustment Calculation

$\frac{(\%\ IO2xP1)}{P2}$ = % IO2 for altitude

Effective Performance Time

The amount of time a FCM is able to perform useful flying duties in an inadequately oxygenated environment

Time of Useful Consciousness The elapsed time from exposure to Oxygen deprived environment to the point where deliberate function is lost

ACID BASE BALANCE & OXYGENATION MANANGEMENT

Cellular metabolism

Aerobic Energy

Glycolysis ››› Pyruvic acid (2 ATP)

Pyruvic acid ››› Acetyl Coenzyme A

- Crebs Cycle
- Electron Transport Chain (32-34 ATP)

Anaerobic Energy

Glycolysis ››› Pyruvic acid

Pyruvic Acid ››› Lactic Acid

ATP production

Clean or aerobic metabolism of ATP requires glucose and O2; the bi-products are CO2 and H2O

Lack of glucose or O2 causes an alteration in ATP metabolism; the bi-product is lactic acid

Oxygen Delivery

Cellular aerobic metabolism is dependent on adequate O2 delivery (FIO2)

- ability to carry O2 (hemoglobin)
- ability to move O2 (cardiac output)
- ability to extract O2 to cells

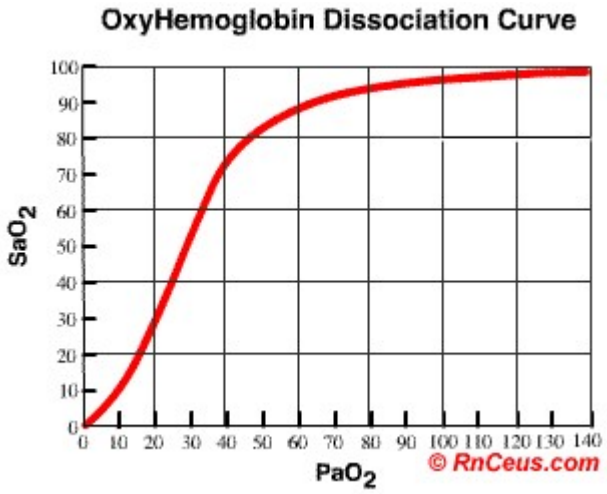
Shock

Shock most simply defined is inadequate tissue oxygenation

Acid Base balance – Primary Buffering Systems

- Carbonic Acid-bicarbonate buffer system operates in the blood, lungs, and kidneys
- Normal relationship between bicarb and carbonic acid is 20:1

- $H^+ + HCO3 \leftrightarrow H_2CO_3 \leftrightarrow CO_2 + H_2O$



The lungs can adjust the amount of carbonic acid by blowing off or holding on to CO2

The Kidneys can reabsorb or excrete bicarb or H^+ PRN; the kidneys also make new bicarb

Does ETCO2 = PCO2?

Consider

- CO2 production and diffusion
- Cardiac Output
- Ability to diffuse CO2 into alveoli
- Minute Volume

Acid Base Balance – Secondary Buffering Systems

- Protein buffering system
- Primarily ICF (phosphates) but ECF also
- Hemoglobin binds with H^+ *and* CO2 bound hgb becomes weak acid
- Bone exchanges calcium, phosphate, and releases carbonate; regulates within hours to days of imbalance

Ionic shifts involve the exchange of intracellular K^+ and NA^+ for H^+

- Regulates within 2-4 hours of imbalance
- *K^+ will change approximately 0.6 mEq for every 0.1 change in pH*

Normal ABG Parameters

	Arterial	*Mixed Venous*
• *PH*	*7.35-7.45*	*7.31-7.41*
• *CO2*	*35-45*	*40-50*
• *HCO3*	*22-26*	*22-26*
• *PaO2*	*80-100*	*35-45*
• *SaO2*	*>95%*	*70-75%*
• ~~*BE*~~	$2/+2$	$-2/+2$

ABG interpretation – General Rules

HIGH CO2 – suggests Acidosis

HIGH HCO3 – suggests Alkalosis

- Look at pH: Always tells you the PRIMARY problem.

  - Normal 7.35 - 7.45
  - Acidosis < 7.35
  - Alkalosis > 7.45

- Look at pCO2 to confirm or eliminate a respiratory problem
  - **Normal 35 – 45**
  - **Alkalosis < 35**
  - **Acidosis > 45**

- Look at Bicarbonate
  - **Normal 22 – 26**
  - **Acidosis < 22**
  - **Alkalosis > 26**

- Look at pO2
  - **Normal > 80**
  - **Hypoxemia < 60**

Adjustment for aging 80 – (age-60)

Critical Care Review

Step 1

Look at pH, get Last name from the pH

Acidosis/Alkalosis

- pH 7.22
- pH 7.47
- pH 7.28
- pH 7.50
- pH 7.37

Step 2

Look at the PCO2 and HCO3, which value represents a condition consistent with the ABG's last name

	Respiratory	Metabolic
• pH 7.19	PCO2 66	HCO3 25
• pH 7.27	PCO2 38	HCO3 15
• pH 7.58	PCO2 12	HCO3 22
• pH 7.48	PCO2 42	HCO3 33
• pH 7.09	PCO2 60	HCO3 10

Compensation vs. Mixed Disturbances

Look at HCO_3 and CO_2:

- do they move in opposite directions (normal compensation)?

- is the magnitude of compensation reasonable (expected in a non mixed disorder) or is it more or less than expected.

Mixed disturbances are suggested by:

- pCO_2, HCO_3 moving in the same direction
- Greater than expected compensation
- Less than expected compensation
- High anion gap

Base deficit reflects the end point of resuscitation

BE = -5/+3

Golden Rules of ABG Analysis

1. For every 10 mmHg change in CO2, the pH will change 0.08 in the opposite direction.
2. For every change in bicarbonate of 10 mEq, the pH will change 0.15 in the same direction.
3. Formula for calculating bicarbonate replacement

0.1 x Wt in Kg x Base deficit = mEq bicarb needed

Respiratory Acidosis

pH < 7.40 with a CO2 > 45

See Rule #1

The result of failure to remove CO2!

Rapid Causes:

- Failure to get CO2 to lungs
- Failure to remove CO2 from the lungs

Slow Causes:

- Bronchospasm and COPD
- V/Q Mismatch
- Pulmonary Edema

Ventilator Management:

1) Ve = Vt x Rate

Minute Volume; appropriate Vt

2) Formula 6-10cc/Kg Lean body mass

Vt – Dead Space = volume of alveolar ventilation

Monitor PIP/PAP

If the PAP/PIP does not change, ALWAYS increase Vt before rate!

Consider increasing the rate.

Respiratory Alkalosis

pH greater than 7.40 with a CO2 less than 35

Rule # 1 still applies

Usually result of alveolar hyperventilation

Causes:

Tissue Hypoxemia

a) Decreased Supply
    - Anoxia
    - Anemia
b) Increased Demand
    - Gram-negative sepsis
    - Hyperthermia

Early Salicylate poisoning

Mechanical Hyperventilation

Anxiety

Treatment of Respiratory Alkalosis

- Control Anxiety
- Administer anxiolytics PRN
- Decrease Minute Volume – Ve

Metabolic Acidosis

pH is less than 7.40 with bicarb less than 22

See Rule #2 and #3

Causes:

Increased organic acid production

- Lactic acidosis
- Ketoacidosis

Decreased organic acid removal

- Renal Failure

Addition of exogenous acids

- Poisoning

Decreased bicarbonate

- Diarrhea
- Renal tubular acidosis
- Hyperchloremia

Anion Gap

The anion gap is a measure of the difference between Cations and Anions

Na^+ - (CI^- + HCO_3^-) = Anion Gap

Normal = 9-12

Cations

Calcium	Ca^{2+}
Magnesium	Mg^{2+}
Potassium	K^+
Sodium	Na^+
Ammonium	NH_4^+

Anions

Chloride	CI^-
Bicarb	HCO_3^-
Nitrate	NO_3^-
Phosphate	PO_4^{3-}
Cyanide	CN^-

The larger the anion gap the more severe the acidosis

Causes:

Methanol	**P**ropylene glycol
Uremia	**I**soniazide/Iron
DKA	**L**actate
	Ethylene Glycol
	Salicylate

Treatment:

- Treat underlying Cause
- Use Rule #3 to replace bicarb
- Correct by 50% with a bolus then the other 50% over an Hour

Metabolic Alkalosis

pH is greater than 7.40 with bicarb greater than 26

Too little H^+ or too much HCO_3

Usually a result of losing H^+, K^+, Na^+, CI^-

Causes:

GI loss of chloride

- Emesis
- NG suctioning

NOTES

Renal loss of Cations

- Diuretic therapy
- Steroid Therapy
- Aldosteronism

Cation deficiencies

Antacid poisoning

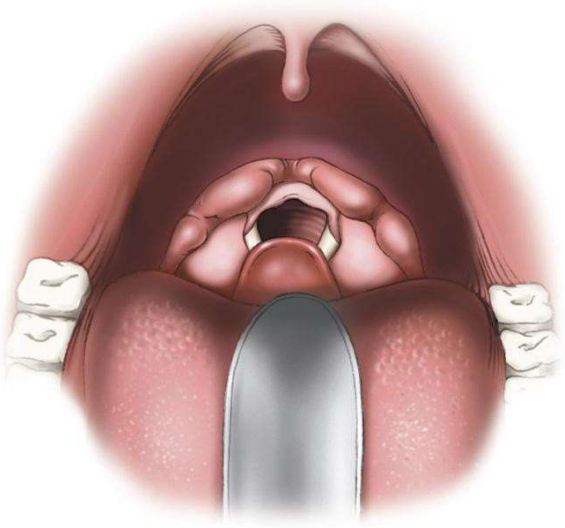
Treatment of Metabolic Alkalosis

K+, Mg++, Na+, Ca++ replenishment

- Kidneys stop excreting H+ ions and excrete K+ and Cl-

Treat underlying Causes

AIRWAY



Indications for Intubation

Airway patency

-"Gag reflex" vs. Ability to swallow

-Oxygenation or ventilation failure

-Expected clinical course

All algorhythms lead to **FAILED**

Inappropriate RSI may be the most costly error made

Any patient near death or any patient with a predicted difficult airway is **not** an RSI candidate

RSI - CRASH AIRWAY

-Who needs intubation?

The first 2 key questions

-Is my patient near death?

-Is my patient a predicted difficult airway?

1. Will you need pharmacologic agents?
2. How likely are you to "get the tube?"
3. What about when you don't?

DIFFICULT AIRWAY - FAILED AIRWAY

-Maintaining SaO2 over 90% is "key"

The 2 key questions

-Am I going to be able to ventilate?

-Am I going to able to intubate?

Can't intubate?

Can't ventilate? =100% mortality rate

Rescue airway vs. Surgical?

Is SaO2>90% and maintainable?

SaO2<90% = CRIC!

IF SaO2>90% = RESCUE AIRWAY until someone rescues you…

Rapid Sequence Induction

7 Ps

1. **P**reparation
2. **P**re-oxygenation
3. **P**retreatment
4. **P**aralysis with induction
5. **P**rotection and positioning
6. **P**lacement with proof
7. **P**ost-intubation management

Preparation

-Less invasive means vs. rapid success

-Anatomical or medical contraindications

Predicting Difficulty

Look

Evaluate 3-3-2 rule

Mallampati

Obstructions

Neck Mobility

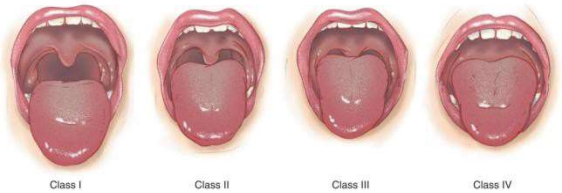
Cormack and Lahene Grades

GRADE I - entire glottis opening visible

GRADE II - arytenoids cartilages or posterior portion of glottic opening is visible

GRADE III - Epiglottis only is visible

GRADE IV – Tongue and/or soft palate only is visible



Malampatti Scoring

Class I – soft palate, uvula, anterior & posterior tonsillar pillars

Class II – tonsillar pillars hidden by tongue

Class III – only the base of the uvula can be seen

Class IV – uvula cannot be visualized at all

BVM Issues - Cric Issues

-**S**uction Mask Seal

-**O**xygen Obesity

-**D**rugs Age

-**A**irways No Teeth

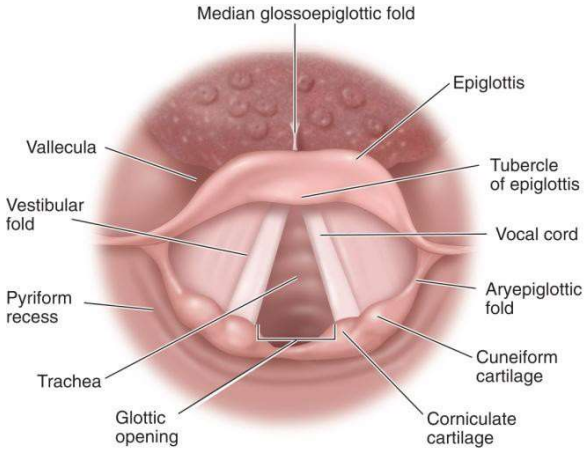
-**S**tiff

-**S**urgery

-**H**ematoma

-**O**besity

-**P**re-oxygenation



Optimal techniques avoid positive pressure ventilation (NRB vs. BVD)

Pretreatment: (LOAD)

Lidocaine 1.5mg/kg

Opiates for analgesia (Fentanyl 3mcg/kg)

Atropine in the Pedi patient 0.02 mg/kg

Defasciculating neuromuscular blockade

-10% normal RSI dosage of NMBA

Paralysis with induction

Wait three minutes after premedication, then administer the induction agent followed by paralytics.

Medication Dosage Onset Duration

Etomidate 0.3mg/kg 30-60 sec/ 3-5 minutes

Fentanyl 15-30 mcg/kg 60 sec /30-60 minutes

Ketamine 1.0-2.0 mg/kg 30-60 sec /15 minutes

Midazolam 0.2-0.3 mg/kg 1-2 min /1-2 hours

Thiopental 3-6 mg/kg 20-40 sec /5-10 minutes

Medication Dosage Onset Duration

Succinylcholine

1-2 mg/kg 1-2 min 4-6 min

Rocuronium

0.5-1.0 mg/kg 1-2 min 20-40 min

Vecuronium

0.1 mg/kg 2-3 min 20-40 min

Atracurium

0.4-0.5 mg/kg 2-3 min 20-45 min

Pancuronium

0.01- 0.04 mg/kg 3-5 min 60-100min

Pharmacology

Succinylcholine –depolarizing (non competitive) neuromuscular blocking agent

Mechanism of action - Stimulates motor end plate acetylcholine receptors causing persistent depolarization

Indications - Paralysis to facilitate intubation

- limit the possibility of aspiration

Contraindications - Penetrating eye injuries, narrow angle glaucoma, Hx of malignant hyperthermia (Dantrolene), burns greater than 24 hours old, hyperkalemia

Duchenne's muscular dystrophy in children

Protection and Positioning

Protect -Apply cricoid pressure to prevent passive regurgitation. If active regurgitation occurs, release cricoid pressure and place patient in recovery position/log roll.

"Active regurgitation" vs. "Silent regurgitation" Positioning

C-spine issues

-Kyphosis

-Bull-neck

Sizes

Women 7.0-8.0 ETT

Men 8.0-9.0 ETT

Peds Age + 16 / 4

Miller/straight blade - Inserted under the epiglottis

Mac/curved blade - Inserted in the vallecula

Securing at the teeth

-Women 21cm

-Men 23 cm

Proof

Primary

-Visualized placement

-Lung/Stomach sounds

Secondary

- Aspiration devices

-Cholormetric devices

-Capnometry/Capnography

-X-ray

Post-intubation Management

-Don't forget to ventilate them

-Secure ETT with commercial restraint device

-Continued sedation

-Consider continued paralysis

Monitoring

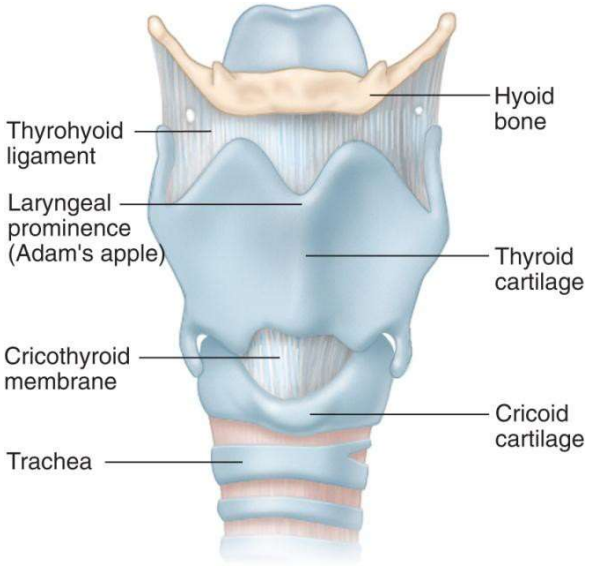
-ECG

-SaO2

-ETCO2

-NIBP

-Temperature



Needle & Surgical Airways

-Used to secure an airway when other means fail

-Surgical Cric more preferable than Needle Cric

Contraindications

Landmarks – must be able to palpate landmarks

Procedure

1. Identify landmarks
2. Stabilize
3. Make skin incision
4. Identify Cricothyroid membrane
5. Make horizontal incision completing Translaryngeal airway

NOTES

Hemodynamic Monitoring

Levels of hemodynamic monitoring

Arterial, Central Venous, Pulmonary Artery

PA catheters allow for monitoring of vascular tone, myocardial contractility, intracardiac pressures, cardiac output, and fluid balance.

Basic Hemodynamic Monitoring

PA pressure waveforms reflect the mechanical events in the cardiac cycle.

Nervous system control

The kidneys receive 25% of cardiac output each minute

In response to decreased blood flow the bodies will always vasoconstrict

• (unless it can't)

CHF -

Major revisions in care targeted toward a decrease in ventricular remodeling

Ventricle dysfunction will result in over distention. Over distention promotes the release of BNP a measurable peptide

Divided in systolic and diastolic failure syndromes

Systolic

-Failure of ventricular muscle to generate adequate contractility to promote forward flow of blood

Diastolic

-Failure of non distendable ventricle to fill during diastole

Goals of therapy -Reduce cardiac workload

Decrease preload

-Nitro, MsO4, Lasix

Decrease after load

-Nipride, Natracor

Decrease rate

-Beta Blockade --- Carvidolol only!

Carvedilol is both a beta blocker (β_1, β_2) and alpha blocker (α_1):

Improve contractility

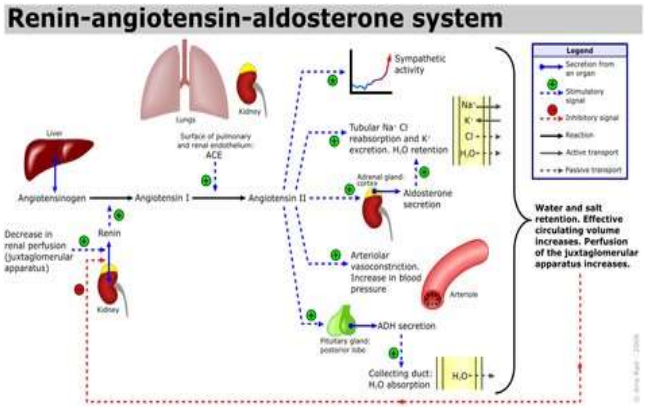
Inotropic Agents

-Dobutamine, Amrinone, Milrinone

Prevent ventricular remodeling

-ACE Inhibitors

- Enalapril, Captopril, Zestril



Cardiogenic Shock — inadequate cardiac pumping resulting in decreased cardiac output and hypotension.

Pharmacologic management includes

-Dobutrex, dopamine and Inocor to improve cardiac output

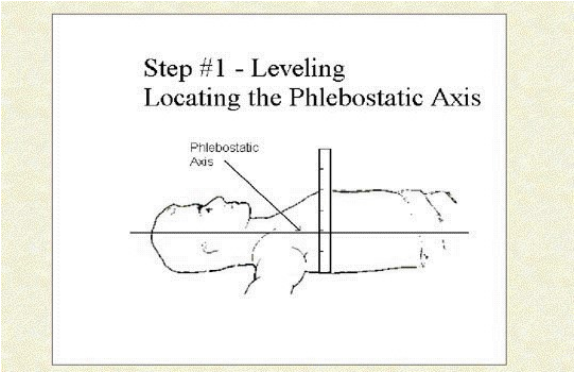
-Vasodilators such as Nipride and nitro and vasoconstrictors such as Levophed

-IABP and LVAD therapy

Types of Hemodynamic Catheters

Pressure Monitoring Setup

1-Purge and Flush Line, pressurize fluid, place transducer at phlebostatic axis, attach tubing, close stopcock to patient, zero, and open to patient and fast flush



Arterial Lines

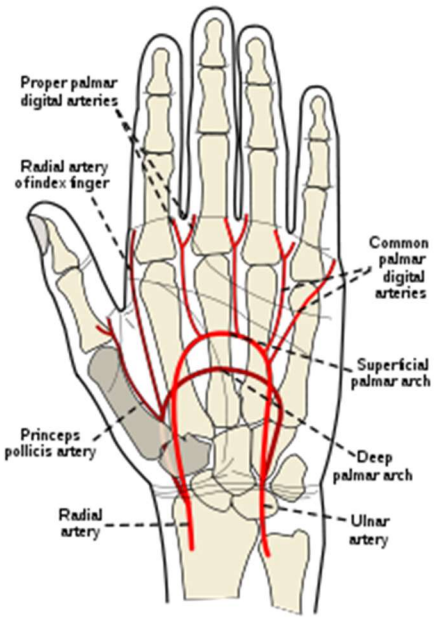
Usually placed in radial or femoral arteries

Allows continuous BP monitoring and rapid recognition of problems requiring intervention from the flight crewmember

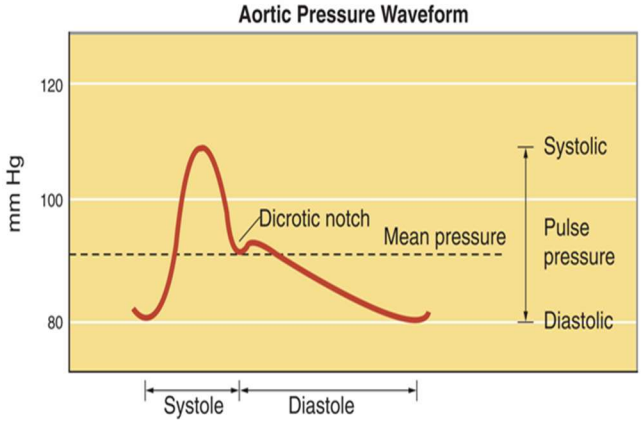
Easy access to blood gases and labs through A-line without sticking the patient

Slurring of the dicrotic notch occurs with aortic valve disease.

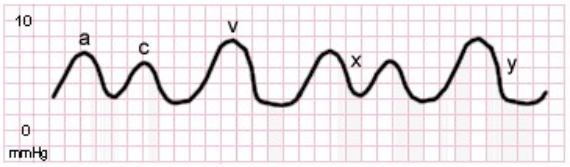
Utilize **Allen Test** prior to establishing A-line



Arterial Line Waveform



CVP/RAP



Measured in the great veins/right atrium, it is a reflection of right atrial pressure.

Normal is 2-6 mmHg.

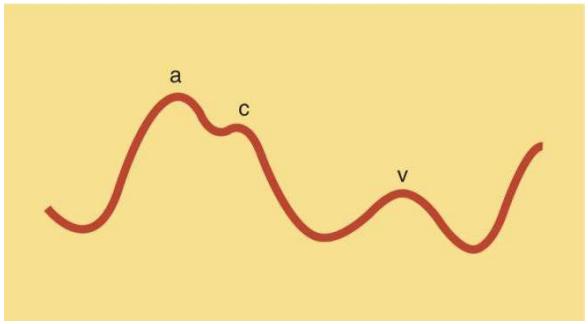
Decreased

- Hypovolemia, vasodilatation, decreased venous returns, & negative pressure ventilation

Increased

- Hypervolemia, right-sided heart failure, cardiac tamponade, positive pressure ventilation, COPD, pulmonary HTN, pulmonary embolus, pulmonic stenosis and tricuspid stenosis/regurgitation.

CVP or RAP waveform

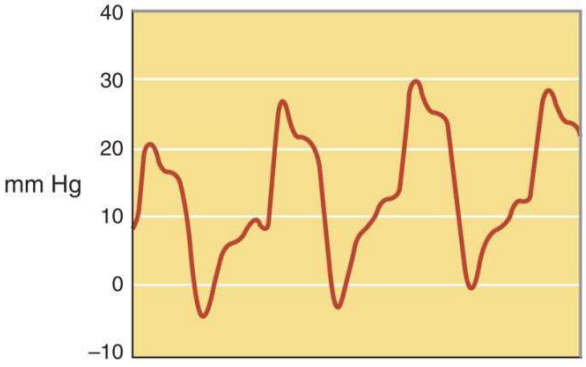


"a wave"—atrial contraction.

"c wave"—closure of the tricuspid valve

"v wave"—passive atrial filling. Tricuspid valve bulges back into atrium during right ventricular systole.

RV Waveform



RVP — not typically monitored, but seen with insertion of the PA catheter

Increased pressures seen in:

Pulmonary HTN—caused by left heart failure, mitral regurgitation, stenosis, and cardiomyopathy
Pulmonary disease—COPD and pulmonary embolus

Normal

-Systolic pressures are 15-25 mmHg

- Diastolic pressures are 0-5 mmHg

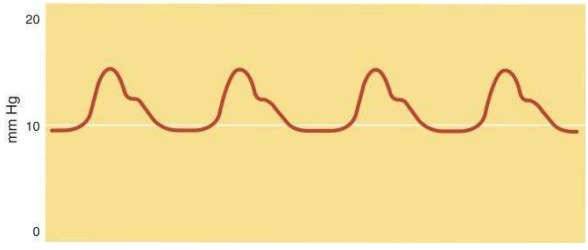
-No dicrotic notch seen on the waveform

-RV waveform has taller, sharper upstrokes

-The diastolic dip on the downstroke reflects ventricular relaxation.

-At end-diastole, atrial kick produces a small rise in pressure.

PA Waveform



PA pressures reflect right and left sided heart pressures.

Increased PA pressures seen in:

- Fluid overload, atrial or ventricular defects, pulmonary problems, LV failure, & mitral stenosis/regurgitation

Normal

-Pulmonary systolic pressure is 15-25 mmHg.

-Pulmonary diastolic pressure is 8-15 mmHg.

Reflects LVEDP and LV function

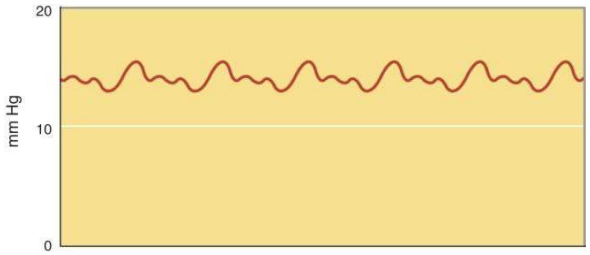
Usually 2-4 mmHg higher than mean PCWP

-May be used to estimate PCWP if unable to obtain an accurate wedge pressure.

-Sharp rise in waveform is systole

-Dicrotic notch is closure of the pulmonic valve and signals the end of systole.

PCWP Waveform



-Obtained by inflating the balloon until waveform changes.

-Occludes blood flow, eliminating the right side of the heart

-Looks at the left side of the heart

Elevated

- Left Ventricular failure, constrictive pericarditis, mitral stenosis, regurgitation, and fluid overload.

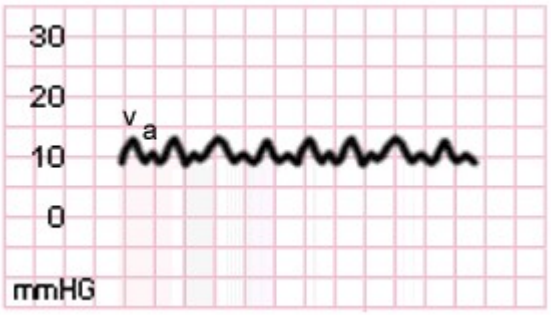
Decreased

- Hypovolemia and venodilating drugs

Normal

-PCWP is 8-12 mmHg.

PCWP Waveform



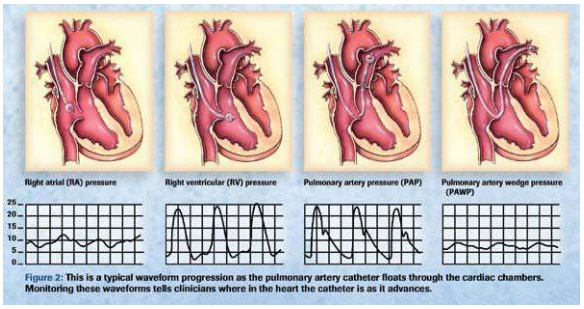
"a wave"—left atrial contraction/systole

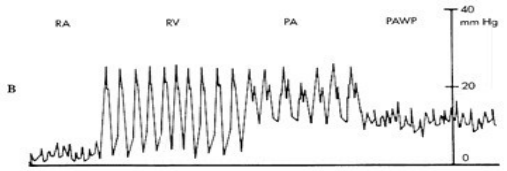
"c wave"—closure of the mitral valve

"v wave"—passive atrial filling. Bulging of the mitral valve during left ventricular systole

"x descent"—left atrial diastole

"y descent"—left atrial emptying. Reflects volume and pressure decline following the opening of the mitral valve and blood emptying into the left ventricle





PA insertion sequence

TABLE 14-2 Normal Ranges and Values for Hemodynamic Measurements	
Assessment Parameter	**Normal Range/Value**
CVP, mm Hg	2 to 6
SBP, mm Hg	100 to 120
DBP, mm Hg	60 to 80
MAP, mm Hg	70 to 105
Pulse pressure, mm Hg	40 to 60
Heart rate, mm Hg	60 to 100
Pulmonary artery pressure, systemic	15 to 30
Pulmonary artery pressure, diastolic	8 to 15
PCWP	4 to 12
CO, L/min	4 to 8
Pulmonary vascular resistance, dyne-sec/cm^{-5}	100 to 250
SVR, dyne-sec/cm^{-5}	800 to 1,200
SV, mL/beat	60 to 100
Svo_2, %	60 to 80
$Scvo_2$, %	≥ 70
Cardiac index, L/min/m^2	2.5 to 4.2
Stroke volume index, mL/beat/m^2	30 to 50

Abbreviations: CO, cardiac output; CVP, central venous pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; PCWP, pulmonary capillary wedge pressure; SBP, systolic blood pressure; $Scvo_2$, mixed central venous oxygen saturation; SV, stroke volume; Svo_2, mixed venous oxygen saturation; SVR, systemic vascular resistance.

Cardiac Output

CO=HR x SV

Normal values 4-8 liters per minute.

Cardiac index is based on body surface area and is a more accurate, individual measurement.

Normal cardiac index is 2.5-4.2 liters per minute per meters squared.

-Heart rate is the quick, easy way to increase cardiac output. Initial compensatory reaction to decreased cardiac output

-Detrimental effects of increased heart rate include increase myocardial oxygen demand & decreased diastolic filling.

Stroke volume

-the amount of blood ejected from the ventricles during systole

-Normal stroke volume is 60-135 cc.

-Normal stroke index is 25-45 ml per meters squared. Components of SV are preload, afterload, and contractility

Preload

-is defined as the amount of stretch on the ventricles at end-diastole. The amount of blood returned to the heart. **Frank Starlings Law**

RA pressure — measures preload for the right side of the heart.

PCWP — measures preload for the left side of the heart.

Decreased preload

-Hypovolemia or vasodilation

Increased preload

-Hypervolemia, CHF, or renal failure

Medications that decrease preload:

-NTG

-MSO4

-Lasix/diuretics

Medications that increase preload:

-Vasoconstrictors

-Fluid

Afterload — the resistance the heart must pump against to eject blood from the ventricles

PVR —Pulmonary Vascular Resistance

-measures afterload for the right heart

-Normal PVR - 50-250 dynes

- PVRI 225-315 dynes.

Increased

-pulmonary disease and hypoxia

SVR — Systemic Vascular Resistance

-measures afterload for the left heart

-Normal SVR - 800-1200 dynes

-SVRI 1100-3000 dynes.

Decreased

-distributive shock states and vasodilators

I**ncreased**

- HTN, IHSS, aortic stenosis and vasoconstrictors

Medications that decrease SVR:

-Nipride

-Corlopam

Calcium channel blockers

-Dobutrex

-Natreocor

Medications that increase SVR:

-Dopamine

-Neosynephrine

-Levophed

-Epi

Contractility is the ability of the myocardium to effectively contract.

Decreased

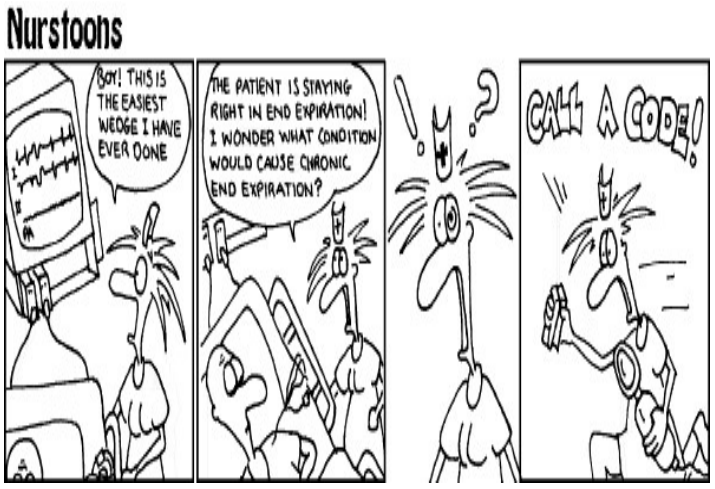
-electrolyte imbalances, hypoxia, acidosis, ischemia and necrosis

Increased

-high levels of Calcium, sympathetic nervous system stimulation and medications such as Digoxin or Dobutamine

Invasive Line Transport Considerations

1. Make sure all air is removed from the system to prevent air embolus and dampened waveforms.
2. Tape the transducer at the phlebostatic axis or on the bicep during transport.
3. Limit the amount of tubing to decrease the chances of dislodgment and artifact due to aircraft vibration.
4. Boyle's law will cause changes in pressure bag necessitating close attention of the flight crew members
5. Needs to be re-zeroed with changes in altitude to ensure accuracy of readings.



Cardiac Pharmacology

Beta Blockers

- Blocks body's response to adrenergic stimuli

-Decreases oxygen consumption of the heart.

Indications

-maintaining HR 60-80 in MI

Contraindications

-heart failure, 1st degree block with PR>.20, 2nd and 3rd degree HB

Dobutamine

- Primarily B1 effects, but produces B2 effects that cause mild vasodilation. More effective than Dopamine for increasing contractility

Dopamine

-has both alpha and beta effects

-low doses (2-5 mcg/kg/min), increases renal perfusion and urinary 0utput

- Moderate doses (5-10 mcg/kg/min), increases cardiac contractility, HR, and CO

-high doses, (10-20 mcg/kg/min), primarily alpha effects & increases SVR

Levophed

- has alpha and beta 1 effects

-Increases coronary artery blood flow

Indicated - profound hypotension.

Contraindicated - patients with occlusive vascular problems and patients with a hypersensitivity to sulfites

Initial dose is 2-12 mcg/min, titrate to desired effect.

Neo-Synephrine

-Potent Alpha stimulator used to increase SVR in profound vasodilatory redistributive shock states such as sepsis

May be given by bolus or steady infusion

Bolus 0.1-0.5mg slow IV q 10-15m PRN

Gtt 100-180mcg/min titrated to BP

- Maintenance gtt of 40-60mcg/min

Use caution - in patients with cardiac disease

Nipride

- used to reduce preload and afterload by dilation of arterioles and veins

Indicated -HTN crisis, refractory congestive heart failure, increased vascular resistance

Contraindicated - decreased cerebral perfusion and hypersensitivity

Use with caution - in renal disease, hepatic disease (increased risk of thiocyanate accumulation), and geriatric patients (increased hypersensitivity.)

-Severe peripheral vascular disease

Intra-Aortic Balloon Pump

Goals of IABP therapy are:

-Decrease the work of the heart

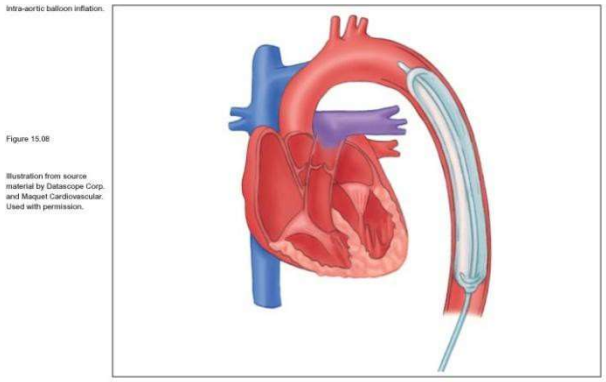
-Decrease myocardial oxygen demand

-Increase coronary perfusion

-Improve cardiac output

-Limits myocardial ischemia

-Prevent cardiogenic shock, limit size of infarctions



IABP uses:

-Support in acute MI with cardiogenic shock

-Circulatory support in post-CABG patients

-Support in high-risk catheterizations

-In severe ischemia as a bridge to revascularization

-Intractable chest pain refractory to conventional care

IABP Therapy

-Balloon inserted percutaneously in the femoral artery.

-Balloon sits in the descending aorta, just distal to the left subclavian artery and above the renal arteries

-Balloon inflates and deflates based on patient's EKG or arterial pressure waveform

-Ventricular systole, the balloon is deflated

-Ventricular diastole, the balloon is inflated

Contraindications:

-Aortic insufficiency

-Severe aortic disease

Complications:

-Ischemia of limb distal to insertion site

-Aortic dissection

-Thrombocytopenia

-Septicemia

-Infection

-Renal complications

-Air/gas emboli

Timing errors:

Late deflation — the balloon is inflated during the beginning of ventricular systole. This increases the work load of the left ventricle. Very harmful for the patient

Early inflation — the balloon is inflated before aortic valve closure. Causes reflux of blood into the left ventricle. Decreases cardiac output and increases SVR, Harmful

Late inflation — results in suboptimal augmentation because there is minimal displacement of blood back toward the coronary arteries

Early deflation — vacuum effect and afterload reduction is lost. This occurs because by the time

the aortic valve opens, the pressures in the aorta has equalized

NOTES

Transport Considerations

-Due to aircraft vibration, the trigger mode may need to be the arterial pressure line. The ECG may have too much artifact to effectively trigger the balloon inflation/deflation

-Balloon will purge on ascent due to Boyle's Law and gas expansion with increasing altitude

-Balloon will purge again on descent due to gas contraction with decreasing altitude.

Watch for patient decompensation during balloon purges. Be prepared to treat patient.

-Make sure that all air is out of the art line to minimize dampening of the waveform since this may be a trigger source in the event of EKG trigger failure.

-In the event of cardiopulmonary arrest in the patient, place the trigger mode on arterial pressure or on internal trigger mode.

-In the event of power failure, the balloon needs to be manually pumped every 30 minutes to prevent thrombus formation on the balloon.

TAKE THE SYRINGE!

-Take extra helium tanks. With altitude changes and balloon purging, you may need those extra tanks.

-Before leaving a facility assure balloon placement by verifying the distal tip of the balloon is 2 cm below the aortic arch, and the proximal end of the balloon does not occlude the renal arteries. Get a **RECENT** chest film.

-*Watch Urinary output and distal pulses closely in transport – renal arteries are occluded*

- *Loss of left radial pulse indicates the balloon has migrated upward covering the subclavian artery*

Environmental

The Body's Response to Cold

Peripheral vasoconstriction

Skeletal muscle vasodilation

Increased cardiac output

Shivering

Limited by available glycogen stores

Loss of shivering at 32°C

Physiologic Response to Hypothermia

Initially metabolism increases to produce heat

Metabolism decreases by half for every drop in core temp of 10°C

Patients BGL will be normal or low with mild chronic hypothermia

BGL is increased with severe hypothermia as insulin stops working

Respiratory minute volume decreases

Respiratory rate is inadequate to blow off CO2 thus a respiratory acidosis follows

Decreased CO

Increased SVR

Capillary leak decreases intravascular volume

Metabolic acidosis related to anaerobic metabolism

Hypothermia Stages

Mild 32-36°C

Increased BMR, CO, HR begins to fall at 32°C

Moderate 29-32°C

Shivering stops, LOC and SVR fall, acidosis, hyperglycemia, CO falls

Severe < 28°C

Hypotension Prolonged PR, QRS, QT intervals, coma, hypoventilation, apnea

VF risk highest at 22°C

Osborne waves clear at 25°C

Hypothermia Treatment

Remove cold and wet environment

Manage airway consider intubation risk for V-fib

Handle gently

Only start CPR if monitor shows VF or Asystole

Defer meds till core temp > 30°C

Passive External Rewarming

Patient allowed to rewarm self with blankets and transport vehicle heater

Active External Rewarming

Heat placed on external body surfaces

Watch for after drop

Active Internal Rewarming

Heat directly to core

IVF, Hemodialysis, gastric lavage, ECMO

Heat Pathophysiology

Cellular breakdown

Vasodilation necessitates CO thus increasing O2 demand

Increased sweat evaporation can lead to loss of 1-3L/Hr.

High output failure can lead to MI

Respiratory alkalosis

Neurologic problems related to cerebral edema, Seizures increase metabolism raising temp

Kidneys retain sodium and release K+, hypoperfusion leads to ATN

Liver is hypoperfused leading to hypoglycemia

Muscle death causes an increase in CK and rhabdomyolysis

ARDS and DIC result from lysosomal enzymes

Methods of Heat Transmission

Conduction-Surface to surface transmission

Convection-Loss of heat by air or water movement across the body surface

Radiation-Loss to atmosphere

Evaporation-Loss of heat by dissipation of sweat

Hyperthermic response

Heat increases

Related to increased metabolic processes

Related to environmental increase

Body compensation

Sweating

Increased respiration

Vasodilation

Heat Related Problems

Heat Cramps

Occur in heavily exercised muscles in high heat
Caused by hyponatremia associated with only water replacement

Cool and rehydrate with salt containing solutions

Heat Exhaustion

Defined as an increase in core temp without neurological impairment

Temp is lower and symptoms less severe than

Heat stroke

Patient retains ability to sweat

Treatment is aimed at cooling as well as fluid and electrolyte replacement

Heatstroke

A life threatening emergency characterized by failure of the body to dissipate heat effectively

LOC is altered

Core temp. 42° C

Oxygen demand exceeds supply

Respiratory alkalosis. Metabolic acidosis

High output cardiac failure

Heatstroke Treatment

COOL, COOL, COOL

Soak patient and provide fanning

Aggressive airway management

Don't under ventilate

Expose

IV fluids consider Thorazine 25-50mg

Prevent shivering

Place NG consider H2 blockers

Consider phenothiazines (poikilothermic properties)

Place a Foley, Monitor labs- ABGs for acidosis

Watch clotting factors for early onset of DIC

Monitor liver enzymes

Watch urinary output for rhabdomylosis

Watch sodium for hyponatremia

Rhabdomyolysis

Treat by alkalinizing the urine with NaHCO3

Hyponatremia

Replace by correcting sodium slowly

Potassium

Hypokalemia usually the result of respiratory alkalosis

Hyperkalemia usually the result of metabolic acidosis

Malignant Hyperthermia caused by Anesthetics and skeletal muscle relaxants such as Succinylcholine (Anectine)

Calcium is released into muscle fibers causing sustained contraction

A hyper metabolic state occurs increasing temp

May prevent mouth opening

Relieved by reversal of anesthetic and administration of 1-3mg/kg of Dantrolene Sodium (Dantrium)

NOTES

Critical Care Review

OB/GYN EMERGENCIES

GENERAL TRANSPORT CONSIDERATIONS

1. Place patient in a left lateral recumbent position
2. Note the patient's temperature
3. Note fetal heart tones, fetal movement, contractions
4. Start a large bore IV of LR
5. O2 to maintain sat of 98-100%
6. Note and quantify any bleeding or leaking of fluid

MATERNAL PHYSIOLOGICAL CHANGES

1. HR increases 15-20 bpm
2. BP decreases in 2nd trimester 5-15mm/Hg
3. BP increases in 3rd trimester 10mm/Hg
4. Cardiac output increases
5. Circulating plasma volume increases 40% thus increasing clotting factors
6. RBCs increase but not to the same percentage as plasma resulting in a dilutional Anemia
7. Heart elevates and rotates
8. WBCs elevate
9. Progesterone relaxes sphincters
10. Decreased functional/residual lung volume

TABLE 21-1 Cardiovascular Changes in Normal Pregnancy

	Nonpregnant Patient	Pregnant Patient
Cardiac output	5,000 mL	6,000 mL (20%-40% increase)
Stroke volume	80 mL	110 mL (approximately 30% increase)
Pulse rate	80 beats/min	10%-20% increase
Red blood cell mass	5 million cu mm	33% increase
Systemic vascular resistance	1,500 dyne-sec/cm^{-5}	20% decrease
Mean arterial pressure	85 mm Hg	No change
Systolic blood pressure	120 mm Hg	120 mm Hg
Diastolic blood pressure	70 mm Hg	80 mm Hg

Adapted from: Sodré PM. Maternal physiology changes during pregnancy. *Obstet Gynecol*. Available at: http://www.medstudents.com.br/ginob/ginob5.htm. Accessed February 12, 2009.

OBSTETRICAL TERMINOLOGY – "Secret Handshake"

Dilation- Refers to the extent of cervical dilation as judged by palpation. Full dilatation occurs at 10 cm

Effacement- Relates to the thickness of the cervix and is expressed as a %. The cervix is normally about 2 cm thick and thins during labor. Thus when thinned to 1 cm the patient would be said to be 50% effaced

Station- Refers to the fetal head in relation to the mothers pubic bone and is expressed as a – or + number as measured in cm

FETAL MONITORING

TABLE 21-3 Heart Rate Parameters	
Rate	**Beats/min**
Normal	120-160
Abnormal • Tachycardia • Bradycardia	< 100 > 160 < 120

Baseline FHT's are 120-160

Variability- Single most important predictor of fetal well-being

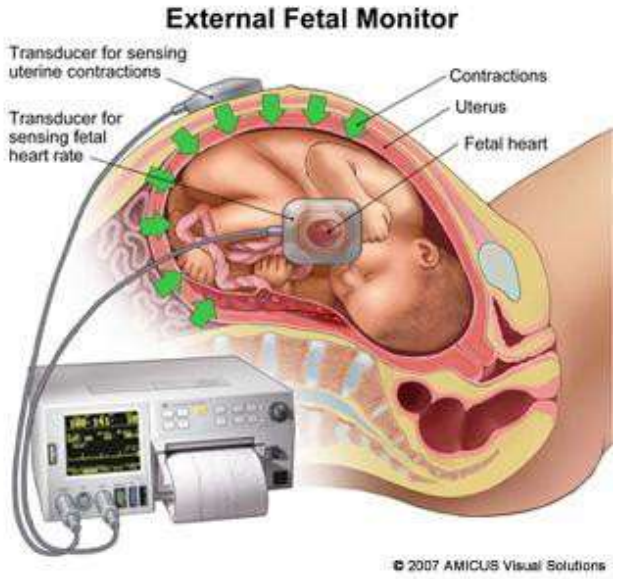
Poor variability caused by

1. Fetal hypoxia
2. Administration of meds to mom
3. Smoking
4. Extreme prematurity
5. Fetal sleep

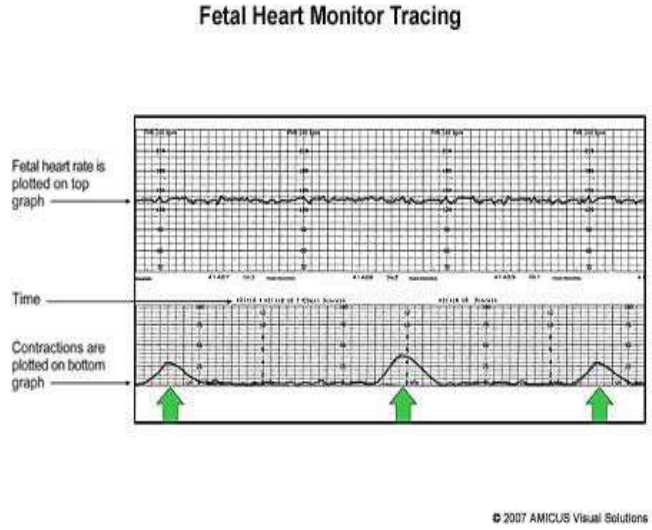
What are Accels?

What are Decels?

How do they correspond to labor?



FETAL MONITORING



-Top waveform signifies fetal HR

-Bottom waveform signifies maternal contractions

Variability is judged by erratic waves of the fetal heart tracing and is separated into short and long term variability (suggests neurologically intact)

Accelerations (**Usually GOOD**)

-Usually associated with fetal movement

-Hypoxic fetus w/ metabolic acidosis cannot accelerate its HR

Variable Decelerations (**Not good**)

-Common during contractions, typically V or W shaped Caused by cord compression

"Shouldering" is common

-Look for 'cord' problems (prolapsed, short, entanglement, nuchal)

Late Decelerations (BAD)

-Always mean uteroplacental insufficiency causing the fetus to experience a hypoxic Bradycardia

- Associated with PIH, DM, Smoking and late deliveries

Sinusoidal (VERY BAD)

-Typical of fetal Hypovolemia or anemia

-Commonly associated with accidental tap of the umbilical cord during amniocentesis, fetomaternal transfusion, placental abruption

Bradycardia

FHR<120 for 5-10 minutes

Tachycardia

FHR>160 for >10 minutes

FACTORS CONTRIBUTING TO FETAL DISTRESS

1. Assure fetal oxygenation give mom High flow O2 by NRB
2. Maternal Hypotension- Initiate 250-500cc IVF bolus, correct supine hypotension. "Tank her up!"
3. Hypertonic or tetanic contractions- Discontinue oxytocin infusion.
4. Rule out cord prolapse
5. Assess for placental abruption
6. Change positions

TABLE 21-2 Conditions Associated With Fetal Distress During Labor

Source	Condition
Umbilical cord	Hematoma Knot in cord Nuchal cord Prolapsed cord Cord compression
Placenta	Infarction Abruption
Uterus	Tetanic contractions Hyperstimulation
Fetus	Anemia Infection
Maternal	Hypertension Hypotension Severe anemia Seizures

MATERNAL COMPLICATIONS

SIGNS/SYMPTOMS OF IMMINENT DELIVERY

-Vaginal bleeding

-Contractions greater than q 10 min

-Increasing intensity of contractions

-Urge to push

-Crowning

PRE-TERM LABOR

1. Look for signs of imminent delivery
2. Determine the status of the amniotic membranes
3. Determine cervical status
4. Maintain the patient in left lateral recumbent
5. Assess for infection
6. Prophylactic antibiotics for group B strep
7. Administer tocolytics:

-**MgSO4**- watch for toxicity, DTRS, Respirations

-Indomethacin

-Terbutaline

-Monitor fetal heart tones, movement

MATERNAL MEDICATIONS

MEDICATION DOSAGE INDICATION

Terbutaline

0.25mg sq q 15 min Pre-term labor

Magnesium Sulfate

4-6g bolus/15-30min 2g/hr maintenance

Preeclampsia, Pre-term labor

Oxytocin

20-40 units/1000cc at 125cc/hr Post-partum hemorrhage

Hydralazine

2mg iv q 5min to DBP 90-100mmHg

Or 5-10mg q 20min PIH, Preeclampsia

ECTOPIC PREGNANCY

1. Every woman of child bearing age with acute abdominal complaints is an ectopic pregnancy until proven otherwise
2. Treatment is usually surgical
3. Maintain ABC's
4. Treat for hypovolemic shock
5. Cross matched blood
6. Give Rhogam if mom is Rh-

MATERNAL HYPERTENSIVE SYNDROMES

Pregnancy Induced HTN (PIH) relates to any new onset HTN with pregnancy

Who's at risk?

Preeclampsia is characterized by HTN, proteinuria, and edema

HELLP Syndrome refers to patients with a myriad of symptoms including Hemolysis, Elevated Liver enzymes, and a Low Platelet count

PIH, PREECLAMPSIA & HELLP

1. Watch for late decels and decreased variability.
2. Monitor fetal movement.
3. MgSO4 4-6g bolus followed by 1-2g/hr
4. Steroids (Celestone) as soon as possible
5. Consider Labetalol 20mg SIVP
6. Consider Hydralazine 2mg SIVP
7. Give Valium prn for seizure activity with MgSo4

TRAUMA IN PREGNANCY

<u>The best way to maximize fetal viability in trauma is to aggressively treat mom like you treat other trauma patients</u>

High Index of Suspicion

-MOI

-Seat Belt

After benign collision Transport in full spinal precautions with board tilted to left

Because of the increase in circulating volume, 30-35% of the blood volume may be lost before signs and symptoms of Hypovolemia surface

Vigorous fluid replacement, High flow O2

PLACENTA PREVIA

Characterized by Placental attachment over the cervical opening

Common w/ uterine scarring, multiparity w/ short intervals, post D&C, etc

Often presents as painless bright red vaginal bleeding

1. *High flow O2, vitals, fluids*
2. *Assess for contractions, fetal movement/heart tones, hemorrhage.*
3. *Consider tocolytics and blood products.*
4. Watch for signs of DIC

PLACENTAL ABRUPTION

Abruption is characterized by disruption of the uterine wall-placental interface with tearing of vasculature

Often presents as ripping or tearing pain with dark or no evident blood loss

There is a twofold threat for Exsanguination

Placental insufficiency

Blood is irritating to the uterus, thus it will initiate contractions

1. High flow O2, vitals, fluids
2. Assess for contractions, fetal movement/heart tones, hemorrhage.
3. Continually reassess fundal height
4. Consider tocolytics and blood products.
5. Watch for signs of DIC

UTERINE RUPTURE

Rapid Recognition is Key!

1. May be misdiagnosed as Placenta Abruptio
2. Serial fundal height measurements
3. Look for signs of previous Cesarean
4. Rapid transfer with supportive care
5. Oxytocin, 20-40 units in 1000cc may reduce bleeding

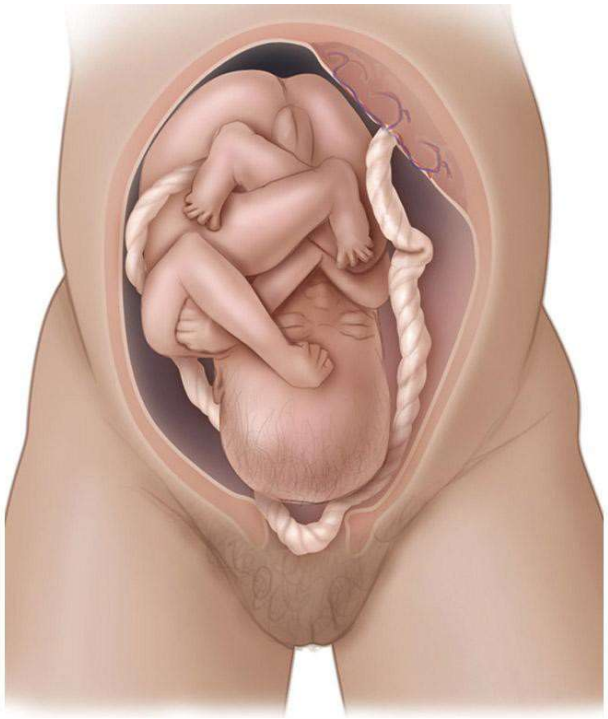
DELIVERY COMPLICATIONS

NUCHAL CORD

-Variable Decelerations

-Gently loosen and draw down over head

-Clamp & cut before shoulders are delivered if it is too tight to remove

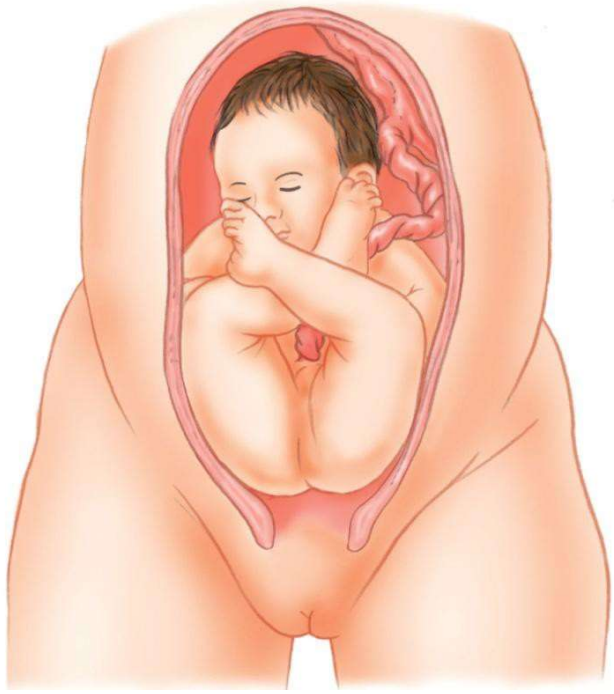


UMBILICAL CORD PROLAPSE

-Elevate presenting part off the cord with a hand in the vagina to prevent cord compression.

-Trendelenburg or knee to chest position

-Tocolytics to reduce pressure on the cord during contractions.



BREECH PRESENTATION

-Fetus should not be touched until the umbilicus has spontaneously delivered.

-Palpate cord for FHR.

-After shoulders have delivered, rotate baby's trunk so that the back is anterior and apply gentle downward traction.

-Apply suprapubic pressure to facilitate delivery of the head **(Mauriceau's maneuver).**

-Do not attempt to deliver a footling breach

MECONIUM DELIVERY

POSTPARTUM HEMORRHAGE

1. Vigorous fundal massage
2. Rapid infusion of 20-40 units oxytocin Methergine (methylergonovine)
3. Assess blood loss
4. Replace fluids, blood
5. Bimanual uterine compression

UTERINE INVERSION

1. Manual replacement if the uterus has not yet contracted down and cervix has not constricted
2. Tocolytics if the uterus has contracted to relax the uterus and allow replacement
3. Oxytocin
4. **DO NOT REMOVE THE PLACENTA!**

Anaphylactoid Syndrome of Pregnancy

The old theory - Amniotic fluid created a pulmonary embolus

The current research - Antibody response to a fetal antigen (anaphylaxis)

Treatment

1. High flow 02, Intubate as needed
2. PEEP may be required
3. Fluid resuscitation
4. Hemodynamic monitoring
5. Blood product replacement as needed FFP, Platelets, Cryo
6. Anticipate severe fetal distress if syndrome presents prepartum
7. Prepare for emergent postmortum c-section

DIC- Disseminated Intravascular Coagulopathy

-Overstimulation of clotting cascade

2 ways to make a clot

Intrinsic – endothelium injury
Causes: ARDS; Septic Shock; Hypotension
Clotting Factors: 12, 11, 9, and 8

Extrinsic – Tissue Injury
Clotting Factors: 7
Release of Tissue Thromboplastin, begins clotting cascade

Why are Mothers so susceptible?

- Highest levels of Tissue Thromboplastin are located in the Brain and Placenta

Labs-

Decreased Fibrinogen level

Increased PT/PTT interval
Increased Fibrin Spit Products (FSP)
Increased D-dimer

Treatment

1. Airway Management
2. Heparin administration

   -inhibits conversion of prothrombin to thrombin

   -inactivates circulating thrombin

   - inhibits conversion of fibrinogen to fibrin
3. Stop ongoing major hemorrhage

   - Get rid of triggering event
4. Volume Replacement
5. Minimize needle sticks
6. Blood Products (FFP, Platelets)

   - Begin administration after heparin and you have reached a PTT between 2 – 2.5

Heparin Induced Thrombocytopenia (HIT)

- Some people do not have Anti-thrombin 3 Heparin will not work with these people will actually do opposite and will lengthen PT/PTT

- These patients need anticoagulant, **Argatroban**

Trauma & Injury –

Mechanism of Injury

Newton's First Law—an object in motion will remain in motion and an object at rest will remain at rest, unless acted upon by a force.

Newton's Second Law of Motion —Force=mass x acceleration F=ma

Newton's Third Law of Motion—for every action there is an equal and opposite reaction.

Head-On Collisions

Predictable patterns

-Up and over

-Down and under

What gets hurt with each?

Predictable Injuries

-Ruptured spleens

-Lacerated livers

-Ruptured thoracic aortas

Rear-End Collisions

Predictable Injuries

-C2 fx of neck

-T12-L1 back injuries

-Femur fx

-Tib/fib fx

-Ankle fx

Evaluate for 2nd impact of vehicle and the predictable injury patterns associated.

T-Bone or Lateral Impacts

Predictable injury patterns

What gets hurt?

Don't forget secondary injuries

Patient propelled to opposite side of car

Injuries of the opposite side of body as well

Motorcycle Crashes

Head-on injury patterns

Up and over

What gets hurt?

Side impact or 'lay it down' injury patterns

-Open fx of the femur

-Pelvic fx's

-Trapped arm breaks ribs

Rollover collision

-axial loading injuries, multiple injury patterns

-this form of accident causes the most lethal injuries

Trauma Management

Airway

-secure c-spine

-Conscious ability to swallow

-Oxygenation or ventilation failure

-Expected clinical course

Breathing

-Adequate oxygenation Sp02 > 90%

-Adequate ventilation CO2 or ETCO2

Adequate mechanics

-Pneumothorax

-Flail chest and pulmonary contusion

-Hemothorax

Circulation

-Assess cardiac output

-HR more important than BP

-BP won't fall until blood loss > 30-40%

-Think tamponade

-Look for bleeding

The five places to look for blood loss

1. The chest
2. The abdomen
3. The muscles around long bones
4. The retroperitoneum
5. The floor

Secure IV access

Disability

- Quick neuro exam

-Perform GCS

-PMS x 4

-Pupils

Support cerebral perfusion

No hypoxia allowed!

No hypotension allowed!

Manage increasing ICP

-Watch for localized signs of herniation

Mildly Hyperventilate @ rate or 20-24bpm

Diuretics

Barbiturates/paralytics

Expose and evaluate

Look for occult injury

Fahrenheit

-Get temp and keep them warm

- Cold Blood will not Clot

Get a thorough set of vitals

-Trends are more important than isolated numbers

Head to toe

-Continuously reassess

Farming Accidents

1. Delayed arrival of medical care and extrication often increase mortality
2. Tractors are much heavier than autos
3. Tractors have a high center of gravity making them prone to rollovers
4. End over end rollovers have a greater potential for patient entrapment
5. High potential for HAZMAT involvement

Specific Small Vehicle Crashes

Rapid Vertical Deceleration

Primarily in adults and children under 5

Children —impact on their head

Adults—"Don Juan injury"

Projectile Penetration

- Impalements/stab wounds

-Firearms

-High Velocity bullets travel at >2000fps

Blast Injuries

Primary—initial air blast

Injuries to air containing organs

Secondary—victim being struck by projectiles from blast force

Tertiary—victim impacting the ground or another object

Burn Management

-Airway early

-Mechanism of the burn

-Carbon Monoxide poisoning

- Cyanide toxicity

Calculate Burn Area then cover with dry dressings

-Urinary output of 30-50ml/hr

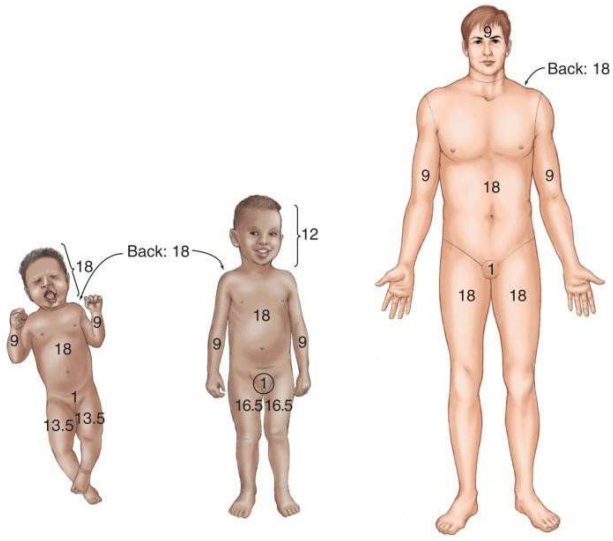
-1-2ml/kg/hr in children

Parkland formula — 4ml/kg/%BSA burned

-Give ½ over first 8 hours post burn

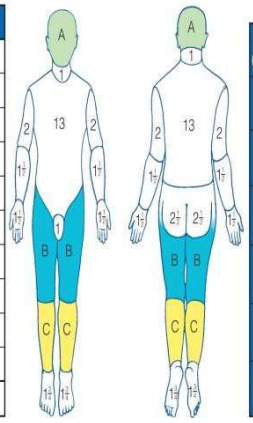
-Don't calculate 1st degree burns

Review your Rule-of-9's



Lund and Browder Chart

Region	%
Head	
Neck	
Ant. Trunk	
Post. Trunk	
Right arm	
Left arm	
Buttocks	
Genitalia	
Right leg	
Left leg	
Total burn	



Relative percentages of body surface area affected by growth

Age (years)	A ($\frac{1}{2}$ of head)	B ($\frac{1}{2}$ of one thigh)	C ($\frac{1}{2}$ of one leg)
0	$9\frac{1}{2}$	$2\frac{3}{4}$	$2\frac{1}{2}$
1	$8\frac{1}{2}$	$3\frac{1}{4}$	$2\frac{1}{2}$
5	$6\frac{1}{2}$	4	$2\frac{3}{4}$
10	$5\frac{1}{2}$	$4\frac{1}{4}$	3
15	$4\frac{1}{2}$	$4\frac{1}{2}$	$3\frac{1}{4}$
Adult	$3\frac{1}{2}$	$4\frac{3}{4}$	3

Classifications of Burns

TABLE 12-1 Classification of Burns

Depth	Color and Vascularity	Surface Appearance and Pain	Swelling, Healing, and Scarring
Superficial (first degree)	Erythematous, pink and red	No blisters Dry and tender	Slight edema Heals easily without scarring
Superficial to partial thickness (second degree)	Erythematous, bright pink/red, mottled Blanches with brisk capillary refill	Intact blisters, moist when removed Weeping wounds and extremely painful	Moderate edema Easily heals but with skin discoloration
Deep to partial thickness (second degree)	Red, waxy-white Blanches with slow capillary refill	Broken blisters, wet Sensitive to pressure, but not to light touch	Marked edema Heals slowly with hypertrophic scars
Full thickness (third degree)	White, black to red/tan No blanching, vessels thrombosed Poor distal circulation	Dry, leathery Anesthetic Hairs pull out easily	Skin grafting required Scarring likely after healing
Subdermal (fourth degree)	Charred	Obvious subcutaneous tissue involvement Anesthetic	Skin grafting and/or flap required Scarring after healing

Chemical Burns

-Most chemical burns should not be neutralized

Hydrofluoric Acid

Copious water

Alkali Metal burns (NA and K)

-React w/ water

-Absorb heat with oil

Acids

Most acids can't penetrate deep into tissue because they cause coagulative necrosis

Acids denature cellular proteins and cause cellular coagulation

Alkalis

Dissolve protein and collagen causing dehydration and saponification

Alkalis tend to cause more severe burns

HAZMAT

Two modes of EMS operations at Hazardous Material scenes

1. "Fast break"
2. "Long-term decision making"

"Two-Step" Decon Process

-Usually at a "fast break" scenario

1. Remove all clothing, shoes and jewelry
2. Wash and rinse patient with soap and water making sure they do not stand in runoff
3. Reposition out of runoff and repeat a second time

Electrical Injuries

-Myoglobinuria is often a problem due to massive muscle damage

-If untreated, will result in Acute Tubular Necrosis and renal failure

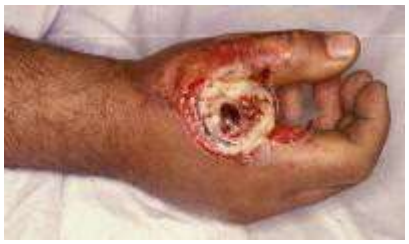
-Maintain urine output of at least 100 ml/hr with fluids

Osmotic diuretics / Loop diuretics

- Furosemide

-Mannitol

Urine alkalinization increases the rate of myoglobin clearance and can be achieved using sodium bicarbonate titrated to a serum pH of 7.5.



START Triage

When your mind starts racing…remember your

RPM's.

START Triage Assessment Parameters

-A form of primary triage focused on three assessment parameters and three levels of triage

Assessment

-Respirations

-Perfusion

-Mentation (LOC)

A couple of other quick rules

1. 15-30 second assessment per patient
2. If patient is apneic, reposition head once
3. Check capillary refill centrally

All penetrating trunk trauma is Immediate

All rescuers injured on scene are Immediate

Walking Wounded = Green Tag = Priority 3

Minor wounds = Yellow Tag = Priority2

Severe injury = Red Tag = Priority 1

Dead = Black Tag = Priority 0

Hematology

Blood Composition

Water exists in the body in 3 areas

-Intracellular – 75%

-Interstitial – 17%

-Intravascular – 8%

Fluid Replacement

3 liters of crystalloid to one liter of blood loss

Initial resuscitation should include an isotonic crystalloid

After crystalloids other options include hypertonic saline, albumin, or dextran.

Maintain MAP 80-90 mmHg with fluid

The kidneys are the poor man's swan ganz- use them to guide fluid replacement

The clotting cascade in response to insult the body

-Vasospasms

-Activates Platelets

-Initiates the Clotting Cascade

-Forms a clot

-Breaks the clot down

Disseminated Intravascular Coagulopathies (DIC)

-Over stimulation of the clotting cascade secondary to massive tissue damage

Treatment options

-Blood component replacement- controversial due to the possibility of perpetuating the abnormal DIC feedback loop

-Heparin- inhibits the conversion of prothrombin to thrombin, slowing the coagulation cascade

-Anti-thrombin III- Prevents the conversion of multiple clotting factors to their active form

Blood Products -

Packed Red Blood Cells-

-Given to increase O2 carrying capacity of blood

-Given over 2-4 hours normally, can be given fast emergently

-ABO antigens are present on RBCs, watch for hemolytic reactions

-Rh factor should be considered

-One unit of PRBCs equals approximately 330cc

-Peds dosing 10cc/kg

-Watch for citrate toxicity with multiple units

-One unit of PRBCs should increase the H/H by 1 and 3

Fresh Frozen Plasma

-Given as a volume expander or for coagulation factors

-Watch for transfusion reactions secondary to viral or antigen responses

-Dosing for coagulation is driven by coags (Pt/Ptt)

Cryoprecipitate NOTES

-A mixed blood product obtained by separating specific clotting factors (Factor VIII:C, Factor XIII, von Willebrand factor, and fibrinogen) from FFP

-The end result is a concentrated fluid with high levels of fibrinogen and three other primary factors

-Given in DIC, Hemophilia A, Von Willebrand's Disease

-Administered at 10 ml min

-Normal adult dosing is 10 units

-Most often given emergently for massive PRBC infusions or TPA related bleeding

Platelets

-Given for platelet deficiency

-Usually not given till platelet count falls to <20K

-Usually given as a six pack

-Should anticipate one unit will increase the platelet count by 12k

-Can be pushed quickly by syringe

Endocrine Emergencies

SIADH – syndrome of inappropriate anti-diuretic hormone

-Dilusional Hyponatremia

Hypothalamus controlled, ADH stored in the posterior pituitary gland

Retention of H2O- fluid overload, CHF

NORMAL SERUM OSMOLARITY 275 – 295
approximately double the normal Sodium level

Causes:

1. Oat Cell Carcinoma
2. Viral Pneumonia
3. Head problems – trauma, tumors, meningitis, strokes, encephalitis

ADH works in the ***distal convoluted tubules***

Need to watch for *Seizure Activity* due to cerebral edema

Hyponatremia = Sodium < 135

Serum Osmolarity = < 270

Treatment:

1. Fluid restriction
2. Hypertonic Solution – 3% NaCl 25cc/hr

Hypotonic solutions causes water to inter the cells because of change in Osmotic pressure

Normal Saline = 0.9% NaCl – any fluid below this is considered Hypotonic

D5W = Osmolarity of 252

- Isotonic in container, however, after administration glucose is rapidly metabolized, leaving only water behind, thus becoming a hypotonic solution. This will cause movement of H2O into cells leading to increased cerebral edema.

Diabetes Insipidus (DI)

Opposite of SIADH- LOW ADH

Loss of up to 6 – 24 liters/day

Labs –

Increased Sodium
Increased Serum Osmolarity > 295
Increased urinary output (Clear)
Specific gravity of 1.001 – 1.005

Causes:

1. Head Problems
2. Dilantin

Resulting in:

1. Hypovolemia
2. Shock

Treatment:

1. Give ADH – IV, IM, SQ, Nasal Spray
2. Monitor Specific Gravity of urine
3. Monitor EKG for Cardiac Ischemia

HHNK

Hyperglycemic Hyperosmolar Nonketotic Coma

These people still have insulin!

Who gets HHNK?

1. Old People – tired pancreas
2. Diet controlled Diabetes – usually Type II
3. TPN – total parenteral nutrition
4. Pancreatitis – 95% mortality rate

Other causes:
Overuse of thiozides – they hold on to glucose
Overuse of Steroids – they lead to insulin resistance

- No acidosis as a result of still having insulin

- Blood Sugar ranges of 1000 – 2000

-Higher levels of BS due to no development of Ketones, no acidosis, long for symptoms to present themselves.

- Severe Dehydration – 8 – 10 liters/day

Treatment:

1. Fluids
   -NS, ½ NS, and D5 ½ NS
2. Insulin
3. Glucose

Always make sure you check a Sugar!!

DKA

These people have no insulin!

Dehydrated 4 – 6 liters behind

Glucose level = 400 – 900 usually reason why level is not as high as HHNK is due to ketoacidosis an patient becoming unconscious, going into coma after around 5 days

State of Acidosis – the body breaks down fatty acids for energy, by product of breakdown is Ketones, which leads to Acidosis.

Ketones cause Fruity Breath

KUSSMAUL Breathing – Increase Rate, Increase Depth, designed to blow off excess CO2

On cellular level, body plays "Hide the Hydrogen" H+ ions leave serum and hide in the cells; this pushes K+ out of the cell, leading to a high Serum K+ level

DKA patients – you want to see High K+ levels
- Low K+ levels your patient is going to die!

Potassium and Hydrogen have a reciprocal relationship

For every change of .1 of H+ ions = change of K+ ions of .6 in the opposite direction

PH	**K+**
7.45	4.5
7.35	5.1
7.15	6.3
7.05	6.9

Treatment:

- Fluids
  - NS, ½ NS, D5 ½ NS
- Insulin

Correct Sugar and Acidosis should correct itself

Acidosis always increases Serum K+ level

DKA – Most common cause of death is Hypokalemia

Insulin - Under certain circumstances, injection of insulin can kill patients because of its ability to acutely suppress plasma potassium concentrations.

NOTES

Pancreatitis

Not an infection of pancreas – it is auto digestion of pancreas

Causes:

1. Obstruction of Pancreatic ducts – Most common!
2. ETOH abuse
3. Biliary stone disease
4. Steroids, antibiotic, thiazide use
5. Infections (viral/bacterial)

Presentation

1. **Hypocalcemia** -Pancreas needs Ca to auto digest itself

2. **HHNK**
   - Pancreas is too busy eating itself, it cannot produce insulin

3. Elevation of the left diaphragm w/ **Left base atelectasis**

4. **Left pleural effusions**

5. **Bilateral Rales**

6. Concomitant **ARDS**
   -Phospholipase A travels through blood to lungs and kills the Type 2 alveoli cells that make surfactant

7. **Renal failure**

8. **Cullen's sign** – Black and Blue bleeding around umbilicus

Treatment

1. –Fluid resuscitation
2. –NPO and place OG/NG
3. –Meperidine over MS - due to Sphincter of Odi spasticity
4. –Antibiotics for sepsis - Rocephin, Ampicillin

Liver

Functions:

1. Detoxify blood – 1500 cc/min
2. Make Bile
3. Synthesis amino acids
4. Makes albumin, prothrombin, fibrinogen
5. Converts glucose into glycogen
6. Converts ammonia into urea

*Decrease in albumin production leads to **ASCITES***

*Increase in ammonia levels leads to **Hepatic Encephalopathy***

LIVER Disease

- Never want a decrease in K+ level – the kidneys will retain K+ and also ammonia ions

- Must utilize K+ sparring diuretics

-Portal Hypertension results in esophageal varices and gastrointestinal bleeding

Renal

Anatomy

1. Proximal Tubular
2. Loop of Henley
3. Convoluted Tubule

Proximal Tubular –

Located in the nephron

Reabsorbs everything

Electrolytes

Loop of Henley –

Responsible for the concentration and dilution of urine

Convoluted and collecting tubules –

ADH affects this area and causes retention of H2O

Acute Renal Failure

Decrease in urinary output < 400 cc/24 hr period

3 Stages

1. ***Pre renal***
   - Decrease in urine output < 400cc/24hr period
   - Causes: decrease blood flow to kidneys
   - CHF; Hypotension
2. ***Renal***
   -Decrease in urinary output < 400cc/24hr period
   - Causes: Kidney damage
   - Parenchyma; Nephrons
3. ***Post Renal***
   - Urethral obstruction
   -Bladder Obstruction

ATN – Acute Tubular Necrosis

For every 100 people that come into ED with ATN, 50 out of them will drop dead. Out of the remaining 50, 25 of them will have dialysis for the rest of their life, and the remaining 25 will survive

3 stages of ATN

Oliguric Stage- 10 – 17 days

No urine output
Increased BUN
Increased Creatinine
Increased K+
fluid overload

Polyuric Stage – 2 weeks – 3 Months

Begin to make urine
Good Quantity
Bad Quality

Increased BUN
Increased Creatinine

Decreased K+ due to fluid depletion

Recovery Stage – 3 Months – 1 year

Hope that kidneys regenerate themselves during this stage

Pre-renal vs. Renal Stage

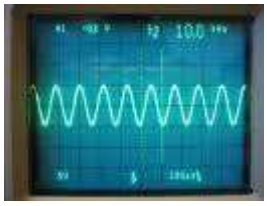
Pre-renal	Renal Stage
Urinary Na+ < 20	Urinary Na+ 40 – 100
hold on to H2O	Hold on to H2O
Hold on to Na+	Nephron leak Na+
BUN/Creatinine Ratio 20/1	**BUN/Creatinine Ratio** 10/1
Fluids/Lasix given Increase in urine	**Fluids/ Lasix given** No change in urine output

Electrolytes

Potassium

Hyperkalemia – increase in K+

Signs	K+
Peaked "T" Wave	6.5 – 6.8
Prolonged PR interval	7
Lose of "P" wave	>7
Widening QRS	8
Sin Wave	DEATH



Treatment:

1. ***Calcium Gluconate*** – works in 1-3 mins
-Only give if you have EKG changes
-Does not lower K+ level

Allows electricity to go through heart muscle normally

2. ***Insulin*** – IVP 10-15 units with 50cc of D50

- pushes K+ into the cells [temporary]

3. ***Glucose*** – Do not want patient to become more hypoglycemic

4. ***Bicarbonate*** – beneficial in cases of metabolic acidosis

- 1 amp given over 5 minutes

- will stimulate an exchange of cellular H^+ for Na^+, thus leading to stimulation of the sodium-potassium ATPase

5. ***Kaexylate*** – Binds with K+ ions and causes excretion decreasing potassium

Calcium

Calcium and phosphate exist in a reciprocal relationship

Patients with Kidney disease always have a decrease in Ca++

- Kidneys make Vitamin D

- We utilize Vitamin D to reabsorb Ca++ from the Gut

- No Vitamin D = No reabsorbed Ca++

Hypocalcaemia = Hyperphosphatemia

Manifestations of Hypocalcaemia and Hyperphosphatemia

- Chvostek's signs
- Trousseau Signs

Treatment of Hyperphosphatemia

Amphojel - binds with phosphate and decreases levels

"Twitch, Twitch; Seize, Seize; Chvostek's and Trousseau"

Weight gain of 1 pound within 24 hrs indicate fluid retention of 1000cc

Toxicology

Arrhythmias secondary to Cardiotoxins / Electrolyte Disturbances

Tricyclic Antidepressants (TCA's)

Due to the location of norepinephrine receptors all over the body, many physical signs are also associated with a TCA overdose[1]:

1. Anticholinergic effects: altered mental status (e.g., agitation, confusion, lethargy, etc.), resting sinus tachycardia, dry mouth, mydriasis (pupil dilation), fever
2. Cardiac effects: hypertension (early and transient, should not be treated), tachycardia, orthostasis and hypotension, arrhythmias (including ventricular tachycardia and ventricular fibrillation, most serious consequence)/ECG changes (prolonged QRS, QT and PR intervals)
3. CNS effects: syncope, seizure, coma, myoclonus, hyperreflexia
4. Pulmonary effects: hypoventilation resulting from CNS depression
5. Gastrointestinal effects: decreased or absent bowel sounds

Treatment:

NaHCO3

Beta blockers Overdose (Propanolol, Atenolol, Inderal, Metoprolol)

Bradycardia w/ resultant hypotension

Look for escape rhythms - Junctional ; Ventricular

-Look for respiratory compromise as well

Treatment:

Atropine rule out/parasympathetic tone prophylaxis

TCP ASAP

Glucagon 1mg IVP

Dopamine prn for hypotension

Isuprel gtt

Ca+ channel blockers (Verapamil, Nifedipine, Diltiazem)

-Look for severe Bradycardia & AV dissociation

-Look for concomitant hypotension

Treatment:

Calcium chloride/gluconate

TCP

Digitalis toxicity (Digoxin, Digitoxin, Foxglove, Oleander)

Commonly see Bradycardia, SVT, VT, blocks

Treatment:

-Digibind

-Assure normal electrolytes

-TCP

Lidocaine, Magnesium & Dilantin for tachyarrthmias

-Caution with electricity

Hypokalemia (<3.5)

-Loop diuretic misuse/OD

-Serum potassium important as well as pH

-ECG: Peaked P's, Flattened/slurred T's, and appearance of U's

Treatment:

-Potassium Chloride, Potassium Phosphate

Hyperkalemia (>5.0)

-Profound acidosis (DKA, Vent mismanagement, etc...)

-Potassium supplement OD

-ECG: Flattening/slurring of P waves and Peaked T waves

Acetaminophen Poisoning

Stage 1 -

-Occurs within 30 minutes to 24 hours

-Nausea, vomiting & anorexia

-Malaise

-Pallor

-Diaphoresis

Stage 2 -

-24-48 hours

-Right upper quadrant pain/tenderness

-Increased liver enzymes

-Increased serum bilirubin

-Increased prothrombin time (PT)

-Oliguria as result of ATN

Stage 3 -

-72-96 hours

-Peak for liver function abnormalities

-Return of anorexia, nausea, vomiting & malaise

-Jaundice becomes apparent

-Hepatic encephalopathy

-DIC

-Death due to fulminate hepatic necrosis

Stage 4 -

-4 days to two weeks

-Resolution period

-Liver functions return to normal baseline values, patients become asymptomatic

Acetaminophen Poisoning Treatment

-Ingestion of 7.5g or 150 mg/kg is typically toxic

-Measure serum levels 4 hour after ingestion

1. GI decontamination

2. Administer N-acetylcysteine (NAC) (Mucomyst) orally 140 mg/kg

3. Intravenous (IV) NAC (Acetadote)

Aspirin (ASA) Poisoning

- Depressed DTR's

-Stupor/coma/convulsions

-Myoclonic jerks

-Hypothermia/low grade fever

-Profound Hypocalcemia

Aspirin (ASA) Poisoning Treatment:

1. Activated charcoal
2. Intravenous dextrose and normal saline
3. Sodium bicarbonate
4. Dialysis

Benzodiazepines

-Flumazenil—administer with caution

Narcotics

-Narcan—titrate to effect

TOXIN ANTIDOTE

Carbon Monoxide	Oxygen
Cyanide	Amyl nitrate, Na thiosulfate
Organophosphates	Atropine, 2-pam
Methemoglobinemia	Methylene Blue
Anticholinergic	Physostigmine
Coumadin	Vitamin K, FFP
Heparin	Protamine sulfate
ß & Ca++ Blockers	Glucagon, Calcium

Snakebites

Signs and symptoms

-Localized pain, swelling & edema

-Diaphoresis

-Chills

-Hypotension

-Muscle fasciculation

-Coagulopathies

-Local ecchymosis

-Faintness/weakness

Treatment:

1. Clean and immobilize

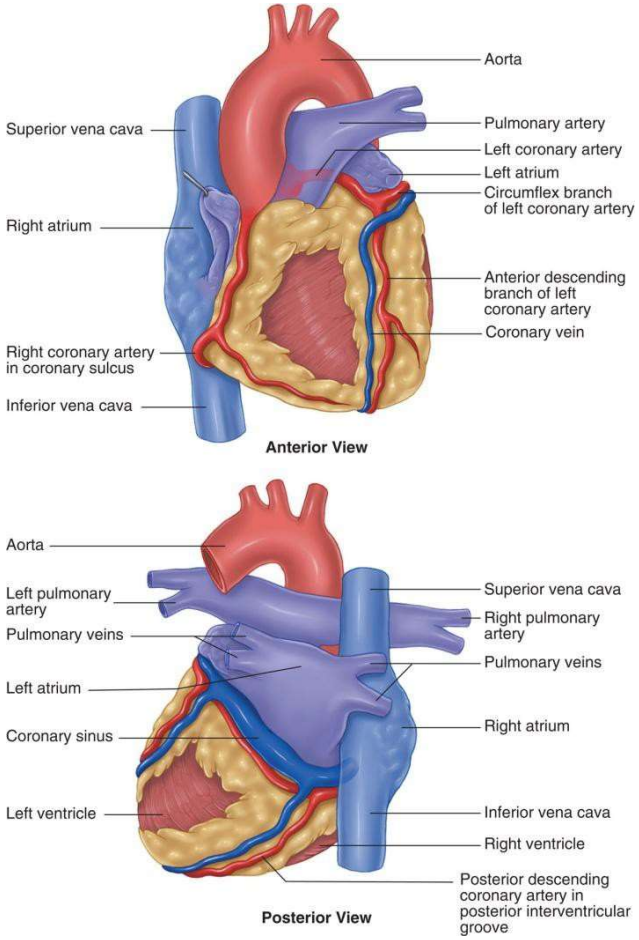
2. Airway, ventilatory & circulatory support IV access

3. Monitor for systemic reaction

4. Severe intoxication—Antivenin (monitor for serum sickness)

Cardiac

Anatomy and Physiology



Pericardium — the double walled fibrous sac surrounding the heart

Heart — contains three layers: epicardium, myocardium & endocardium.

Epicardium—thin, outermost layer

Myocardium—thick, muscular middle layer

Endocardium—thin inner layer

Heart chambers — four chambers divided by septums and valves — two types: atrioventricular and semilunar valves.

AV valves — located between atria and ventricles Open as a result of lower ventricular pressures and close as a result of increased ventricular pressures.

Tricuspid valve — located between the right atrium and right ventricle, three cusps.

Mitral valve — located between the left atria and left ventricle, two cusps.

Semilunar valves — located between the ventricles and the great arteries.

Pulmonic valve — separates the right ventricle from the pulmonary artery.

Aortic valve — separates the left ventricle from the aorta.

Coronary Circulation

Circulation consists of right and left coronary arteries that arise from the coronary Ostia at the aortic root.

Left coronary artery (LCA) bifurcates into the LAD and LCX.

LAD supplies the anterior surface of the heart, the anterior two-thirds of the septum, and part of the lateral wall.

LCX primarily supplies the lateral wall of the left ventricle.

RCA becomes the PDA

These supply the right atrium, right ventricle as well as the inferior and posterior walls of the left ventricle.

Overview of blood flow through the heart

Right atrium—tricuspid valve—right ventricle—pulmonic valve—pulmonary artery—lungs—pulmonary vein—left atrium—mitral valve—left ventricle—aortic valve—aorta—systemic circulation—vena cava— right atrium

Blood flow through the heart.

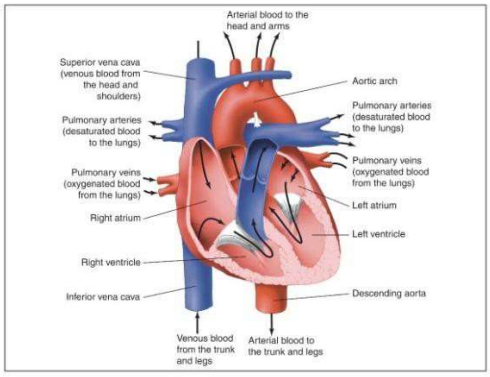


Figure 13.05

Valve order T-P-M-A

Types of Infarctions

Infarcts are divided into Transmural and non-Transmural, or subendocardial MI's.

Transmural — extends through the full thickness of the myocardium and includes the endocardium and epicardium.

A. If the Q wave is more than one third the total height of the QRS, it is a pathological Q. B. If the Q wave is more than 30 milliseconds wide, it is a pathological Q.

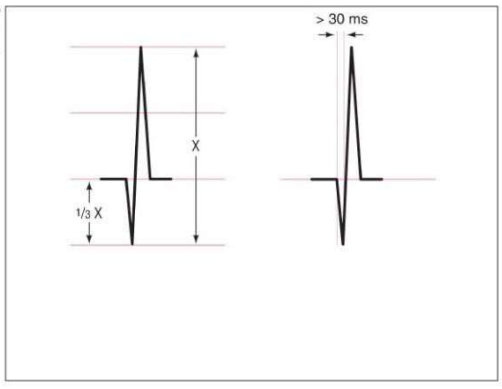


Figure 13.54

Q Waves Formation

Greater risk of complications due to loss of functional myocardium

Subendocardial — necrosis is limited to the subendocardial surface.

The relationship between leads and views of the heart.

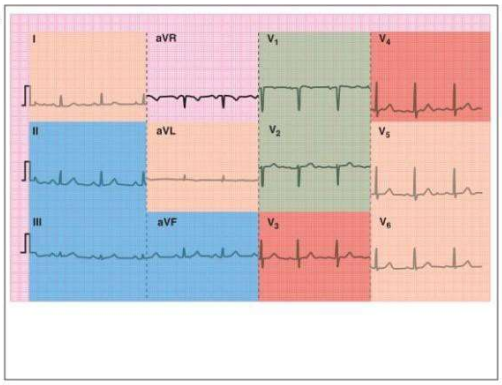


Figure 13.16

ACS Classifications

Unstable angina -Diagnosed positive cardiac markers and negative 12-lead changes

Non-STE MI -Diagnosed positive cardiac markers and ST depression or dynamic T wave changes

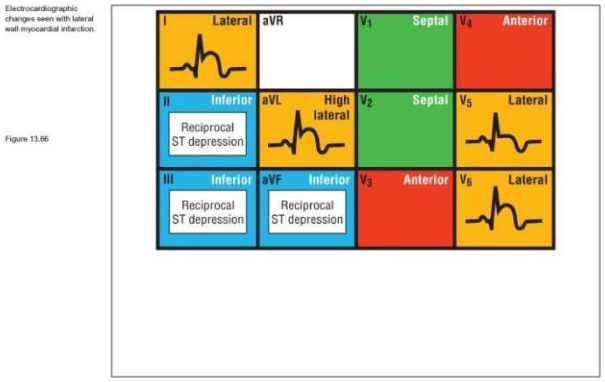
STE MI -Diagnosed positive cardiac markers and identified STE pattern on 12-lead

Cardiac Enzyme Studies

Critical Care Review

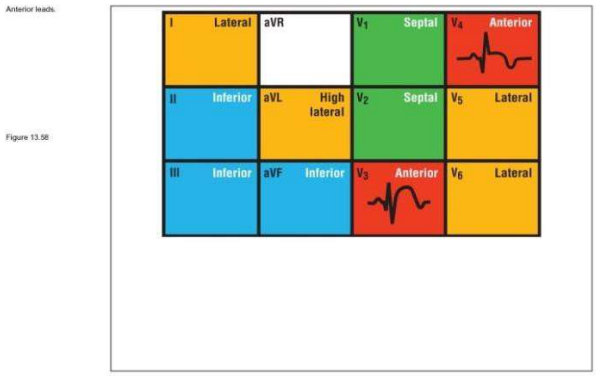
Types and Locations of Infarcts

TABLE 13-8 Reciprocal Leads and Their Corresponding Locations		
Location	**Facing Leads**	**Reciprocal Leads**
Anterior	V_3, V_4	None
Inferior	II, III, aVF	I, aVL
Lateral	I, aVL, V_5, V_6	II, III, aVF
Septum	V_1, V_2	None
Posterior	None	V_1, V_2



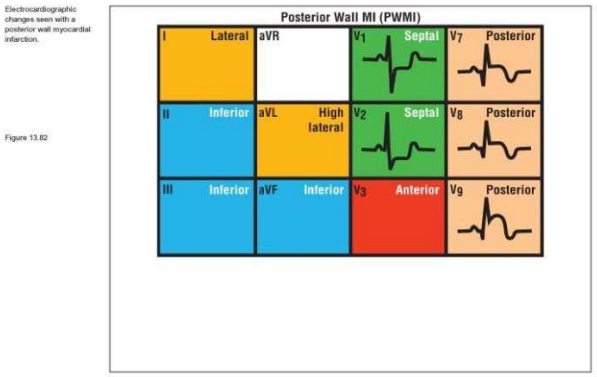
Lateral MI

Obstruction of the LCX



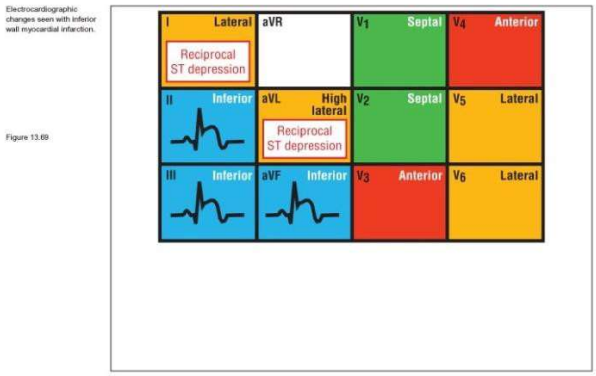
Anterior MI

Obstruction of LAD Infarction of the regions of LV, parts of septum and papillary muscles.



Posterior MI

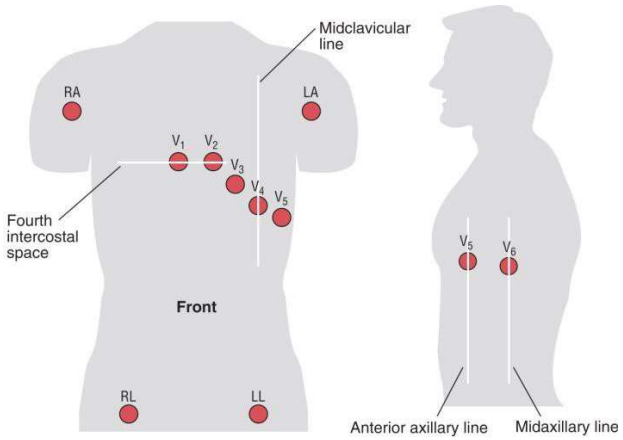
Obstruction of the LCX or the RCA



Inferior MI

Obstruction of the RCA, also infarcts the papillary muscles

What exactly are we looking for on the 12-Lead?



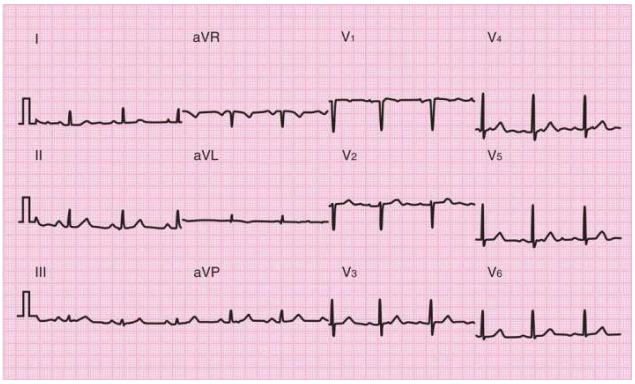


TABLE 13-5 Possible Causes of Axis Deviation	
Axis Deviation	**Possible Cause**
Right	RVH LPH Chronic obstructive pulmonary disease Dextrocardia Ectopic beats and rhythms Normal in children
Left	LAH LVH IWMI Ectopic beats and rhythms Obesity Pregnancy

Abbreviations: IWMI, inferior wall myocardial infarction; LAH, left anterior hemiblock; LPH, left posterior hemiblock; LVH, left ventricular hemiblock; RVH, right ventricular hypertrophy.

First –ST Elevation

1mm in two anatomically contiguous leads

Second –Q waves

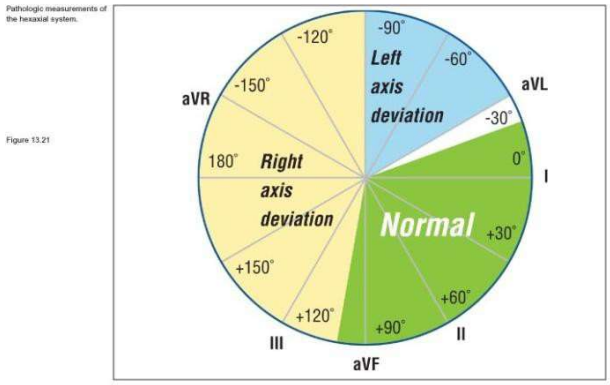
"True" Q's

Pathological vs. physiological

Third –ST Depression

Reciprocal finding from elevated leads

Axis Deviation



Angina

Stable -Onset with physical exertion or emotional stress, Pain lasts 1-5 minutes and is relieved by rest, predictable.

Unstable -Stable angina that has changed in frequency, quality, duration or intensity, pain lasts longer than 10 minutes despite rest and NTG therapy.

Variant-Spontaneous episodes of CP frequently noted at rest or on early rising, circadian pattern and relieved by NTG.

Silent-Objective evidence of ischemia in asymptomatic patients

Mixed-Combination of stable and variant angina

Treat angina

Nitrates, Beta Blockers, ASA, and MSO4

Inhibit Clotting

G2B3A Inhibitors, Heparin

Management and Treatment of AMI/USA

Pre-load reduction

Nitrates-improve coronary blood flow through smooth muscle relaxation, venous pooling decreases cardiac output and O2 consumption

NTG dosage 5-200 mcg/min

Morphine-vasodilator that decreases preload and afterload, also decreases sympathetic tone causing a decrease in heart rate and reduces O2 demand. Dosage is 2-4 mg every 5-15 minutes.

CPP = DBP - PCWP

Heart rate & myocardial O2 demand reduction B-blockers-decrease heart rate and contractility, and increases diastolic filling time.

Calcium Channel Blockers

Produces dilation of the coronary arteries and collateral vessels, a decrease in myocardial contractility and

Clot prevention and "busting"

ASA/G2B3A Inhibitors

Inhibit platelet aggregation (prevention)

Heparin/LMWH

Inactivates thrombin and factors

IX, X, XI and XII and prevents the conversion of fibrinogen to fibrin. (Prevention)

Fibrinolytic-work by various mechanisms to activate plasminogen to plasmin, resulting in fibrin degradation ("Busting")

Thrombolytics

Indications

New onset ST segment elevation MI

Complications

Bleeding, intracranial hemorrhage, dysrhythmias, cardiac tamponade, & pulmonary edema

Relative contraindications

HTN, recent trauma, pregnancy

Absolute contraindications

Active internal bleeding, suspected aortic dissection, known intracranial neoplasm and previous hemorrhagic stroke at any time or any stroke within the last year.

Angioplasty

Evidence suggests Equal mortality for patients who receive thrombolytics within 30 minutes and patient who undergo angioplasty within 90 minutes

Post procedure

-watch for re-occlusion

-Continuous lead specific EKG monitoring

-Maintain sheath and vessel patency

-Distal Pulses and occult bleeding

Sheath removal

-maintain direct pressure for 30 minutes

-Leg straight, HOB < 30 Degrees

Cardiovascular Disease and Anomalies

TABLE 13-7 Common Toxic Drug Effects

Drug	Possible Toxic Effects
Class I antiarrhythmics	Lengthened QRS and QTc intervals Possible AV blocks Slowed or completely blocked SA node Arrhythmias
Calcium channel blockers	Blocked AV node primarily, but extent of block varies significantly among different drugs in this class
Beta-blockers	Slowed automaticity of the SA node and the Purkinje system Blocked AV node
Amiodarone (Cordarone)	Slowed conduction everywhere: the SA node, atrium, AV node, Purkinje system, and ventricles
Phenothiazines and tricyclic antidepressants	Widened QRS and QTc interval T-wave abnormalities Arrhythmias common in overdoses

Abbreviations: AV, atrioventricular; SA, sinoatrial.

Source: Garcia T, Holtz N. Figure 16-3. In: *12-Lead ECG: The Art of Interpretation*. Sudbury, MA: Jones and Bartlett Publishers; 2001:507.

Dysrhythmias — treat if symptomatic.

Symptomatic Bradycardia, second degree AV blocks Mobitz II, high grade AV block and complete heart block require placement of a pacemaker.

A commonly used monitor-defibrillator.

Figure 13.101

Courtesy of Physio-Control, Inc.

Complications of pacing

-Failure to sense

-Failure to capture

Cardiomyopathy

Classified as 3 Stages

Dilated — ventricular dilation & contractile dysfunction

Managed with cardiac glycosides, inotropes and diuretics

Hypertrophic — (IHHS) Idiopathic Hypertrophic Sub aortic Stenosis

-Inappropriate ventricular hypertrophy-Big fat ventricular septum hangs out into L ventricle

IHSS – limits blood volume entering L Ventricle to 30cc

Reduction if CO during Systole

Managed with B-blockers – allow heart to stay in Diastole longer, decreasing contractility

-No Nitrates
-No Digoxin, Dobutamine, Dopamine

Restrictive — myocardial scarring of ventricle with impaired diastolic filling.

-Managed with diuretics, anticoagulants,

Aortic Dissection

An intimal tear in the aorta, three types:

Type I — occurs in the ascending aorta and extends distally beyond the aortic arch.

Type II — process is limited to the ascending aorta. (Aortic valve incompetence & Marfan's)

Type III — dissection distal to the origin of the LSC artery & extends distally to abdominal aorta

Management

-Lower SBP to 100-110 mmHg with vasodilators

-Beta blockers to slow the heart rate and decrease the ejection fraction

-Pain relief

Normal Assorted Lab Values

HTN crisis

3 Classifications

1. Essential Hypertension
2. Accelerated Hypertension
3. Malignant Hypertension

LOOK AT DIASTOLIC PRESSURE!

> 120mmHg	Accelerated
> 140mmHg	Malignant
242/142	**Malignant Hypertension**
230/130	**Accelerated Hypertension**

Management includes the use of Nipride and Hyper stat to lower the BP to patient's normal within 30-60 minutes

Adult Lab Values

Normal Parameters for Electrolytes

Sodium	Na+	135-145 mEq/L
Potassium	K+	3.5-5.5 mEq/L
Calcium	Ca++	8.8-10.4 mg/dL
Magnesium	Mg+	1.5-2.5 mEq/L
Chloride	CL-	95-105 mEq/L
Phosphorus		3.0-4.5 mg/dL
Bicarbonate	HCO3	21-28 mEq/L
Blood Urea Nitrogen	BUN	6-23 mg/dL
Creatinine		0.6-1.4 mg/dL
Glucose		70-110 mg/dL
Serum Osmolality		275-295 mOsm/kg water
Carbon Dioxide	CO2	24-30 mEq/L
Anion Gap		12 +/- 4

CBC

Hemoglobin	12-18 g/dL
Hematocrit	36-52%
Platelets	140-400 x 109/L
WBCs	4,500-10,500 cells/mm3
Bands	0-3%
PT	11-13 Seconds
INR	1
APTT	21-35 Seconds
Fibrinogen	200-400mg/dL
Fibrin Split Products	< 10mg/L
D-Dimer	<250mcg/L
BNP	<100pg/mL
Lactate	<2.2mEq/L
Troponin I	<0.5 ng/mL
CK Total	0-120 ng/mL
CK MB	0-3 ng/mL

Neurological Emergencies

Cerebral Perfusion Pressure (CPP)
Normally ICP = 0-10 mmHg
Transducer must be located at Foramen of Monro
CPP = 70-90 mmHg CPP=MAP-ICP
Very important to maintain MAP when ICP is increased
Normal MAP = 80-100 mmHg

Neuro Anatomy
Pia mater – soft inner covering, adheres to the brain's surface
Dura Mater – tough outer covering Epidural Space – potential space between skull and dura
Subdural Space – potential space between the dura and the arachnoid

Signs of Increased ICP

- Change in LOC

- Change if pupil size and reaction

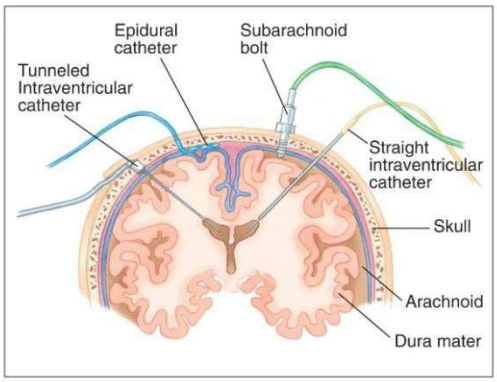
- Abnormal motor response

Decorticate posturing-flexion of Upper Extremities towards the CORE

Decerebrate posturing-extension and hyperpronation of Upper Extremities

Cushing's Reflex –increased systolic pressure resulting in:

1. Widened pulse pressure
2. Bradycardia
3. Respiratory changes (Cheyne Stokes)



Treatment:

1. Positioning of patient
2. 15 – 30 degrees reverse trendelenburg
3. Limit suctioning procedures
   -Noise
   -Atmospheric pressure changes
4. Maintain euvolemia and normothermia
5. Maintain normal electrolytes
6. Sedation – narcotics, bezo's, propofol, NMBA

Subdural Hematoma

Blood between dura and arachnoid layers

Usually venous in nature

High morbidity and mortality rate

3 Types:

Acute – onset within 24 hrs

Subacute – onset within 2 -10 days

Chronic – onset after 2 weeks

Elderly – larger subdurals with slowly developing sysmptoms due to crebral atrophy

Younger person – rapid onset of sysmptoms with marked increased ICP

Pediatrics – generally occur in children under the age of 18 months

-Bulging fontanelles

-Separation of sutures

-Shock

- Retinal Hemorrhages

Treatment approach to herniaton

1. Intubate and mildly hyperventilate to a PCO2 of 28 – 32
2. Assure adequateHgb and cardiac output
3. Adequate Fluid resuscitation, then Mannitol, Hypertonic Solution
4. Maintain serum sodium 155
5. Serum Osmolarity less than 320
6. Barbituate therapy

Mannitol –osmotic diuretic thought to work by decreasing the viscosity of blood and improving microcirculation (0.5 – 1.5 gms/kg)

Lasix – loop diuretic and alternative to Mannitol

Vasopressors to maintain MAP and blood pressure

Epidural Hematoma

Bleeding between the skull and the dura mater

Usually arterial, but can be venous

Laceration of Middle meningeal artery in the temporal lobe area

Classic signs – transient loss of consciousness followed by a period of lucidity

Uncal herniation – will result in dilation of the ipsilateral pupil with contralateral Neuro deficits/posturing – brain shifts laterally

Acute – usually an arterial bleed, onset of symptoms in a few hours

Subacute – usually a venous bleed with onset taking longer

Subarachnoid Hemorrhage

Bleeding between the arachnoid mater and the pia mater

Trauma is the most common cause

May occur with other injuries or as the only evidence of trauma

Signs/Symptoms

1. Severe headache
2. Vomiting, stiff neck, leg pain
3. Confusion, lethargy
4. Loss of consciousness

Avoid lumbar punctures due to the possibility of Uncal herniation (Uncus of the tentorium)

Intracerebral Hematoma

Hemorrhage in the brain parenchyma

Produced from shearing and tensile forces

Frequently occurs in the white matter of the frontal an temporal regions

May be single of multiple and is associated with contusions, SDH and DAI

Less common injury with delayed onset of symptoms (24 hours or more after injury)

Intraventricular Hemorrhage

Bleeding into the ventricles as a result of severe brain trauma

Result of shearing forces

Greatly increased mortality rate

Usually found in frontal or temporal lobes

Cerebrovascular Accident (CVA)

3 Classifications of stroke

1. **Embolic** – most common
2. **Hemorrhagic**-bleeding into the brain tissue
3. **Thrombotic** – least common

Treatment:

-Prevent additional insult

-Maximize CPP

-Control increased ICP and associated conditions

-Thrombolytic therapy within 3 hours of neurologic deterioration onset

Skull Fx

Linear – a line that extends toward the base of the skull

Linear Stellate – Multiple fractures that radiate from the compressed area

Diastatic – involves a separation of the bones at a suture line of a marked separation of bone fragments

Depressed Skull – May be closed or open

Basilar Skull – fracture of the base of the cranial vault

Battle sign – bruising behind the ear on the mastoid bone

Periorbital ecchymosis (Raccoon Eyes) – bruising around the eyes

Otorrhea – bleeding from the ear with CSF leak

Rhinorrhea – bleeding from the nose with CSF leak

Complications of Skull Fractures

-Intracranial infections

-Hematomas

-Meningeal and brain tissue damage

-Pneumocephalus

-Nerve damage and palsies

Spinal Cord Syndromes

Brown-Sequard – hemisection of the cord (usually cervical region) very rare, ipsilateral loss of motor, position, and vibratory sense with contralateral loss of pain of temperature perception

Central Cord Syndrome – greater motor weakness in upper extremities than lower with varying degrees of sensory loss (children shopping analogy)

Anterior Cord Syndrome – complete motor, pain, temperature loss below the lesion with sparing proprioception, vibration, and touch

Complications

Be aware of SCIWORA

Neurogenic shock-areflexia with flaccid paralysis immediately or shortly after injury

Transection of the cord will result in parasympathetic dominance below the lesion

Classic Signs of Neurogenic Shock – includes hypotension, warm red skin, and absence of tachycardia (Bradycardia)

Autonomic Dysreflexia or Neurogenic Bladder – Potential life threatening HTN caused by transection of spinal cord usually above T6 – check Foley for kinks and drain slowly

Pediatric-Neonatal Emergencies

Anatomical Differences

1. Rib cage is more elastic and flexible
2. Lung tissue is more fragile
3. Mediastinum is more mobile
4. Bones of the skull are soft and separated by cartilage until the age of 5
5. Liver and Spleen are proportionately larger and more vascular
6. Bones are softer
7. Fontanelles-anterior closes at 12-18 months, posterior closes by 2 months

Physiological Differences

- Children have higher cardiac output per kg of body weight than adults

- Heart rate is increased and stroke volume is smaller

- Children with trauma will not demonstrate hypotension until acute blood loss totals 25% of the circulating volume

- Circulating blood volume in children is 75-80 mL/kg

- Fluid resuscitation= 20cc/kg

- Glucose needs = 2cc/kg D25

- Larger ratio of surface area to volume

Respiratory – Airway differences

Trachea is narrow with elastic rings

Tongue large in comparison to mouth

Epiglottis is short, narrow and angled away from the long axis of the trachea

Larynx is higher and more anterior

The vocal cords have a lower attachment

< 10 years old, narrowest portion of airway is at the cricoid cartilage

Oxygen consumption in infants is 6-8ml/kg compared to 3-4ml/kg in adults

Pediatric Assessment

1. *LOC* – activity and responsiveness
2. *Perfusion Status* – Color; distal pulses
3. *Work of Breathing (WOB)* – Retractions; Rate/Depth

Resuscitation

Airway, Airway, Airway!

ETT size (age/4) + 4

(Age+16)/4

Depth of insertion (age/2) +12

IV access

Cardioversion: 0.5-1.0 j/kg “sync”

Defibrillation: 2j/kg then 4j/kg

Cerebral resuscitation

Common Injuries

Solid Organs

- Brain, lung, liver-spleen, pancreas, kidney

Hollow Organs

- Proximal jejunum, duodenum, bladder

Fractures of long bones

Waddell's Triad: common injuries when a child is struck by a motor vehicle; injuries include those to the head, trunk, and extremities

The single largest cause of traumatic deaths is motor vehicle related accidents

Orthopedic Injuries

Comminuted – Bone is broken into fragments

Compound – Bone is broken and piercing the skin

Compressed – One bone is forced against another

Displaced – The ends of the bone are not aligned

Greenstick – Periosteum divided on only one side

Pathological – Occurs because of a bone defect

Simple – Fracture is straight and in good alignment

Spiral – Fracture resulting from twisting motion

Isolette Use

AAP less than 10lbs or 30 days

-Thermoregulation issues

-Ventilator management strategies

-Sound, Light and Stimulus Discipline

Thermoregulation Assessment

- Temperature; continuously via skin and rectal probe

-Neurologic status

- Perfusion Status

- Vital signs

Interventions

- Incubator; <30 days or <5kg

- Pre-warm; check battery; keep transport environment warm;

Incubator Temps

Birth weight (grams)	Clothed	Exposed
>2500	28C	33C
1501-2500	31C	35.5C
<1500	33C	36.5C

Each 1C above 37C

-Heart rate increases 20bpm

-Respiratory rate increases 10rpm

Mechanical Ventilation

Pressure cycle mode for uncuffed tubes; SpO2 and ETCo2 are standard of care; Correlate gas interpretation with SpO2 and ETCo2

Infant Ventilator Settings

FiO2	100%
Inspiratory Time	>.5 sec
PIP	20 – 30 cmH2O
Rate	20 – 30
PEEP	3 – 5 cm

Child Ventilator Settings

FiO2	100%
I: E ratio	1:1
Tidal Volume	6-10 cc/kg
PIP	Up to 40 cm H2O
Rate	16 – 20
PEEP	3-5 cm

Sound, Light, and Stimulus

- Over stimulus promotes HTN

- Think about your local NICU

Quiet; dark; like where a baby comes from

-Use isolette covers

- Stay out of isolette as much as possible

-Gentle handling/loading

Seizures

-Alert to signs of neonatal/infant seizures

-Don't forget to rule out common causes

-Hypoglycemia

-May be primary or secondary

-Due to immaturity of nervous system rarely demonstrate classic generalized tonic-Clonic activity

Subtle – repetitive mouth/tongue movement, bicycling, eye deviation, repetitive blinking

Clonic – repetitive jerky movements of limbs

Tonic – May resemble posturing or tonic extension seen in older patients

Myoclonic – Multiple jerking motions, usually of the upper extremities

NEONATAL EMERGENCIES

Diaphragmatic Hernia

-Intubation

-PPV

-OGT w/ suction

-NPO

Choanal Atresia

-Obligate nose breathers

-Intubate/OPA

Aspiration Pneumonia

Lethargy?

-Intubation

-Suctioning

Brisk?

-Monitor only

Coarctation of the Aorta

-Narrowing of aortic arch typically just distal of left subclavian bifurcation

-Decreased aortic flow results in increased LV pressures and failure

Commonly associated with VSD

Treatment -Surgical dilatation

Tetrology of Fallot

-VSD

-PA outflow obstruction

-RV hypertrophy

-Rightward displacement of aorta exploited by VSD

Treatment

Severe hypoxia?

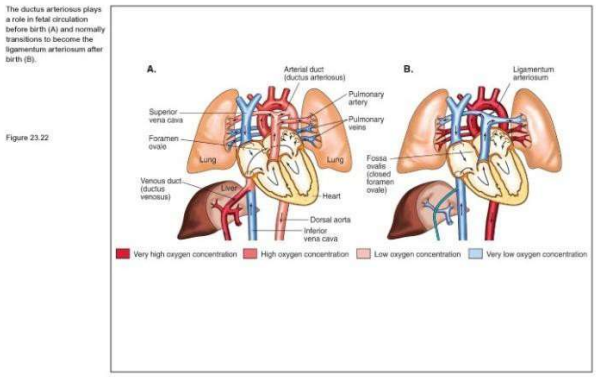
-PGE1 for PDA patency mgt

Surgical

-Cath PA and dilate

- "Patch" VSD

- "Tet spell" management



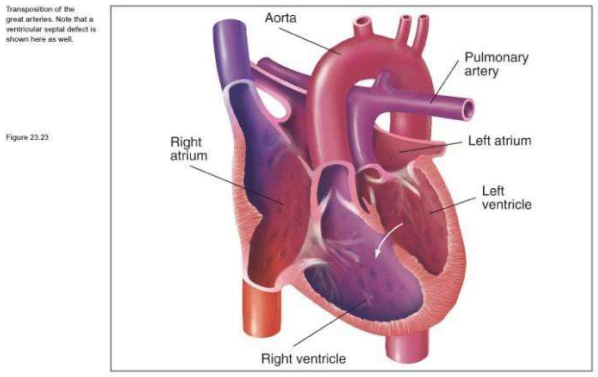
Patent Ductus Arteriosus (PDA)

-Potential for gross pulmonary edema and respiratory failure

-Patency with prostaglandin may be necessary with specific CHD's

-Oxygen administration issues for therapeutic

-PDA patency



Transposition of the Great Vessels

-"Survival" via foramen ovale and PDA and/or VSD

-Progressive hypoxemia will ensue

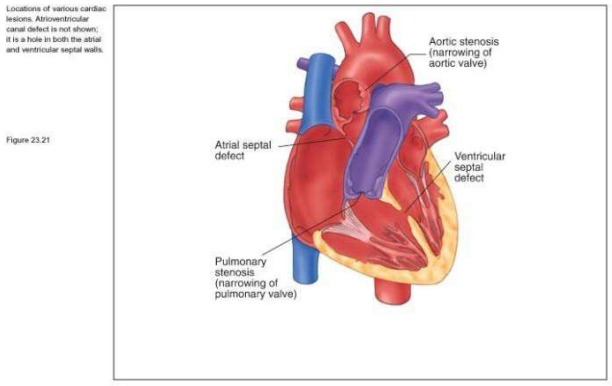
-Extreme caution with air embolus

-Prostaglandin administration (PDA patency management)

Surgery

-VSD created (temp)

-Jantene ("arterial switch")



Ventricular Septal Defect (VSD)

-Dependent on size and location

-Majority close spontaneously

Treatment

Meds

Indomethacin for closure

Prostaglandin for patency Coil

References

Alspach, J. (Ed.). (1998). *Core curriculum for critical care nursing* (6th Ed.). Philadelphia, PA: Saunders Elsevier

American Heart Association & American Academy of Pediatrics. (2000). *Neonatal resuscitation textbook* (4th Ed.). Elk Grove Village: American Academy of Pediatrics

Fischbach, F. (2004). *A manual of laboratory and diagnostic tests* (7th Ed.). Philadelphia, PA: Lippincott Williams & Wilkins

Guyton, A., & Hall, J. (2000). *Textbook of medical physiology* (10th Ed.). Philadelphia, PA: W.B. Saunders Elsevier

Hazinski, M. (Ed.). (2002). *PALS provider manual*. Elk Grove Village: American Academy of Pediatrics & the American Heart Association

Holleran, R. (Ed.). (2005). *Air and surface patient transport: Principles & practice* (3rd Ed.). Philadelphia, PA: Elsevier Health Sciences.

Holleran, R. (Ed.). (2010). *Air and surface patient transport: Principles & practice* (4th Ed.). Philadelphia, PA: Elsevier Health Sciences.

Pillitteri A. (2007). *Maternal & child health nursing* (5th Ed.). Philadelphia, PA: Lippincott, Williams & Wilkins

Pollack, Andrew. (Ed.). (2010). *Critical Care Transport*. Sudbury, MA: Jones and Bartlett Publishers

Sanders, M. (2007). *Mosby's paramedic textbook* (3rd Ed.). St Louis, MO: Elsevier Health Sciences

Urden, L., Stacy, K., & Lough, M. (2005). *Thelan's critical care nursing diagnosis and management* (5th Ed.). Maryland Heights, MO: Elsevier Health Sciences

Walls, R., Murphy, M., Luten, R., & Schneider, R. (2008). *The manual of emergency airway management*. (3rd Ed.). Philadelphia, PA: Lippincott Williams & Wilkin