Surgical Critical Care Syllabus 1

GI HEMORRHAGE & INTRA- ABDOMINAL PROBLEMS IN THE ICU

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Courtesy of Fahim Habib, MD and Vishnu Mani, MD

UPPER GI HEMORRHAGE

- Definition: Proximal to Ligament of Treitz
- Severe UGI Hemorrhage
  - Shock
  - Orthostatic Hypotension
  - Decrease in Hgb by 3 – 4 g/dL
  - Transfusion of at least 2U prbcs
- Overall Mortality: 5 – 10%

ETIOLOGY

CAUSE	FREQUENCY
Erosive Gastritis	5 – 25%
Duodenal Ulcer	24%
Gastric Ulcer	21%
Varices	9 – 21%
Esophagitis	2 – 8%
Erosive Duodenitis	5 – 9%
Mallory-Weiss Tear	11 – 14%
Malignancy	3%

HISTORY

- Specifics
  - Bright Red/Coffee Ground/Melena
  - Prior GI Bleeding
  - Retching/Vomiting: Mallory-Weiss tear
- PMH
  - Peptic Ulcer Disease
  - Renal or Liver Disease
  - Alcohol Abuse
- PSH
  - AAA Repair
  - Instrumentation of GI Tract
- Medications
  - NSAIDs
  - Anti-coagulants

PHYSICAL EXAM

- Hemodynamic Status
- Liver Disease
- Malignancy
- Laboratory
  - Electrolytes
  - CBC
  - Coags
  - LFTs
  - T & C

INITIAL MANAGEMENT

- Resuscitation
  - Large Bore IVs
  - Crystalloid/pRBCs
  - Monitor CVP
  - Serial H&H
  - Correct Coagulopathy
- NGT (may omit if EGD soon)
- H-2 Blocker/PPIs
- Gastric Lavage (may omit if EDG soon)
- Foley Catheter

UPPER ENDOSCOPY

- Indications
  - Resuscitated Patient
  - Active Hemorrhage
  - Requires Transfusion
  - Known/Suspected Portal Hypertension
  - Suspected Aortoenteric Fistula
  - Rebleed
- Radiologic Contrast Studies
  - No therapeutic benefit
  - May interfere with endoscopy
- Accuracy: Highest in first 12 – 18 hours (approx 90%)
- Success Rate: Approx 90%

ENDOSCOPY & REBLEED RISK

ENDOSCOPIC FINDING	RISK OF REBLEEDING
Arterial/Pulsatile Bleeding	85%
Nonbleeding visible vessel	40 – 50%
Adherent Clot	20 – 30%
Oozing without visible vessel	< 20%
Flat blood spot at ulcer base	< 10%
Clean base	< 5%

ANGIOGRAPHY – UPPER GI

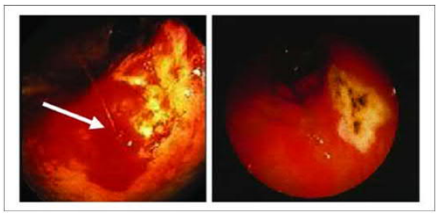
- Identification of Source
- Therapeutic Embolization
- Success Rate: 75 – 80%
- Rebleed: Up to 50%

EMERGENCY SURGERY

- Failure to control bleeding with nonoperative means
- Severe rebleeding despite two attempts at endoscopic hemostasis
- Lesion inaccessible to endoscopy
- Severe shock/to prevent exsanguination
- Complication of endoscopic therapy

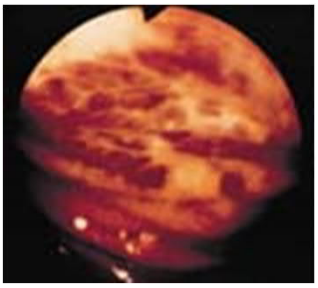
GASTRIC/DUODENAL ULCERS

- Bleeding Risk: 15%
- Rebleed: Gastric > Duodenal
- Endoscopic Interventions
  - Electrocautery/Heater Probes
  - Injection Therapy
  - Laser Photocoagulation
- Rebleeding Options
  - Repeat Endoscopy
  - Angiographic Embolization
  - Surgical Therapy



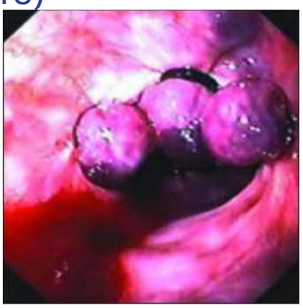
STRESS ULCER/GASTRITIS

- Risk Factors
  - Mechanical Ventilation ≥ 48 hrs
  - Coagulopathy
  - Increased ICP (Cushing's)
  - Burns (Curling's)
  - Sepsis
  - Trauma
  - Renal Failure
  - Liver Failure
  - Multi-Organ System Failure



ESOPHAGEAL VARICES

- Mortality: 15-40%
- Second Episode Risk: 70% (without portal pressure lowering procedure)
- Therapy
  - Endoscopy
  - Pharmacology
  - Shunt

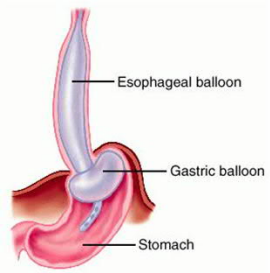


ESOPHAGEAL VARICES
ENDOSCOPY

- Diagnosis & Therapy
  - Nonvariceal Source: Seen in up to 50% with portal hypertension
  - Bleeding Varices: Sclerotherapy or Variceal Ligation
- Success
  - Up to 90%
  - Rebleed: Up to 66%
  - Mortality & Rebleed: Ligation >> Sclerotherapy

ESOPHAGEAL VARICES
TAMPONADING TUBES

- Types
  - Sengstaken-Blakemore
  - Minnesota
- Indications
  - Fail endoscopic therapy
  - Require stabilization
  - Awaiting radiologic or surgical therapy
- Success
  - Up to 80%
  - Short-lived/temporizing
  - Requires experience



ESOPHAGEAL VARICES
PHARMACOLOGY

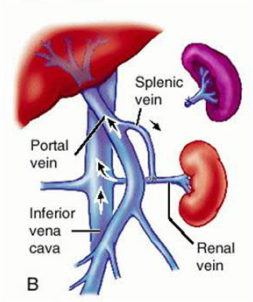
- Vasopressin Infusion
  - Help control hemorrhage in 52%
  - No change in mortality
- Somatostatin + Sclerotherapy or Ligation
  - Reduces transfusion requirements
  - Reduces rebleed rates
- Propranolol
  - Proven prophylaxis against variceal hemorrhage
  - No role in acute management

ESOPHAGEAL VARICES
SHUNT PROCEDURES - TIPS

- Indications
  - Bleeding varices due to portal hypertension
  - Do not respond to endoscopic or pharmacologic therapy
- Actions
  - Rapidly lower portal pressure
  - Achieve immediate control of variceal bleeding
  - New/worsening hepatic encephalopathy: 10-30%
  - Stenosis: Approx 50% at 6-12 months
- Contraindications
  - Elevated bilirubin or creatinine levels
  - Hepatic encephalopathy refractory to medical management

ESOPHAGEAL VARICES
SHUNT PROCEDURES - SURGICAL

- Indications
  - TIPS not available
  - Refractory variceal bleeding
- Distal Splenorenal is preferred
- Dependent on:
  - Severity of Liver Disease
  - Presence of Ascites
  - Degree of encephalopathy
  - Surgeon Experience



LOWER GI HEMORRHAGE

- Definition: Distal to the Ligament of Treitz
- Facts
  - Overall Incidence (annual): 20.5 in 100,000
  - Resolution (spontaneous): 80%
  - Intervention: 10-15%
  - Rebleed Rate: 25%
  - Mortality: 10-15%
- Hematochezia: Most common cause is Upper GI Hemorrhage

ETIOLOGY

Etiology	Incidence (%)
Diverticular Disease	40
Inflammatory Bowel Disease	21
Neoplasia	14
Coagulopathy	12
Benign Anorectal Disease	11
Arteriovenous Malformation	2

HISTORY

- Specifics
  - Bright Red/Melena
  - Prior GI Bleeding
  - Pain: Rectal or Abdominal
- PMH
  - Hemorrhoids
  - Diverticular Disease
  - Malignancy
- PSH
  - AAA Repair
  - Instrumentation of GI Tract
- Medications
  - NSAIDs
  - Anti-coagulants

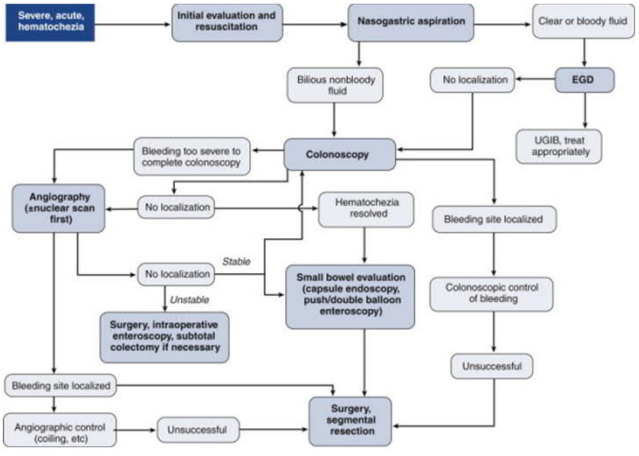
PHYSICAL EXAM

- Hemodynamic Status
- Abdominal Exam
  - Pain out of proportion; A-Fib; Recent MI
  - Pulsatile Mass
  - Liver Disease Stigmata
- Rectal Exam/Anoscopy/Proctoscopy
- Laboratory
  - Electrolytes
  - CBC
  - Coags
  - LFTs
  - T & C

INITIAL MANAGEMENT

- Resuscitation: IVs, Crystalloid, pRBCs
- NGT with Lavage: False Negative = 15%
- Foley Catheter
- ICU Admission
- Serial H&H
- GI Prophylaxis
- Stop Anticoagulants
- Diagnostic Workup

DIAGNOSTIC ALGORITHM



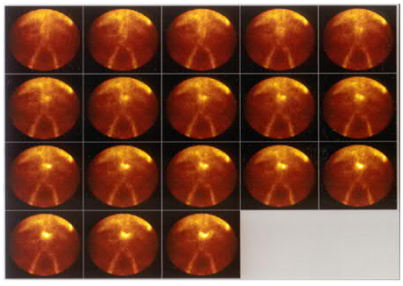
Cameron, *Current Surgical Therapy, 10th Ed*

MODALITIES

- Diagnostic Only
  - Nuclear Scintigraphy
- Diagnostic & Therapeutic
  - Colonoscopy
  - Mesenteric Angiography
  - Operative Management

NUCLEAR SCINTIGRAPHY (Tagged RBC)

- Detects Hemorrhage as low as 0.1 mL/min
- Sensitivity: 80-98%
- Specificity: 41-94% (about 48%)



NUCLEAR SCINTIGRAPHY (Tagged RBC)

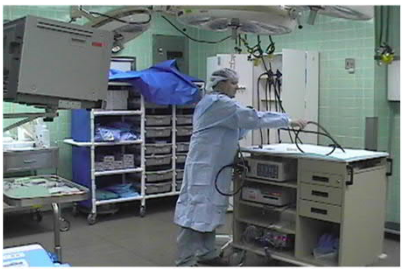
- Advantages
  - Long half-life (24-48 hrs)
  - Sensitivity
- Disadvantages
  - Localization not optimal (specificity)
  - Preparation required
  - Not for critically ill patients
  - Non-therapeutic

NUCLEAR SCINTIGRAPHY (Tagged RBC – Predictive Value)

- Pennoyer, et al
  - Retrospective Review: 131 angiograms, 54 Tag RBC studies prior to angiogram
  - Positive bleeding scan DID NOT increase percentage of a positive angiogram
  - Neither did prior history of GI bleed, transfusions, orthostatic hypotension, or tachycardia
  - Conclusion: Nuclear medicine scans should not be used routinely as a screening test for angiography
  - *Dis Colon Rectum* 1997; 40:1014-1058.
- Ng, et al
  - Retrospective Review: 160 Tag RBC studies for massive lower GI bleeding; 86 positive scans; 47 angiograms; (39 colonoscopy/obs)
  - Immediate Blush (< 2 mins): PPV 75%
  - Delayed Blush (> 2 mins): NPV 93%
  - Conclusion: Patients with immediate blush required urgent angiography; those with delayed blush/negative scan may be observed or evaluated with colonoscopy
  - *Dis Colon Rectum* 1997; 40:471-477

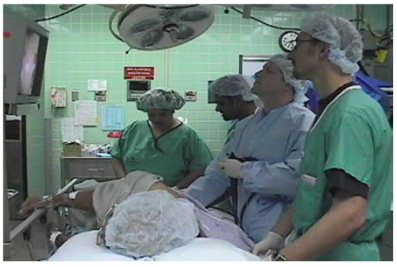
COLONOSCOPY

- Procedure for mild/moderate/ceased bleeding + hemodynamically stable/stable with resuscitation
- Sensitivity: 70 – 100%
- Specificity: 86%



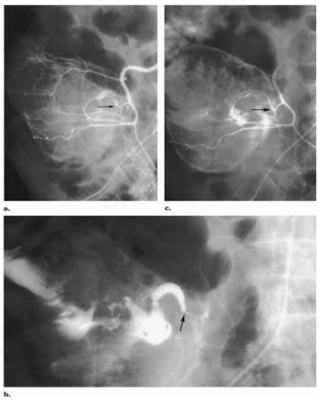
COLONOSCOPY

- Advantages
  - Diagnostic and Therapeutic
  - Non-invasive
  - Low Complication Rate
- Disadvantages
  - Diminished visibility in aggressive bleeding
  - Prep time: 2 – 4 hours
- Options: Metal clips, Elastic bands, Coagulation, Epinephrine injection, Inking



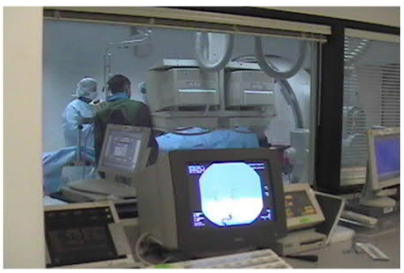
MESENTERIC ANGIOGRAPHY

- Detects hemorrhage as low as 0.5 – 1.0 mL/min
- Sensitivity: 40 – 86% (about 47%)
- Specificity: 86 – 100%
- Rebleed Rate: 22%



MESENTERIC ANGIOGRAPHY

- Advantages
  - Diagnostic & Therapeutic
  - Low Complication Rate
- Disadvantages
  - Arterial access-related complications
  - Intestinal ischemia/infarction
- Options
  - Vasopressin Infusion
  - Embolization
  - Inking



VASOPRESSIN INFUSION

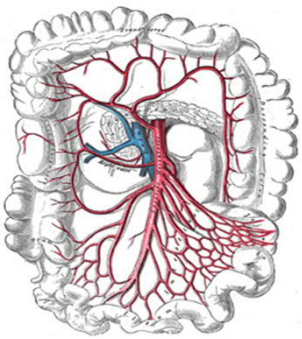
- Purpose: Arteriolar vasoconstriction and bowel wall contraction
- Procedure
  - Catheter in main feeder artery (SMA, IMA, etc)
  - Infusion for 20-30 minutes; repeat angio
  - With cessation, infusion for 6-12 hours; reduce dosage 50%; infusion another 6-12 hours
  - Repeat angio; bleeding stops; remove catheter

VASOPRESSIN INFUSION

- Success Rate: 60-90%
- Rebleed Rate: 22-71% (about 50%)
- Complications (rate up to 43%)
  - Myocardial ischemia/infarction
  - Intestinal ischemia/infarction
  - Bradycardia
  - Hypertension
- Contraindications: CAD, PVD

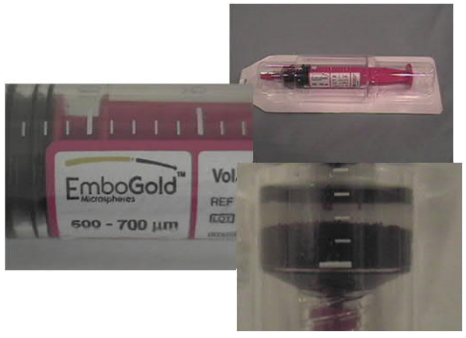
SELECTIVE EMBOLIZATION

- Procedure
  - Catheter placed as distal as possible into bleeding branch
    - Vasa Recta
    - Marginal Artery
  - Embolization agent released to lodge distally



SELECTIVE EMBOLIZATION (AGENTS)

- Gelfoam (temporary)
- Spheres



- PVA Particles

- Coils

SELECTIVE EMBOLIZATION

- Success Rate (immediate): 90-100%
- Rebleed Rate: 0-12% (most recent 0%)
- Complications (rate up to 5%)
  - Intestinal ischemia/infarction
  - Angiography related complications
- Super Selective Embolization
  - More technically advanced catheters, guide wires, and digital angiography
  - Intestinal ischemia/infarction rates: < 5%

OPERATIVE THERAPY

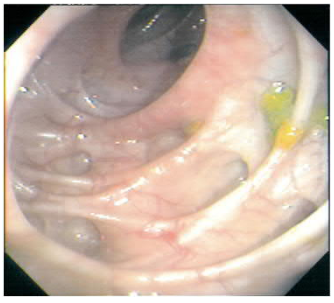
- Rate: 10-25% (acute lower GI bleeding)
- Indications
  - Hemodynamic instability
  - Transfusion requirement
  - Persistent/recurrent hemorrhage
- Threshold
  - 10 total units (48 hrs); 6 units (24 hrs) and ongoing hemorrhage
  - Massive bleeding + Hemodynamically unstable + Unresponsive to resuscitation
  - EGD is mandatory to r/o UGI

OPERATIVE THERAPY

- Plan
  - Preoperative localization: Segmental resection
  - Without localization: Intraoperative localization, colonoscopy, enteroscopy, EGD
  - Blind Segmental Colectomy
  - Subtotal Abdominal Colectomy

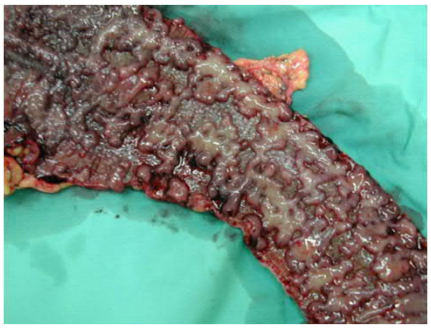
DIVERTICULAR DISEASE

- Bleeding Risk: 4-48% (largest review 17%)
- Resolution (spontaneous): 80%
- Rebleed Rate: 25%



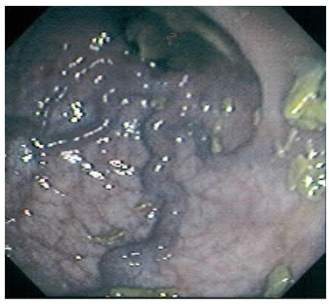
INFLAMMATORY BOWEL DISEASE

- Bleeding Risk: 6% (Crohn's & UC)
- Resolution (spontaneous): 50%
- Rebleed Rate: 35%



ARTERIOVENOUS MALFORMATION

- Bleeding Risk: 2%
- Resolution (spontaneous): 85-90%
- Rebleed Rate: 25-85%



INTRA-ABDOMINAL INFECTION

- Sterile Space Contents
  - Viscera
  - Serous Fluid (50 ml)
- Defenses
  - Translymphatic diaphragmatic absorption – diaphragmatic pores to thoracic duct
  - Resident Macrophages: Phagocytosis/Microbe Killing

PERITONITIS

- Primary
  - Microbial infection in absence of perforation of hollow viscus
  - Single organism: E coli, S aureus, E faecium/faecalis
- Secondary
  - Subsequent to perforation of hollow viscus and spillage of endogenous microflora
  - Multiple organisms: GN Aerobes + Anaerobes
  - E coli, K pneumonia, Bacteroides, Peptostreptococcus
- Tertiary
  - Secondary disease plus persistent microbial contamination, even with antimicrobial therapy
  - Low virulence pathogens: S epidermidis, E faecium/faecalis, Candida albicans

DIAGNOSIS

- General: Fever, Leukocytosis, Lactic Acidosis
- Source of Infection
  - Neurologic – Mental status changes
  - Pulmonary –Minute ventilation, Hypoxemia, Hypercarbia
  - Cardiovascular – Hypotension, Tachycardia
  - Hepatic – Increased bilirubin and LFT's
  - Renal – Decreased urine output, Inc bun/creatinine
  - Gastrointestinal – Pain, Ileus, Tube feed intolerance
  - Endocrine: Glucose intolerance
  - Hematologic – Thrombocytopenia, Coagulopathy

DIAGNOSIS

- Inciting Event
  - Abdominal surgery
  - Endoscopy
  - Percutaneous procedure
- Host Defense Status
  - Persistent/Progressive Sepsis
  - Unexplained Organ Failure
- Rule-Out Other Sources
  - Urinary tract infection
  - Sinusitis
  - Pneumonia
  - Catheter related sepsis

DIAGNOSIS

- Plain X-ray
  - Acute abdominal series
  - Free air; Ileus; SBO; Ogilvie's
- U/S
  - Biliary Tree; Gallbladder
- Computed Tomography
  - Most sensitive/specific
  - Therapeutic: Percutaneous drainage, etc.
- DPL/Paracentesis
  - Eval for Spontaneous Bacterial Peritonitis
  - Sensitive for detecting inflammation/not specific for diagnosis

ACUTE PANCREATITIS

- Pathophysiology: Enzyme activation/ autodigestion of pancreas
- Etiology
  - Gallstone/Alcoholic: 90%
  - Other: Trauma, hyperparathyroidism, hyperlipidemia, infectious, medications (HIV), pregnancy, scorpions
- S/S: Fever, tachycardia, abdominal pain, leukocytosis, elevated amylase, elevated lipase

ACUTE PANCREATITIS

- CT Scan: Most sensitive/specific
- Therapy
  - Supportive
  - Pain Control
  - NG Tube Decompression: PRN/Not shown to alter course
  - Prophylactic antibiotics: For severe/concern for/evidence of necrotizing pancreatitis
  - Enteral Feeds vs TPN

ACUTE PANCREATITIS

- Time Course
  - Mild/Self-Limiting
  - Severe/Multiorgan Failure
- Ranson's Criteria – Classic Prognostic Indicator
  - Measured at Admission and 48 hrs
  - Mortality (# Criteria Met)
    - 1 – 2: < 1%
    - 3 - 4: 10 – 15%
    - 5 – 7: 40 – 50%
    - > 7: Up to 90%

RANSON'S CRITERIA

- At admission
  - Age > 55
  - WBC > 16,000
  - Glucose > 200
  - LDH > 350
  - AST > 250
- Within 48 hours
  - Hct decrease > 10
  - BUN Increase > 5
  - Ca < 8
  - PaO_2 < 60
  - Base deficit > 4
  - Fluid req > 6L

NECROTIZING PANCREATITIS

- Fluid Resuscitation
- Nutritional Support
  - Gut preferable
  - May need TPN; watch lipids (triglycerides < 300)
- Antibiotics – Severe Pancreatitis
  - Reduced sepsis & mortality
  - Imipenem & Quinolones: Found in therapeutic concentrations in pancreatic secretions

NECROTIZING PANCREATITIS

- Surgical Debridement
  - Sterile Necrosis vs Infected Necrosis
    - CT Evidence: Nonenhancing; air in tissue
    - FNA Aspirates
  - Clinical Picture: Worsening vs Improving
- Operative Strategy
  - Wide Debridement
  - Temporary Packing
  - Extensive Drainage

ACUTE CHOLECYSTITIS

- Cholecystitis – Gallstone Etiology
  - Still most common
  - Secondary bacterial infection : 90% Positive
- Acalculous Cholecystitis
  - Etiology: Bile stasis/sludge formation/distension
  - Secondary bacterial infection
    - 40% Positive
    - Mostly chemical inflammation
  - Risk factors: DM, Male, Ileus, Sepsis, Narcotics, TPN

ACUTE CHOLECYSTITIS

- Clinical (ICU): Fever, RUQ Pain, Increased LFTs, Leukocytosis
- Diagnosis
  - Ultrasound: Gallbladder dilation/wall thickening, Pericholecystic fluid, +/- Stones
  - HIDA: Non-filling of gallbladder
- Treatment
  - Fluid and Antibiotics
  - Surgical: Cholecystectomy
  - Cholecystostomy Tube: Open vs Percutaneous

BOWEL COMPLICATIONS

- Perforated Peptic Ulcer Disease
  - Diagnosis
    - Epigastric Pain; sudden onset; initially intense
    - Peritonitis: Chemical then bacterial (12 – 24 hr)
    - Radiologic: Pneumoperitoneum (20% neg)
  - Therapy
    - Resuscitation
    - Broad-Spectrum Antibiotics
    - Surgical Intervention: Definitive vs Damage Control

BOWEL COMPLICATIONS

- Small Bowel Obstruction
  - Diagnosis
    - Abdominal pain/distention, emesis, tube feed intolerance
    - Laboratory Studies: CBC, Electrolytes, Lactate
    - Radiologic Plain Films/CT Scan
      - Mechanical vs Functional
      - Complete vs Incomplete
      - Transition Point?

BOWEL COMPLICATIONS

- Small Bowel Obstruction
  - Therapy
    - NG Decompression
    - Fluid resuscitation
    - Correct Electrolyte Abnormalities
    - Operative vs Non-Operative Management
      - Complete obstruction
      - Persistent pain, tenderness (closed loop obstruction)
      - Incarcerated hernia
      - Free air and/or pneumatosis
      - Bowel necrosis?: Fever, tachycardia, localized tenderness, leukocytosis, acidemia

BOWEL COMPLICATIONS

- Mesenteric Ischemia
  - Nonocclusive bowel ischemia (NOBI)
    - Hypovolemia/Hypoperfusion common
    - Gradual onset
    - Thickened bowel with pneumatosis
  - Arterial occlusion
    - Embolism – 75% originate in heart
    - Thrombosis – Preexisting vascular lesion
  - Mesenteric venous thrombosis
    - Hypercoagulable conditions
    - Treatment: Anticoagulation

BOWEL COMPLICATIONS

- Missed Bowel Injury - Characteristics
  - Blunt Trauma
  - Delayed Diagnosis
  - Non-operative management of liver/spleen injuries
  - 1% of admitted patients
  - Seatbelt sign
  - CT with no solid organ injury and intra-abdominal fluid
  - Worse outcomes for adults but not children

BOWEL COMPLICATIONS

- Diverticulitis
  - Diagnosis
    - Fever; LLQ pain; nausea/emesis; peritonitis (free perforation)
    - CT Scan: Diverticula/Free Air/Abscess
    - Laboratory Studies: CBC, Lactate
  - Therapy
    - Bowel rest
    - Antibiotics
    - Resuscitation
    - Operative vs Non-Operative Management
    - Percutaneous Drainage (Abscess)

BOWEL COMPLICATIONS

- Large Bowel Obstruction
  - Carcinoma: 20% have near or complete obstruction
  - Diverticulitis
  - Volvulus – axial twist or folding
    - Sigmoid (50-70%) – Loop of bowel in right upper quadrant
      - Stable patient: Decompress colon
      - Emergent surgery: Perforation or necrosis
      - Operative Strategy: Resection preferred/low recurrence

BOWEL COMPLICATIONS

- Large Bowel Obstruction (continued)
  - Volvulus – axial twist or folding
    - Cecal (20-30%) – Loop of bowel in left upper quadrant with small bowel obstruction
      - Decompress colon – Not always successful/possible
      - Surgical resection for both gangrenous or viable bowel
  - Ogilvie's syndrome (pseudo-obstruction)
    - Dilated colon – Associated with medical conditions
    - Endoscopic Decompression: For cecum > 12cm
    - Neostigmine – Monitor for bradycardia

QUESTIONS

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ABDOMINAL TRAUMA
PART ONE
Surgical Critical Care

David Hampton MD, MEng, FACS

September 2025

Courtesy of Fahim Habib, MD and Vishnu Mani, MD

EPIDEMIOLOGY

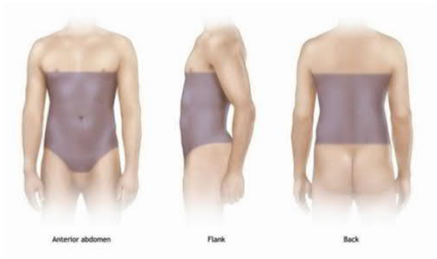
- Blunt Abdominal Trauma
  - Spleen: 40 – 55%
  - Liver: 35 – 45%
  - Small Bowel: 5 – 10%
- Penetrating Abdominal Trauma
  - Stab Wounds
    - Liver: 40%
    - Small Bowel: 30%
    - Diaphragm: 20%
    - Colon: 15%
  - Gunshot Wounds
    - Small Bowel: 50%
    - Colon: 40%
    - Liver: 30%
    - Abdominal Vascular Structures: 25%

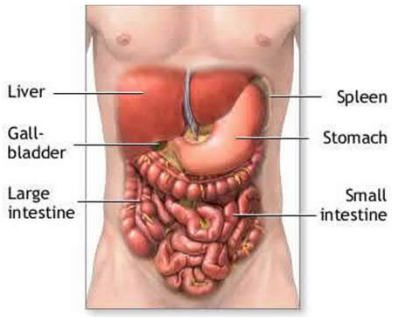
EPIDEMIOLOGY

- Blunt Trauma
  - Compression or Crushing
  - Shearing
- Penetrating Trauma
  - Low Velocity: Laceration or cutting
  - High Velocity
    - Laceration or cutting
    - Cavitation

ANATOMY

- External
  - Anterior abdomen
  - Flank
  - Back
  - Thoracoabdominal
- Internal
  - Peritoneal cavity
  - Pelvic cavity
  - Retroperitoneum





INITIAL EVALUATION

- Primary Survey
  - ABCs – ATLS Standard
  - Cardiac monitor and pulse oximeter
  - Concurrent resuscitation
- Secondary Survey
  - Head to toe exam
  - FAST
  - NGT
  - Foley Catheter
- Tertiary Survey
  - After addressing life-threatening/serious injuries
  - Commonly Missed: Vertebral/spinal, Extremity fractures, Reperfusion/Compartment Injuries

PHYSICAL EXAM

- External Signs
  - Abrasions/Contusion/Ecchymosis
  - Seatbelt
- Abdominal/Flank Exam
- Pelvis – Stability
- Perineum/Rectum – Urethra, Prostate
- Roll the patient – check the back!!

CONFOUNDERS

- Significant Abdominal Injury: 40% have no peritoneal signs
- Positive Physical Findings: 20% have no injury
- **Unreliability**
  - Closed Head Injury
  - Spinal Cord Injury
  - Altered Mental Status (i.e. intoxication, etc.)
  - Distracting Injury

DIAGNOSTIC EVALUATION

- DPL
- FAST
- CT SCAN
- Assessment of Pelvis
  - Exam
  - Plain film

DPL

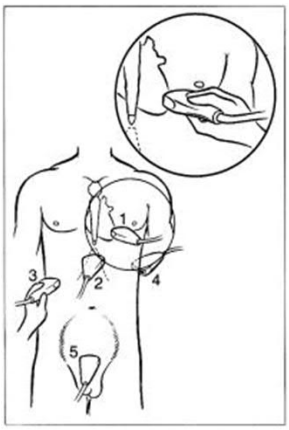
- Indications
  - Hemodynamically abnormal patients with Blunt Abdominal Trauma
  - Selected patients with Penetrating Abdominal Trauma
  - Looking for presence of Blood (as little as 2-10ml)
- How
  - Place NGT and Foley catheter
  - Decision: Open or Percutaneous; Infra or Supra-umbilical/fundal
  - Aspiration followed by instillation of 1L NS (10 cc/kg for children)
  - Positive – Blunt Trauma
    - 10 cc gross blood on aspiration
    - RBCs $\geq$ 100,000 cells/ml
    - WBC $\geq$ 500 cells/ml
    - Elevated Amylase or Bilirubin
    - Presence of GI contents, urine

DPL

- How (continued)
  - Positive – Penetrating Trauma (controversial)
    - 5 cc gross blood
    - RBCs > 5,000 cells/ml
    - WBC > 500 cells/ml
    - Amylase > 100 units
    - Gross Bile or GI Contents
- Advantages
  - High Sensitivity (98%)
  - Rapid
  - No transport required
- Disadvantages
  - Invasive
  - Low specificity
  - Misses diaphragm and retroperitoneum
  - Compromises follow-up CT-imaging/interpretation

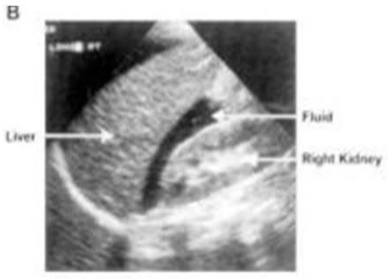
FAST

- Indications
  - **Hemodynamically abnormal patients with Blunt Abdominal Trauma**
    - Looking for presence of fluid in abdomen, pericardial sac (as little as 100ml)
  - Peritoneal, Pericardial, Thoracic Cavities
  - Also Dx: Pneumothorax, Solid Organ Injury, Sternal Fracture
- How
  - Perform w/o Foley Catheter
  - 3.5 Mhz convex or curvilinear array transducer (abdomen); 7 – 7.5 MHz linear array transducer (chest)



FAST

- Advantages
  - Rapid
  - No transport required
  - Noninvasive
  - Repeatable
  - Real-Time Data



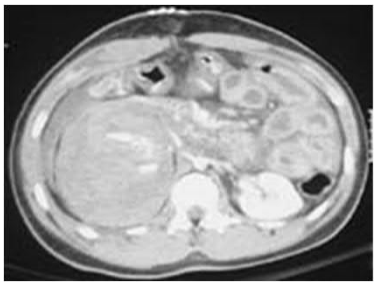
- Disadvantages
  - Operator-dependent
  - Low ability to identify source of fluid
  - Distortions by bowel gas, subcutaneous air, obesity
  - Misses diaphragm, bowel, retroperitoneal injuries

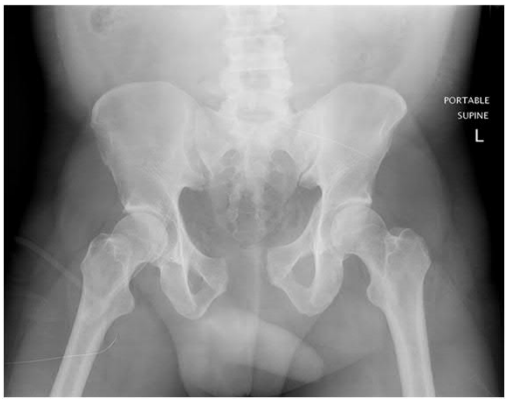
CT SCAN

- Characteristics
  - Study of choice in hemodynamically normal patient with blunt abdominal trauma or penetrating trauma to the back/flank (“triple contrast CT”)
  - Document injuries
  - Can direct work-up and therapy
    - Findings don’t necessarily mandate O.R.
    - Can direct towards angiography/embolization

CT SCAN

- Advantages
  - Most specific
  - High sensitivity
  - Magnitude of Injury
  - Associated Injuries
- Disadvantages
  - Increased cost and time
  - Transport required
  - Can miss diaphragm and bowel injuries (although there are stigmata suggestive)
  - Not appropriate for hemodynamically abnormal patient





PELVIC FRACTURES- EPIDEMIOLOGY

- 3% of All Fractures
- Occur in 10-20% of all trauma patients
- Source of Life-Threatening hemorrhage
- Mortality
  - All fracture types: 5 – 30%
  - Closed + Hypotension: 10 – 42%
  - Open: 50%
- Associated with Other Injuries: 50%

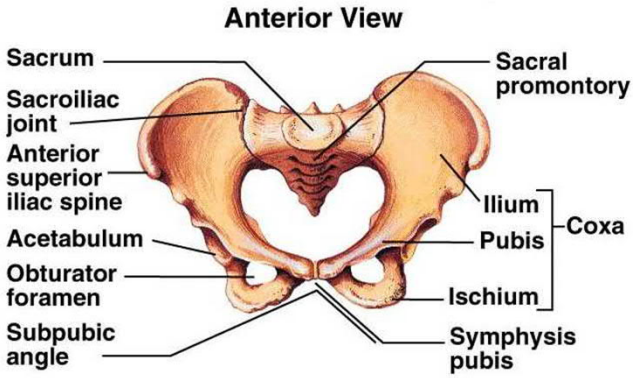
ETIOLOGY

- Motor Vehicle Collision: 60%
- Motor Vehicle vs Pedestrian: 12%
- Motorcycle Collision: 9%
- Falls: 10%
- Crush: 9%

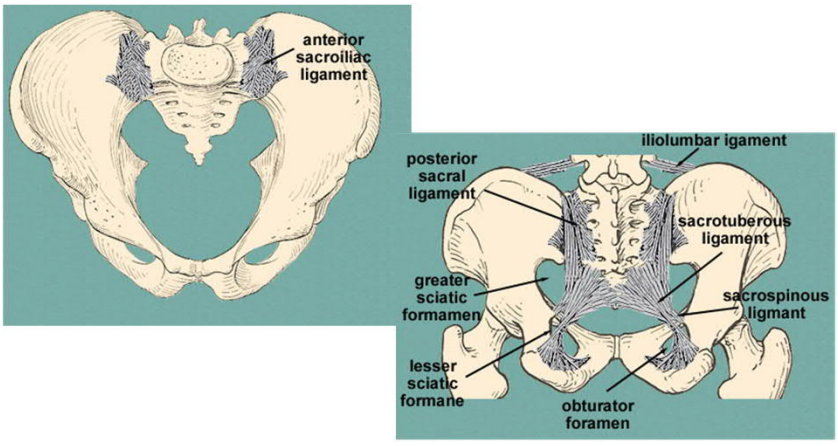
ASSOCIATED INJURIES

- TBI: 51%
- Thoracic: 20%
- Abdominal: 17%
- Urologic: 25%
  - Bladder: 9%
  - Urethral: 13%
- Neurologic/Spinal (L4/5, S1-3): 10-21%
- Rectal: 5%
- Vaginal: 3%

ANATOMY



ANATOMY – PELVIC LIGAMENTS



PELVIC FRACTURE
YOUNG-BURGESS CLASSIFICATION

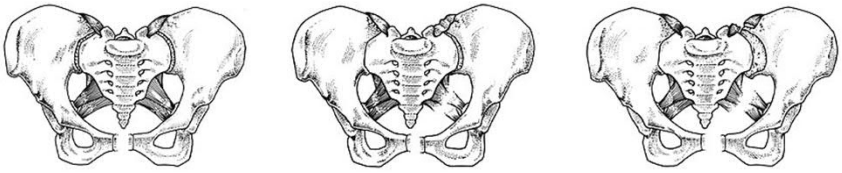
- Vector of Force Applied
- Antero-posterior Compression (APC)
- Lateral Compression (LC)
- Vertical Shear

PELVIC FRACTURE CLASSIFICATION
ANTERIOR-POSTERIOR COMPRESSION (APC)

- Forces: Applied in the Sagittal plane
- Incidence: 20 – 30%
- Involve degree of:
  - Pubic symphyseal diastasis
  - Posterior ligamentous injury
- Seen in:
  - Head-On MVC
  - Pedestrian struck Head-On
- Hemipelvis Displacement: External Rotation
- Associated Injury
  - Higher incidence of Thoracic, Abdominal, and Pelvic Arterial
  - APC-III: highest incidence of neurologic deficit and massive hemorrhage

PELVIC FRACTURE CLASSIFICATION
ANTERIOR-POSTERIOR COMPRESSION (APC)

Category	Characteristics
Type I	Pubic diastasis <2.5 cm
Type II	Pubic diastasis > 2.5 cm; Anterior SI joint disruption
Type III	Type II + Posterior SI joint disruption



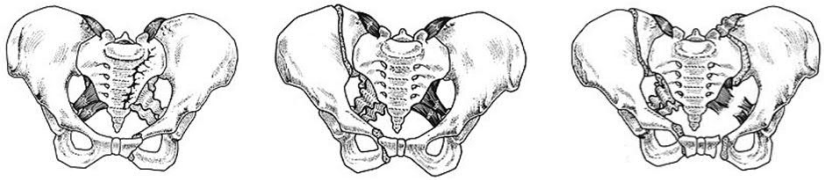
APC-I **APC-II** **APC-III**

PELVIC FRACTURE CLASSIFICATION
LATERAL COMPRESSION (LC)

- Force: Applied in the Horizontal plane
- Incidence: Most common (50%)
- Hemipelvis Displacement: Internal Rotation
- Seen in:
  - MVC with lateral impact
  - Pedestrian struck on Side
- Associated Injury: TBI

PELVIC FRACTURE CLASSIFICATION
LATERAL COMPRESSION (LC)

Category	Characteristics
Type I	Ipsilateral sacral buckle fractures Ipsilateral horizontal pubic rami fractures
Type II	Type I + Ipsilateral iliac wing fracture or posterior SI joint disruption
Type III	Type I or Type II on side of impact Contralateral open-book (APC) injury



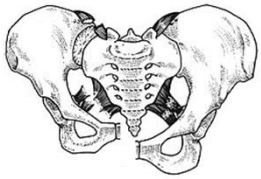
LC-I **LC-II** **LC-III**

PELVIC FRACTURE CLASSIFICATION
VERTICAL SHEAR (VS)

- Hemipelvic shearing of anterior and posterior ligaments
- Complete destabilization of injured side
- Hemipelvis Displacement: Vertical
- Etiology
  - Fall: Most Common
  - Head-On MVC
  - Heavy object to back or shoulders

PELVIC FRACTURE CLASSIFICATION
VERTICAL SHEAR (VS)

Category	Characteristics
	Vertical pubic rami fractures SI joint disruption ± adjacent fractures



VS

SOURCE OF BLEEDING

- Venous
  - Majority of Bleeding
  - Controlled with reduction of pelvic volume and stabilizing fracture segments
- Arterial
  - 20% of Pelvic Fx with hemorrhage
  - Superior Gluteal, Internal Pudendal, Obturator, Lateral Sacral
- Fracture Fragments

HEMORRHAGE CONTROL

- Early Stabilization
  - Reduces pelvic volume
  - Provides tamponade
  - Decreases Mortality: 41% to 21%
  - Decreases Transfusion: 8U to 4U

EVALUATION – HEMODYNAMICALLY NORMAL PATIENT

- Physical Exam
  - Gentle AP and Lateral compression of Iliac wings
  - Perineal Exam (Urethra/Vagina)
  - Rectal/Prostate Exam
  - Lower Extremity Deformity: Femoral Neck Fx or Hip Dislocation
  - Neurologic Assessment
  - Vascular Assessment
- Radiologic
  - Plain Film: AP Pelvis
  - CT Scan: Fine Cuts (3 – 5 mm)

EVALUATION-HEMODYNAMICALLY ABNORMAL PATIENT

- ATLS Guidelines
  - Injuries to chest/abdomen cause more hypotension than pelvic fractures
  - Spine/SCI may cause hypotension
- Radiographic
  - Rapid plain films of chest, pelvis, C-spine
  - FAST or DPL: R/O Abdomen as source of hypotension

MANAGEMENT – HEMODYNAMICALLY ABNORMAL PATIENT

- External Compression Device: Binder, Sheet
  - Stabilizes pelvis to stop movement of fractured elements
  - Decreases retroperitoneal volume
  - Pitfalls
    - Placement too high
    - Indiscriminate pulling of strings leads to excess compression
    - May compromise viability of skin, subcutaneous tissue, and muscle if left too long
  - Guideline: Maximum 24-hours in place
- Temporary Stabilization: External Fixator
  - Stabilizes fractured elements
  - Decreases pelvic volume
  - Do **after** workup for bleeding is complete
- If Stabilized – get CT Scan to:
  - Further evaluate thoracoabdomen
  - Further define fracture pattern
  - Evaluate vasculature (**if patient remains hypotensive, go directly to angiography)**

MANAGEMENT – HEMODYNAMICALLY ABNORMAL PATIENT

- Unable to Stabilize
  - Isolated Pelvic Fx: Angio-embolization or Operating Room
  - Multiple Hemorrhage Sources (+ FAST/+DPL/+CXR)
    - Operating Room
    - Address priorities simultaneously
- Operative/Post-Operative
  - Pre-peritoneal Pelvic Packing
  - Hypogastric Vessel Ligation
  - Close abdomen using Damage Control Techniques
  - Angio-embolization

OPEN PELVIC FRACTURE

- Definition: Direct communication between a fracture and either the rectum, vagina, or skin of the groin or perineum
- Incidence: 5% of pelvic fractures
- Mortality: 50%
- Early Death: Due to uncontrolled bleeding
- Management
  - Operating Room: Control hemorrhage from open wounds
  - Angio-embolization

DEFINITIVE MANAGEMENT

- LC-I and APC-I: Protected weight bearing
- LC-II and LC-III: Anterior and Posterior fixation
- APC-II: Anterior reduction/stabilization (ORIF)
- APC-III
  - External fixation to control hemorrhage
  - Posterior percutaneous iliosacral screws
- VS
  - Depends on posterior fracture location
  - Reduction with traction
  - Posterior percutaneous iliosacral screw fixation and anterior stabilization

NON-OPERATIVE MANAGEMENT of BLUNT ABDOMINAL TRAUMA

- Stable Patient
- Normal Mental Status
- No Distracting Injuries
- Absence of Multi-Trauma
- Available/Experienced Personnel
- Ability to perform frequent exams

What would be your criteria for taking a patient being managed non-operatively to the OR?

Development hemodynamic instability

-not just drop in hemoglobin

Development of peritonitis

Test documenting intraperitoneal hollow viscus injury

- Will discuss non-operative management in more detail in part II

EXPLORATORY LAPAROTOMY

- When to Go Without Fancy Work Up
  - Hypotension with other etiologies ruled out
  - Evisceration
  - Peritonitis
  - Free Air on imaging
  - Ruptured Diaphragm (may be seen on plain x-ray ± NGT)
  - Retained stabbing implement

TRAUMA LAPAROTOMY
PRIORITIES

- First: Control Hemorrhage
- Second: Control/contain gross bowel contamination
- Third: Inspect entire abdomen
- Fourth: Definitive Repair vs Damage Control

PENETRATING ABDOMINAL TRAUMA
ANTERIOR ABDOMEN

- Stab Wound
  - Wound Exploration
  - Exploratory Laparotomy
  - Laparoscopy
  - Serial Physical Exams
- Gunshot Wound
  - Exploratory Laparotomy
  - CT Scan

STAB WOUNDS
ANTERIOR ABDOMEN

- Local Wound Exploration
  - Hemodynamically normal patient
  - Evaluation for peritoneal cavity intrusion
- Exploratory Laparotomy
  - For any hemodynamically abnormal patient with no other etiology for hypotension

STAB WOUNDS
ANTERIOR ABDOMEN

- Laparoscopy
  - Specific role not codified
    - Diaphragm injuries
    - Prevention of bile peritonitis from hepatic trauma
    - Evaluation of peritoneal penetration
  - Can be difficult to evaluate retroperitoneum
  - Surgeon experience
- Serial Physical Exams
  - Asymptomatic patient
  - Positive local wound exploration

GUNSHOT WOUNDS
ANTERIOR ABDOMEN

- Exploratory Laparotomy
  - Symptomatic or Asymptomatic patients
  - Path transverses abdomen, pelvis, or retroperitoneum
  - Stable or unstable patients
- CT Scan – No Defined Role
  - Asymptomatic, Stable patient
  - Sensitivity: As low as 14%
  - False Negative: As high as 86%

PENETRATING ABDOMINAL TRAUMA
THORACOABDOMINAL/FLANK/BACK WOUNDS

- Thoracoabdomen
  - Inferior to trans-nipple line & infra-scapular line
  - Superior to costal margins
  - Includes diaphragm, liver, spleen, and stomach
- Back
  - Inferior to scapular tips, superior to iliac crest
- Flank
  - Anterior to Posterior Axillary lines
  - Sixth intercostal space to Iliac Crest

PENETRATING ABDOMINAL TRAUMA
THORACOABDOMINAL

- Laparotomy
- Serial Physical Exams
- CT is not very good for isolated diaphragm injuries
- Laparoscopy
  - Peritoneal Penetration
  - Diaphragm Injury

PENETRATING ABDOMINAL TRAUMA
BACK/FLANK

- "Triple Contrast CT" in a stable patient
  - Oral
  - IV
  - Rectal
- May not see an actual injury; look for violation of the retroperitoneum in proximity to colon, kidney, or retroperitoneal organ

QUESTIONS

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ABDOMINAL TRAUMA
PART TWO: Specific Injuries
Surgical Critical Care

David Hampton MD, MEng, FACS

September 2025

Courtesy of Fahim Habib, MD and Vishnu Mani, MD

SPECIFIC INJURIES
CHARACTERISTICS & MANAGEMENT

- Duodenum
- Pancreas
- GU
- Stomach, Small Bowel & Colon
- Rectum & Perineum
- Solid Organ Injury

DUODENUM

- Characteristics
  - Incidence
    - Overall: 0.2-0.3%
    - Patients undergoing laparotomy for trauma: 3-6%
  - Penetrating (85%) > Blunt
  - Mortality
    - Overall: 17%
    - Blunt: < 24 hr – 11%, > 24 hr – 40%
    - With concomitant Pancreas injury: 29%
- Associated Injuries (>90%)
  - Major Vascular: 48%
  - Liver: 44%
  - Colon: 31%
  - Pancreas: 30%
  - Small Bowel: 29%

PANCREAS

- Characteristics – Difficult to Diagnose
  - Low overall incidence: 0.004 – 0.6%
  - Penetrating (75%) > Blunt
  - Mortality
    - Overall: 17%
    - With Associated Injuries (< 48 hrs)
      - O – 1: 4%
      - ≥ 2: 25%
- Associated Injuries (Blunt 65%, Penetrating 90%)
  - Liver: 46%
  - Stomach: 41%
  - Major Vascular: 28%
  - Spleen: 26%
  - Kidney: 22%

DUODENUM/PANCREAS

- Diagnosis – History
  - High index of suspicion
  - Frontal impact, unrestrained OR direct abdominal blow
  - Adult: Steering Wheel
  - Pediatric: Handlebars
  - Injuries apposing the anterior wall against the spinal column

DUODENAL INJURY - AAST

Grade[a]		Description of Injury[b]
I	Hematoma	Involving single portion of duodenum
	Laceration	Partial thickness; no perforation
II	Hematoma	Involving more than one portion
	Laceration	Disruption < 50% of circumference
III	Laceration	Disruption of 50–75% of circumference of D2
		Disruption of 50–100% of circumference of D1, D3 and D4
IV	Laceration	Disruption of > 75% of circumference of D2
		Involving ampulla or distal common bile duct
V	Laceration	Massive disruption of duodenopancreatic complex
	Vascular	Devascularization of duodenum

[a] Advance one grade for multiple injuries up to Grade III
[b] Based on most accurate assessment at autopsy, laparotomy, or radiological study

DUODENUM

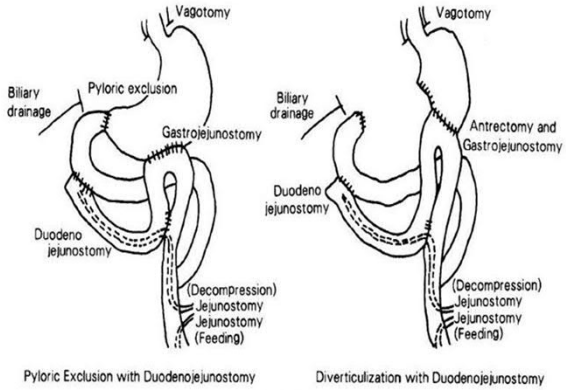
- Duodenal Hematoma (Grade I, II)
  - If No Laparotomy (blunt trauma)
    - Observation, IV hydration, NGT decompression
    - Most resolve in 3 weeks
    - If after 7 – 10 days; repeat CT; consider exploration
  - With Laparotomy/Incidental Finding
    - Controversial
    - Small Hematoma/Mild luminal compromise: Conservative/NG suction/Distal Feeding Access
    - Large Hematoma/Mass Effect & Luminal compromise: Explore/Evacuate/Distal Feeding Access

DUODENUM/PANCREAS

- Diagnosis – PE/Lab/Radiologic
  - Subtle findings to persistent abdominal pain
  - Missed duodenal injury = Peritonitis in a few days
  - Amylase/Lipase: Not diagnostic; serial elevations warrant evaluation/exploration
  - CT Scan
    - Early suspicion BEST evaluated by CT with oral/IV contrast
    - Peri-duodenal/pancreatic fluid; contrast extravasation; transection; phlegmon; hemorrhage
  - ERCP/MRCP: To exclude ductal injury
  - Most discovered at time of laparotomy
  - Cholangiopancreatography: Done at time of laparotomy to evaluate duct

DUODENUM – OPERATIVE STRATEGY

- Simple Laceration (Grade I, II)
  - Debridement
  - Primary Closure – Two layers
  - External Drainage
- Complex Laceration (Grade III, IV)
  - Some Grade III amenable to primary closure
  - Roux-en-Y Duodenojejunostomy
  - Jejunal mucosal patch
  - External Drainage
  - Pyloric Exclusion with Gastrojejunostomy
  - Duodenal diverticulization/Tube duodenostomy



DUODENUM – OPERATIVE STRATEGY

- Grade V: Whipple/Staged Management
- Feeding jejunostomy: essential adjunct

DUODENUM - COMPLICATIONS

- Complications (50%)
  - Duodenal Leak
  - Infection
  - Multi-System Organ Failure
- Duodenal Leak/Fistula Prevention
  - External Drainage
  - Retrograde Duodenostomy
  - Distal Feeding Access
  - Pyloric Exclusion

PANCREAS INJURY - AAST

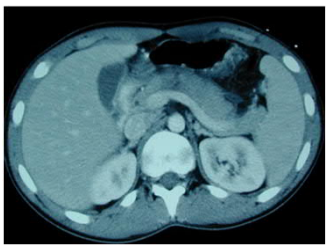
Grade[a]		Description of Injury
I	Hematoma	Minor contusion without duct injury
	Laceration	Superficial laceration without duct injury
II	Hematoma	Major contusion without duct injury or tissue loss
	Laceration	Major laceration without duct injury or tissue loss
III	Laceration	Distal transection or parenchymal injury with duct injury
IV	Laceration	Proximal[b] transection or parenchymal injury involving ampulla
V	Laceration	Massive disruption of pancreatic head

[a] Advance one grade for multiple injuries up to Grade III
[b] Proximal pancreas is to the patient's right of the Superior Mesenteric Vein

PANCREAS – OPERATIVE STRATEGY

- Governing Principles
  - Status of Main Pancreatic Duct
  - Relationship of injury to Superior Mesenteric Vessels
  - Pancreatic Insufficiency: Significant risk with resection of more than 85% of pancreas
  - Goal: Leave at least 20% of pancreas
- Damage Control
  - Packing (back in 24 hrs, NLT 72 hrs)
  - Temporary Closure
  - Further Diagnostics (e.g. ERCP, MRCP, etc)
  - Definitive Management

PANCREAS – OPERATIVE STRATEGY



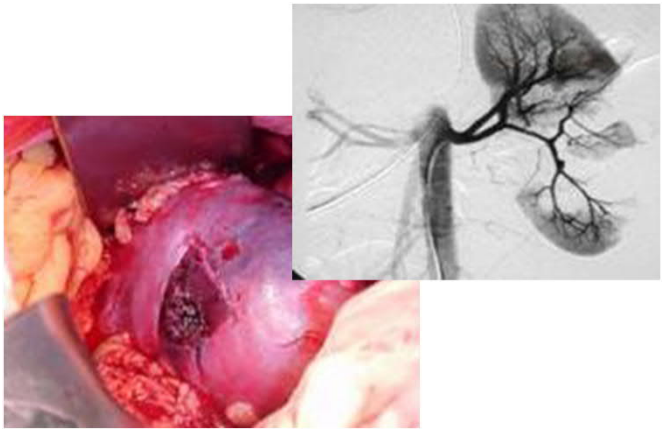
- Grade I – II
  - Non-Operative
  - At Laparotomy: External Drainage
- Grade III
  - Distal Pancreatectomy
  - External Drainage
- Grade IV
  - Extended distal pancreatectomy
  - Proximal Pancreatectomy with Roux-en-Y pancreaticojejunostomy
  - Damage Control/ERCP Stenting
- Grade V
  - Whipple (delayed)

PANCREAS - COMPLICATIONS

- Pancreatic Fistula (7-20%)
  - Low Output: Close within about 2 weeks
  - High Output: May close over 2 – 6 weeks; if not, consider intervention
  - Adjuncts: TPN, Octreotide (not studied in trauma)
- Delayed Hemorrhage (2-5%) – Due to inflammation/ enzymatic leak/erosion into vessels/rupture of pseudoaneurysm
- Pancreatic Insufficiency – Rare (with 15-20% of gland preserved)

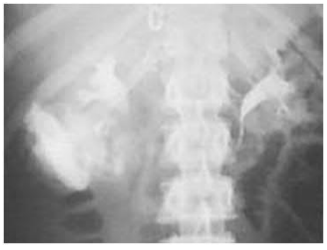
GENITOURINARY

- Kidneys
- Ureters
- Bladder
- Urethra



KIDNEY/URETER

- Characteristics – Kidney
  - Blunt (90%) >> Penetrating
  - Presence of Multiorgan injury: 80%
  - Most common GU injury
- Characteristics – Ureter
  - Penetrating > Blunt
  - 17% of GU Trauma
- Diagnosis
  - Triage Tool: Urine
  - Hematuria
    - Gross: Indicator of either bladder injury or major renal trauma
    - Microscopic: Present in 95% of renal injuries
    - NO correlation between degree of hematuria and severity of injury



KIDNEY/URETER

- Diagnosis (continued)
  - CT Scan
    - Most sensitive/specific eval for kidneys
    - Essentially has replaced IVP
    - Hemodynamically stable patient
  - Indications for Imaging
    - Penetrating Trauma
    - Blunt Trauma + Gross Hematuria
    - Blunt Trauma + Microscopic Hematuria + SBP < 90 mm Hg
  - Important Findings (CT Scan)
    - Presence/absence of contrast extravasation – collecting system or parenchyma
    - Trajectory (if penetrating)

KIDNEY

- Therapy
  - Non-Operative Management: About 85%
  - Urinary Leak: About 87% stop without any intervention
  - Indications for Intervention
    - Hemodynamic Instability
    - High Grade Injury
    - Renovascular Pedicle Injury
    - Falling Hemoglobin/Hct
    - Active Extravasation
- Selective Embolization
  - Excellent renal salvage

RENAL OPERATIVE STRATEGY

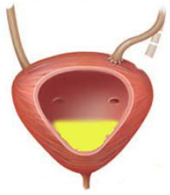
- Nephrectomy
  - Procedure of last resort
  - Hypotensive patient
  - Multiple injuries
  - Severe destruction of renal pelvis
  - Unable to control bleeding with renorrhaphy
- Renorrhaphy
  - Consider after control of hypotension
  - Options: Surface hemostasis, partial nephrectomy, mesh wrap
- Renovascular Trauma
  - Primary repair/interposition graft may be possible if patient is stable
  - Thrombosis: Operative vs. Endovascular option
  - Warm ischemia; do not attempt if > 4 hrs
  - Nephrectomy for those developing complications

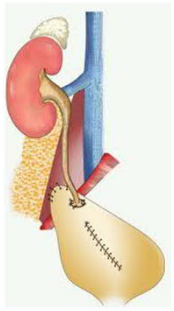
URETER OPERATIVE STRATEGY

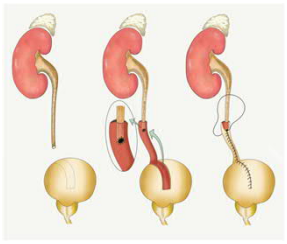
- General Rules
  - Non-Operative management has limited application
  - Early surgical repair favored
  - Spatulate ends to prevent stricture
  - Stent
  - Closed suction drainage
  - Minimal dissection
- High-Mid Ureter
  - Ureteroureterostomy
  - Renal Mobilization
  - Transureteroureterostomy

URETER OPERATIVE STRATEGY

- Low Ureter
  - Reimplantation into Bladder
  - Psoas Hitch
  - Boari Flap





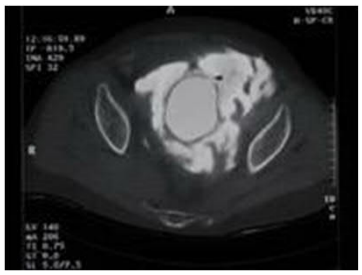


KIDNEY/URETER - COMPLICATIONS

- Early
  - Bleeding
  - Pseudoaneurysm/AV Fistula: Embolization
  - Persistent Hematuria: Eval for nontraumatic source
  - Urinoma: Cystoscopic stent ± perc drainage
  - Infection (Urinoma/Hematoma): Perc drainage
- Late
  - RV Hypertension: Medical Management
  - Ureteral Obstruction/Hydronephrosis
  - Renal Insufficiency

BLADDER

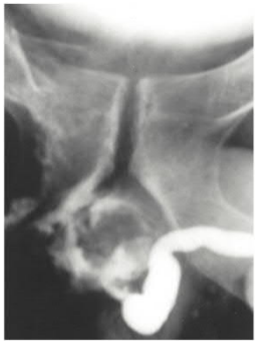
- Characteristics
  - Blunt (85%) > Penetrating
  - Blunt: 10% with Pelvic Fx
  - Contusion
  - **Extraperitoneal/Intraperitoneal**
- Diagnosis
  - Hematuria: may be present
  - Stress Cystogram
    - Plain Film
    - CT
    - 300 – 400 cc contrast (adult)



- Management
  - Extraperitoneal: Nonoperative, Foley 10 – 14 days, then cystogram
  - Intraperitoneal: Operative Primary Repair

URETHRA

- Characteristics
  - Mostly Males
  - Associated with Pelvic Fx
  - Blunt > Penetrating
- Diagnosis
  - Blood at Meatus; Prostate Exam; Inability to Void; Perineal Hematoma
  - Retrograde Urethrogram (30 cc contrast)
- Management
  - Cystoscopic Foley Placement
  - Non-Operative vs Operative Repair



Transpelvic Gunshot Wounds

- Work up in a hemodynamically normal patient (CT scan may give you much of the info you need)
  - **GU tract**
    - Retrograde urethrogram and/or cystogram
  - **Peritoneal GI tract**
    - DPL
  - **Vasculature**
    - CT of iliacs
  - **Rectum**
    - Proctoscopy
  - **Vagina**
    - Vaginal vault exam

STOMACH & SMALL BOWEL

- Characteristics
  - Incidence
    - 1 – 5% of Blunt Admissions
    - 10 – 50% of Penetrating Admissions
  - Mechanism vs Injury
    - Gunshot Wound: 80%
    - Stab Wound: 30%
    - Blunt: 1%

STOMACH & SMALL BOWEL
PENETRATING INJURIES

- Operative Management – Laparotomy
  - Hemodynamic Instability
  - Peritonitis/Peritoneal Signs
  - Positive DPL/Wound Exploration
  - Evisceration
  - Gross Blood in NGT/Rectum/Emesis
  - Clinical Judgment

STOMACH & SMALL BOWEL
BLUNT INJURIES

- Factors
  - Abdominal Tenderness/Peritonitis
  - Examination – Confounders
    - Alcohol
    - Illicit Drugs/Medications
    - CHI
    - Distraction Injury
    - Spinal Cord Injury
      - **Question on a patient whose vital signs suggest neurogenic shock: DON'T FORGET TO RULE OUT HEMORRHAGE**

STOMACH & SMALL BOWEL
BLUNT INJURIES

- Abdominal Wall Ecchymoses
- Seat Belt Sign
- Chance Fracture (Hyperflexion of lumbar spine over seat belt)
- CT Findings
  - **Free Fluid in Absence of Solid Organ Injury**
  - **Mesenteric Stranding**
  - **Bowel wall thickening or pneumatosis**
- Unexplained Findings
  - **Tachycardia**
  - **Hypotension**
  - **Hyperamylasemia**
  - **Metabolic Acidosis**
  - **Leukocytosis**

STOMACH & SMALL BOWEL
BLUNT INJURIES

- Management of CT Free Fluid (No Solid Organ Injury) – No Consensus
  - Observation
  - Repeat CT
  - Evaluate with DPL
  - Diagnostic Laparoscopy
  - Laparotomy
- Greater than Trace (> 50 ml) Fluid: Evaluation recommended

STOMACH INJURY MANAGEMENT

- Inspect Anterior/Posterior Walls/GE Junction
- Anterior Hole = Look for a second hole
- Small Perforations
  - Close in 1 or 2 layers
  - Hand-sewn or GIA stapler
- Extensive Wounds
  - Partial Gastrectomy
  - Billroth I or II reconstruction

SMALL BOWEL INJURY MANAGEMENT

- Partial Thickness: Primary Repair
- Full Thickness (< 50%): Primary Repair
- Indications for Resection/Anastomosis
  - Multiple large full-thickness defects in short segment
  - Associated devascularizing mesenteric injury
- Stapled vs Hand Sewn Anastomosis
  - No clear data
  - Complication rate similar
  - Consider edema with stapled anastomosis

STOMACH & SMALL BOWEL
COMPLICATIONS

- Wound Infection
- Anastomotic Breakdown/Fistula/Leak
- Abscess
- Delayed Hemorrhage
- Prolonged Ileus
- Missed Injury
- Small Bowel Obstruction

COLON

- Epidemiology
  - Penetrating >> Blunt
  - Gunshot Wound: 27% (undergoing laparotomy)
  - Blunt: 0.5% overall
- Most Common Location
  - Gunshot Wound: Transverse Colon
  - Stab Wound: Left Colon

COLON INJURY
DIAGNOSIS & MANAGEMENT

- Diagnosis
  - Most made intraoperatively
  - CT Scan with Contrast
    - Sensitivity: 90%; Specificity: 96%
    - Pneumoperitoneum
    - Unexplained free peritoneal fluid
    - Thickened colonic wall
- Management
  - Injury < 50% bowel wall/no devascularization: Primary repair
  - Injury > 50% bowel wall or devascularization: Resection
- Colostomy vs Anastomosis
  - Hypotension at Admission
  - PATI > 25
  - Severe fecal contamination
  - Blood Transfusion > 6 units
  - Delay of operation > 6 hrs

COLON INJURY
COMPLICATIONS

- Abdominal Septic Complications
  - Incidence: 16 – 33%
  - Risk Factors
    - Multiple blood transfusions (≥ 4 Units)
    - Severe fecal spillage
    - Operative Delay (timing not clear)
- Wound Infection
  - Skin Closure, especially with Fecal Spillage
- Colon Leaks
  - Incidence: 2.2%
  - Resection/Anastomosis >> Simple Repair
  - Most managed nonoperatively with adequate drainage

RECTUM & PERINEUM

- Etiology
  - Penetrating: > 90% (GSW – 85%)
  - Blunt: 5 – 10%
  - Open Pelvic fracture: 1 – 2% have rectal trauma
- Diagnosis
  - Digital Rectal Exam
  - Rigid Proctosigmoidoscopy
  - CT Scan

RECTUM & PERINEUM

- Intraperitoneal Injury
  - Most are amenable to Primary Repair
  - Colostomy: Severe contamination, stability, transfusion
- Extraperitoneal Injury
  - Diversion
    - Loop Colostomy vs Hartmann's
  - Debridement and Primary Repair
    - +/- Colostomy
    - Small Perforations: ?No colostomy
  - Drainage- Presacral Drains
  - Distal Washout

SOLID ORGAN INJURY – LIVER

- Characteristics
  - Most Common **solid** organ injured in Penetrating Trauma
  - MVC: Most common etiology for liver injuries
  - Associated Injuries: Up to 2/3 of those with Penetrating Trauma
  - Diaphragm Injury: 3 – 5% of patients

SOLID ORGAN INJURY – LIVER

- Diagnosis
  - Hemodynamically Normal
    - CT Scan
      - Gold Standard
      - Classifies injury
      - Evidence of associated organ injuries
      - Degree of hemoperitoneum
      - Evidence of Active Hemorrhage
  - Hemodynamically Abnormal
    - FAST: Presence of intraperitoneal fluid
    - DPL: Presence of intraperitoneal blood

LIVER INJURY - AAST

Grade[a]		Description of Injury
I	Hematoma	Subcapsular, < 10% surface area
	Laceration	Capsular tear, < 1 cm parenchymal depth
II	Hematoma	Subcapsular, 10 – 50% surface area; intraparenchymal < 10 cm diameter
	Laceration	Capsular tear < 1 – 3 cm parenchymal depth; < 10 cm in length
III	Hematoma	Subcapsular, > 50% surface area of ruptured subcapsular or parenchymal hematoma; intraparenchymal hematoma > 10 cm or expanding
	Laceration	> 3 cm parenchymal depth
IV	Laceration	Parenchymal disruption involving 25 – 75% hepatic lobe or 1 – 3 Couinaud's segments
V	Laceration	Parenchymal disruption involving > 75% of hepatic lobe or > 3 Couinaud's segments within a single lobe
	Vascular	Juxtahepatic venous injuries; ie. retrohepatic vena cava/central major hepatic veins
VI	Vascular	Hepatic Avulsion
[a] Advance one grade for multiple injuries up to Grade III		

MANAGEMENT – LIVER

- Non-Operative
  - Hemodynamically Stable
  - Absence of Peritoneal Signs
  - Absence of Other need for Surgery
  - Absence of Injury precluding Abdominal Exam
  - Limited Transfusions
  - Success Rate: 85%
- Use of Angioembolization
  - Hemodynamically Stable
  - CT Scan with Contrast Blush

LIVER
COMPLICATIONS – NON-OPERATIVE MANGEMENT

- Predictors
  - Significant Coagulopathy
  - Grade V Injury
- Specific Treatment
  - Delayed Hemorrhage: Angioembolization
  - Abscess: CT-Guided drainage
  - Biloma: CT-Guided drainage
  - Hemobilia: Angioembolization = Dx and Therapy
  - Hepatic Necrosis: Operative Debridement
  - Acalculous Cholecystitis: Cholecystectomy

MANAGEMENT – LIVER

- Operative
  - Hemodynamic Instability
  - ?Penetrating Trauma
  - ? Increasing Transfusion Requirement (> 4 units pRBCs)
  - Newly diagnosed hollow viscus injury/peritonitis
  - Failed Angio-Embolization

MANAGEMENT – LIVER

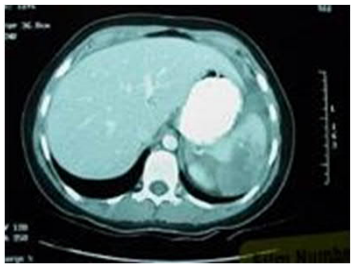
- Hemorrhage Control
  - Packing
  - Pringle Maneuver
  - Selective HA Ligation (if Pringle works)
  - Resectional Debridement
  - Tractotomy/Vessel Ligation
  - Partial Lobectomy
  - Local procoagulant agents
  - Hepatic Sutures
  - Topical Hemostatic Agents
  - Shunt/Vascular Isolation

LIVER
COMPLICATIONS –OPERATIVE MANGEMENT

- Specific Treatment
  - Bleeding: Angioembolization or Operative Intervention
  - Abdominal Compartment Syndrome: Decompression
  - Hemobilia: Angioembolization
  - Biliary Fistulae: Fistula control
  - Hepatic Necrosis: Operative Debridement

SOLID ORGAN INJURY – SPLEEN

- Characteristics
  - Most Common solid organ injured – **Blunt** Trauma
- History/Physical
  - LUQ Mechanism
  - Asymptomatic
  - Kehr's Sign
  - Left Rib Fractures



SOLID ORGAN INJURY – SPLEEN

- Diagnosis/Management
  - Hemodynamically abnormal
    - FAST/DPL or laparotomy
    - Angiography
  - Hemodynamically normal:
    - CT Scan
    - Consider Non-operative Management

SPLENIC INJURY - AAST

Grade[a]		Description of Injury
I	Hematoma	Subcapsular, < 10% of surface area
	Laceration	Capsular tear, < 1 cm parenchymal depth
II	Hematoma	Subcapsular, 10 – 50% surface area
	Laceration	Capsular tear, 1 – 3 cm parenchymal depth that does not involve a trabecular vessel
III	Hematoma	Subcapsular, > 50% surface area or expanding; ruptured subcapsular or parenchymal hematoma; intraparenchymal hematoma ≥ 5 cm or expanding
	Laceration	> 3 cm parenchymal depth or involving trabecular vessels
IV	Laceration	Laceration involving segmental or hilar vessels producing major devascularization (> 25% of spleen)
V	Laceration	Completely shattered spleen
	Vascular	Hilar vascular injury which devascularized spleen
[a] Advance one grade for multiple injuries up to Grade III		

MANAGEMENT - SPLEEN

- Criteria For Non-Operative Management
  - No indications for laparotomy
  - Hemodynamically stable after resuscitation
  - No factors precluding reliable exam of abdomen
  - Transfusion Limitations
- Non-Operative Strategy
  - NPO
  - Bedrest
  - Serial Physical Examination
  - Serial hemoglobin
  - Monitored Setting
  - ? Repeat CT 24 – 72 hours (for Gr III and above)

MANAGEMENT – SPLEEN

- Use of Angioembolization
  - CT Scan with Contrast Blush
- Results – Non-Operative Management
  - Initial Candidates: 60%
  - Success
    - Adult: 80 – 94%
    - Pediatric: > 95%
  - Failures: Most within 72 hours

MANAGEMENT – SPLEEN

- Factors Associated With Failure of Non-Operative Management
  - Age > 55
  - Volume of Hemoperitoneum (S/M/L)
    - Moderate: 50% failure
    - Large: 75% failure
  - **Grade of Injury – EAST Failure Rate**
    - **Gr I & II: 10%**
    - **Gr III: 20%**
    - **Gr IV: 33%**
    - **Gr V: 75%**
  - Presence of Pseudoaneurysm
    - On Admission or Follow-Up CT Scan
    - 25% are seen on Admission
    - 75% are seen on Follow-Up

MANAGEMENT – SPLEEN

- Indications for Operative Management
  - Hemodynamic Instability
  - Laparotomy for Other Injuries
  - Failed Non-Operative Management
- Operative Strategy
  - Splenorrhaphy: Stable; salvageable spleen
  - Splenectomy
- Post-Operative
  - Complications: Bleeding, infection, pneumonia, abscess
  - OPSI: Up to 2% splenectomized pts; overall mortality 0.6%
  - Vaccination: Pneumococcal, meningococcal, HiB

DAMAGE CONTROL SURGERY

- Lethal Triad
  - Coagulopathy
  - Hypothermia
  - Acidosis
- Principles of Damage Control
  - Control Hemorrhage
  - Control Contamination
  - Packing/Temporary Abdominal Closure
  - Further Resuscitation in ICU

DAMAGE CONTROL SURGERY

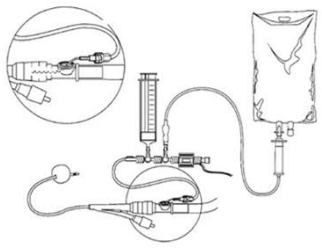
- Definitive Therapy Principles
  - Return to OR
    - Stabilized, normothermic patient, metabolically normal patient
    - Timing: As short as 6 hours or up to 24 – 48 hours
  - May require additional operations over time
  - Definitive Therapy/Reconstruction
  - Abdominal Wall Closure

ABDOMINAL COMPARTMENT SYNDROME

- Intra-abdominal Hypertension (IAH) Leads to:
  - Decreased Cardiac Output/Stroke Volume/Venous Return
  - Respiratory Failure: High Insp Pressure, Hypoxia, Hypercarbia, Decreased Compliances
  - Renal Failure: Decreased RBF, GFR, Increased Renin, Increased Aldosterone
  - Gut Mucosal Acidosis: Splanchnic hypotension
- Risk Factors
  - Preoperative hypovolemic shock and massive fluid resuscitation
  - Increased intraabdominal fluid accumulation
  - Mechanical increase in abdominal pressure

ABDOMINAL COMPARTMENT SYNDROME

- Prophylaxis
  - "Open Abdomen" at initial operation
  - Monitor IAP in high-risk patients
  - Intervene if IAH occurs
- Intraabdominal Pressure
  - Normal: < 10 cm H_20
  - Surrogate Method: Bladder Pressure Measurement
- Classification
  - Grade I: 10 – 15 cm H_20
  - Grade II: 15 – 25 cm H_20
  - Grade III: 25 – 35 cm H_20
  - Grade IV: > 35 cm H_20



ABDOMINAL COMPARTMENT SYNDROME

- When To Intervene
  - Most Grade III and All Grade IV
  - Clinical Judgment
- Therapeutic Approach
  - May need to sedate ± paralyze to get accurate reading
  - Decompressive Laparotomy
  - Re-opening of Temporary Closure
  - Replacement of Temporary Closure

QUESTIONS

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ENDOCRINE EMERGENCIES

Surgical Critical Care

September 2025

Updated by Gaby Iskander, MD, MS. FACS

Calcium physiology

- Decrease Ca & Mg and β stimulation will stimulate PTH release
- PTH has N terminal (higher activity and shorter half lifetime). Carboxyl terminal measure

PTH: 3 Sites of Action

- Bone: calcium resorption with calcium elevated in blood
- Kidney: acts on distal tubules to increase Ca absorption and decrease Phosphorous absorption, helps Vitamin D hydroxylation
  - Calcium will be high in blood and urine in hyperparathyroidism
  - In Familial hypercalcemic hypocalciuria, Calcium will be low in urine (used to be most common cause of negative neck exploration) Always check urine Calcium
- GI Tract PTH increases Ca absorption

Causes of Hypercalcemia

- Malignant tumors (ICU patients)
- Hyperparathyroidism (primary and tertiary) not secondary
- Drugs (hydrochlorothiazide- lithium)
- Sarcoidosis
- Thyrotoxicosis
- Milk alkali syndrome
- Paget's disease of bone
- TB

Causes of Hypercalcemia in Hyperparathyroidism

- Primary
  - Single adenoma 80%
  - Adenoma 4%
  - Hyperplasia 2%
  - Cancer 1% = mass, lethal
- Tertiary
  - Persistent stimulation of parathyroid by hypocalcemia
  - After successful renal transplantation
- Familial Hypercalcemia hypocalciuria
- Sarcoidosis

EKG in hypercalcemia

- Short QT interval
- Increase PR and QRS interval
- T wave becomes flat
- AV block

Treatment

- Saline IV
- Diuretics
- Dialysis
- Bisphosphonate: Osteoclast inhibitors, pamidronate 60 gm IV over 4 hours – one dose every seven days
- Calcitonin
- Plicamycin
- Corticosteroids
- Calcium channel blockers
- Urgent neck exploration after sestamibi scan

Hyperparathyroidism Diagnosis

- Increase Calcium in blood and urine
- Increase Phosphorus in urine and decrease phosphorus in blood
- Chloride / Phosphorus ratio > 33
- Increase HCO3 in Urine
- Almost similar to mild renal tubular acidosis

Thyrotoxicosis

- TSH is low, high T3, T4
- Causes
  - Grave's disease
  - Toxic multinodular goiter
  - Toxic adenoma
  - Sub acute thyroiditis

Severe Thyrotoxicosis / Thyroid Storm - Symptoms

- Life threatening
- Fever out of proportion for infection
- Atrial tachycardia
- Hypotension resistant to inotropic support
- 50% mortality if unrecognized

Treatment of Thyroid Storm

- Immediate hydration. Saline until urine output improves
- β blockers to control tachycardia
- Hypotension is treated with direct vasocontrictor agents
- Propylthiouracil

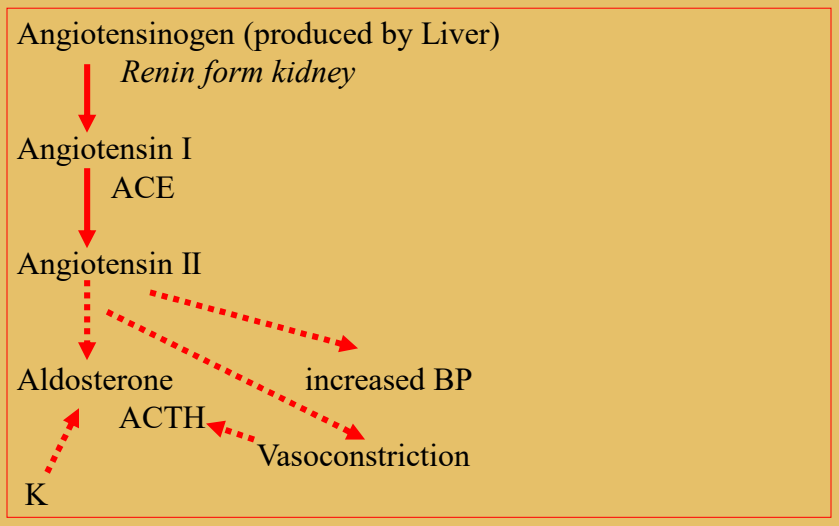
Hypothyroidism

- Hypothyroidism = Low T4, T3 with high TSH
- **Sick Euthyroid state:** ICU patient. Low T4,T3 but normal TSH

Adrenal gland physiology

- ZONA GLOMERULOSA: Aldosterone production
- Factors stimulate production are:
  - angiotensin II from renin pathway
  - Hyperkalemia
  - hyponatremia
- Factors inhibit production are:
  - Atrial nartiuretic hormone
  - Dopamine
  - ACTH plays some role in Aldosterone

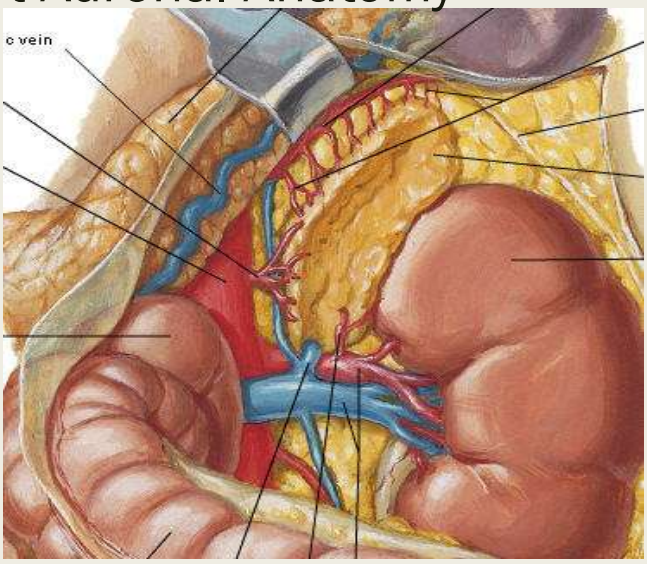
Regulation of Aldosterone secretion



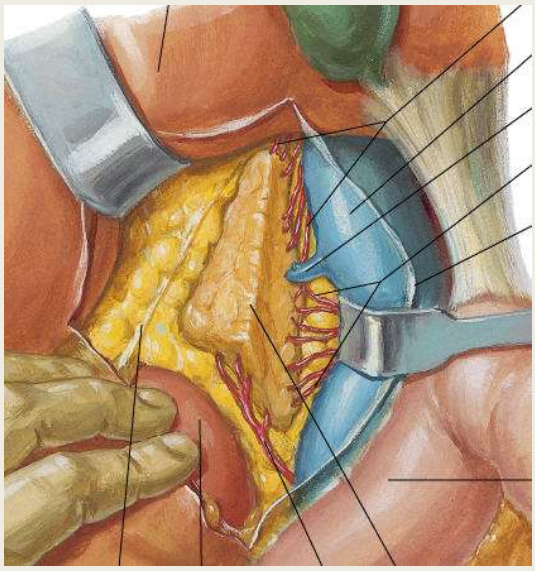
Adrenal Gland Anatomy

- Multiple small arteries mainly from aorta from celiac, SMA, inf phrenic, renal
- One single vein to IVC on right and to left renal vein on left

Left Adrenal Anatomy



Right adrenal anatomy



Hyperaldosteronism: 2 Types

- Primary = adrenal gland overproduction by an adenoma
  - Conn syndrome (surgically treated)
  - Hyperplasia (medically treated)
- Secondary = adrenal gland is normal
  - Causes are renovascular hypertension (RVH), liver cirrhosis, CHF

	Primary Hyperaldosteronism	Secondary
Causes	Adrenal gland • Adenoma • Hyperplasia	Renovascular HTN Liver cirrhosis CHF
RENIN Level	**LOW and remains low even with provocative tests**	**Always high**
Hypertension	Mild. Easy to treat	Severe, several BP medications
Other features	Alkalosis Hypokalemia	RVH, young with high BP

Decreased Adrenal Function

- Causes:
  - Drugs: etomidate, ketoconazole
  - Adrenal hemorrhage
  - Tumors
  - Infections
  - Auto immune disorders
- Clinically: hypotension, weakness, hyponatremia, hyperkalemia

Decreased Adrenal Function

- Random cortisol level:
  - Less than 15 ug/dl in the ICU setting is almost diagnostic of hypoadrenalism
  - If more than 34 ug /dl. It almost rules out hypo-adrenalism
  - ACTH stimulation test is done if the random cortisol level is between 15 - 34

Decreased Adrenal Function

- Treatment:
  - IV Hydrocortisone 50 mg every 6 hours
  - Treatment of hypovolemia and hypoglycemia
  - In septic, critically care patients, glucocorticoid therapy improves hemodynamics
  - After unilateral adrenalectomy, supplemental therapy may be needed for few months until the other gland compensates

Pheochromocytoma

- 90% adrenal – 10% extra adrenal
- 90% Benign – 10% Malignant
- 90% Unilateral – 10% Bilateral
- Symptoms:
  - Intermittent HTN, Headache, Palpitations
- Norepinephrine > epinephrine
- VMA in urine
- MIBG I131 Localization

Antidiuretic hormone

- Secreted by the hypothalamus and is stored in the posterior pituitary gland
- Level after trauma or stress correlates to the degree of stress
- Disturbances lead to Diabetes insipidus or SIADH
- PEEP increases ADH secretion

Antidiuretic hormone

- ADH is released by decrease in blood volume or increase in serum osmolarity.
- ADH then acts on
  - distal tubules to reabsorb water(Aquaporins) Inserted into the cell membrane under the influence of ADH
  - the liver causing glycogenolysis and gluconeogenesis to increase the circulating plasma volume
- Normally ADH persists for few days after any surgery
- When prolonged=SIADH
  - highly concentrated small volume urine
  - dilution hyponatremia
    - serum osmolarity < 275,urine osmolarity > 100

Serum Osmolarity (calculated)

- $2(Na) + \frac{Glucose}{18} + \frac{BUN}{2.8}$
- Normal serum osmolarity 280
- Urine osmolarity 200 ---1200

	Serum osmo	Urine osmo
Dehydration	increased	increased
Overhydration	decreased	decreased
SIADH	decreased	increased
Diab Insipidus	increased	decreased

Diabetes insipdus

- Loss of ADH could be central or nephrogenic.
- Head trauma, tumors, are some causes of central
- Sickle cell disease, lithium are common causes of nephrogenic

Diabetes insipdus

- Dilute, hypo-osmolar urine
- > 400 ml/h in the setting of pituitary surgery or head trauma
- Simultaneous serum and urine osmolarity values:
  - serum osmolality is high (310-320)
  - urine is inappropriately diluted (less than 200)
  - Serum Na levels are elevated

Diabetes insipdus

- Treatment is DDAVP, side effects are hyponatremia and coronary artery disease
- In nephrogenic type: mild salt restriction and thiazide diuretics

SIADH

- Tumors, trauma
- High urine osmolarity (higher than serum value)
- Urine sodium concentration >20 meq/l
- Serum sodium concentration <130
- Serum Na level less than 110 → cerebral edema, seizures
- Treatment:
  - Fluid restriction (800-1000) ml /day = primary treatment for mild and moderate SIADH
  - Role of hypertonic saline is limited to symptomatic patients with coma or seizures

Carcinoid tumor/syndrome

- Small bowel carcinoid
- Liver metastasis are present
- Octreotide is the first line
- Debulking of hepatic metastasis and resection of primary
- Common symptoms:
- Flushing
- Wheezing
- Diarrhea
- Fatigue

ONCOLOGIC EMERGENCIES AND TUMOR LYSIS

Learning Objectives

- Identification of 3 major oncologic emergencies
- Management of tumor lysis syndrome
- Management of neutropenic fever
- Management of spinal cord compression

Oncologic Emergencies

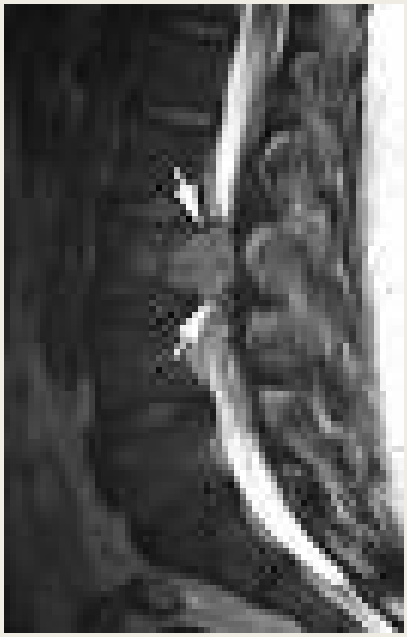
- 4 Major types
  - Metabolic emergencies (hypercalcemia, hyponatremia, hypoglycemia, adrenal failure, lactic acidosis)
  - Hematologic emergencies (hyperleukocytosis, DIC, thrombosis)
  - Infectious / Inflammatory emergencies (typhlitis, pancreatitis, chemo infiltration, hemorrhagic cystitis)
  - Mechanical emergencies (cerebral herniation/status epilepticus, cardiac tamponade, SVC syndrome?)

Acute Spinal Cord Compression

- Tumors with high chance of spinal cord compression are:
  - Breast Ca
  - Lung Ca
  - Malignant melanoma
  - Multiple myeloma
  - Prostate cancer
- Acute cord compression is caused by epidural mets
- Rarely by intramedullary mets

LOCATION:

- Thoracic spine: 60%
- Lumbosacral spine: 30%
- Cervical spine: 10%



Acute Spinal Cord Compression

- Paralysis, muscle weakness, sensory loss, bowel and bladder symptoms
- Pain in the area is usually present some time before and is exaggerated by coughing or Valsalva
- 80% of patients have weakness
- 50% have sensory loss
- MRI is the test of choice, before surgery and has replaced Myelogram

Acute Spinal Cord Compression

- Diagnosis of epidural compression mandates immediate therapy even before completion of radiological examination especially in patient with rapidly evolving neurologic signs
- Dexamethasone 100 mg IV followed by 24 mg every 6 hours for 72 hours and then tapered over 2 weeks offers significant pain improvement in few hours for most patients
- Definite therapy is by surgical decompression or radiotherapy with almost equal results

Acute Spinal Cord Compression

- Recurrence after radiotherapy is treated by surgical decompression by anterior approach

Neutropenic Enterocolitis

- Also known as typhlitis, necrotizing enterocolitis, ileocecal syndrome
- Associated with fever, diarrhea, abdominal pain and distention in oncologic patients with neutropenia
- Leukemia is the most common malignancy
  - followed by other hematological cancers
  - rarely solid tumors

Neutropenic Enterocolitis

- Mucosal inflammation of terminal ileum, caecum and ascending colon
- Lymphoma cells or leukemic cells precipitates in the mucosa
- Prior use of antibiotics lead to selection and overgrowth of bacteria

Neutropenic Enterocolitis

- Differential Diagnosis:
  - Appendicitis, C. difficile colitis, ischemic colitis
- Symptoms:
  - Fever
  - Abdominal pain
  - Loose bowel movements
- It is a diagnosis of exclusion

Neutropenic Enterocolitis

- Medical therapy:
  - Bowel rest
  - TPN
  - Broad spectrum antibiotics
- Surgical exploration is done to rule out perforation
- Measures to raise WBC may help
- Recurrence is common

Acute Tumor Lysis Syndrome

- Definition:
  - Acute metabolic and renal complication from acute dissolution of bulky tumors which are highly sensitive to radiation therapy or chemotherapy
  - Intracellular products when rapidly released into the circulation can produce acute hyperkalemia, hyperphosphatemia, hypocalcemia and most importantly hyperuricemia (uric acid more than 18 mg/dl) with or without acute renal failure.
  - The degree of renal dysfunction depends on the base line renal function and the degree of tumor dissolution

Tumor Lysis Syndrome

- Metabolic derangements caused by the massive and abrupt release of cellular components into the blood after the rapid lysis of malignant cells. (↑phos, ↑K, ↑uric acid, ↓Ca)
- **Uric acid crystals** and/or **$CaPO_4$** in renal tubules = impaired renal function, ARF, even death
- **↑phos** leads to **↓Ca**: tetany, seizures, arrhythmia
- **↑K** = life-threatening arrhythmia

Tumor Lysis Syndrome

- High tumor cell proliferation rate
- Large tumor burden
- Tumor chemosensitivity
- ALL, AML, NHL, Burkitt's Lymphoma (heme malignancies)
- Small cell >>> Hodgkin's disease
- Multiple Myeloma, Solid Tumors (breast, GI, prostate etc.)

Tumor Lysis Syndrome

- Most common Tumor is Burkitt's lymphoma and other lymphoma
  - Also seen in leukemia
- Acute renal failure most commonly occurs from intratubular precipitation of uric acid crystals or calcium phosphate crystals
- Post tubular obstructive uropathy with uric acid stones blocking the renal pelvis or ureter
  - may also occur if the release of uric acid is seen more slowly

Tumor Lysis Syndrome

- Signs and Symptoms are non-specific
  - Can occur before chemo, but usually within 12 to 72hrs after starting chemo

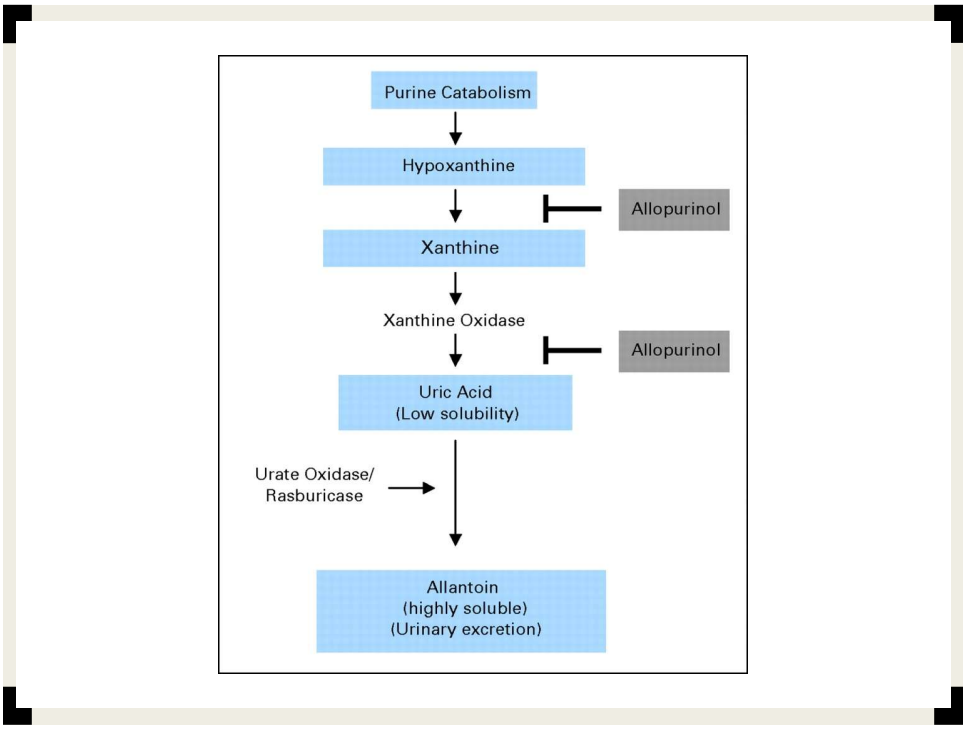
Nausea	Lethargy
Vomiting	Edema
Diarrhea	Fluid overload
Anorexia	Cramps
Syncope	Sudden death

Tumor Lysis Syndrome

- Usually develops after chemotherapy (paclitaxel, fludarabine, etoposide, thalidomide, bortezomib, and hydroxyurea)
- Can occur after radiation therapy, corticosteroids, chemoembolization, intrathecal chemotherapy, rarely from spontaneous necrosis
- LDH is considered by some a measure of tumor load and a marker of TLS risk

Tumor Lysis Syndrome Prevention & Management

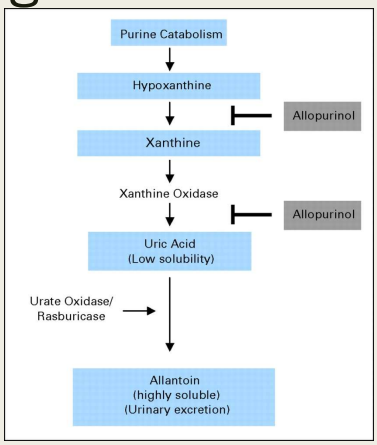
- "*The best management is prevention*."
- FLUIDS and HYDRATION:
  - Aggressive hydration and diuresis
  - Improve intravascular volume, renal blood flow, GFR (decrease [solute] in distal nephron/renal microcirculation)
  - +/- diuretics (contraindicated in hypovolemia and obstructed uropathy)



Tumor Lysis Syndrome Prevention & Management

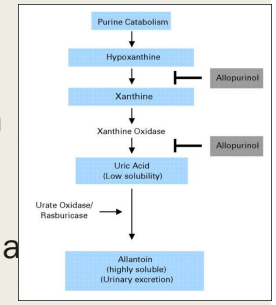
ALKALINIZATION OF URINE:

- Uric acid > 10x's more soluble in pH of 7.0 compared to pH of 5.0
- Xanthine/hypoxanthine is also significantly more soluble in basic urine
- Historically used, but not based on evidence-based practice.
- Complications of alkalinization outweighs benefits (calcium phosphate precipitation, metabolic alkalosis)



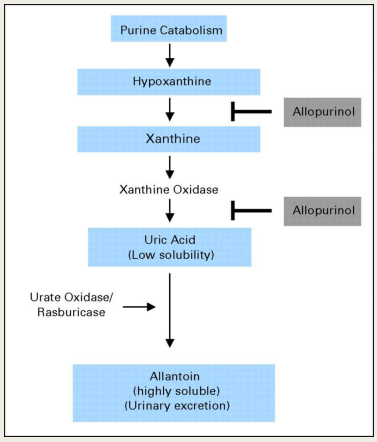
Tumor Lysis Syndrome Prevention & Management

- **ALLOPURINOL:**
  - Competitive inhibitor of xanthine oxidase which decreases conversion of purine metabolites to uric acid. Used prophylactically for TLS
  - Prophylactic option for patients with a medium risk of TLS
    - Limitations:
      1) *ineffective in reducing uric acid levels before chemoTx*
      2) *Xanthine and hypoxanthine precipitate → obstructive uropathy*
      3) *reduces clearance of some chemoTx (azathioprine & 6-mercaptopurine)*



Tumor Lysis Syndrome Prevention & Management

- **RASBURICASE** (recombinant urate oxidase):
  - promotes catabolism of uric acid:
    - Uric acid → allantoin (10x more soluble than uric acid)
  - 100 adult pt (w/ aggressive NHL) got 3 to 7 days of rasburicase beginning day 1 of chemo:
    1) Uric acid levels decreased w/i 4 hrs of rasburicase
    2) Normalized uric acid levels maintained throughout chemo
    3) No increase in creatinine observed
    4) No patient required dialysis



Clinical Management

- Patients with bulky abdominal tumor, elevated plasma lactate dehydrogenase level, evidence of renal dysfunction or metabolic alterations require careful monitoring and renal protection during the induction of antineoplastic therapy
- If the metabolic burdens appears great or ARF ensues, dialysis is initiated.
- The mainstay in prophylaxis against ARF is vigorous hydration and maintaining urine output of at least 2ml/kg/h
- This is mainly achieved by saline
- If saline hydration can not do it, add dopamine at 2ug/kg/min

Hypercalcemia

- Hydration
- Diuretics
- Bisphosphonate most specific for hypercalcemia of malignancy, given IV. Response occurs in 2-4 days (zoledronate)
- Calcitonin: rapid within 12 hours and can be used in renal failure where others are contraindicated
- Mithramycin: used to be first choice

Malignant Effusion

- Commonly associated with:
  - Lung ca
  - Breast ca
  - Ovarian ca
- Typically presents with rapid effusion and respiratory symptoms
- Life expectancy is variable depending on primary tumor and disease stage

Malignant Effusion

- If lung is trapped and effusion loculated, symptoms are worse and intervention is needed
- PleurX: tunneled valve catheter has allowed many patients to be treated outside

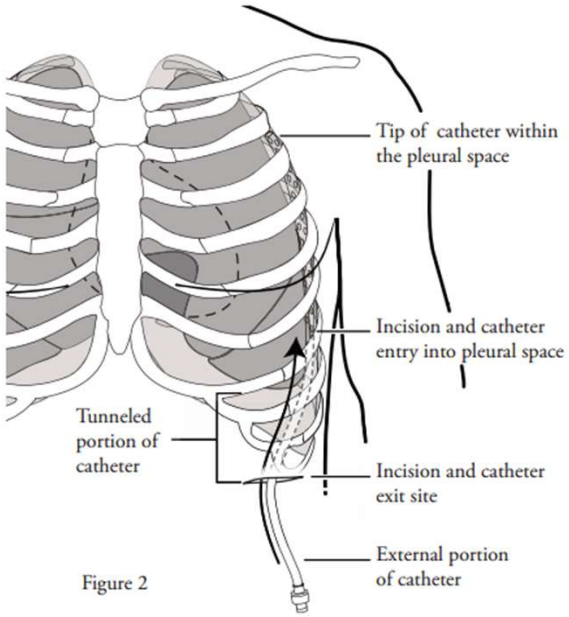


Figure 2

Infection in Cancer Patients

- Neutropenia is a risk factor
- Indwelling venous ports,
- Gram positive (gm +)
- Fungus, Gram negative (gm-)

Febrile Neutropenic Patient

- One of the following is acceptable monotherapy:
  - Ceftazidime
  - Cefepime
  - Imipenem
  - Meropenem
  - Piperacillin/tazobactam (a broad-spectrum antipseudomonal penicillin)

Febrile Neutropenic Patient

The addition of vancomycin should be reserved for specific settings such as:

1) clinically apparent, catheter-related infection:
2) blood culture-confirmed presence of a Gram-positive bacterium before identification and susceptibility testing occurs:
3) known colonization with methicillin-resistant S aureus (MRSA) or with penicillin/cephalosporin-resistant pneumococci: (4) hypotension or septic shock without an identified pathogen:
4) b-lactamase allergies

Febrile Neutropenic Patient

- Empiric antifungal therapy
  - febrile neutropenia of unknown cause unresponsive to 4 to 7 days of appropriate antibiotic therapy
- Agent active against Candida and Aspergillus species
  - Amphotericin B formulations
  - Itraconazole (cyclodextrin formulation)
  - Voriconazole
  - Caspofungin

Question 1

Which of the following works to lower hypercalcemia of malignancy before all others treatment

A. Lasix
B. lactated ringer
C. calcitonin
D. bisphosphonate

Answer

C: calcitonin

- Calcitonin lower calcium in 4 hours but does not last for long and biphosphonate has to be given simultaneously
- All other drugs take longer time to act

Question 2

Most common cause of carcinoid crisis in patients with carcinoid tumors is seen in

A. appendix tumor
B. foregut carcinoid
C. midgut carcinoid
D. lung carcinoid

Answer

C: midgut carcinoid

- Midgut tumors have the worst prognosis and commonly have associated tumors with the syndrome develop in 30% of these tumors
- Somatostatin is drug of choice for treatment of crisis

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ASA Classes

1- normal healthy patient

2- mild systemic disease

3- severe systemic disease

4- severe systemic disease with constant threat to life

5- moribund patient not expected to survive without operation

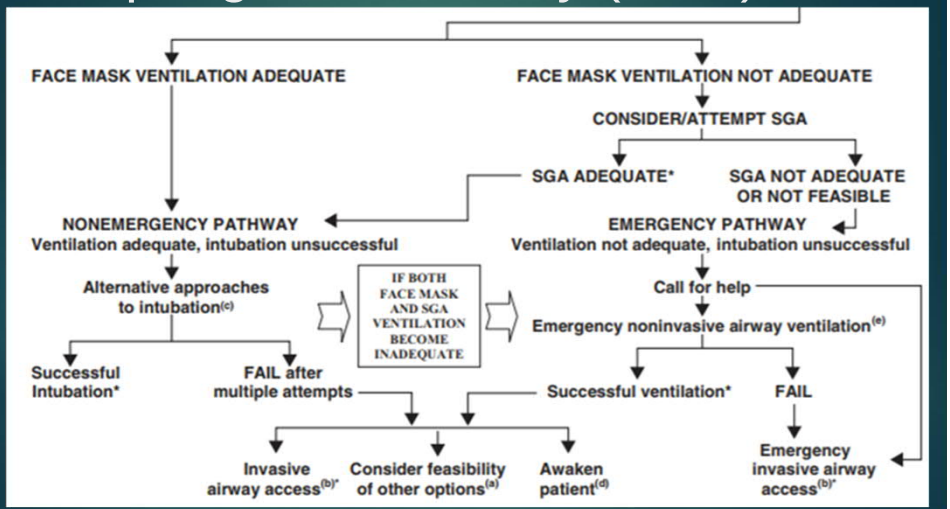
6- declared brain dead patient whose organs are being removed for donor

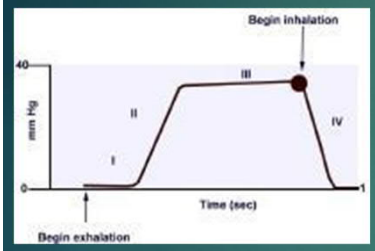
E is added for emergency

Video-assisted Laryngoscopy

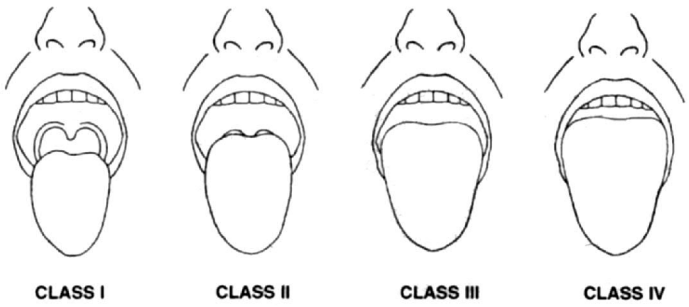
- Meta-analyses of RCTs comparing video-assisted laryngoscopy with direct laryngoscopy in patients with predicted or simulated difficult airways
- Video-assisted laryngoscopy associated with:
  - Report improved laryngeal views
  - Higher frequency of successful intubations
  - Higher frequency of first attempt intubations

Supraglottic Airway (SGA)





- Phase I of the capnogram represents baseline inspired gas (normally devoid of carbon dioxide).
- Phase II represents expired air with an upward sloping curve This phase displays the transition from dead space air flow to alveolar air flow.
- Phase III, the plateau, represents the partial pressure of carbon dioxide exchanged at the alveoli. This plateau number represents end-tidal carbon dioxide.
- Phase IV, downward deflection, represents the inspiratory point in the ventilatory cycle. This phase displays the transition from alveolar air flow to fresh air inflow, or "scrubbed" air in a closed circuit.



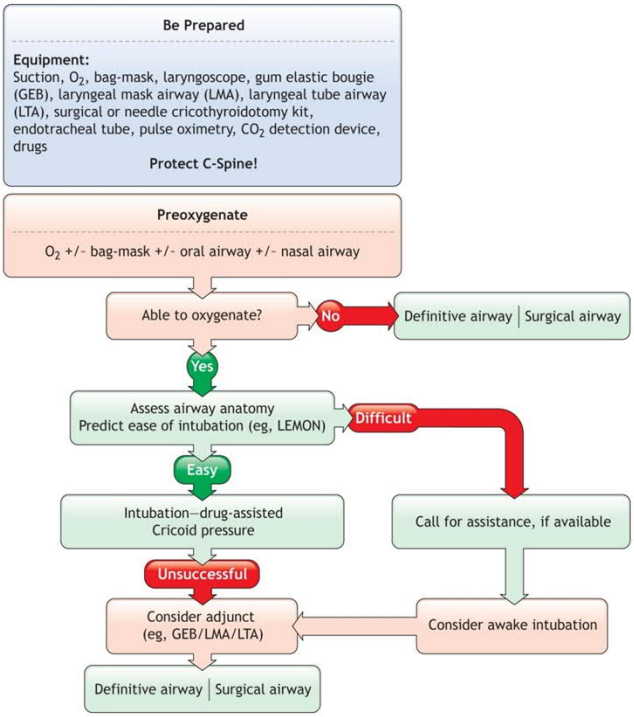
Mallampati Scoring System

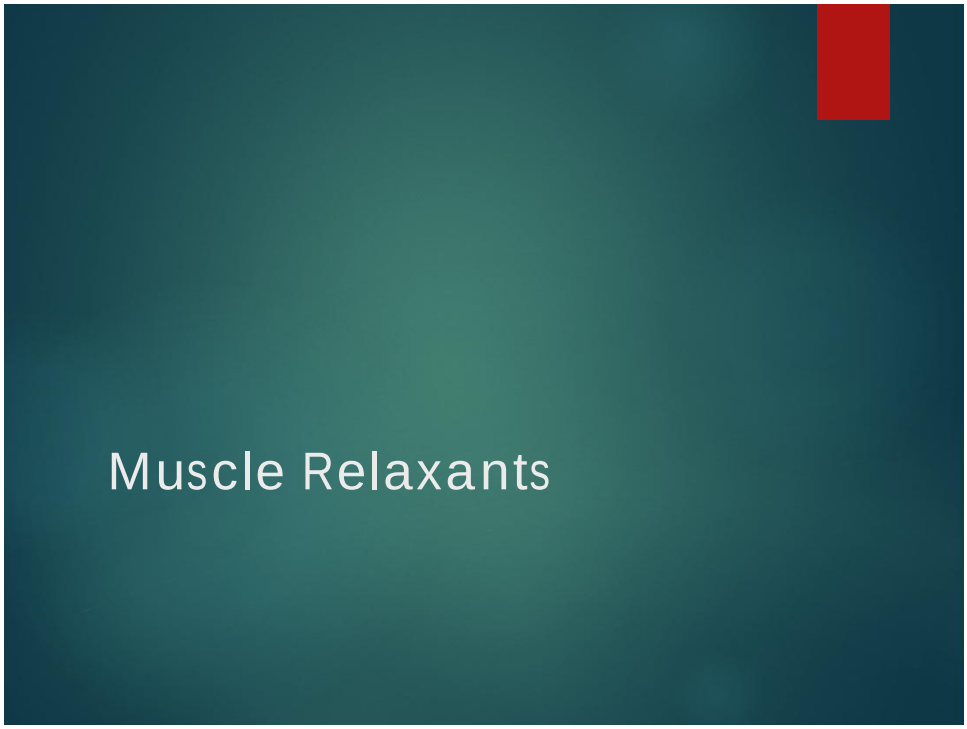
Pediatric ET Tube Size

Age	Size (mm)
Premature	2.5
Term infant	3.0
1 - 4 months	3.5
4 months - 1 year	4.0
3 years	5.0
6 years	6.0

Pediatric Airway

- Larynx is anterior, short and more cephalad
- Cricoid is the narrowest part
- Trachea is short
- Large occiput
- Epiglottis is long and narrow
- Size = age/4 + 4 = mm of ET tube





Depolarizing

- Succinylcholine
- Competitive inhibition
- Fasciculation then flaccid paralysis
- Cannot be reversed
- Short half-life
- Eliminated by pseudocholinesterase in serum
- Side effects:
  - increase intraocular pressure
  - increase K (Hyperkalemic cardiac arrest)
  - increase intracranial pressure
  - bradycardia (especially in children)

Malignant Hyperthermia

- Autosomal dominant
- Response to halogenated anesthetic gases (halothane, isoflurane) and/or succinylcholine
- Sudden rise in end tidal CO_2
- Fever, muscle rigidity, myoglobinuria
- Treatment:
  - Immediate discontinuation of anesthetic agent
  - Dantrolene for 3 days (side effect is liver failure)
  - Treat fever
  - Increased ventilation to reduce end tidal CO_2

Nondepolarizing

- Watch for prolonged blockage with aminoglycosides, clindamycin, hypermagnesemia, myasthenia gravis, hypothermia
- Atracurium
- Cisatracurium
- Rocuronium
- Vecuronium
- Pancuronium

Atracurium and Cisatracurium

- Metabolized to laudanosine which can cross the blood brain barrier leading to seizure especially in children.
- Degraded by Hofmann elimination
  - Can be used in liver failure / renal failure
- Cisatracurium has no histamine effect
- Atracurium associated with histamine release

Rocuronium, Vecuronium, Pancuronium

- Metabolized by the liver and eliminated by the kidney
- Prolonged effect in renal failure
- Free of cardiac effects
- May lead to profound paralysis secondary to blocking binding of acetylcholine to cholinergic receptors

Pancuronium

- Longest duration
- Rarely used in any surgery today because of long duration of action
- Cleared by kidneys
- Reversal with neostigmine or edrophonium (anticholinesterase)

Train of Four (TOF)

- Way to assess the degree of paralysis
- In long term use of paralysis the goal is to keep train of four at 2 twitches

Number of Twitches	Degree of paralysis
4	0-5 %
3	65 – 75 %
2	85 %
1	95 %
0	100 %

Sedation and Analgesia

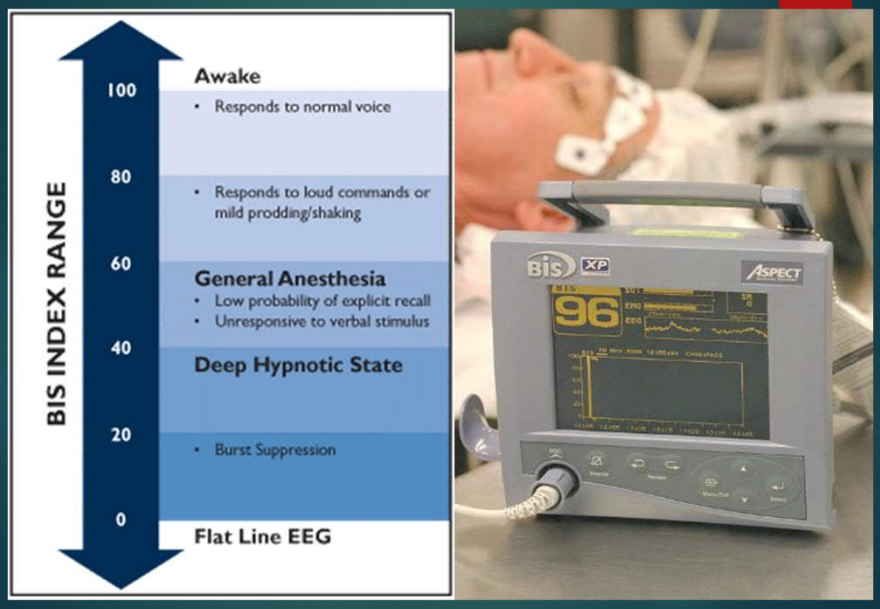
Sedation

- To improve patient comfort
- Reduce stress
- Facilitate interventions
- Allow effective ventilation

Sedation-Analgesia Medications

- IV Anesthetics:
  - Propofol
  - Thiopentone
  - Ketamine
  - Etomidate
- Benzodiazepines:
  - Diazepam
  - Midazolam
  - Lorazepam
- Other:
  - Dexmedetomidine

Bispectral Index



Propofol

- Non-analgesic sedative hypnotic drug
  - Acts on $GABA_A$ receptors in the brain
- Rapid onset of action (dose dependent)
- Duration of action partially dependent on duration and dose of administration
  - Infusion: 25 – 75 mcg/kg/min (1.5 – 4.5 mg/kg/hr)
  - Initial half-life = 40 min
  - Lipid emulsion, prolonged duration can accumulate in peripheral tissues
  - Provides 1.1 kcal/ml, consider caloric contribution with long term or high dose administration
  - Monitor triglycerides
- Clearance by liver (60%) and kidneys (40%)

Propofol: Therapeutic Effects

- Dose-dependent decrease in consciousness
  - Moderate sedation to general anesthesia
- CNS
  - Decrease in cerebral blood flow, ICP, cerebral blood oxygenation
  - Suppress seizure activity
- Respiratory
  - Bronchodilation

Propofol: Side effects

- Pain at injection site
- Cardiovascular
  - Vasodilation ➔ Hypotension
  - Mild depression of myocardial contractility
- Respiratory depression, apnea
- Propofol Infusion Syndrome

Propofol Infusion Syndrome (PRIS)

- Rare, possibly fatal complication
- Risk factors:
  - Prolonged infusion / high dose
  - Young age
  - Critical Illness
  - Excess lipid state (low carbohydrates)
  - Corticosteroid use
  - Patients with inborn errors of metabolism
- Presentation:
  - Metabolic acidosis
  - Hyperkalemia
  - Hyperlipidemia
  - Rhabdomyolysis
  - Arrhythmias
  - Bradycardia, decreased myocardial contractility, asystole
  - Renal Failure

Propofol Infusion Syndrome (PRIS): Treatment

- Best management is prevention
- Discontinue propofol
- Overall supportive care
- Acidosis: Sodium bicarbonate, hemodialysis
- Cardiac: Pacing, inotropes, vasopressors
- Refractory cardiogenic shock – can consider VA-ECMO

Benzodiazepines

- Act on benzodiazepine receptors in CNS which form GABA receptors
  - When stimulated cause open channel allowing chloride to enter cells
  - large flow of chloride hyperpolarize the neuron and lead to CNS depression

Benzodiazepines

- Diazepam
  - Long action
  - Requires hepatic clearance
  - Produces active metabolites
- Lorazepam
  - Long action
  - Does not need hepatic clearance
  - Metabolites are not active
- Midazolam
  - Short action
  - Needs hepatic clearance
  - Produces active metabolites

Haloperidol (Haldol)

- Excellent choice for agitated patients who are not in pain
- Drug of choice in delirium
- May lead to rare complication of tardive dyskinesia (extra pyramidal symptoms) – torsade de pointes, neuroleptic malignant syndrome
- Similar drugs are: risperidone, clonazepam… Are all antipsychotic drugs with results similar to Haldol

Neuroleptic Malignant Syndrome

- Drug induced, similar to malignant hyperthermia
- Causative agents:
  - Haldol, metoclopramide, cocaine, lithium, tricyclic antidepressants are few examples
- Stop the drug, dantrolene, bromocriptine

Epidural Analgesia

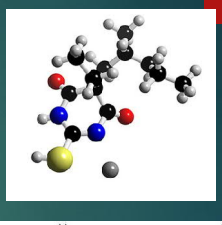
- A combination of local anesthetic and opioid will provide synergistic pain relief and diminish complication of each drug
- Complications:
  - Hypotension
  - Urinary retention
  - Hematoma
  - Causes vascular dilatation

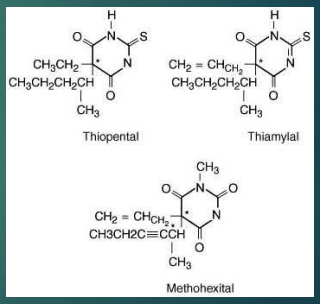
Local / Regional Anesthesia

- Inadvertent intravascular injection of Marcaine or lidocaine
- Lead to tinnitus, twitches, seizure… Cardiac arrest
- Treatment:
  - Lipid injection (create lipid sink) to pool the drug there, until metabolized by the body
  - 20% Intralipid bolus with infusion to follow

IV Anesthetics: Thiopentone

- Acts on the $GABA_A$.
- Zero order kinetics (accumulation)
- Provides a cerebral protection effect
- Main uses in ICU:
  - High ICP
  - Status epilepticus





Ketamine

- NMDA Receptor Agonist
- Dissociative anesthetic
- Hypnosis and analgesia
- No respiratory depression
  - Airway smooth muscle relaxant
- May block development of tolerance to opioids
- Dose:
  - Continuous sedation: 1 – 10 mcg/kg/min
    - < 5 mcg/kg/min for pain for awake patients
  - Procedural sedation: 10 – 20 mg bolus

Sedation-Analgesia Medications

- Opioids:
  - Morphine
  - Fentanyl
  - Remifentanil
- α_2 receptors agonists: (No level 1 evidence yet)
  - Clonidine
  - Dexmedetomidine

α_2 Agonists

Clonidine	Dexmedetomidine
• Selectivity: α_2:α_1 200:1	• Selectivity: α_2:α_1 1620:1
• $t_{1/2}$ β 8 hrs[1]	• $t_{1/2}$ β 2 hrs
• PO, patch, epidural	• Intravenous
• Antihypertensive	• Sedative-analgesic
• Analgesic adjunct	• Primary sedative
• IV formulation are in trials in Europe	• Only IV α_2 available for use in the US

α_2 Agonists

- Selectivity: α_2:α_1 200:1
- $t_{1/2}$ - 8 hrs
- PO, patch, epidural
- Antihypertensive
- Analgesic adjunct

- Selectivity: α_2:α_1 1620:1
- $t_{1/2}$ - 2 hrs
- Intravenous
- Sedative-analgesic
- Primary sedative

Dexmedetomidine (Precedex)

- Short acting alpha 2 agonist(8-10x increased binding than clonidine)
- Anxiolytic, anesthetic, hypnotic and analgesic
- Rapid onset: 6 min
- Elimination: 2 hours
- Pts can be arousable/alert with stimulation
- Very useful in alcohol, cocaine and marijuana withdrawal patients
- Does NOT inhibit respiration (Can be extubated while infusing)

Dexmedetomidine (Precedex)

- Sedation with less lethargy & less reduction in level of arousal
- Dose:
  - loading infusion for 1mg/kg for 10 min
  - maintenance of 0.2 to 0.7 mcg/kg/hr
- Side effects:
  - Hypotension
  - Bradycardia
  - High doses can have alpha 1 agonist effect
  - Long term used may be associated with rebound hypertension similar to clonidine withdrawal. OK for 3 or 4 days but no longer

Treatment of Pain: Opioids

Drug	Dosing	Onset (minutes)	Peak (hrs)	Duration (hrs)	Adverse Effects
Morphine	2-10mg IV or drip titrated to pain control	<5	0.5-1	3-7	Histamine release Hypotension GI effects Tolerance and withdrawal **Delirium** Rash, AW
Dilaudid	1-4mg IV	15-30	0.5-1	4-5	No active metabolites No histamine Tolerance/WD GI effects
Fentanyl	25ug-50ug q 15-30min or drip 50-200ug/hr	1.5	1.5	1.5	No histamine release Less hypotension GI effects Tolerance/WD

Question1

Ketamine, is contraindicated in

A. head injury patients
B. asthma patients
C. children
D. bradycardia patients

Answer 1

A: head injury patients

– Because of increase in ICP it may not be a good choice in head injury but it increases cerebral blood flow

Question 2

In patients with failed first endotracheal intubation, the second recommended choice is

A. repeat endotracheal attempt one more time
B. video laryngoscopy intubation
C. nasotracheal if breathing
D. bronchoscopic intubation

Answer 2

B – video laryngoscopy intubation

– Video assisted intubation using video laryngoscope has been now recommended as second and sometimes first in anticipated difficult air way cases

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NUTRITION & METABOLISM
Surgical Critical Care

Gregory B. York, MD, FACS
Trauma/Critical Care

Presented by Laura Crankshaw, MD
Surgical Critical Care 2025

ORGANIC FUELS & ENERGY YIELD

- Lipid
  - 9.1 kcal/gm
  - RQ: 0.70
- Protein
  - 4.0 kcal/gm
  - RQ: 0.80
- Glucose
  - 3.7 kcal/gm
  - RQ: 1.00

RQ REVIEW

- Respiratory Quotient (RQ) = VCO_2 / VO_2
- Healthy Subject: RQ = 0.8
- RQ = 0.7
  - Lipid is sole substrate
  - STARVATION
- RQ > 1.0
  - Net Lipogenesis
  - OVERFEEDING

HARRIS-BENEDICT

- Basal (Daily) Energy Expenditure
  - Heat production of basal metabolism
  - At Rest
  - Fasted State
- BEE (kcal/day)
  - Men: 66 + (13.7 x Wt) + (5.0 x Ht) - (6.7 x Age)
  - Women: 655 + (9.6 x Wt) + (1.8 x Ht) - (4.7 xAge)
  - Wt = IBW in kg; Ht = Inches; Age = Years
- BEE Adjustments
  - Fever: BEE x 1.1 (for each ^{o}C above normal)
  - Stress: Mild x 1.2; Moderate x 1.4; Severe x 1.6

NUTRITION ASSESSMENT
SERUM ALBUMIN

- Measure of Protein Depletion
  - Mild – Moderate: 2.7 – 3.5 g/dL
  - Moderate – Severe: 2.0 – 2.6 g/dL
  - Profound: < 2.0 g/dL
- Negative acute phase reactant
- Determinate of Acute Protein Deficiency?
  - Half-Life: 2 – 3 weeks
  - Large (> 50%) extravascular pool – difficult to interpret levels
  - Non-nutritional factors affect serum concentration
    - Lower: Hepatic dysfunction/injury, burns, chronic renal disease
    - Higher: Dehydration, blood transfusion

NUTRITION ASSESSMENT
TRANSFERRIN

- Measure of Protein-Calorie Malnutrition
  - Mild: 150 – 200 mg/dL
  - Moderate: 100 – 150 mg/dL
  - Severe: < 100 mg/dL
- Negative acute phase reactant
- Determinate of Acute Protein Deficiency?
  - Half-Life: 8 – 10 days
  - Non-nutritional factors affect serum concentration
    - Lower: Hepatic dysfunction/injury, B_{12} deficiency
    - Higher: Iron deficiency, hemochromatosis, hemosiderosis, estrogen/oral contraceptive use

NUTRITION ASSESSMENT
SERUM PREALBUMIN

- Measure of Protein-Calorie Malnutrition
  - Mild: 10 – 15 mg/dL
  - Moderate: 5 – 9 mg/dL
  - Severe: < 5 mg/dL
- Negative acute phase reactant
- Determinate of Acute Protein Deficiency?
  - Half-Life: 2 – 3 days
  - Non-nutritional factors affect serum concentration
    - Lower: Hepatic dysfunction/injury, hyperthyroidism, zinc deficiency, burns
    - Higher: Chronic renal disease, corticosteroid use

NONPROTEIN CALORIES (NPCs)

- Daily energy requirement should come from:
  - Carbohydrates: 70%
  - Lipids: 30%
- Proteins - Used to maintain stores of:
  - Essential enzymes
  - Structural proteins
- NPC:Nitrogen Ratio
  - Goal: 150 kcal NPC:1 gm Nitrogen
  - 1 gm Nitrogen is in 6.25 gm Protein

PROTEIN REQUIREMENTS

- Goal: Protein Intake = Protein Catabolism
- Normal: 0.8 – 1.0 gm/kg/day
- Nitrogen Balance
  - Goal: + 2 to + 4 gms/day
  - Balance (g) = ((Protein intake (g)/6.25) – (UUN + 4)
  - UUN = Urinary Urea Nitrogen (g) in 24 hrs
- Critical Illness
  - May require 1.0 to 1.5 gm/kg/day
  - High catabolic patients (e.g. burn, etc.) may need 2 gm/kg/day

CALORIC INTAKE

- Desire positive nitrogen balance
- Goal: Provide enough NPCs to spare proteins from being degraded to provide energy
- Maintaining Positive Nitrogen Balance
  - May need to increase NPCs vs Protein

VITAMINS

- Essential
  - Vitamin A
  - Vitamin B_{12}
  - Vitamin C
  - Vitamin D
  - Vitamin E
  - Vitamin K
  - Thiamine (B_1)
  - Riboflavin (B_2)
  - Pyridoxine (B_6)
  - Pantothenic Acid (B_5)
  - Biotin
  - Folate

VITAMIN A

- Essential Roles
  - Vision: Phototransduction
  - Cellular differentiation (especially in the eye)
  - Immune system function
- Sources
  - Orange/yellow fruits & vegetables
  - Tuna
  - Leafy greens
- Deficiency
  - Xeropthalmia (cornea/conjunctiva abnormalities)
  - Night or complete blindness
  - Dermatologic problems (hyperkeratosis, etc)
  - Impairment of humoral & cell-mediated immunity (phagocytes & T cells)

THIAMINE (VITAMIN B_1)

- Roles
  - Essential cofactor in carbohydrate metabolism
  - Nerve impulse propagation
  - Many cellular metabolic activities
- Sources
  - Pork
  - Whole Grains
- Deficiency
  - Beri-beri: Cardiac & neurologic dysfunction
  - Wernicke-Korsakoff Syndrome
    - Wernicke's Encephalopathy: Nystagmus, ophthalmoplegia, ataxia
    - Korsakoff's Syndrome: Chronic neurologic disorder (follows WE)
  - Requires Magnesium: Low Mg = functional form of thiamine deficiency

RIBOFLAVIN (VITAMIN B_2)

- Roles
  - Coenzyme component in energy producing respiratory pathways
  - Mitochondrial function
  - Red blood cell production
- Sources
  - Meats and fish
  - Eggs and milk
- Deficiency
  - Pharyngeal edema/sore throat/glossitis/stomatitis
  - Anemia (normocytic-normochromic)

NIACIN (VITAMIN B_3)

- Roles
  - Used to form NAD and NADP
  - Crucial in synthesis of carbohydrates, proteins, and fatty acids
- Sources
  - Poultry
  - Fish
  - Veal
- Deficiency
  - Pellagra
    - Symmetric hyperpigmented rash
    - Diarrhea, vomiting, red tongue, other non-specific symptoms
    - Neurologic symptoms

PANTOTHENIC ACID (VITAMIN B_5)

- Roles
  - Converted to Coenzyme A
  - Crucial in molecular synthesis
  - Coenzyme A is in first step of TCA cycle
- Sources
  - Major: Egg yolk, broccoli, and milk
  - Chicken and beef
  - Potatoes
  - Whole grains
- Deficiency
  - Rare in humans
  - Distal paresthesias
  - Gastrointestinal distress

PYRIDOXINE (VITAMIN B_6)

- Roles
  - Schiff base formation
  - Amino acid & neurotransmitter synthesis
  - Gluconeogenesis
- Sources
  - Meats: Poultry, fish
  - Plants: Potatoes, bananas
  - Whole grains
- Deficiency
  - Rare in humans
  - Stomatitis, glossitis, cheliosis
  - Confusion and depression
  - Increased homocysteine concentrations/atherosclerosis

CYANOCOBALAMIN (VITAMIN B_{12})

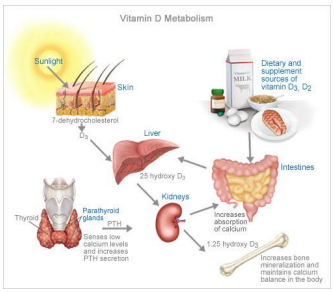
- Roles
  - DNA synthesis
  - Red blood cell production
  - Myelin formation
- Sources
  - Meat products
  - Dairy products
  - Fish
- Deficiency
  - Megaloblastic anemia
  - Distal neuropathy (> arms): Paresthesias, ataxia, weakness
  - Osteoporosis/bone loss/spine & hip fractures

VITAMIN C

- Roles
  - Fatty acid transport
  - Collagen synthesis
  - Norepinephrine and prostaglandin synthesis
- Sources
  - Citrus fruits
  - Vegetables: Potatoes, cauliflower, broccoli, cabbage, spinach
  - Strawberries, tomatoes
- Deficiency
  - Scurvy (syndrome of disordered connective tissue)
    - Ecchymoses, bleeding gums, hyperkeratosis
    - Arthralgias
    - Impaired wound healing

VITAMIN D

- Roles
  - Stimulates intestinal absorption of calcium and phosphorous
  - Stimulates mobilization of calcium from bone
  - Stimulates the kidney to reabsorb calcium
- Sources
  - Dairy products
  - Fish
  - Sunlight
- Deficiency
  - Hypocalcemia and hypophosphatemia
  - Rickets, osteomalacia, osteoporosis
  - Secondary hyperparathyroidism



VITAMIN E

- Roles
  - Free radical scavenger/antioxidant
  - Inhibition of cell proliferation and platelet aggregation
- Sources
  - Meats
  - Eggs
  - Leafy vegetables
- Deficiency
  - Neuromuscular symptoms: ataxia, hyporeflexia, sensory loss
  - Hemolysis
  - Decreased RBC lifespan

VITAMIN K

- Roles
  - Cofactor for activation of factors VII, IX, X, and prothrombin
  - Cofactor for activation of Proteins C and S
  - Cofactor for proteins in bone mineralization
- Sources
  - Leafy green vegetables
  - Tomatoes
  - Eggs
- Deficiency
  - Coagulation disorders manifested by:
    - Easy bruising
    - Melena or hematuria
    - Prolonged bleeding

BIOTIN

- Roles
  - Essential component of several enzymatic complexes
  - Carbohydrate and lipid metabolism
  - Protein and DNA synthesis
- Sources
  - Yeast
  - Egg yolks
  - Soy beans
- Deficiency
  - Increased lactate and fatty acid production
  - Myalgias, dysesthesias, mental status changes
  - Anorexia and nausea

FOLATE

- Roles
  - DNA synthesis
  - Red blood cell production
- Sources
  - Leafy vegetables
  - Cereals and grains
  - Meats
- Deficiency
  - Megaloblastic anemia
  - Neural tube defects

REVIEW

A 57 y/o female is intubated in the ICU following a stroke. She is on ICU day 10 and her course is complicated by a small bowel ileus. She is currently receiving TPN, which consists of 80 gm of protein per day. A 24-hour urine urea nitrogen (UUN) is 6 gm. What can you say about her current protein intake?

a. Her nitrogen balance is -6.8 indicating a catabolic state of severe stress.
b. Her nitrogen balance is -2.8 indicating a catabolic state of moderate stress.
c. Her nitrogen balance is +2.8 indicating an acceptable anabolic state.
d. Her nitrogen balance is +6.8 indicating an excess of protein intake.

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TRACE ELEMENTS

- Essential
  - Chromium
  - Copper
  - Iodine
  - Iron
  - Manganese
  - Selenium
  - Zinc

ZINC

- Roles
  - Amino acid binding/protein structure maintenance
  - Cell division and apoptosis
  - Approximately 250 proteins contain zinc
- Sources
  - Meat and chicken
  - Nuts and lentils
- Deficiency
  - Growth retardation/impotence/hypogonadism
  - Immune dysfunction/impaired wound healing
  - Skin lesions

IRON

- Roles
  - Heme proteins: hemoglobin, myoglobin, cytochrome c
  - Metalloenzymes used in protein synthesis
  - Cofactor in gluconeogenesis, TCA cycle, DNA synthesis
- Sources
  - Meat, poultry, and fish
  - Vegetables and fruit
- Deficiency
  - Microcytic, hypochromic anemia
  - Lethargy and decreased work performance
  - Developmental delays

SELENIUM

- Roles
  - Form over 30 proteins
  - Antioxidant defense
  - Thyroid hormone production
- Sources
  - Meat and seafood
  - Whole grains and nuts
- Deficiency
  - Cardiomyopathy (Keshan disease)
  - Skeletal muscle dysfunction
  - Immune dysfunction

ENTERAL NUTRITION

- Trophic Effects
  - Bowel Atrophy
    - Evident after just a few days of bowel rest
    - NOT prevented by parenteral nutrition
    - Glutamine: Principal metabolic fuel for intestinal epithelial cells
  - Translocation: Documented during periods of bowel rest
- Delivery
  - Gastric: Bolus or Continuous Infusion
  - Small Bowel: Continuous Infusion ONLY

ENTERAL FEEDINGS

- Contraindications – Absolute
  - Circulatory shock
  - Intestinal Ischemia
  - Complete mechanical bowel obstruction
  - Ileus
- Contraindications – Relative
  - Partial mechanical bowel obstruction
  - Severe diarrhea
  - Pancreatitis
  - High-Output EC Fistulas

ADDITIVES

- Glutamine
  - Not an essential protein
  - Easily depleted in hypermetabolic, stressed patient
- Fiber
  - Helps alleviate diarrhea
  - Fermentable fiber is nutrition for large bowel
- BCAAs (Isoleucine, Leucine, Valine)
  - Trauma: As fuel source, sparing degradation of muscle proteins
  - Hepatic encephalopathy: Reduces uptake of aromatic AAs and thus their breakdown to false neurotransmitters – implicated in HE

COMPLICATIONS

- Tube Obstruction
  - Warm water lavage
  - Pancreatic enzymes
- Aspiration
  - Beyond Ligament of Treitz
  - HOB 45^{o}: Can reduce/not eliminate risk
  - Lowest Risk = Beyond LT + HOB > 30^{o}

COMPLICATIONS

- Diarrhea
  - In approx 30% of patients receiving enteral feeds
  - Etiology
    - Sorbitol-containing medicinal elixers
    - Clostridium difficile enterocolitis

PARENTERAL NUTRITION

- Caloric Daily Requirements (Estimate)
  - Non-Protein Caloric Estimate: 25 kcal/kg/day
  - Protein: 0.8 – 1.0 gm/kg/day
  - NPCs: 70% Carbohydrate; 30% Lipid
- Volume Requirements (Estimate)
  - 4:2:1 Rule
  - 30 – 35 ml/kg/day
  - 100:50:20 Rule

TPN EXAMPLE

- 70 Kg Adult
  - NPC: 25 kcal/kg/day x 70 kg = 1750 kcal/day
  - Carbohydrate: At least 1750 x .7 = 1225 kcal/day
  - Lipid: Up to 1750 x .3 = 525 kcal/day
  - Protein: 1 gm/kg/day x 70 kg = 70 gm/day
- Resultant Needs
  - 1750 kcal/day (mixture of carbohydrates & lipids)
  - 70 gm/day protein
  - Volume: Approx 2450 - 2640 ml/day

LIPIDS

- Oxidation-Prone
- Promote oxidant-induced cell injury
- Restrict use in critically ill patients
- Bottom Line
  - Some feed lipids every day
  - Some feed lipids 3 times/week
  - Some feed lipids 1 time/week
- Essential Fatty Acid Requirements: Minimum of 3% of NPCs must be given as lipid

ELECTROLYTES

- Most commercial solutions contain daily requirements
- Rule-of-Thumb Estimation
  - K: 1 – 2 meq/kg/day
  - Na: 3 – 4 meq/kg/day
  - Cl: 5 – 6 meq/kg/day

REVIEW

A 45 y/o 80 kg male is admitted to the ICU following a house fire with 45% TBSA. He has completed resuscitation and has stabilized. He is intubated on minimal ventilator settings (SIMV RR 12 bpm, TV 450 ml, Peep 5 cm H_20, PS 10 mm Hg, FiO_2 40%, SaO_2 100%). What is an appropriate nutritional intake to start for this patient?

a. Total Calories: 2200 kcal/day; Protein: 80 gm/day; Volume: 2800 ml/day.
b. Total Calories: 1600 kcal/day; Protein: 160 gm/day; Volume: 2000 ml/day.
c. Total Calories: 3000 kcal/day; Protein: 80 gm/day; Volume: 2800 ml/day.
d. Total Calories: 2200 kcal/day; Protein: 160 gm/day; Volume: 2800 ml/day.

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MAINTENANCE IV FLUIDS

- 70 kg Adult Daily Requirements (Estimate)
  - K: 70 – 140 meq/day
  - Na: 210 – 280 meq/day
  - Cl: 350 – 420 meq/day
  - Dextrose: Approx 50 – 100 gm/day (prevent muscle breakdown)
  - Volume: Approx 3 L/day
- IV Fluid
  - Na Needs: 70 – 90 meq/L/day
  - K Needs: 20 – 40 meq/L/day
  - Fluid Choice: $D_5$1/2NS + 20 meq/L KCl

CARBOHYDRATE COMPLICATIONS

- Hyperglycemia
  - Glucose intolerance: Common TPN complication
  - May require addition of Insulin to TPN
- Hypophosphatemia
  - Cumulative serum phosphate decline over 8 – 10 days
  - Glucose entry into cells causes enhance phosphate uptake by cells
  - Used to form thiamine pyrophosphate – cofactor in carbohydrate metabolism

CARBOHYDRATE COMPLICATIONS

- Fatty Liver
  - Seen with glucose calorie excess
  - Excess calories undergo lipogenesis and fatty infiltration of the liver
- Hypercapnia
  - Excess carbohydrates promote CO_2 retention in patients with respiratory insufficiency

GI COMPLICATIONS

- Mucosal Atrophy
  - Absence of nutrients in contact with bowel mucosa
  - May predispose to translocation
- Acalculous Cholecystitis
  - Absence of lipids in bowel prevents contraction of gallbladder
  - Bile stasis occurs

REVIEW

One liter of D_5½NS + 20 meq/L KCl will contain:

a. 500 grams of Dextrose, 145 meq of NaCl, and 40 meq of KCl.
b. 50 grams of Dextrose, 77 meq of NaCl, and 20 meq of KCl.
c. 50 grams of Dextrose, 145 meq of NaCl, and 20 meq of KCl.
d. 500 grams of Dextrose, 77 meq of NaCl, and 20 meq of KCl.

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REFEEDING SYNDROME

- Etiology
  - Fluid and electrolyte shifts during the nutritional rehabilitation of a chronically malnourished patient
- At Risk Populations
  - Malnourished elderly
  - Anorexia Nervosa
  - Oncology patients undergoing chemotherapy
  - Homeless or alcoholic patients

REFEEDING SYNDROME

- Manifestations
  - Clinical
    - Hemolysis
    - Rhabdomyolysis
    - Heart Failure
    - Volume Overload
  - Chemical
    - Hypophosphatemia
    - Hypokalemia
    - Hypomagnesemia
    - Vitamin and trace mineral deficiencies
- Treatment
  - Correct electrolyte abnormalities
  - Lower initial caloric intake/gradual increases to target

SPECIFIC DISORDERS
RENAL INSUFFICIENCY

- Susceptibilities
  - Hyperkalemia
  - Hyperphosphatemia
  - Hypermagnesemia
- Protein Restriction
  - To prevent steep increase in BUN if NOT on dialysis
  - 0.6 to 1.0 gm/kg/day
  - Restriction NOT recommended in acute renal failure or if receiving dialysis

SPECIFIC DISORDERS
HEPATIC FAILURE

- Susceptibilities: Zinc, Vits A, D, E, K deficiencies
- Generally tolerate normal recommendations
- Overt Encephalopathy
  - Limit protein
  - Limit amino acids
  - If responsive to lactulose – may give normal protein provisions (1 gm/kg/day)
- BCAA Formulations
  - Not routinely recommended
  - Recommended if encephalopathy is refractory to lactulose

SPECIFIC DISORDERS
RESPIRATORY FAILURE

- High Carb Diet = Inc RQ = Inc VCO_2
- Promotes Hypercapnia in patients with:
  - COPD
  - Alveolar Ventilation Limitations
  - Increased Dead-Space Ventilation
- Calorie Restriction: May be needed in cases of severe hypercapnia refractory to mechanical ventilation or other respiratory therapies

REVIEW

A 64 y/o female with a history of alcoholism and malnutrition has been admitted to the ICU for the past three weeks following a ileal resection for a small bowel obstruction. Her return of bowel function has been delayed and on hospital day six she is started on TPN. Two days later she is noted to be lethargic, tachycardic, and mildly hypotensive. Which electrolyte is her TPN most likely deficient?

a. Calcium.
b. Chloride.
c. Phosphate.
d. Magnesium.

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QUESTIONS

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BLEEDING AND COAGULATION
Surgical Critical Care

Gregory B. York, MD, FACS
Trauma/Critical Care

Presented by Laura Crankshaw, MD
Surgical Critical Care 2025

INTRODUCTION

- Biology of hemostasis
- Coagulation pathway
- Hypercoagulable states
- Evaluation of the bleeding patient
- Management of anemia
- Blood products
- Complications of transfusion

BIOLOGY OF HEMOSTASIS

- Capillary vasoconstriction
- Platelet plug formation
- Fibrin formation
- Fibrinolysis

CAPILLARY VASOCONSTRICTION

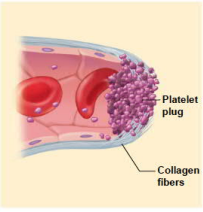
- Reflex contraction of local smooth muscle occurs
  - Before platelet plug formation
- Thromboxane A2
  - Released from platelets
  - Causes vasoconstriction + further platelet aggregation
- Prostacyclin
  - Released from endothelium
  - Causes vasodilatation + inhibits platelet aggregation

PLATELET PLUG FORMATION

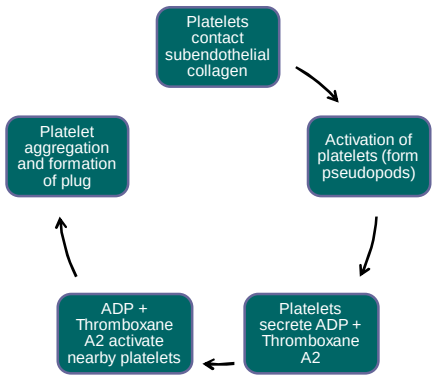
- Platelets are fragments of megakaryocytes
  - Life span = 8 days
  - 1/3 are sequestered in the spleen

PLATELET PLUG FORMATION

- Platelets are exposed to subendothelial collagen
  - Platelets adhered to collagen by vWF (cofactor VIII)
  - GP Ib is the platelet receptor for vWF
- Platelets are activated and change shape
  - Develop pseudopods to become “sticky”
- Platelets release mediators
  - Thromboxane A2 and ADP
    - Activate nearby platelets
    - Uncover fibrinogen receptors on platelet surfaces
- Fibrinogen links the platelets together via GP IIb

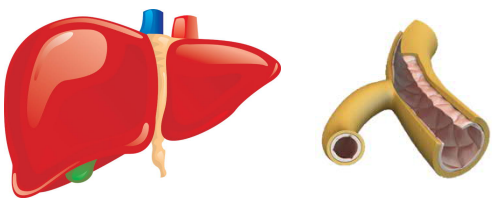


PLATELET PLUG FORMATION



COAGULATION FACTOR SYNTHESIS

- All coagulation factors are formed in the liver
  - Except factors VIII and vWF (cofactor VIII)
    - Produced in the endothelium



EXTRINSIC PATHWAY

- Initiated by damaged tissue outside the blood vessel
  - Release tissue factor = factor III = thromboplastin
    - Primary physiologic starter of coagulation
- Thromboplastin then activates factor VII
- Activated factor VII + activated factor III w/Ca^{2+} (factor IV)
  - Join on the surface of the platelet → activate factor X
    - Starts the final common pathway
- Evaluated by the prothrombin time (PT)

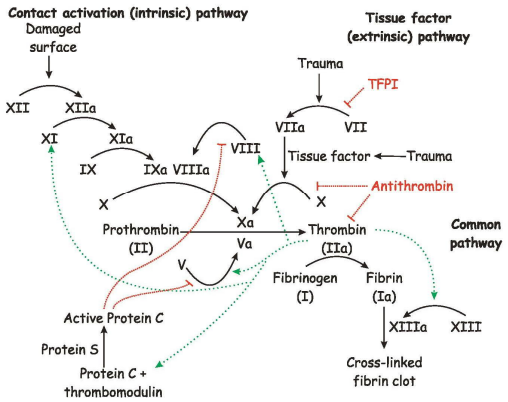
INTRINSIC PATHWAY

- Initiated by injury to the blood vessel
  - Release of clotting factors from the circulation
- Factor XII is activated (XIIa)→ activates factor XI (XIa)
  - Needs kininogen and accelerated by prekallikrein
- XIa then activates factor IX (IXa)
- Factor IXa + factor VIIIa w/Ca^{2+} will activate factor X
  - Starts the final common pathway
- Evaluated by the activated partial thromboplastin time (aPTT)

FINAL COMMON PATHWAY

- Fibrin and clot formation (coagulation)
- Xa + Va + Ca^{2+} on the platelet surface
  - Converts prothrombin (II) → thrombin (IIa)
- Thrombin (IIa) converts fibrinogen (I) → fibrin (Ia)
- XIII → XIIIa = stabilizes fibrin by cross links → clot

COAGULATION CASCADE



FIBRINOLYSIS

- Physiologic anticoagulants
- Occurs simultaneous to clotting
- Plasminogen (from liver) is converted to plasmin
  - Action of kallikrein, factor XII, TPA, streptokinase, and urokinase
- TPA (from endothelium) = primary signal of fibrinolysis
- Plasmin cleaves fibrin into FDPs (D-dimer is one)
  - Inhibit platelet aggregation
- Inhibitors of fibrinolysis
  - Alpha-2 antitrypsin, macroglobulin, aprotinin, and amicar

FIBRINOLYSIS

- Endothelium produces prostacyclin
  - Inhibits platelet activation
- Endothelium produces thrombomodulin
  - Inhibits thrombin
- Antithrombin III
  - Produced in the liver
  - Inactivates thrombin and factor X
  - NOT vitamin K dependent
  - Is activated 10,000 times by heparin

CONGENITAL HYPERCOAGUABLE STATES

- Antithrombin III deficiency
  - Congenital (autosomal dominant)
  - Acquired (liver disease, nephrotic syndrome, protein enteropathy)
  - Causes massive recurrent thrombosis in arterial or venous system
  - Resistant to heparin therapy
- Treatment = FFP

CONGENITAL HYPERCOAGUABLE STATES

- Protein C deficiency
  - Inhibits factors V + VIII
  - 2nd most common cause of congenital hypercoagulability
  - Recurrent thrombosis in early teenage years
  - Coumadin → skin necrosis in patients w/protein C deficiency
- Protein S deficiency
  - Cofactor for protein C

FACTOR V LEIDEN MUTATION

- Most common cause of congenital hypercoagulability
- Mutation in a part of factor V
  - Renders factor V resistant to protein C
  - Factor V is not inhibited (regulated) by protein C
- Presents as recurrent thrombosis
  - 5 x more common than normal population
- Prophylaxis = LMWH

HEPARIN INDUCED THROMBOCTOPENIA (HIT)

- Incidence = 2-6%
- Immune mediated by IgG and PF 3
- May occur w/heparin flush
- Thrombosis occurs at arteries and veins
  - Bleeding is less common
- Takes up to one month for antibodies to disappear
- Alternative treatments
  - Direct thrombin inhibitor (argatroban and bivalirudin)

THE BLEEDING PATIENT

- Evaluation
- Technical Causes
- Initial Assessment
- Coagulopathy Diagnoses
- Perioperative Evaluation
- Management of Anemia

THE BLEEDING PATIENT
EVALUATION

- Surgeon is often 1st person called w/ongoing bleeding
- Need to identify the cause/source
- Two main categories
  - Loss of vascular integrity
  - Derangement of the hemostatic process (focus)

All bleeding stops... eventually.

BLEEDING
TECHNICAL CAUSES

- Most common causes of post-op bleeding are technical
  - Unligated vessel or unrecognized injury
- Need to exclude a surgically correctable cause
- May be surprisingly difficult to diagnose
  - Healthy, young pts can maintain a normal BP until EBL > 40% of total blood volume (roughly 2L)
- Bleeding from a laceration on the extremity is obvious
  - Internal bleeding may have few physiologic signs

BLEEDING
TECHNICAL CAUSES

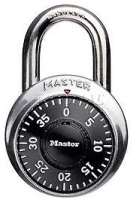
- Even when excluded, need to reconsider possibility throughout assessment
- Unresuscitated or underresuscitated → vasospasm
  - As resuscitation proceeds, bleeding may recur

INITIAL ASSESSMENT

- 1st step = draw a blood sample
  - CBC and coagulation studies (purple and blue tops)
- Note patient's temperature
  - Hypothermic coagulopathy = coagulation slows with ↓ temp
  - Hypothermic → actively rewarm patient
- Obtain personal and family history
  - Eval for congenital or hereditary bleeding disorder

COAGULOPATHY – DIAGNOSIS
INR & aPTT COMBINATIONS

- Normal INR and normal aPTT
- Normal INR and prolonged aPTT
- Increased INR and normal aPTT
- Increased INR and prolonged aPTT

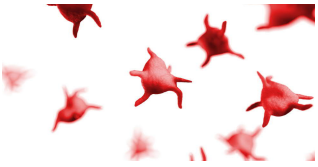


NORMAL INR/NORMAL aPTT

- May have impaired platelet activity
- Manifestations
  - Persistent oozing from wound edges
  - Low volume bleeding (exsanguination rare)
- Reasons
  - Insufficient number of platelets (thrombocytopenia)
  - Platelet dysfunction

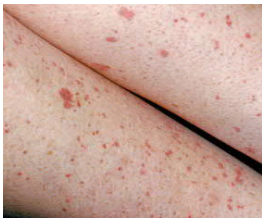
THROMBOCYTOPENIA

- Causes
  - ↓ production (marrow failure)
  - Platelet sequestration (hypersplenism)
  - ↑ destruction (autoantibodies, prosthetic valves, DIC)
- Platelet > 20,000
  - Usually adequate for hemostasis
- Platelet < 10,000
  - Spontaneous bleeding



IDIOPATHIC THROMBOCYTOPENIC PURPURA (ITP)

- Diagnosis of exclusion
  - R/O other causes of thrombocytopenia
- Bone marrow = ↑ megakaryocytes
- Initial treatment = steroids
- Last treatment = splenectomy



PLATELET DYSFUNCTION

- Platelet count normal → think platelet dysfunction
  - More common now w/routine administration of ASA
    - Causes irreversible platelet dysfunction via cyclooxygenase pathway (↓ TXA2)
    - Last for approximately 10 days
  - NSAIDs – reversible dysfunction (does not last as long)
  - GP IIb-IIIa inhibitors
    - Block this platelet surface receptor which binds platelets to fibrinogen
- DDAVP can reverse this platelet dysfunction
- Less common causes
  - Factor XIII deficiency; hypofibrinogenemia or dysfibrinogenemia

NORMAL INR/PROLONGED aPTT

- Likely a drug-induced coagulation defect
  - Most common = unfractionated heparin
- Protamine → reverses the heparin effect
  - Use caution: Can induce a hypercoagulable state
  - Also use caution in diabetics (can cause anaphylaxis if sensitized)
    - From impurities in protamine-containing insulin formations
- Low-molecular-weight heparin (LMWH)
  - aPTT does not accurately measure its activity
  - Need anti-Xa activity assay
  - No role for protamine

NORMAL INR/PROLONGED aPTT

- Important points to remember
  - Heparin and LMWH block activity of antithrombin
    - Need adequate antithrombin levels for effect
    - Acquired antithrombin deficiency
      - Seen in trauma and other critical illnesses
      - Most common reason for LMWH to have an inadequate effects
  - FFP will not correct the effect of heparin or LMWH
    - Actually contains antithrombin → could worsen anticoagulation effect

NORMAL INR/PROLONGED aPTT

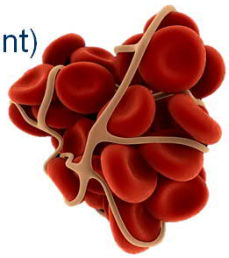
- Direct thrombin inhibitors (lepirudin, argatroban, etc.)
  - Cause ↑ aPTT
  - Effects are irreversible
    - FFP must be given to correct the aPTT

Von Willebrand disease (vWD)

- Normal INR and frequent (but not always) ↑ aPTT
  - Due to variable clinical expression
  - Also have ↑ bleeding time
- 2nd most common congenital coagulopathy
- Autosomal dominant
- Deficiency of vWF production by endothelial cells
- Test circulating factor levels + platelet function analysis
- Treatment
  - vWF
  - DDAVP → stimulates VW production from endothelium
  - Cryoprecipitate (has vWF)

HEMOPHILIA

- Normal INR and Prolonged aPTT
  - Bleeding time = normal
- Spontaneous bleeding
- Prolonged bleeding after trauma or surgery
- Rare in the absence of a personal or family history
- X-linked recessive
- Involve factor deficiencies (all procoagulant)
  - Hemophilia A (most common) = factor VIII
  - Hemophilia B = factor IX
  - Hemophilia C = factor XI



HEMOPHILIA

- ↓ in these procoagulant factors not clinically evident until severe
  - No abnormal labs until factor levels ↓ 60% from normal
  - No significant clinical abnormalities until ↓ 90%
- Test for specific factors if diagnosis is suspected
- Treatment = give deficient factor(s)
  - For type A → can give FFP or cryo (both contain VIII)
  - If h/o significant transfusion → pt may have Abs
    - Treat with recombinant activated factors (rVIIa)

INCREASED INR/NORMAL aPTT

- More ominous finding
- Multiple causes (all based on factor deficiency)
  - Cirrhosis (most serious cause)
    - ↓ in production of all clotting factors (except VIII)
    - Defective platelet plug formation (thrombocytopenia from hypersplenism)
    - TPA is increased → fibrinolysis
- Treatment = factor replacement with FFP (cryo as needed)
  - If variceal bleeding → portal decompression (TIPS)
  - If life-threatening → activated factor VII (20-40 µg/kg)
- Mild elevation of INR + not actively bleeding = observe

INCREASED INR/NORMAL aPTT

- Vitamin K
  - Required by the liver to produce factors II, VII, IX and X
  - Also required for proteins C and S
- Vitamin K deficiency
  - Bile obstruction
  - Coumadin (warfarin) therapy
- Treatment = vitamin K (FFP as needed)

INCREASED INR/NORMAL aPTT

- Vitamin K
  - Required by the liver to produce factors II, VII, IX and X
  - Also required for proteins C and S
- Vitamin K deficiency
  - Bile obstruction
  - Coumadin (warfarin) therapy
- Treatment = vitamin K (FFP as needed)

INCREASED INR/NORMAL aPTT

- Warfarin
  - Causes a pure factor deficiency
  - Disrupts vitamin K metabolism
  - Treatment = give vitamin K
    - If actively bleeding → vitamin K + FFP
  - Disadvantages of vitamin K
    - Reanticoagulation w/warfarin in near future will be difficult
    - Possible anaphylaxis with IV administration

INCREASED INR/PROLONGED aPTT

- Worst combination of all
  - Likely multiple factor deficiencies
- Causes
  - Dilutional coagulopathy
    - Severe hemodilution
    - Renal failure with severe nephrotic syndrome
  - DIC (consumptive coagulopathy)
- If pt is asymptomatic → consider lab error

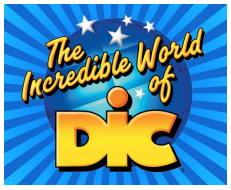
DILUTIONAL COAGULOPATHY

- Severe hemodilution (rare)
  - Pt receives a large volume of PRBCs (> 10), but NO coagulation factors
    - PRBCs stored > 24 h become defective in factors V, VII, and VIII
- Renal failure
  - Platelet dysfunctional by uremic toxins
    - DDAVP is the treatment of choice (also dialysis)
  - Nephrotic syndrome
    - Loss of protein (including coagulation proteins) from the kidneys

DISSEMINATED INTRAVASCULAR COAGULATION (DIC)

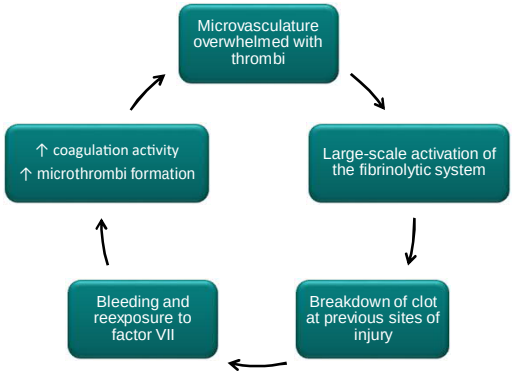
- Increased INR and Prolonged aPTT
- Diffuse, disorganized activation of the clotting cascade w/in vessels
- Consumptive (vs. dilutional) process
- Causes
  - Overwhelming clotting stimulus (massive crush injury or overwhelming infection)
  - Moderate clotting stimulus + shock
- Check D-dimer
  - < 1,000 ng/ml → DIC unlikely
  - > 2,000 ng/ml → DIC likely (in absence of any other clear explanation)

DISSEMINATED INTRAVASCULAR COAGULATION (DIC) – LEVELS



- Mild
  - Microthrombi form but are cleared effectively
  - May escape recognition
- Moderate
  - Microthrombi are ineffectively lysed → occludes microcirculation
    - Examples: ARDS, renal failure, and hepatic failure
- Severe
  - Microthrombi overwhelm the microcirculation
  - Bleeding from fibrinolysis + depletion of coagulation factors

DISSEMINATED INTRAVASCULAR COAGULATION (DIC) – CYCLE



DISSEMINATED INTRAVASCULAR COAGULATION (DIC) – TREATMENT

- Multifaceted approach
  - Remove clotting stimulus (debride dead tissue, drain abscess, etc.)
  - Correct hypothermia
  - Replace blood loss (based on H/H) → PRBCs
  - Replace clotting factor deficits (based on INR) → plasma
  - Possible role for activated factor VII
- Approach is only moderately successful
- Mortality is near 100% for some pts (head injuries)
  - More likely related to underlying pathology

INCREASED INR/PROLONGED aPTT

- Other causes
  - Various isolated factor deficiencies
    - Congenital deficiencies of X, V, and prothrombin (all rare)
    - Acquired factor V deficiency (autoimmune disorders)
    - Acquired hypoprothrombinemia (some lupus pts w/abnormal bleeding)
    - Factor X deficiency (amyloidosis)
  - Stabilized warfarin therapy
  - Animal venoms

BLEEDING DISORDERS
PERIOPERATIVE EVALUATION

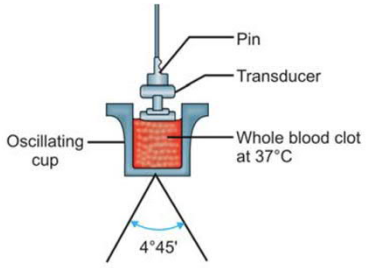
- History and PE is the most single important factor
- VWD = aPTT and bleeding time are prolonged
- ITP = bleeding time is prolonged
- Hemophilia = aPTT is prolonged
- ACT = used to assess degree of heparinization
- FDPs = DIC

THROMBOELASTOGRAPHY (TEG)

- TEG
  - Test of efficiency of blood coagulation
  - Based on viscoelastic properties of whole blood
  - Dynamic assessment of clot:
    - Development
    - Stabilization
    - Dissolution
  - PT, INR, PTT, Fibrin, FDP – Static measurements

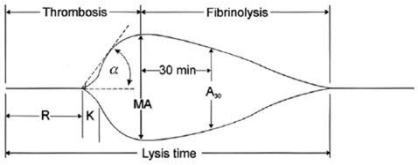
THROMBOELASTOGRAPH

- Oscillating Sample Cup
- Clot Formation
- Measurement of Rotations Forces



TEG WAVEFORM

- R Time
  - "Reaction" time to initial clot formation
- K Time
  - Initial clot formation to 20 mm amplitude.
  - Speed of clot formation
- Alpha angle (α)
  - Angle between baseline and curve tangent
  - Speed of clot formation
- Maximum Amplitude (MA)
  - Reflection of clot strength
- G Value
  - Calculated value of clot strength
- LY30
  - Clot lysis at 30 mins following MA



TEG VALUES

TEG Value	Normal	Measures
R Time	5 – 10 min	Clotting factors (Intrinsic Pathway)
K Time	1 – 3 min	Fibrinogen, Platelet Number
α Angle	53 – 72 degrees	Fibrinogen, Platelet Number
MA	50 – 70 mm	Platelet Number & Function
G Value	5.3 – 12.4 dynes/cm^2	Coagulation Cascade
LY 30	0 – 3%	Fibrinolysis

TEG TRANSFUSION STRATEGY

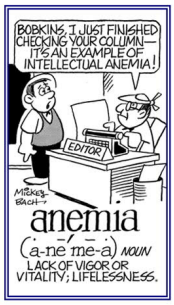
TEG Value	Strategy
R Time > 10	FFP
K Time > 3	Cryoprecipitate
α Angle < 53	Cryoprecipitate +/- Platelets
MA < 50	Platelets, DDAVP
LY 30 > 3%	TXA

TEG TRANSFUSION STRATEGY

Problem With	TEG	R	K or α	MA	Strategy
		N	N	N	
Hemodilution Clotting Factors		H	L or N	L or N	FFP
Fibrinogen Deficiency		H or N	L	L or N	Cryoprecipitate
Platelets		N	N	L	Platelets, DDAVP
Primary Fibrinolysis		N	N	L	TXA
Secondary Fibrinolysis		L	H	H	Treat DIC
Thrombosis		L	H	H	Anticoagulants

ANEMIA
MANAGEMENT

- Common among hospitalized surgical patients
  - EBL from primary condition or surgery
  - Serial blood draws
  - Diminished erythropoiesis
- Treatment changed substantially in the 1990s
  - PRBCs have significant immunosuppressive effects
  - Possible transmission of fatal diseases



ANEMIA
MANAGEMENT

- Need to consider more than just the Hgb level
  - Eval current and predicted need for additional O_2 carrying capacity
- PRBCs should be aggressively transfused
  - ↑ risk for active bleeding (massive liver injury or GI bleed)
    - Establish enough reserve oxygen-carrying capacity
  - Acute coronary artery ischemic syndromes (unstable angina or MI)
- May be a role in certain neurologic conditions (TBI, SCI, or CVA)
  - Clinical evidence is insufficient

ANEMIA
MANAGEMENT

- Consider PRBCs if symptomatic (fatigue, ↑ HR, ↑ RR)
- Asymptomatic = observation (acceptable)
  - For acute anemia → accept Hgb ↓ 6 to 7 g/dl
  - If Hgb < 6, benefits of transfusion outweigh risks
  - If Hgb < 5, ↑↑ mortality (esp in elderly and CV disease)
    - Cellular metabolism cannot be maintained

BLOOD PRODUCTS

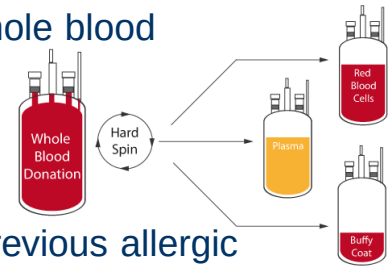
- Whole Blood
- PRBCs
- Platelets
- FFP
- Cryoprecipitate

WHOLE BLOOD

- Rarely used
- Only for acute massive blood loss
- O negative in emergency situations
- CPD = Citrate Phosphate Dextrose and store at 4°C
- Shelf life = 35 days
- Platelets, V, VII, and VIII are lost in 24 hours

PACKED RED BLOOD CELLS (PRBCs)

- Remove plasma from whole blood
- Hct is 70%
- Shelf life = 42 days
- Improves O_2 capacity
- Use washed PRBCs if previous allergic reactions
- Leukocyte-poor → RBCs by filtration



PLATELETS

- Collected by centrifugation of whole blood
  - Done w/in 6 hours of collection
- Can be stored for 5 days
- Single or multiple donor
- Indications
  - Platelets < 10,000
  - Surgery: Platelets < 50,000
- Hypothermia make platelets unable to produce thromboxane A2

FRESH FROZEN PLASMA (FFP)

- Removed from whole blood within 6 hours of collection
- No RBCs, no platelets
- Frozen to -18°C to protect factors V and VIII
- Requires ABO typing (but not cross match)
- Indicated in coagulopathy of liver disease, vitamin K deficiency, Coumadin therapy, dilutional coagulopathy, and antithrombin III deficiency

CRYOPRECIPITATE

- Plasma is first frozen to -90°C
- Slowly warmed to 4°C
- Precipitate forms
  - Separated w/a small amount of plasma
- Provides
  - vWF in VWD
  - Factor VIII in hemophilia A
  - Fibrinogen in DIC

TRANSFUSION COMPLICATIONS

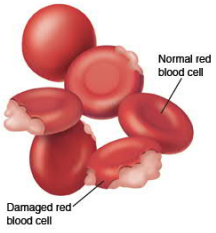
- Hemolytic Reactions
- Delayed Hemolytic Reactions
- Leukoagglutinin Reactions
- TRALI
- Infection Transmission

HEMOLYTIC REACTIONS

- Intravascular Ab and complement-mediated hemolysis
  - From ABO incompatibility (error by personnel)
- Occurs w/first 50 cc of blood
- Symptoms/signs
  - HA, chest pain, SOB, chills, fever, flank pain, bleeding, renal failure
- Treatment
  - Stop transfusion and recheck ABO
  - Hydrate = Keep u/o > 100 cc/hr; mannitol, lasix, $NaHCO_3$

DELAYED HEMOLYTIC REACTIONS

- Due to incompatiblity of minor blood group antigens
- Occurs days later
- Develop fever, chills, and jaundice
- Treatment = same as for acute reactions

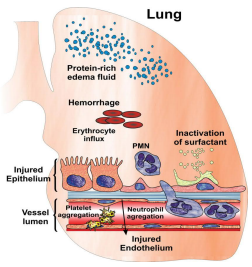


LEUKOAGGLUTININ REACTION

- Febrile transfusion reaction (most common)
- Due to recipient Ab to donor leukocyte antigen
- Presents w/mild fever one hour after the start of transfusion
- Care is supportive

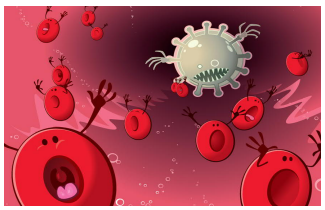
TRANSFUSION RELATED ACUTE LUNG INJURY (TRALI)

- Manifests as acute respiratory failure (ARDS)
- 50% of all blood transfusion mortality
- Due to leukocyte activation by antibodies against HLA
- FFP is the most common offending agent



INFECTION TRANSMISSION

- Hepatitis
  - Most common is B (1 in 843,000 to 1.2 million)
  - Most serious is C (1 per 1.49 million)
  - ↑ risk with cryo and factor concentrate
- HIV
  - Risk is 1 in 1,467,000
- West Nile Virus (WNV)
  - Risk is 1 in 1 million



QUESTIONS

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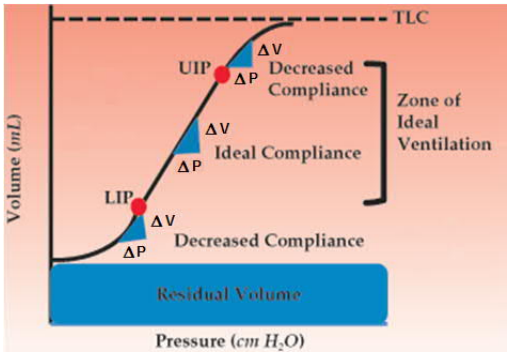
ARDS & RESPIRATORY FAILURE
Surgical Critical Care

Gregory B. York, MD, FACS
Trauma/Critical Care

Presented by Laura Crankshaw, MD
Surgical Critical Care 2025

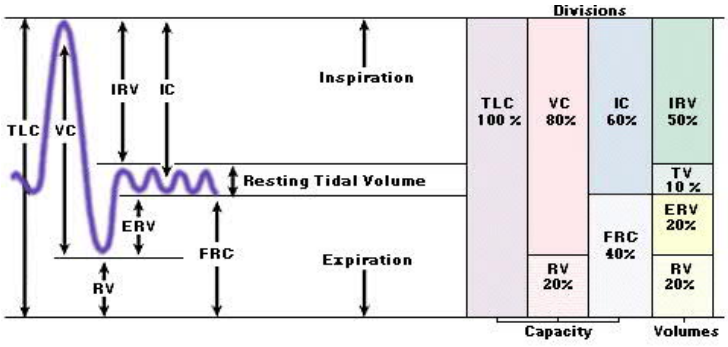
PULMONARY PHYSIOLOGY

- Most Efficient Breathing occurs with:
  - Adequate volume
  - Minimal pressure change
- Compliance
  - $\Delta V / \Delta P$
  - Slope of line
- Work of Breathing
  - Generating the ΔP
  - Area of triangle



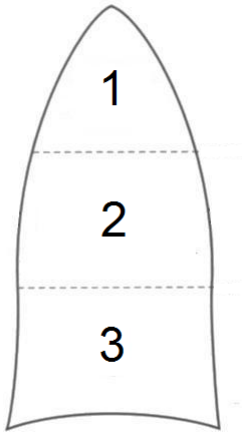
PULMONARY PHYSIOLOGY

- Pulmonary Volumes



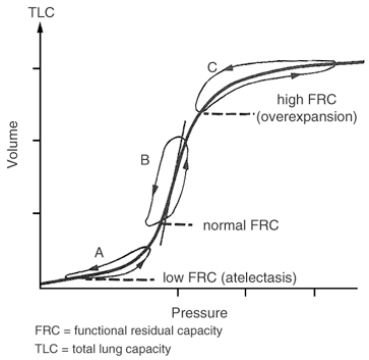
PULMONARY PHYSIOLOGY

- West Zones
  - 1: P_A > Pa > Pv
  - 2: Pa > P_A > Pv
  - 3: Pa > Pv > P_A
- Implications
  - 1: Anatomic dead space
  - 2: Well-matched V/Q
  - 3: Physiologic Shunt



PULMONARY PHYSIOLOGY

- Functional Residual Capacity (FRC)
  - Volume of air in lungs after passive expiration.
  - Functions
    - Oxygen store
    - Buffer for maintaining steady PaO_2
    - Prevents atelectasis
    - Minimize work of breathing
    - Minimize pulmonary vascular resistance
    - Minimize V/Q mismatch
    - Keep airway resistance low
  - Decreased FRC =
    - Increased airway resistance
    - Increased PVR
    - Decreased compliance
    - Increased Work of Breathing

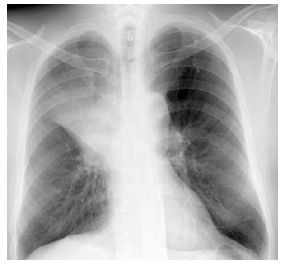


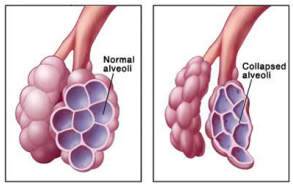
ATELECTASIS

- Pathophysiology
  - Compression: Transmural pressure distending alveoli is reduced (by some mechanism) to a level that allows the alveoli to collapse.
  - Resorption: Obstruction results in trapped gas which continues to be absorbed with eventual alveolar collapse.
  - Surfactant Impairment: Result is increased alveolar surface tension and collapse.

ATELECTASIS

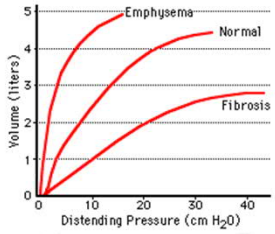
- Effects
  - Decreased Compliance
  - Impaired Oxygenation
  - Increased Pulmonary Vascular Resistance
  - Lung Injury
- Preventing/"Fixing"
  Atelectasis = Recruitment





ALVEOLAR RECRUITMENT

- Depends on:
  - Position ON Pressure-Volume Curve
  - Position OF Pressure-Volume Curve
  - Alveolar Integrity/Status/Damage
  - Number/Percent of Involved Alveoli



ALVEOLAR RECRUITMENT

- Spontaneous Methods
  - Incentive Spirometry
  - Spontaneous Breathing Exercises
- Mechanical Methods
  - Acapella, EzPAP, IPV, etc.
  - CPAP, BiPAP, IPPB
  - Mechanical Ventilation: Recruitment maneuvers
- Adjuncts
  - Bronchodilators
  - Chest Physiotherapy (CPT)
  - Positioning
  - Therapeutic Bronchoscopy

ADULT RESPIRATORY DISTRESS SYNDROME (ARDS)

- Definition: Acute, diffuse inflammatory lung injury, leading to increased pulmonary vascular permeability, increased lung weight, and loss of aerated tissue
- Acute Lung Injury: Term no longer used (Berlin Consensus 2013)

ADULT RESPIRATORY DISTRESS SYNDROME (ARDS)

- Pathophysiology
  - Inflammatory mediator induced injury to pulmonary capillaries and alveolar membrane
  - Capillary leak, alveolar destabilization, interstitial/alveolar inflammation/damage
  - Atelectasis/volume loss, decreased compliance, increased work of breathing, shunting
  - Severe respiratory failure

ARDS

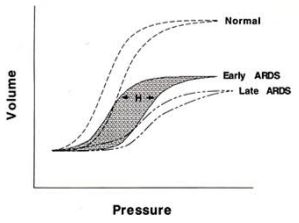
- Inflammatory mediator activation
  - Inciting event
  - Neutrophil activation
  - Release of proteolytic enzymes & toxic oxygen metabolites
  - Membrane damage
  - Inflammatory changes

ARDS

- Capillary leak/alveolar destabilization
  - Large pores in pulmonary capillaries keeps intravascular protein = interstitial protein
  - Membrane inflammation/damage = pore size increases
  - Interstitial edema increases
  - Lymphatic capacity exceeded
  - Alveolar edema results

ARDS

- Atelectasis/Volume Loss
  - Massive fluid/protein influx
  - Unstable geometric shape of alveoli
  - Surfactant denatured
- Pathophysiologic Changes
  - Decreased lung volume(s)
  - Decreased compliance
  - Increased physiologic dead space
  - Higher Inflection Point



ARDS

- Injury or Exudative Phase
  - 1 – 7 days after injury/insult (usually 24 – 48 hrs)
  - Primary Pathophysiology
    - Interstitial & alveolar edema
    - Unstable alveolar geometric shape
    - Surfactant denaturation
    - Atelectasis
  - Pulmonary Physiology
    - Severe V/Q mismatch and shunting of capillary blood
    - Refractory Hypoxemia (unresponsive to increased fiO_2)
  - Pulmonary Mechanics
    - Increased respiratory rate
    - Decreased tidal volume
    - Increased cardiac output
    - Respiratory alkalosis

ARDS

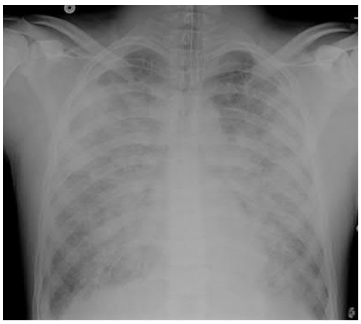
- Reparative or Proliferative Phase
  - 1 – 2 weeks after initial lung injury
  - Primary Pathophysiology
    - Influx of neutrophils, monocytes, lymphocytes
    - Fibroblast proliferation
    - Interstitial fibrosis
  - Pulmonary Physiology
    - Hypoxemia
    - Diffusion limitation/shunting
  - Pulmonary Mechanics
    - Increased pulmonary vascular resistance
    - Pulmonary hypertension
    - Decreased lung compliance
  - Final
    - If reparative phase persists, widespread fibrosis
    - If reparative phase arrested, lesions resolve

ARDS

- Fibrotic or Chronic Phase
  - 2 – 3 weeks after initial lung injury
  - Primary Pathophysiology
    - Lung is sparsely collagenous and fibrous tissue
    - Diffuse scarring and fibrosis
  - Pulmonary Physiology
    - Surface area for gas exchange is significantly reduced
  - Pulmonary Mechanics
    - Hypoxemia continues
    - Pulmonary hypertension
    - Decreased lung compliance

ARDS

- Criteria (Berlin, 2013)
  - Acute onset (< 1 week) of respiratory failure
  - Bilateral opacities c/w pulmonary edema (CT scan or CXR)
  - P/F ratio < 300 (min 5 cm H_2O Peep)
  - Not explained by cardiac failure or fluid overload



ARDS

- Predisposing Conditions
  - Intracranial Hypertension
  - Blood products/catheter sepsis
  - Pneumonia/Pulmonary contusion
  - Cardiopulmonary Bypass
  - Pancreatitis
  - Translocation Endotoxemia
  - Urosepsis/Amniotic Fluid Embolism
  - Long Bone Fracture

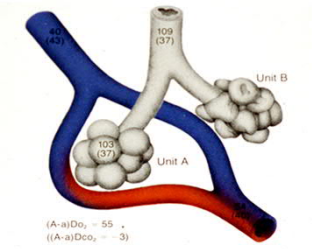
ARDS

- Severity (Berlin, 2013)

Severity	PaO_2/FiO_2*	Mortality
Mild	200 – 300	27%
Moderate	100 – 200	32%
Severe	< 100	45%
* On Peep > 5		

ARDS

- Clinical Progression
  - Early Changes
    - Increased respiratory rate
    - Respiratory alkalosis
    - Chest x-ray clear/mild volume loss
  - Early Failure
    - Dyspnea due to increased work of breathing
    - Chest x-ray: Diffuse infiltrates/moderate volume loss
    - Decreased FRC; decreased compliance
    - Increased shunting (due to atelectasis)



ARDS

- Clinical Progression
  - Late Failure
    - Labored tachypnea
    - Chest x-ray: Extensive infiltrates
    - Respiratory acidosis
    - Severe hypoxemia
    - Consolidation/necrosis/pneumonia

ARDS MANAGEMENT

- Lung-Protective Ventilation
- Fluid Management
- Promoting Oxygen Transport
- Pharmacotherapy

LUNG-PROTECTIVE VENTILATION

- Ventilator-Induced Lung Injury (VILI)
  - Oxygen toxicity: FiO_2 > 60%.
  - Barotrauma: Alveolar injury/rupture related to increased alveolar pressures.
  - Volutrauma: Stress fracture in alveolar-capillary interface related to large tidal volumes.
- Low-Volume Ventilation
  - ARDS Patients
  - Plateau Pressure < 30 cm H_2O.
  - Low V_T(6 ml/kg) vs Conventional V_T (12 ml/kg)
  - Low V_T showed 9% reduction in mortality

LUNG-PROTECTIVE VENTILATION

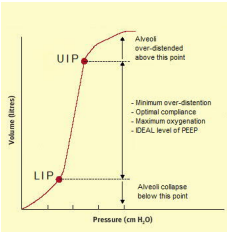
- Permissive Hypercapnia
  - Low volume ventilation = reduced CO_2 elimination.
  - Favoring lung-protective volumes over resultant respiratory acidosis.
  - Limits: Few guidelines – studies show:
    - $PaCO_2$ from 60 – 70 mm Hg.
    - pH between 7.2 – 7.25.
  - Concern
    - Respiratory driven hyperventilation
    - May require paralysis.

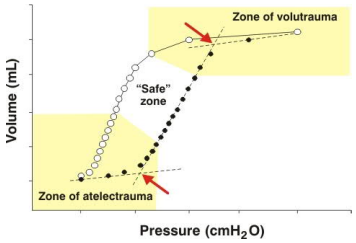
LUNG-PROTECTIVE VENTILATION

- Positive End-Expiratory Pressure (PEEP)
  - Opening/closing terminal airways = Lung Injury
  - Low-level PEEP mitigates.
  - Set PEEP just above lower inflection point.
  - Added benefits
    - Aid to arterial oxygenation.
    - Allows reduced FiO_2 = reduced oxygen toxicity.
  - High Levels
    - Decrease CO
    - No added benefit.

LUNG-PROTECTIVE VENTILATION

- Positive End-Expiratory Pressure (PEEP)
  - Ventilation above UIP = Overdistension = Volutrauma
  - Ventilation below LIP = Underrecruitment = Shearing forces = Atelectrauma

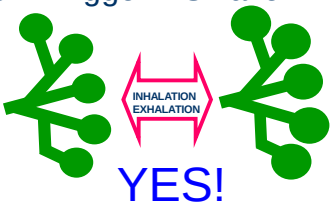




LUNG-PROTECTIVE VENTILATION

- Positive End-Expiratory Pressure (PEEP)
  - Goal: Entire tidal ventilation takes place above LIP.
  - AVOID: Open – Close – Open – Close
  - PROMOTE: Bigger – Smaller – Bigger – Smaller

NO!



YES!

LUNG-PROTECTIVE VENTILATION

- Fluid Management
  - Diuretic Therapy
    - Benefits in lung compliance, gas exchange, length of time on ventilator.
    - Little evidence of survival benefit.
  - Pitfalls of Diuretic Therapy
    - Diuretics remove fluid NOT inflammation.
    - Risk for hemodynamic compromise (especially in positive-pressure mechanical ventilation)

LUNG-PROTECTIVE VENTILATION

- Promoting Oxygen Transport
  - Support each variable in Oxygen Delivery
    - Cardiac Output
      - Goal: 5 – 6 L/min
        - Volume Infusion.
        - Inotropic Agents
          - Avoid Dopamine: Constricts pulmonary veins.
          - Dobutamine favored – less intrapulmonary shunt as seen with vasodilators.
    - Hemoglobin
      - No scientific basis for certain level.
      - Transfuse if evidence of tissue dysoxia.
    - Saturation
      - PaO_2: 55 – 80 mm Hg
      - SpO_2: 88% - 95%.

LUNG-PROTECTIVE VENTILATION

- Pharmacotherapy
  - Glucocorticoids
    - Non-ARDS indications
      - ARDS precipitated by steroid-responsive process
      - Refractory sepsis
    - Moderate to Severe ARDS
      - Early (within 14 days of onset)
      - Persistent/refractory to initial standard therapies
    - Regimens
      - Methylprednisolone: 1 mg/kg x 21-28 days, then taper
      - Dexamethasone: 20 mg IV QD x 5 days, then 10 mg QD x 5 days

ALI/ARDS STRATEGY

- Ventilator Management
  - Set initial VT = 8 ml/kg (PBW).
  - Reduce slowly to 6 ml/kg (PBW).
  - Goal: Pplat < 30 cm H_2O.
- Oxygenation
  - Use FiO_2-PEEP combinations (ARDSNet).
  - Assess P-V curve; set just above LIP.
  - Goal: PaO_2 = 55 – 80 mm Hg or SaO_2 88% – 95%.
- Set respiratory rate
  - Goal: pH 7.30 – 7.45.
  - Permissive Hypercapnia

MISCELLANEOUS TOPICS

- Prone Positioning
- Nitric Oxide
- Heliox
- TRALI

PRONE POSITIONING

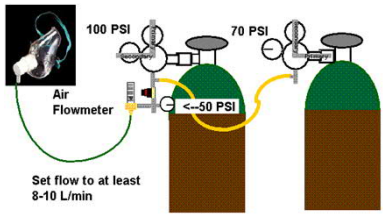
- Rationale
  - ARDS: Localization of lung water in dependent parts of lung.
  - Prone would result in less right-to-left shunting.
  - Prone position gas-filled alveoli would now be in dependent parts of the lung.
- Advantages
  - Alveolar recruitment in dorsal segments.
  - Enhanced secretion drainage.
  - Increased FRC.
  - Enhanced perfusion of uninjured lung.
  - Decreased compression of lung by heart.
- Disadvantages
  - Loss of airway during turning.
  - Pressure ulcers (chin, face).

NITRIC OXIDE

- Rationale
  - NO relaxes (vasodilates) smooth muscle.
  - Selectively increases blood flow to ventilated alveoli.
  - No effect on non-ventilated alveoli – as NO is given by inhalation.
- Advantages
  - Improves PaO_2 transiently.
  - No systemic effects; dissipates quickly.
- Disadvantages
  - No improvement in mortality.
  - High cost.

HELIOX

- Rationale
  - Low density mixture of helium and oxygen.
  - Less dense helium (vs air) flows more readily.
  - Converts turbulent flow into laminar flow.
- Indications
  - Obstructive Lung Disease
  - Asthma exacerbation
  - NOT for patients requiring FiO_2 > 40%.
- Benefits
  - Increased laminar flow.
  - Increased peak expiratory flow (PEFR).
  - Decreased air trapping.
  - Decreased work of breathing.
  - Decreased pulsus paradoxus.



TRANSFUSION-ASSOCIATED ACUTE LUNG INJURY (TRALI)

- Pathophysiology
  - Inflammatory lung injury
  - Antileukocyte antibodies (donor) bind granulocytes (recipient)
  - Promote leukocyte sequestration in lungs
  - Granulocyte-mediated lung injury
- Clinical
  - During or within 6 hours after start of transfusion.
  - More associated with plasma components (platelets, FFP) versus packed RBCs.
  - Dsypnea, hypoxemia
  - Fever is common.
  - Hypotension is reported.
  - Chest x-ray: Diffuse pulmonary infiltrates.

TRANSFUSION-ASSOCIATED ACUTE LUNG INJURY (TRALI)

- Treatment
  - Stop transfusion.
  - This is not pulmonary edema – RBCs do not produce hydrostatic pulmonary edema.
  - Management similar to ARDS.
- Prognosis
  - Incidence: 1:5000 transfusions.
  - Mortality < 10%.
  - Usually resolves 48 – 96 hours.
  - Leading cause of death from blood transfusions.

REFERENCES

- Marino PL. The ICU Book. 3rd Edition. Lippincott Williams & Wilkins. Philadelphia. 2007.
- Parrillo JE, Dellinger RP. Critical Care Medicine. 2nd Edition. Mosby. St Louis 2002.

QUESTIONS

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Hemodynamic Assessment & Monitoring

Presented by Laura Crankshaw, MD, FACS
Surgical Critical Care 2025

THE AMERICAN BOARD OF SURGERY

SURGICAL CRITICAL CARE

CONTENT OUTLINE FOR THE CERTIFYING EXAMINATION (CE) AND CONTINUOUS CERTIFICATION (CC) READMISSIBILITY EXAMINATION

TOPIC	WEIGHT*
A. Initial Resuscitation	6%
B. Cardiovascular Physiology, Pathophysiology, and Therapy	10%
C. Respiratory Physiology, Pathophysiology, and Therapy	9%
D. Fluid and Electrolyte Pathophysiology and Therapy	11%
E. Neurological Physiology, Pathophysiology, and Therapy	5%
F. Metabolic, Endocrinologic, and Nutritional Effects of Surgical Illness	5%
G. Infectious Disease, Pathophysiology, and Therapy	10%

Initial resuscitation: 11 Qs

Cardiovascular Physiology, Pathophysiology & Therapy: 16 Qs

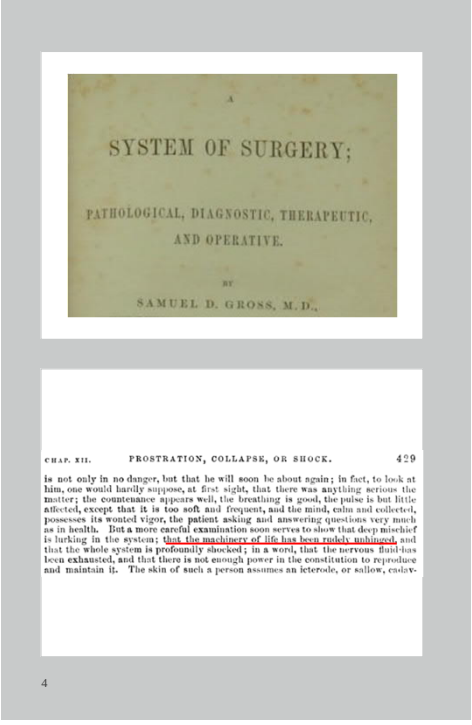
Outline

- Shock
  - Definition
- Hemodynamic Monitoring
  - Waveforms
    - Systemic arterial, central venous, pulmonary arterial catheter
    - Anatomy & analysis of waveforms
- Oxygen Delivery & Consumption
  - Formulas
  - _O2 (Ca, D, V, Sv, Scv)
- Types of Shock
- Use of hemodynamic monitoring data in the management of Shock

Defining Shock

- "the machinery of life has been rudely unhinged…" Samuel Gross 1872
- 1899 Crile: hypotension critical factor in shock
- 1936: Blalock: 4 categories of shock
  - Oligemic (hypovolemic)
  - Cardiogenic
  - Neurogenic
  - Vasogenic
- Surviving Sepsis
- Most widely accepted definition: an acute clinical syndrome that results from INADEQUATE PERFUSION OF TISSUE

Surviving Sepsis Campaign



A

SYSTEM OF SURGERY;

PATHOLOGICAL, DIAGNOSTIC, THERAPEUTIC, AND OPERATIVE.

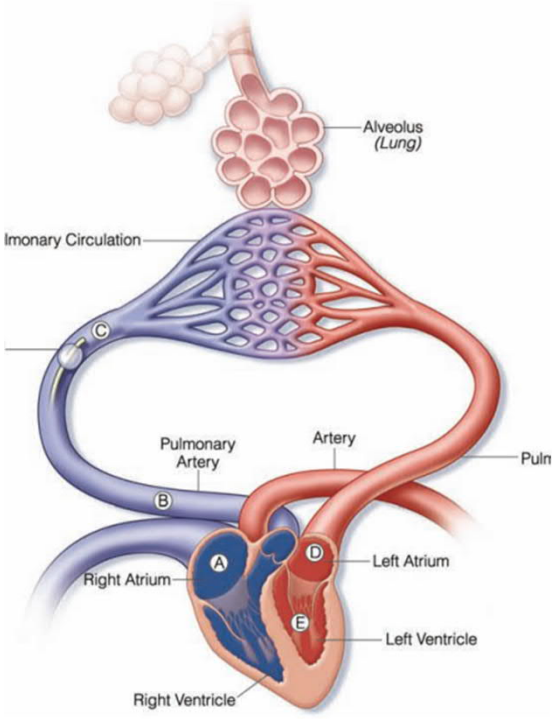
BY

SAMUEL D. GROSS, M.D.,

CHAP. XII. PROSTRATION, COLLAPSE, OR SHOCK. 429

is not only in no danger, but that he will soon be about again; in fact, to look at him, one would hardly suppose, at first sight, that there was anything serious the matter; the countenance appears well, the breathing is good, the pulse is but little affected, except that it is too soft and frequent, and the mind, calm and collected, possesses its wonted vigor, the patient asking and answering questions very much as in health. But a more careful examination soon serves to show that deep mischief is lurking in the system; that the machinery of life has been rudely unhinged, and that the whole system is profoundly shocked; in a word, that the nervous fluid has been exhausted, and that there is not enough power in the constitution to reproduce and maintain it. The skin of such a person assumes an icterode, or sallow, enlav-

4

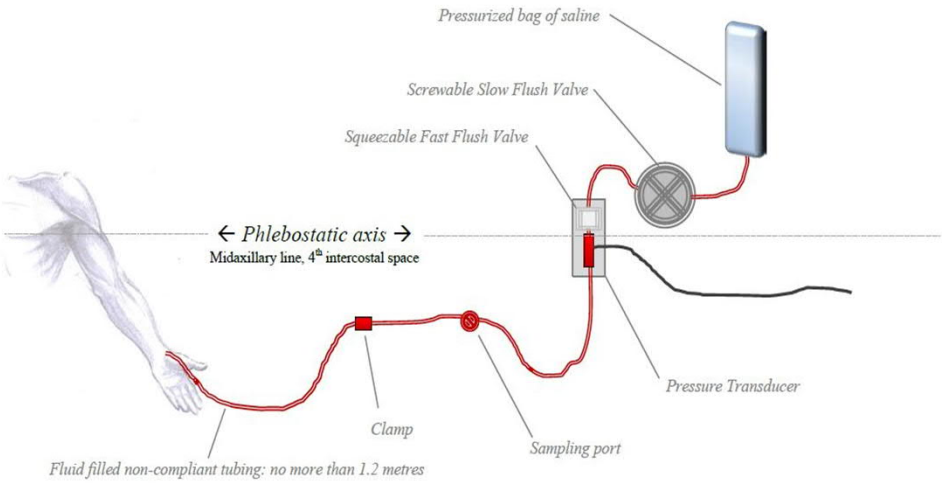


How do we monitor organ perfusion?

- Unfortunately, we can't. At least not directly
- What we can measure:
  - Systemic arterial pressure
  - Central venous pressure
  - Pulmonary arterial pressure
- Pressure is surrogate for volume:
- CVP (RAP) => RV EDV = RV preload
- PCWP (LAP) => LV EDV = LV preload

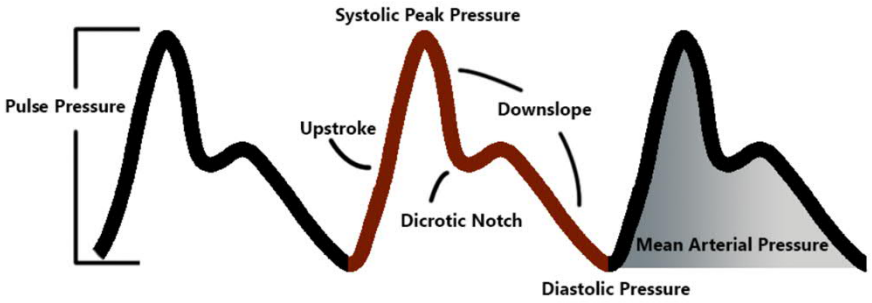
5

Continuous arterial monitoring

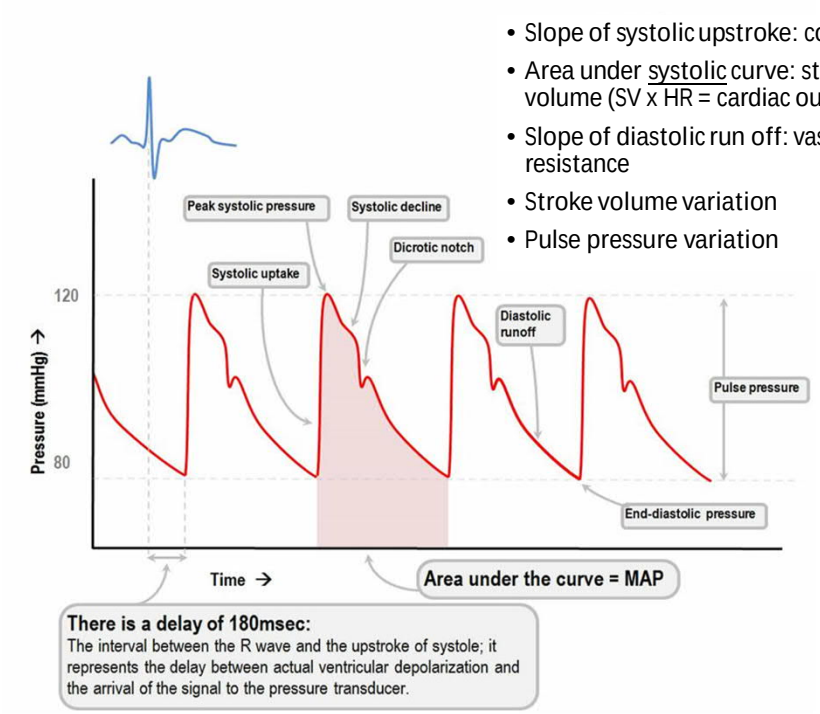


Components of the arterial waveform

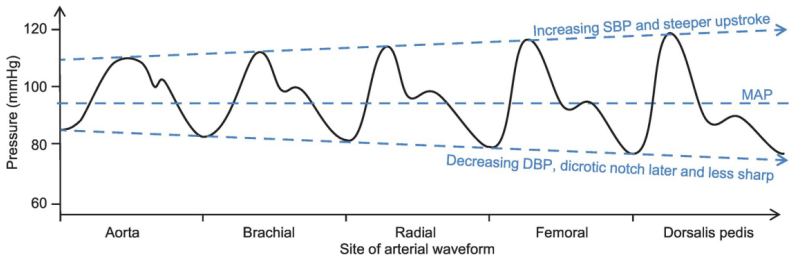
- Upstroke (anacrotic limb): expulsion of blood from LV during systole
- Peak: peak systolic pressure, maximum ventricular contraction
- Dicrotic notch: closure of aortic valve, blood "bounces" off closed AV
- Downslope (dicrotic limb, diastolic run-off): diastole, nadir is DBP
- Mean Arterial Pressure (MAP) = DP + 1/3(SP – DP)



- Slope of systolic upstroke: contractility
- Area under systolic curve: stroke volume (SV x HR = cardiac output)
- Slope of diastolic run off: vascular resistance
- Stroke volume variation
- Pulse pressure variation



Arterial waveform analysis – changes from proximal to peripheral vessels



- From central to peripheral:
  - Upstroke becomes steeper
  - Systolic peak becomes higher
  - Dicrotic notch appears later
  - End diastolic pressure becomes lower
- Peripheral waveforms have:
  - Higher systolic BP
  - Lower diastolic BP
  - Wider pulse pressure
  - MAP relatively unaffected

9

Arterial waveform analysis - Damping

Damping is the interference of the resonant frequency (arterial system or tubing system) on the arterial waveform.

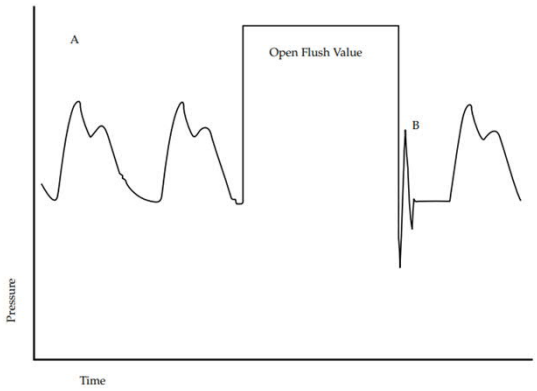
Overdamped
- Falsely ↓ systolic BP
- Falsely ↑ diastolic BP
- Narrow pulse pressure

Underdamped
- Falsely ↑ systolic BP
- Falsely ↓ diastolic BP
- Wide pulse pressure

10

Arterial waveform analysis – Square Wave/Fast Flush/Dynamic Response Test

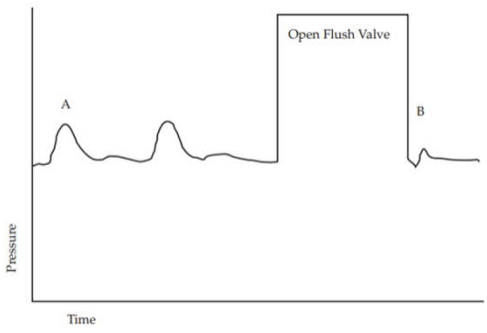
- Optimally damped arterial line system



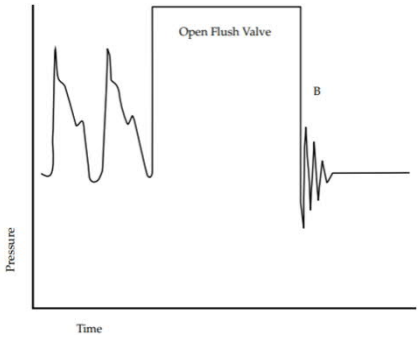
11

Arterial waveform analysis
Over- and underdamped arterial line systems

Overdamped

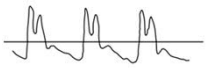
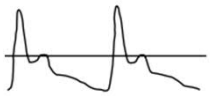
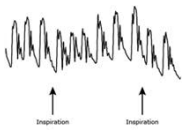


Underdamped



12

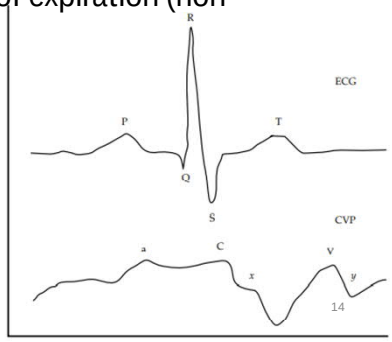
Waveform analysis – Abnormal Arterial Waveforms

Diagnosis		Arterial waveform characteristics
• Aortic stenosis		• Pulsus parvus (narrow PP), pulsus tardus (delayed upstroke)
• Aortic regurgitation		• Wide PP, bisferiens (double peak)
• Hypertrophic cardiomyopathy		• Spike-and-dome pattern (mid-systolic obstruction)
• Left ventricular failure		• Pulsus alternans
• Pericardial tamponade		• Pulsus paradoxus

13

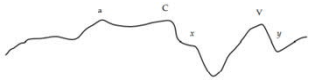
Central Venous Pressure waveform

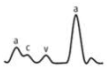
- Benefits: central venous access + allows measurement of CVP, ScvO2
- CVP is surrogate for RA pressure and preload (RV EDV)
  - Influenced by RV compliance, cardiac valve function, intrathoracic pressure (e.g. high PEEP)
- Most accurately measured at end of expiration (non-ventilated and ventilated patients)
- Components of CVP waveform:
- a wave (atrial contraction)
- c wave (isovolumetric contraction)
- x descent (atrial relaxation)
- v wave (systolic filling of atrium)
- y descent (early diastolic filling)



14

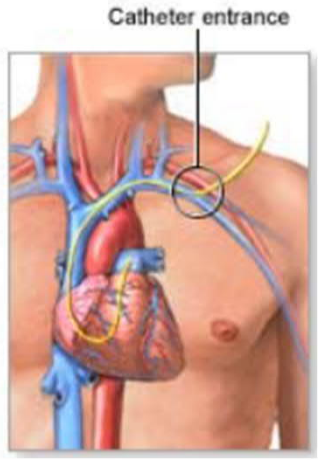
Classic pathologic CVP waveforms

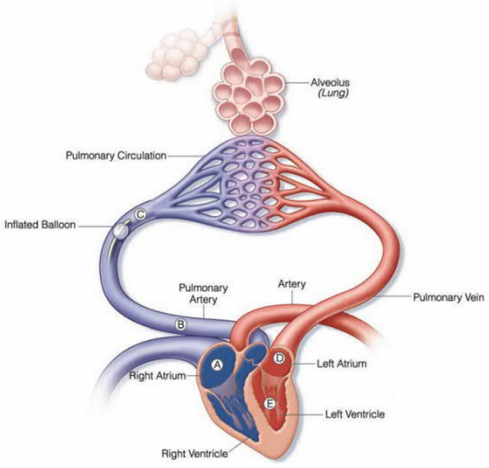


DIAGNOSIS	CVP WAVEFORM CHARACTERISTICS
Atrial fibrillation	Loss of a wave 
Atrioventricular dissociation (junctional rhythms, heart block)	"cannon" a waves 
Tricuspid regurgitation	Loss of x descent 
Tricuspid stenosis	Tall a wave, weak y descent
RV ischemia	Tall a and v waves, steep x and y descents, bifid appearance
Pericardial constriction	Tall a and v waves, steep x and y descents, bifid appearance
Cardiac Tamponade	Loss of y descent 

15

Pulmonary Artery Catheter & Pulmonary Capillary Wedge Pressure





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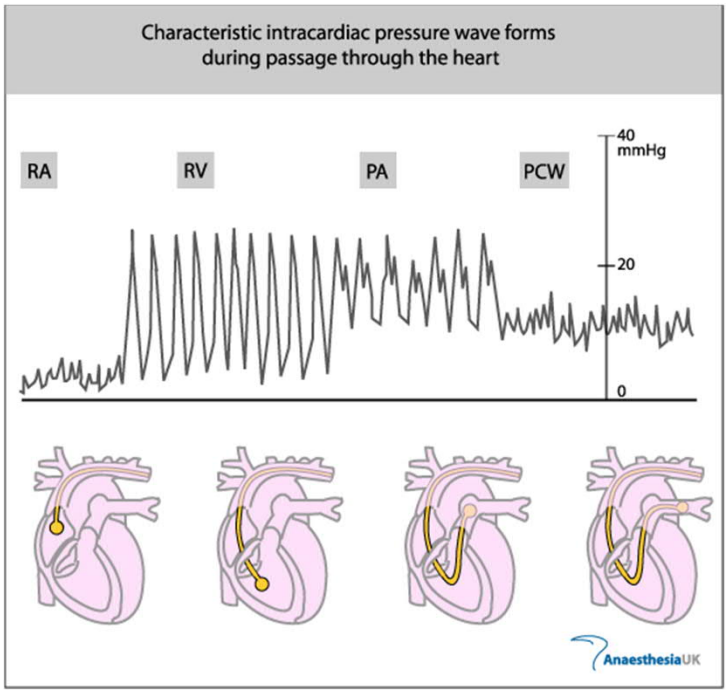
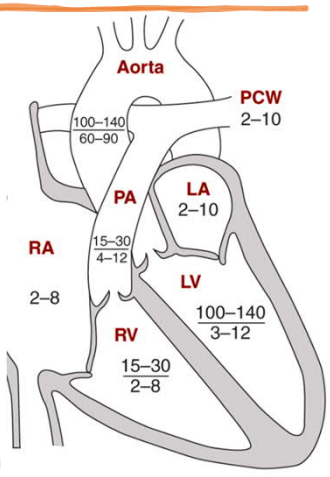


17

Know the Normal Values

- CVP / RA: ~5 mmHg
- RV: 25/5 mmHg
- PA: 25/10 mmHg
- PCWP / LA: 10
- LV: 130/10
- Aorta: 130/80 mmHg

- CO (SV x HR) ~ 4-8 L/min
- CI (CO/BSA) ~ 2.6-4 (L/min)/m2
- SV (CO/HR) ~ 50-100 mL/beat
- SVR (MAP – CVP/CO) x 80 ~ 900-1200 dynes s/cm



18

Interpreting PAC Data to Diagnose Shock States

Types of Shock	CVP	PAWP	Cardiac Index	SVR	Mixed Venous SvO2
Hypovolemic	↓	↓	↓	↑	↓
Distributive (septic, neurogenic)	↓/nl	↓/nl	↑	↓*	↑
Cardiogenic	↑/nl	↑	↓	↑	↓
Obstructive (cardiac tamponade)	↑	↑/nl	↓	↑	↓

19

Pulmonary Artery Catheter (PAC)
Indications and Applications

- Indications for PAC placement:
  - Shock (septic, cardiogenic, traumatic), assessment of response to therapies
  - New onset pulmonary hypertension
  - Perioperative monitoring of patients undergoing cardiovascular surgery
  - Acute MI
- What you can measure directly with a PAC:
  - Heart rate, arterial waveform
  - Cardiac output (thermodilution)
  - PA pressure, RA pressure (CVP), pulmonary arterial occlusion pressure ("wedge pressure"/PAWP)
  - Mixed venous oxygen saturation (SvO2)
- What you can calculate with a PAC:
  - Mean arterial pressure (MAP)
  - Stroke volume
  - Systemic and pulmonary vascular resistance
  - Ventricular stroke work
  - Oxygen delivery and oxygen consumption

20

Pulmonary Artery Catheter
Complications & Contraindications

- Complications specific to PAC:
  - Right heart block
  - Ventricular tachycardia
  - Valvular injury
  - Pulmonary infarction
  - Pulmonary artery rupture
- Absolute contraindications:
  - Infection at insertion site
  - Right atrial or ventricular mass
  - Tetralogy of Fallot
- Relative contraindications:
  - Left bundle branch block (may cause complete heart block)
  - Newly inserted pacemaker wires
  - Coagulopathy

21

OXYGEN DELIVERY & CONSUMPTION

Shock = inadequate perfusion

22

Oxygen Delivery, Consumption & Extraction

Oxygen content (CaO_2)	CaO_2 = (Hgb x 1.34 x SaO_2) + (PaO_2 x 0.003) mL/dL	mL/dL
Oxygen delivery (DO_2)	DO_2 = CO x CaO_2	(x10 to convert mL/dL -> mL/L)
Oxygen consumption (VO_2)	VO_2 = (CO x CaO_2) - (CO x CvO_2) = CO x (CaO_2 - CvO_2) = CO × 1.34 × Hgb × (SaO_2 – SvO_2)*	mL/min
Oxygen extraction ratio (O_2ER)	O_2ER = VO_2 / DO_2 = (SaO_2 – SvO_2) / SaO_2	%
PaO_2 = partial pressure of oxygen dissolved in plasma 1.34 = oxygen carrying capacity of 1g of Hb 0.003 = solubility constant SaO_2 = Hb oxygen saturation (obtain from pulse oximeter) SvO_2 = mixed venous oxygen content (obtain from PAC)		

23

Oxygen Delivery, Consumption & Extraction

Average mixed venous sample of blood from the pulmonary artery is ~75% saturated, thus average O_2 consumption is ~25% of the oxygen being delivered

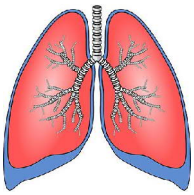
- In normal state: DO_2 = 1000 mL/min of O_2
- VO_2 (25% of 1000 mL/min) = 250 mL/min of O_2
- In shock state: ↓ CO results in ↓ delivery of O_2,
  - The body compensates by increasing the extraction of oxygen (↑O_2ER)
- Different organs have different maximum O_2 extraction rates
  - Heart ~ 100%
  - Kidneys ~ 50%
- If DO_2 is inadequate to meet demand, or O_2 extraction is limited → tissue hypoxia -> cell dysfunction, ↓ ATP production -> organ dysfunction (shock state)

24

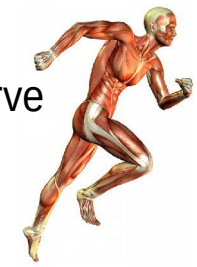
Altered Consumption (SVO_2)

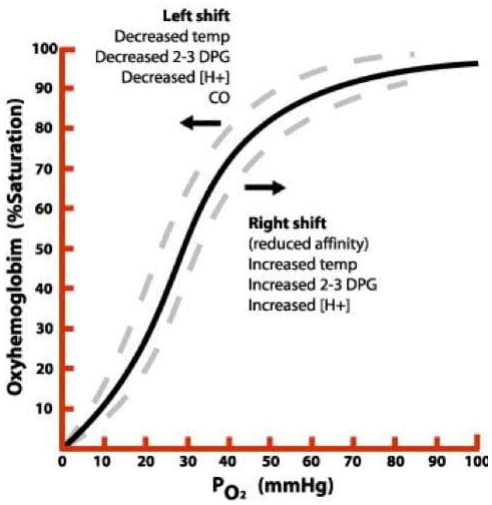
- Factors resulting in INCREASED SvO2:
  - Increase in delivery (DO_2): CO, Hb, SaO_2
  - Decrease in consumption (VO_2): hypothermia, sedation, mechanical ventilation, sepsis, cell death, A-V shunting
- Factors resulting in DECREASED SvO2:
  - Decrease in delivery (DO_2): CO, Hb, SaO_2
  - Increase in consumption: hypermetabolic sepsis, "fighting the vent"
- Beware of ignoring an <u>increased SVO_2</u>
  - Sepsis, shunting, dying

25



Oxyhemoglobin dissociation curve





26

Strategies to Improve Survival in MOSF

- Correct underlying disorder

- Early use of proper monitoring & interpretation of data
  - Arterial line, central venous line, pulmonary artery catheter
  - Focus on trends and responses to your interventions, not just numbers

- Optimize oxygen delivery & utilization
  - Largest increase in DO_2 is achieved through increasing CO!
  - ↓ energy expenditure: ventilator, paralyze, fevers

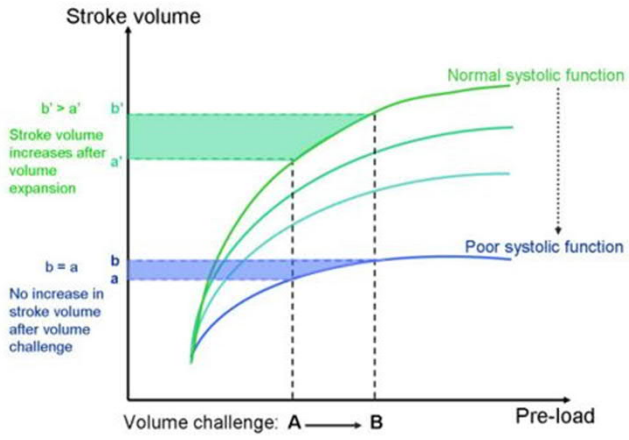
- Appropriate resuscitation
  - Fluid before pressors
  - All shock states should begin with restoring adequate circulating volume

- Avoid secondary insults
  - Nutrition, infection, reperfusion injury

27

Q : How Much Fluid?

A : Just enough to see if they "respond to it"



Improving oxygen delivery = improving cardiac output

- Inotropes & Vasopressors – given based on data obtained from invasive hemodynamic monitoring
  - Catecholamines
    - Norepinephrine (Levophed)
    - Epinephrine
    - Dopamine
  - Dobutamine
    - ↑↑ cardiac O_2 consumption (use in stress myocardial perfusion imaging)

29

Improving oxygen delivery = improving cardiac output

- Phenylephrine (Neo-synephrine)
  - Rapidly treats sudden cardiovascular collapse
  - ↑ MAP in shock patients with aortic stenosis, treat sildenafil and nitrate induced hypotension
- Phosphodiesterase Inhibitors (Milrinone)
  - Prevents breakdown of cAMP in myocardial and vascular smooth muscle -> enhances ventricular contraction and vasodilation
- Vasopressin
  - Acts on V1 receptors in vascular smooth muscle, V2 receptors in nephron
  - No effect on CO

30

Pressor effects on hemodynamics

Inotrope/Vasopressor	Effect on CO	Effect on SVR
Dobutamine beta1 > beta2	BIG increase	decrease (vasodilation)
Milrinone PD inhibitor -> cAMP	increase	decrease
Dopamine D2 receptors, mild beta1, alpha1	increase	increase
Norepinephrine alpha1 > alpha2	Big increase	increase
Epinephrine beta1, alpha1	Big increase	increase
Phenylephrine alpha	Almost no effect	BIG increase

31

Take Away Points

- No single vital sign, physiologic variable, lab marker or invasive hemodynamic measurement will tell us exactly what is happening at the tissue level
- Invasive hemodynamic monitoring techniques can be invaluable in helping diagnose shock states and in guiding fluid resuscitation and vasopressor and inotropic support
- Always use data in conjunction with your clinical exam
- Focus on the trends and responses to your interventions, not just the numbers

32

For the Exam

- Know the arterial, central venous, and pulmonary arterial waveforms
  - Be ready to be given pulmonary artery catheter data and calculate SVR
  - Be ready to diagnose a shock state based on pulmonary artery catheter data
- Know the oxygen delivery equation
  - Be ready to be given values and calculate and O_2ER or DO_2
- Know your inotropes and vasopressors
  - Mechanism of action, effects on hemodynamics and why

33

Questions?

34

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OXYGEN TRANSPORTATION & VENTILATOR MANAGEMENT

Surgical Critical Care

Gregory B. York, MD, FACS
Trauma/Critical Care

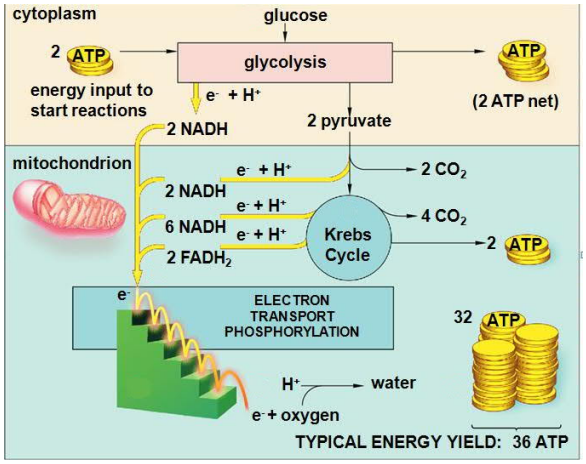
Presented by Laura Crankshaw, MD
Surgical Critical Care 2025

OXYGEN PHYSIOLOGY

- Adenosine Triphosphate (ATP)
  - Phosphate Bonds = High Energy
  - Required for cell function & structure
  - Fundamental to human life
- Oxidative Phosphorylation

$$ADP + Energy \leftrightarrow ATP$$

OXYGEN PHSIOLOGY



OXYGEN DELIVERY

- DO_2 - Rate at which oxygen reaches the systemic capillaries.
- $DO_2 = CO \times CaO_2 \times 10$
- $CaO_2 = (1.34 \times Hb \times SaO_2) + .0031 \times PaO_2$
- Normal DO_2 = 900 – 1,100 mL/min

OXYGEN CONSUMPTION

- VO_2 – Rate at which oxygen leaves capillary blood and moves into tissues.
- Approximates metabolic rate
- Cannot clinically measure
- Different tissues = Different VO_2
- $VO_2 = CO \times (CaO_2 - CvO_2) \times 10$
- Normal = 200 – 270 mL/min
- Highest VO_2 Organ = Heart

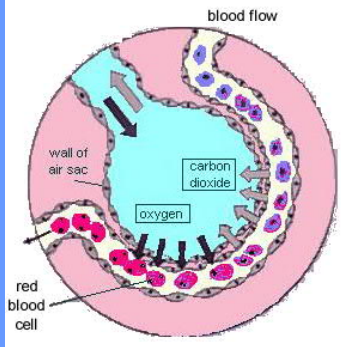
OXYGEN-EXTRACTION RATIO

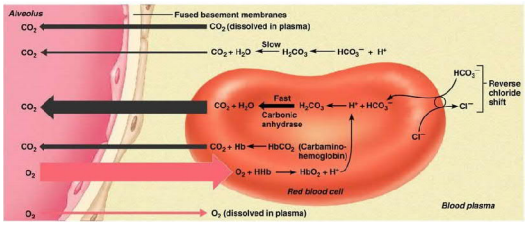
- O_2ER – Fraction of oxygen delivered to the capillaries that is taken up into the tissues.
- $O_2ER = VO_2 / DO_2$
- Normal O_2ER: 0.2 – 0.3
- High Extraction
  - Excessive consumption
  - Poor delivery
- Low Extraction
  - Lack of tissue consumption
  - Diffusion abnormalities

OXYGEN TRANSPORT

- Series of steps from lungs to cells
- There is NO storage of oxygen
- Oxygen Delivery must be continuous
- Process Involves
  - Pulmonary System
  - Circulatory System

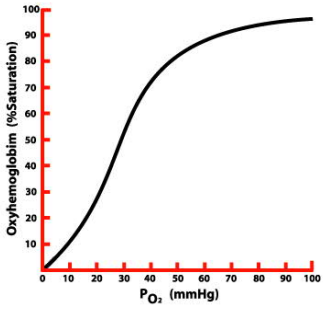
OXYGEN – UPLOADING/TRANSPORT



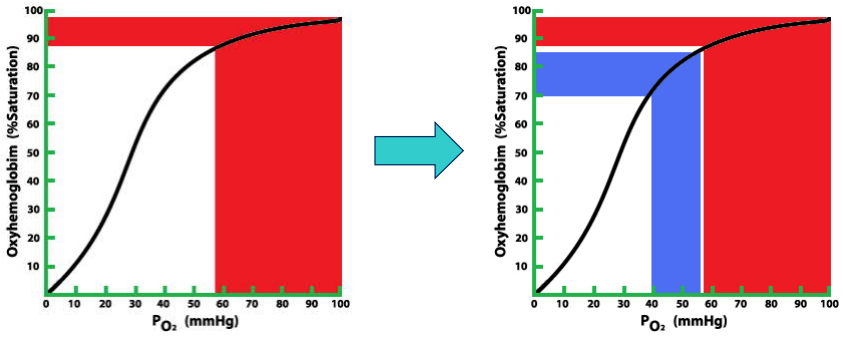


OXYGEN – HOW MUCH?

- Decreased O_2 Affinity (Right shift)
  - Increased Temperature
  - Increased 2,3-DPG
  - Increased pCO_2
  - Decreased pH (acidosis)
- Increased O_2 Affinity (Left shift)
  - Decreased Temperature
  - Decreased 2,3-DPG
  - Decreased pCO_2
  - Increased pH (Alkalosis)

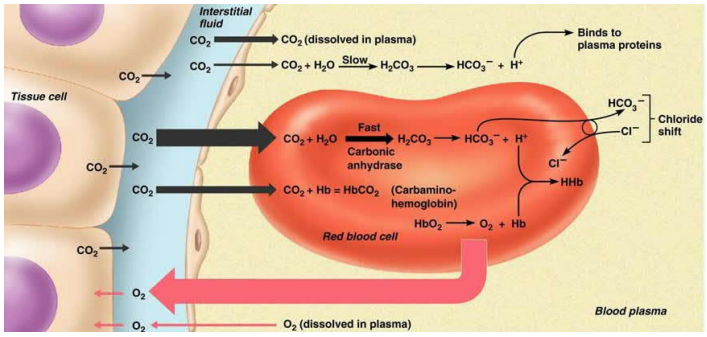


OXYGEN – OFFLOADING

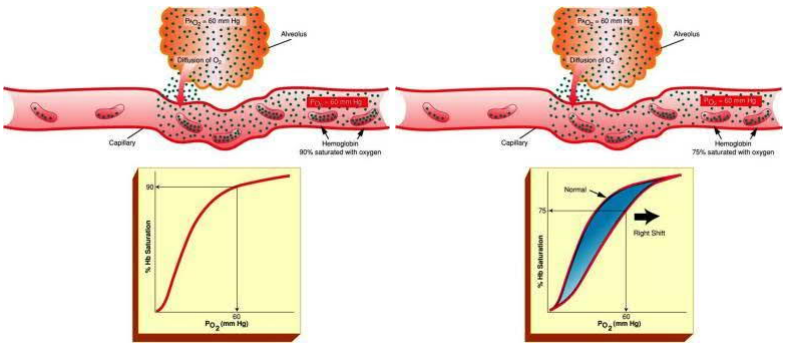


OXYGEN – OFFLOADING

- Cellular Processes



OXYGEN – OFFLOADING

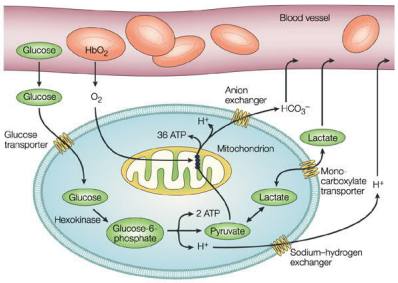


OXYGEN – OFFLOADING

- Requires Hypoxemia
- Generates capillary pO_2 which becomes the O_2 diffusion driving force
- Increased only by:
  - Further decrease in tissue pO_2
  - Right shift in Oxyhemoglobin dissociation curve
- Due to Oxygen gradient between capillaries and cells

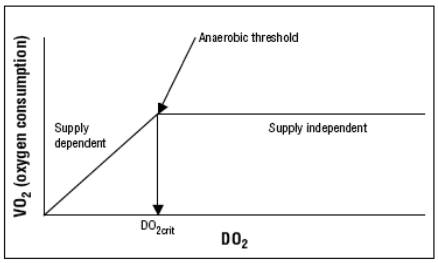
OXYGEN – CRITICAL pO_2

- Critical pO_2
  - Capillary pO_2 REQUIRED to generate an adequate diffusion force
  - No compensatory mechanism exists to correct this physiologic process if it is inadequate enough to generate sufficient mitochondrial pO_2



OXYGEN – CRITICAL DO_2

- Minor DO_2 variability is routine
- Progressive decline = compensatory processes
- Exceeding compensatory processes = Tissue Hypoxia
- Below Critical DO_2 – Physiological support is inadequate



OXYGEN DEBT

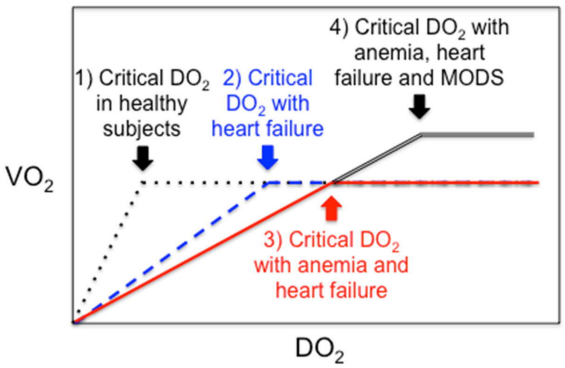
- When aerobic metabolism is insufficient
  - Oxygen debt is created
  - Metabolism changes
- Anaerobic metabolism increases
  - Lactate production rises
  - H^+ production rises
- Oxygen debt = oxygen required to reverse anaerobic metabolism

OXYGEN DEBT

- Effects of Continued Oxygen Debt
  - Metabolism change
  - Deterioration in cell structure/function
  - Pro-inflammatory mediator release – SIRS
  - Endothelial Cell
    - Vascular reactivity – maldistribution of capillary flow
    - Cell aggregation – Neutrophils/platelets/mediator release
    - Cell junction separation – Capillary leak
  - Compensatory responses occur
- Treatment: Oxygen

CRITICAL DO_2 IN CRITICAL ILLNESS

- Critical Illness INCREASES Critical DO_2



OXYGEN DELIVERY – DETERMINANTS

- Hemoglobin/Oxygen Binding
  - Anemia/decreased hemoglobin
  - Hemoglobin dysfunction
  - Atmospheric hypoxia
- Pulmonary
  - Primary/secondary lung disease
  - Hypoventilation
  - V/Q abnormality

$$DO_2 = CO \times CaO_2 \times 10$$

OXYGEN DELIVERY – DETERMINANTS

- Cardiovascular
  - Decreased CO states
  - Peripheral vascular dysfunction
  - Some areas may be OK; some may be critical
- Oxygen Offloading – Rarely causes significant issues

OXYGEN DELIVERY – COMPENSATION

- Mechanisms
  - Usually work well in mild-moderate states
  - Acute vs Chronic Compensation
- Acute Mechanisms
  - CO Increase
  - Arterial flow redistribution
  - Local microcirculatory response
    - Result is to increase flow
    - Autonomics
    - Endothelial cell mediator release (e.g. vasodilators)
  - Increased offloading of oxygen from hemoglobin
  - Intracellular biochemical protective responses
    - Heat shock proteins
    - Glucose regulating proteins
- Chronic Mechanisms
  - Generally organ function changes
  - Primarily cardiovascular or pulmonary
  - Hemoglobin increase

TISSUE OXYGEN – MONITORING

- Use of “Routine” Monitoring
  - Patient appearance
  - Vital signs
  - Hemoglobin level
  - Arterial pO_2
  - Central Venous Pressure
- POOR correlation with tissue O_2 status

TISSUE OXYGEN – MONITORING

- Use of Metabolic Status
  - Lactate
  - Base Deficit/Excess
  - DO_2 & VO_2 Measurements/Calculations
- Fair correlation with tissue O_2 status
- Use only with good understanding/caution

TISSUE OXYGEN – MONITORING

- Use of Venous Oxygen Status
  - Mixed Venous Oxygen Saturation (SvO_2)
  - Central Venous Oxygen Saturation ($ScvO_2$)
- Good correlation with tissue O_2 status
- Recommended methods of use
- Normal Values
  - SvO_2: 68% - 77%
  - $ScvO_2$: Approximately 5% above SvO_2 values

TISSUE OXYGEN – MONITORING

- Issues
  - Non-hypoxic tissue can generate mild lactic acidosis
  - Global versus regional dysoxia
  - "Satisfactory" SvO_2 can occur in setting of hypoxia
    - High DO_2 and High VO_2 = Normal SvO_2
    - Low DO_2 and Low VO_2 = Normal SvO_2

DO_2 DEFICIT – MANAGEMENT

- Hemoglobin/Oxygen Binding
  - RBC Transfusion
  - Increase Oxygenation (ie. FiO_2, Peep, etc.)
    - To effect Saturation
    - To effect PaO_2.
- Pulmonary
  - Treat primary/secondary lung disease
  - Ventilator adjustments (ie. FiO_2, Peep, I:E ratio, etc)

DO_2 DEFICIT – MANAGEMENT

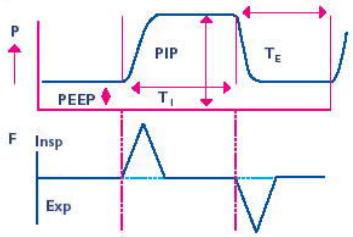
- Cardiovascular
  - Augment Cardiac Output
    - Volume (ie. Crystalloid, colloid, plasma, RBCs, etc)
    - Inotropic agents: Dobutamine, Milrinone
- Other Management
  - Artificial Oxygen Carriers
    - Hemoglobin-based Solutions: PolyHeme, Hemopure, Hemolink
    - Non-hemoglobin Solutions: Perfluorocarbon emulsions
    - Issues
      - FDA Approval
      - Vasoconstriction
      - Increased LFTs
  - Hyperbaric Oxygen
    - Indications: CO Poisoning, Decompression Sickness, Wound Care, Ischemic grafts/flaps
    - Issues: Seizures, Oxygen toxicity, Barotrauma, Myopia

VENTILATOR MANAGEMENT

- Some Basics
  - PEEP
  - Auto-PEEP
- Non-Invasive Modes
  - CPAP
  - BiPAP

POSITIVE END EXPIRATORY PRESSURE (PEEP)

- Application of positive pressure throughout the airway during the expiratory phase of mechanical ventilation.
- Not a ventilatory mode; additional support.



POSITIVE END EXPIRATORY PRESSURE (PEEP)

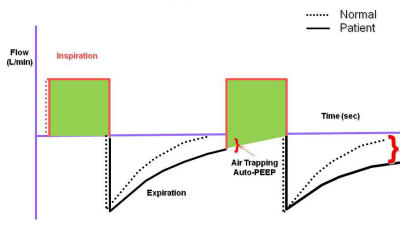
- What PEEP does
  - Increase alveolar surface area.
  - Increase FRC.
  - Increase PaO_2.
  - Decrease shunt fraction.
  - Decrease hypoxic pulmonary vasoconstriction.
  - Decrease Cardiac Output (if high, > 15).
  - No change in $PaCO_2$ or pH.

POSITIVE END EXPIRATORY PRESSURE (PEEP)

- Advantages
  - Provides increased PaO_2 for given FiO_2.
  - Can increase lung compliance.
  - Can decrease work of breathing.
  - Alveolar recruitment maintenance.
- Disadvantages
  - Increases mean pressures, ICPs, PVR.
  - Decreases venous return.
  - Increases barotrauma.

AUTO-PEEP

- Incomplete expiration prior to inspiration resulting in progressive air trapping.
- Effects
  - Lung hyperinflation
  - Decreased right, then left heart filling.
  - Barotrauma.

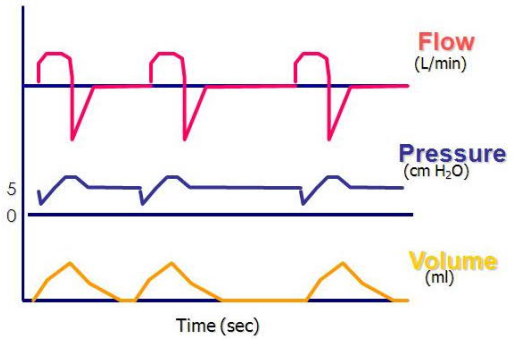


AUTO-PEEP

- How to “Fix”
  - Decrease respiratory rate.
  - Decrease tidal volume.
  - Decrease I:E ratio.
  - Increase PEEP.
  - Increase Inspiratory Flow.
  - Increase sensitivity.

CONTINUOUS POSITIVE AIRWAY PRESSURE (CPAP)

- Application of positive pressure throughout spontaneous breathing cycle.



CONTINUOUS POSITIVE AIRWAY PRESSURE (CPAP)

- Advantages
  - Provides increased PaO_2 for given FiO_2.
  - Can increase lung compliance.
  - Can decrease work of breathing.
  - Alveolar recruitment.
- Disadvantages
  - Increases mean pressures, ICPs, PVR.
  - Requires intact respiratory drive.
  - Decreases venous return.
  - Increases barotrauma.
  - Hypoventilation is a hazard.

BI-LEVEL POSITIVE AIRWAY PRESSURE (BIPAP)

- Patient triggered, pressure-limited breaths.
- Positive pressure throughout expiration.
- Equivalent to PSV + CPAP.
- Tidal volume depends on pressure difference (IPAP – EPAP) and patient effort.

BI-LEVEL POSITIVE AIRWAY PRESSURE (BIPAP)

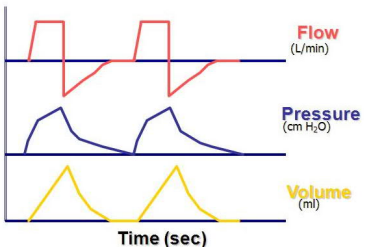
- Advantages
  - Fewer hazards than invasive ventilatory support.
  - Patient controls rate of breathing, inspiratory time and flow.
- Disadvantages
  - Requires cooperative patient with adequate secretion clearance.
  - Most have limited FiO_2 and pressure capability.
  - Airway interface issues (e.g. leaks, discomfort, tissue damage)

VENTILATOR MANAGEMENT

- Categories
  - Controlled vs Assisted
  - Volume Control vs Pressure Control
- Modes
  - CMV
  - A/C
  - IMV/SIMV
  - PSV
  - PCV
  - APRV

CONTROLLED MANDATORY VENTILATION (CMV)

- Patient breaths at rate and time interval set on ventilator.
- No patient triggering.
- Set tidal volume with each breath.

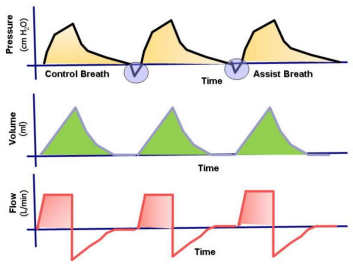


CONTROLLED MANDATORY VENTILATION (CMV)

- Advantages
  - Full control
  - Eliminates work of breathing (as long as no effort occurs by patient)
- Disadvantages
  - Poorly tolerated.
  - Usually results in asynchrony and increased work of breathing.
  - May require heavy sedation and/or neuromuscular paralysis

ASSIST-CONTROL VENTILATION (A/C)

- Ventilator provides set rate.
- Set tidal volume with each breath.
- Patient can trigger additional breaths.
- Spontaneous breaths augmented to receive set tidal volume.

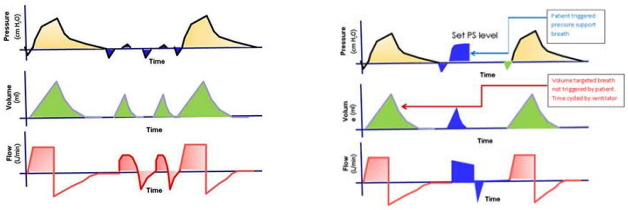


ASSIST-CONTROL VENTILATION (A/C)

- Advantages
  - Patient controls rate & CO_2 level.
  - May avoid need for sedation and/or paralysis.
  - Guaranteed baseline respiratory rate.
- Disadvantages
  - Hyperventilation can occur.
  - Asynchronous breathing can occur.
  - May worsen auto-PEEP in COPD patients.
  - Volutrauma at higher tidal volumes.

INTERMITTENT MANDATORY VENTILATION (IMV)

- Patient breaths spontaneously between machine breaths.
- Full ventilator support at set rates; partial support at lower rates.
- Spontaneous breaths may be pressure supported.
- SIMV – Machines breaths synchronized to patient effort.

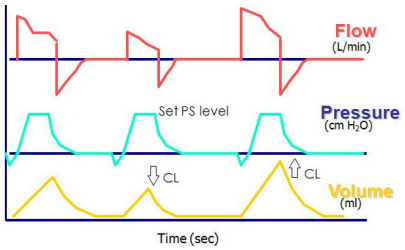


INTERMITTENT MANDATORY VENTILATION (IMV)

- Advantages
  - Graded levels of ventilatory support.
  - Spontaneous breathing allowed.
  - Decreased need for sedatives and/or paralysis.
  - Lower mean pressures than A/C.
  - Less asynchrony.
- Disadvantages
  - Inferior weaning mode.
  - Hypoventilation at low rates.
  - May increase work of breathing.
  - Asynchrony can occur with machine breaths.

PRESSURE SUPPORT VENTILATION (PSV)

- Patient triggered spontaneous breaths
- Pressure-limited & flow-cycled.
- Tidal volume depends on pressure setting and patient effort.
- Inspiratory flow reduction triggers expiratory phase.

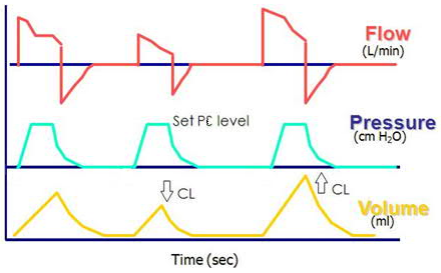


PRESSURE SUPPORT VENTILATION (PSV)

- Advantages
  - Patient controls rate, inspiratory time, and flow.
  - Improves muscle conditioning.
  - Facilitates weaning.
  - Lower rate, higher tidal volumes, less work compared to unsupported breaths.
- Disadvantages
  - Hypoventilation can occur.
  - Variable tidal volumes.
  - Patient must have intact respiratory drive.

PRESSURE CONTROLLED VENTILATION (PCV)

- Patient breaths at set pressure, time (or I:E), and rate.
- Patient determines flow.
- Tidal volume varies with resistance and compliance.

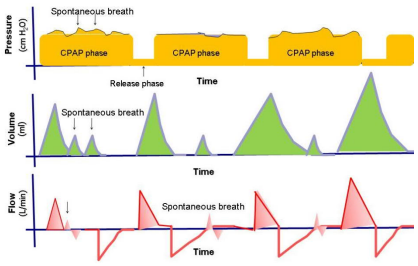


PRESSURE CONTROLLED VENTILATION (PCV)

- Advantages
  - Decreases volutrauma.
  - Alveolar recruitment.
  - Decelerating inspiratory flow pattern – reduces peak airway pressures.
- Disadvantages
  - Hypoventilation in presence of auto-PEEP.
  - Permissive hypercapnia.
  - Inflation volumes vary.

AIRWAY PRESSURE RELEASE VENTILATION (APRV)

- Continuous Positive Airway Pressure – modified form.
- Time-cycled release to lower pressure.



AIRWAY PRESSURE RELEASE VENTILATION (APRV)

- Advantages
  - Lower airway pressure for given tidal volume.
  - Reduces dead space ventilation.
  - Allows for spontaneous breathing throughout.
  - Decreased sedation use.
  - Decreased/eliminated paralytic use.
- Disadvantages
  - Affected by changes in lung compliance – must monitor tidal volumes.
  - Asynchrony may occur – but not yet reported as problematic.

SPONTANEOUS BREATHING TRIAL (SBT)

- Criteria
  - Acceptable oxygenation ($PaO_2 \geq 60$ mm Hg) with:
    - $FiO_2 \leq 50\%$
    - PEEP ≤ 8.
  - Hemodynamically stable
  - Spontaneous breathing.
  - pH ≥ 7.25.
- Mechanics
  - Spontaneous breathing: 30 – 120 minute trial.
  - Ventilator vs T-Piece
  - Allow reduced support: PSV 5; PEEP 5
- Extubation
  - RSBI ≤ 105
  - 70% - 90% success at 48 hours.
- Failure
  - Place on stable, nonfatiguing mode (A/C or PSV)
  - Repeat in 24 hours.

REFERENCES

- Marino PL. The ICU Book. 3rd Edition. Lippincott Williams & Wilkins. Philadelphia. 2007.
- Parrillo JE, Dellinger RP. Critical Care Medicine. 2nd Edition. Mosby. St Louis 2002.

QUESTIONS

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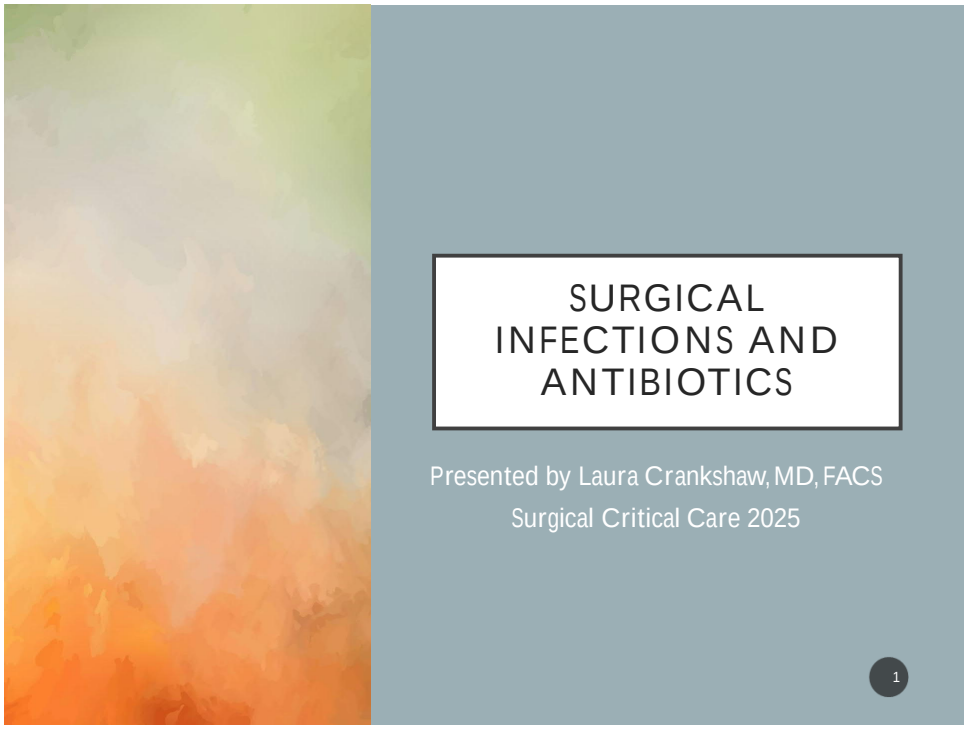
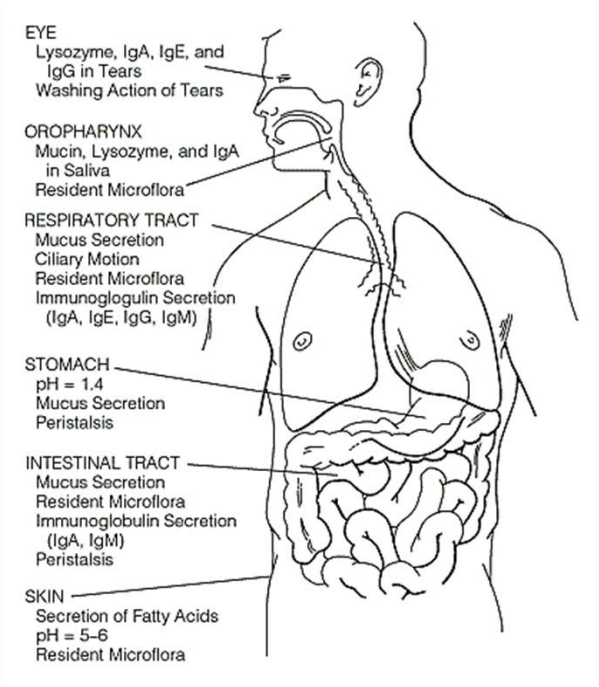


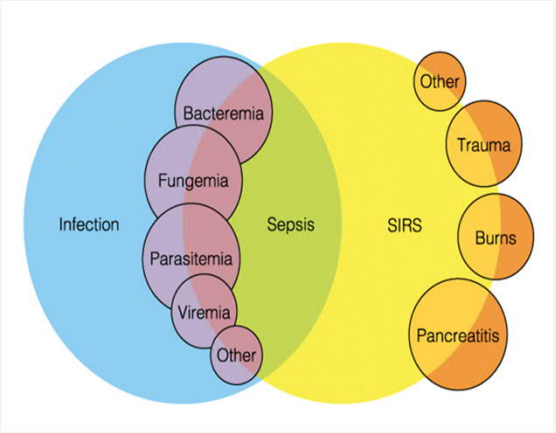
INTRODUCTION

- Definition of Key Concepts
- Surgical Site Infections
- Surgical Wound Classification
- Approach to the Diagnosis of Infection
- Commonly Encountered Organisms
- Antibiotic Selection
- Causes of Peritonitis
- Radiological Examples of Intra-abdominal Abscesses

2



INTERRELATIONS AMONG INFECTION



- Depicted are the interrelations among infection, sepsis, and systemic inflammatory response syndrome

DEFINITION OF KEY CONCEPTS

- Infection
- Bacteremia
- Sepsis
- Severe Sepsis
- Septic Shock
- Systemic Inflammatory Response System
- Multiple Organ Dysfunction Syndrome

5

Bacteremia

Presence of a viable bacteria in the blood.

Infection

Microbial phenomenon characterized by an inflammatory response to the presence of microorganisms or the invasion of normally sterile host tissue by these organisms.

6

SEPSIS

- Systemic response to infection
- Manifested by two or more of the following conditions as a result of infection:
  - T > 38°C or < 36 ° C
  - HR > 90
  - RR > 20
  - pCO_2 < 32 mmHg
  - WBC >12,000 or < 4,000 or bands accounting for more than 10% of neutrophils present.

7

SEVERE SEPSIS

- Sepsis associated with organ dysfunction, hypoperfusion or hypotension.
- May include, but not limited to oliguria, acute alteration of mental status, lactic acidosis.

8

SEPTIC SHOCK

- Sepsis with hypotension, occurring despite adequate fluid resuscitation, along with the presence of perfusion abnormalities.

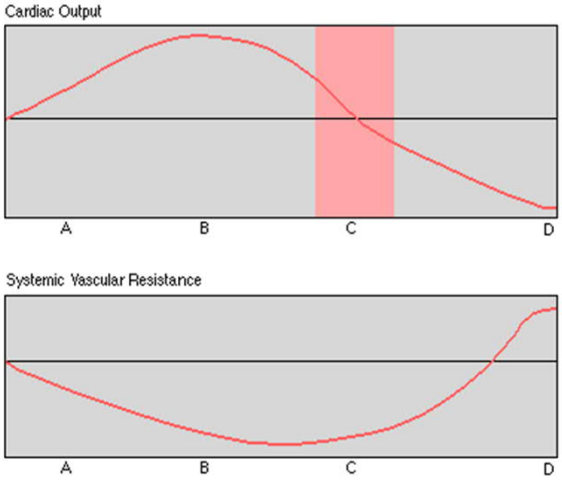
9

SEPTIC SHOCK

- May be the first signal of infection
- Manifestations include tachycardia, hypotension, warm/dry extremities
- Hypermetabolic and hyperdynamic state

10

CARDIAC OUTPUT VS SYSTEMIC VASCULAR RESISTANCE



- A – normal adaptive stress response
- B – early decompensation
- C – clinical septic shock (SVR < 800)
- D – preagonal period before death

11

SYSTEMIC INFLAMMATORY RESPONSE SYNDROME (SIRS)

- Manifested by two or more of the following conditions with no clear infectious cause:
  - T > 38°C (100.4°F) or < 36°C (96.8°F)
  - HR > 90
  - RR > 20 or pCO_2<32 mmHg
  - WBC > 12,000 or < 4,000 or 10% bands

12

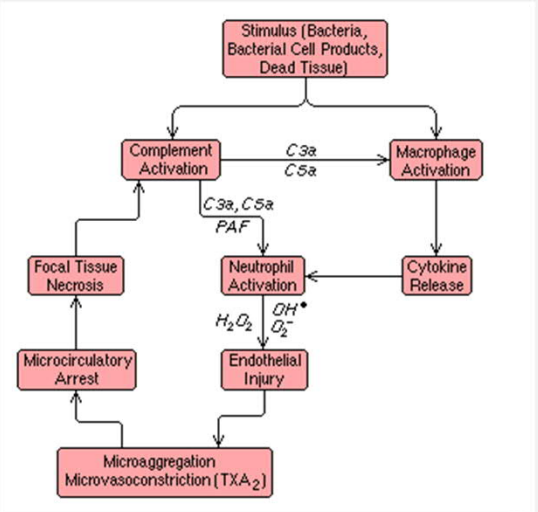
MULTIPLE ORGAN DYSFUNCTION SYNDROME (MODS)

- Presence of altered organ function in an acutely ill patient such that homeostasis cannot be maintained without intervention.

13

TISSUE LEVEL INJURY

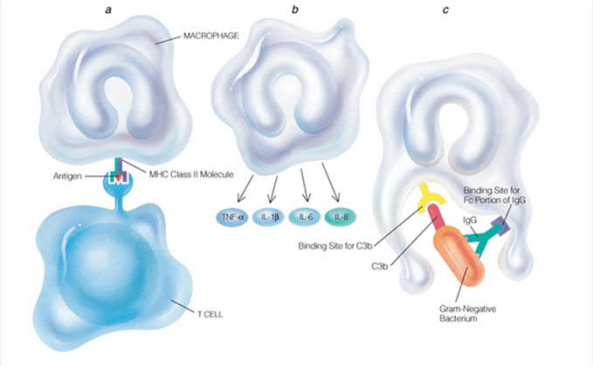
- Self energized cycle of tissue-level injury that becomes end organ injury



14

MACROPHAGES

- Antigen presentations and T cell activation
- Cytokine secretion
- Bacterial phagocytosis and killing



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CARDINAL SIGNS OF INFLAMMATION

- Redness, heat, pain, swelling, loss of function
- Fever >38°C
- Elevated WBC
- Infectious syndrome (unsatisfactory clinical course, failure to thrive)

16

SURGICAL SITE INFECTIONS

- Superficial Incisional
- Deep Incisional
- Organ/Space

17

SURGICAL SITE INFECTIONS

- Involve only the skin or SQ tissue within 30 days of the operation and at least one of the following:
  - Purulent drainage
  - Localized signs of infection requiring opening of the superficial wound
  - Positive wound culture

18

DEEP INCISIONAL SURGICAL SITE INFECTIONS

- Involve the fascia and muscle layers within 30 days of operation and at least one of the following:
  - Purulent drainage from the deep incision
  - Fever of 38°C or greater, localized pain or tenderness, or spontaneous dehiscence
  - An abscess in the deep wound

19

ORGAN/SPACE SURGICAL SITE INFECTIONS

- Involve any part of the anatomy, other than the incision, which was manipulated at time of operation and at least one the following:
  - Purulent drainage from a drain that is placed through a stab wound into the organ/space
  - Positive cultures obtained from the organ/space
  - An abscess in the organ or space

20

SURGICAL WOUND CLASSIFICATION

- Class I – Clean wounds
- Class II – Clean-contaminated wounds
- Class III – Contaminated wounds
- Class IV – Dirty wounds

21

CLASS I WOUNDS

- Wounds without infection or inflammation in which the GI, GU, or respiratory tracts have not been entered during the operation.
- Most common infecting organisms are S. aureus and S. epidermidis
- Risk of infection is 1.5%

22

CLASS II WOUNDS

- Wounds include operations in which the GI, GU, or respiratory tracts have been entered during the operation.
- Risk of infection is 15%.

23

CLASS III WOUNDS

- Wounds include those in which acute inflammation are encountered during time of operation.
- Fresh traumatic wounds
- Major break in aseptic technique
- Risk of infection is 15%

24

CLASS IV WOUNDS

- Old traumatic wounds
- Gross purulence
- Preexisting infection
- Devitalized tissue
- Risk of infection is 40%

25

RISK FACTORS FOR INFECTION

- Age extremes (< 1 yo , > 50 yo)
- Major trauma
- End organ failure
- Multiple medical problems
- Diabetes mellitus (elevated HgA1c)
- Obesity
- Malnutrition
- Presence of malignancy/chemotherapy
- Blood transfusion requirement
- History of cigarette smoking
- Altered immune response

25

GENERAL APPROACH TO DIAGNOSIS OF INFECTION

- History and Physical examination
- Obtain gram stain and cultures of wound tissue, sputum, urine, and drainage effluent.
- Consider percutaneous aspiration and microbiological examination of potentially infected fluid
- Obtain WBC and blood chemistry measurements.
- Obtain CXR; consider U/S or CT scan.

27

LABORATORY SIGNS OF OCCULT INFECTION

- Hyperglycemia and insulin resistance
- Thrombocytopenia
- Immune failure
- Renal, hepatic, or respiratory dysfunction

28

OTHER CLINICAL MANIFESTATIONS OF INFECTION

- Confusion
- Hypotension
- Ileus
- Water retention
- Gastric bleeding
- Delayed wound healing

29

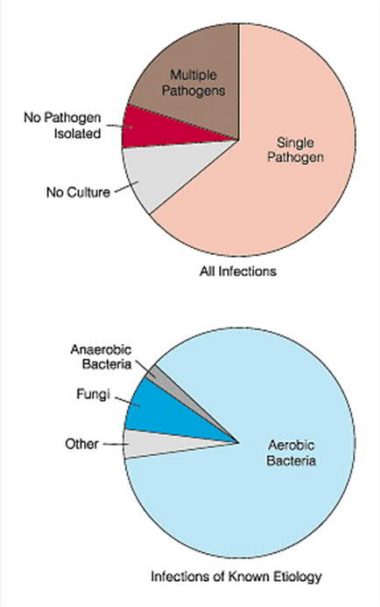
COMMON PATHOGENS

- Gram-Positive Cocci
- Enteric Gram-Negative Bacilli
- Other Gram-Negative Bacilli
- Anaerobic Bacteria

30

NOSOCOMIAL INFECTIONS

- Breakdown of the etiology of nosocomial infections



GRAM-POSITIVE COCCI

- Staphylococcus aureus (wound infections)
- S. epidermidis (catheters, shunts, prosthetics)
- Streptococcus pyogenes (Group A Beta hemolytic – post op wound infections)
- Enterococcus faecalis (peritoneal and pelvic infections)

BACILLI

GRAM-POSITIVE

- Clostridium: metronidazole
- Actinomyces: penicillin
- Nocardia: trimethoprim and sulfa

ENTERIC GRAM-NEGATIVE

- E. Coli
- Klebsiella
- Proteus
- Enterobacter
- Serratia
- Other GNB - Pseudomonas

33

ANAEROBIC BACTERIA

- Bacteroides fragilis (most commonly cultured organism in intra-abdominal infections)
- Clostridium (necrotizing soft tissue infections)

34

MOST COMMON PATHOGENS FROM SURGICAL PATIENTS

- Surgical wound infection (S. aureus, Enterococci, E. coli)
- Bacteremia (Coag – staph, S. aureus, Enterobacter sp)
- UTI (E. coli, Pseudomonas, Enterobacter)
- Respiratory Infections (Pseudomonas, S. aureus, Enterobacter sp)
- Cutaneous infections (S. aureus, Pseudomonas, Enterococci)

35

ANTIBIOTICS

- Empirical antibiotic therapy is always instituted before diagnosis is completed.
- Efficacy of empirical antibiotic therapy is assessed on the basis of the patient's clinical course.
- Empirical antibiotic therapy is modified or discontinued accordingly.

35

STAPHYLOCOCCUS AUREUS

- Causes soft tissue infection, commonly with intravascular device or prosthesis.
- Antibiotic selection:
  - cefazolin or nafcillin
  - Vancomycin if MRSA

37

STAPHYLOCOCCUS EPIDERMIDIS

- Infection of intravascular device presumed until proven otherwise
- Antibiotic selection: cefazolin or vancomycin if MRSA

38

E. COLI, KLEBSIELLA SPECIES

- Infection usually arises in peritoneal cavity, biliary tract, or urinary tract.
- Antibiotic selection: gentamicin, cefotaxime, or ceftizoxime.
- (third generation cephalosporins) but not ceftriaxone

39

ENTEROCOCCUS

- Commonly found in peritoneal and pelvic infections.
- Infection of intravascular device, heart valve, or biliary tract
- Antibiotic selection: piperacillin-tazobactam (Zosyn), or ampicillin-sulbactam (Unasyn)

40

BACTEROIDES, CLOSTRIDIA, ANAEROBIC STREPTOCOCCI

- Intra-abdominal sepsis, polymicrobial soft tissue infection, or female genital tract infection.
- Antibiotic selection: metronidazole or clindamycin.

41

PSEUDOMONAS SPECIES, SERRATIA SPECIES

- Pulmonary sepsis (usually with respiratory failure), urinary tract sepsis, or infection of intravascular device.
- Antibiotic selection: gentamicin or amikacin with additional expanded spectrum piperacillin-tazobactam (Zosyn).

42

CANDIDA SPECIES

- Primary source of infection is infected catheters or GI translocation.
- Antifungal selection: fluconazole or amphotericin B.

43

COMMON NONSURGICAL CAUSES OF PERITONITIS

- Intra-abdominal
  - GI (pancreatitis, congestive hepatomegaly)
  - GYN (PID)
  - UT (pyelonephritis, renal colic, cystitis)
  - Pneumonia, PE
  - MI, pericarditis

44

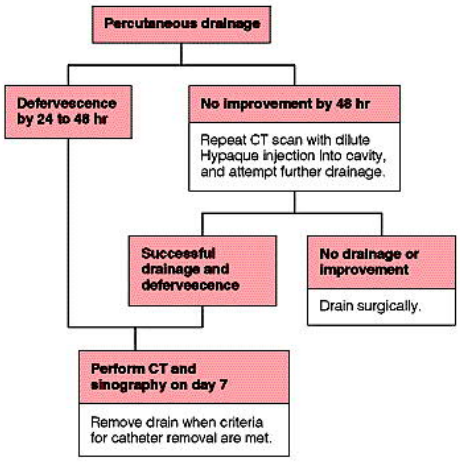
COMMON SURGICAL CAUSES OF PERITONITIS

- Perforated appendicitis
- Cholecystitis
- Postoperative peritonitis
- Perforated duodenal/gastric ulcer
- Perforated diverticulitis
- Small bowel infarction/perforation

45

INTRA-ABDOMINAL ABSCESS

Radiologic Diagnosis and Intervention



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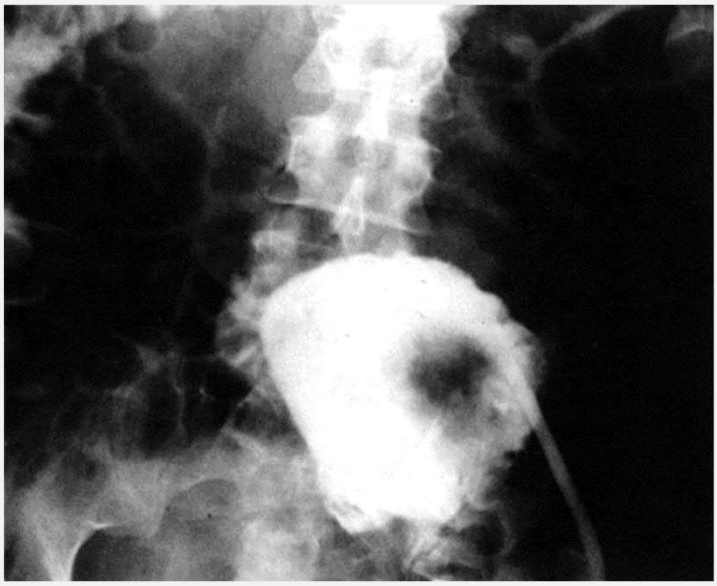

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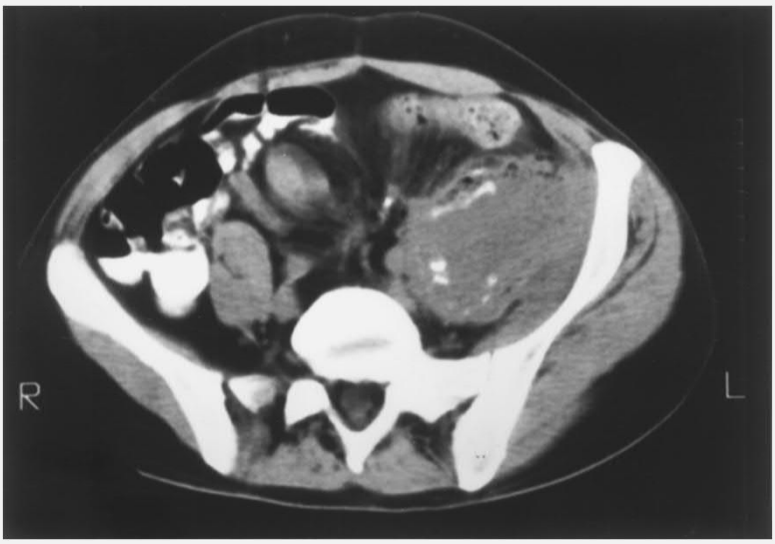

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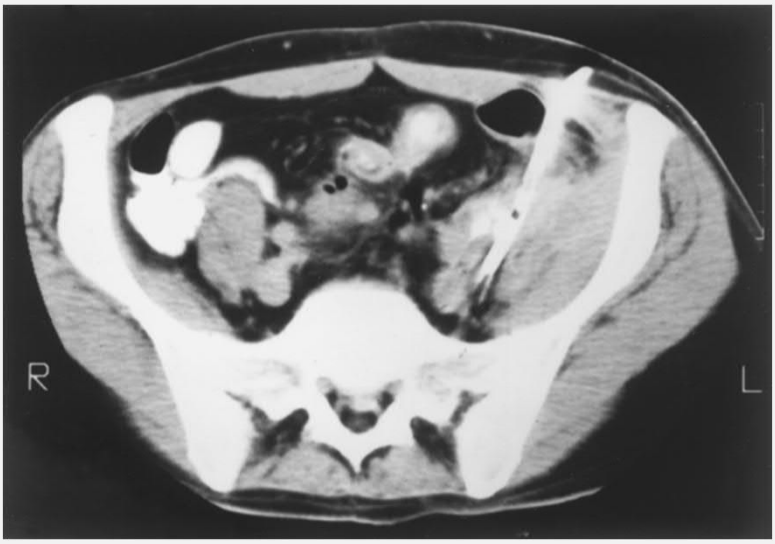

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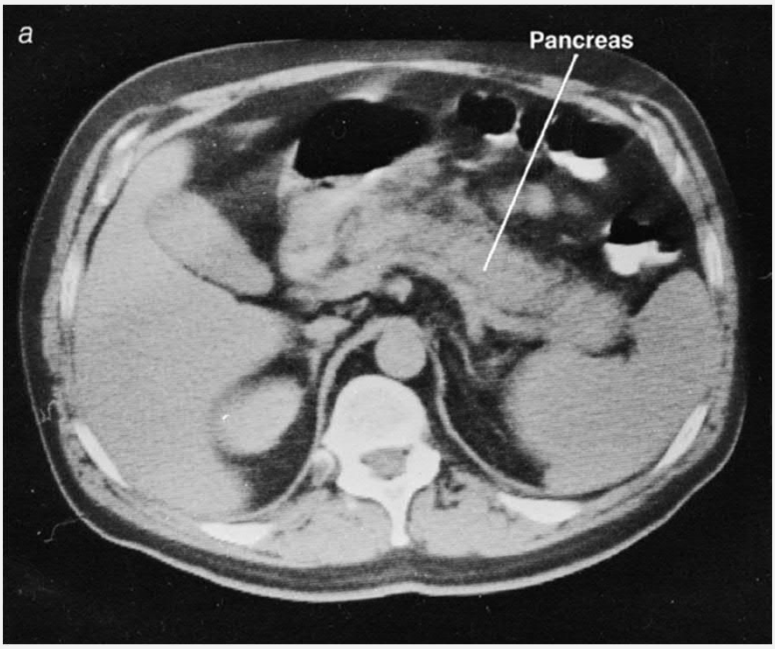

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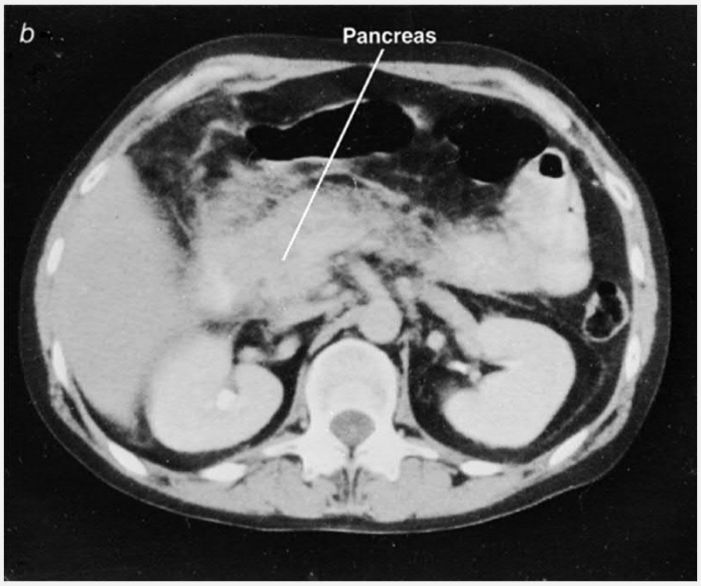

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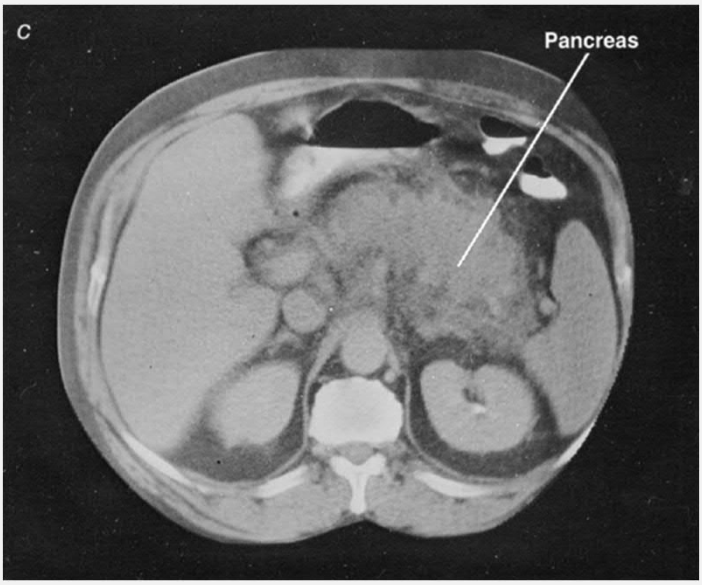

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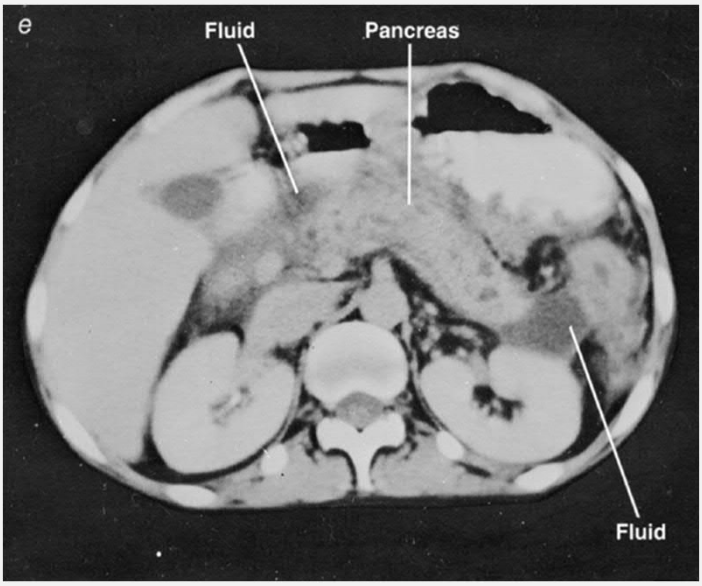

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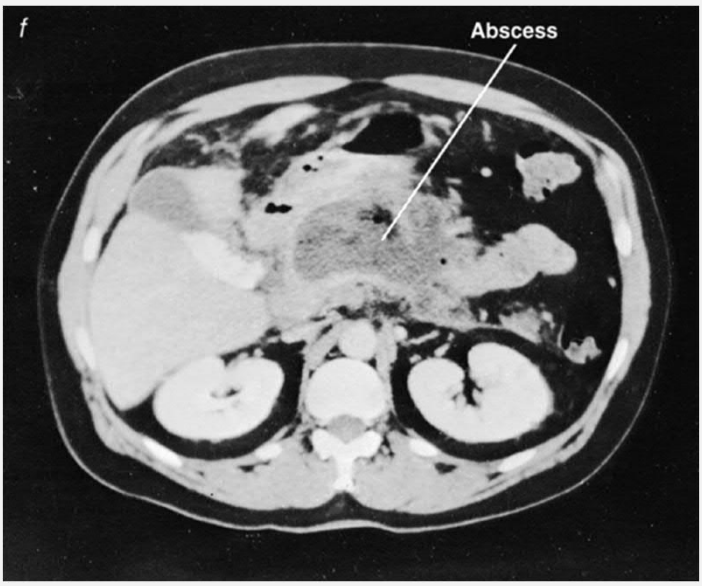

54

PERITONEAL DIALYSIS CATHETER INFECTION

- Rate: 1.4% per patient per year
- Most common organisms:
  - Coagulase negative staph aureus 60%
  - Gram negative bacteria: E. coli 20%
  - Pseudomonas (very rare)
  - Fungus
- Treatment, antibiotics via catheter and systemically.
- Cefazolin with aminoglycosides with or without vancomycin
- Suspect bowel perforation with E. coli infection
- Catheter has to be removed with fungal and most pseudomonas infections

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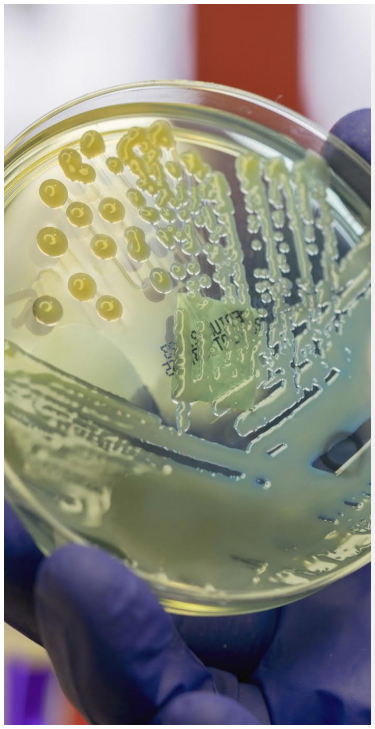


SPONTANEOUS BACTERIAL PERITONITIS

- 25% mortality rate
- Most cultures are negative
- Gram neg bacilli are the most common (E coli)
  - Followed by non-enterococcal streptococci
- Low protein content in the ascites predisposes to infection
- Treatment: first antibiotics (cefoxitin) in first 48 hours. Monitor neutrophil count in the fluid. If remains high then surgical exploration is indicated
- E coli is not indication for surgery alone

56

INFECTION IN IMMUNOCOMPROMISED PATIENTS

- Pseudomonas is the most common in bacteria
- A specific syndrome is (viridians streptococcus shock syndrome) caused by strep viridians and is seen in allogenic bone marrow transplant.
  - Skin lesions of desquamation
  - Previous use of levofloxacin predispose to the selective growth of viridians

57

CHRONIC DISSEMINATED CANDIDA INFECTION

- Hepatosplenic candidiasis
- Unexplained fever and right abdominal pain
- CT shows multiple hypodense defects in the liver

58

RESISTANCE TO VANCOMYCIN

- Enterococci (VRE) and rarely S. aureus are increasing in prevalence
- Linezolid is bacteriostatic and is approved for treatment of VRE.
  - Excellent GI absorption
  - Also effective against MRSA

59

ANTI FUNGAL

- Amphotericin
- Triazoles (Diflucan [fluconazole]) effective against all candida except glabrata and krusei
- Caspofungin is effective against all candida and against Aspergillus
- Treatment of aspergillosis: amphotericin or voriconazole

60

VENTILATOR ASSOCIATED PNEUMONIA (VAP)

- Early: first 3-4 days E. coli
- Late: pseudomonas

61

ANTIBIOTICS

62

B LACTAM ANTIBIOTICS

- Penicillins
- Cephalosporins
- Carbapenems
- Aztreonam

63

CARBAPENEMS

- Broadest antibiotics spectrum
- Beta lactam antibiotic
- Covers all gram positive, negative, anaerobes
- Except MRSA, Enterococcus and pseudomonas
- Includes:
  - Imipenem, Meropenem, Ertapenem

64

THE FOLLOWING DO NOT NEED TO BE ADJUSTED IN RENAL PATIENTS

- Cleared by liver
  - Nafcillin
  - Oxacillin
  - Ceftriaxone

65

LINEZOLID

- Only active against gram positive aerobes (not broad spectrum)
- Used against MSSA and MRSA,
- Vancomycin is first line against for MRSA, but linezolid is the alternative
- Linezolid can be given orally and does not need adjustment with hepatic or renal disease

65

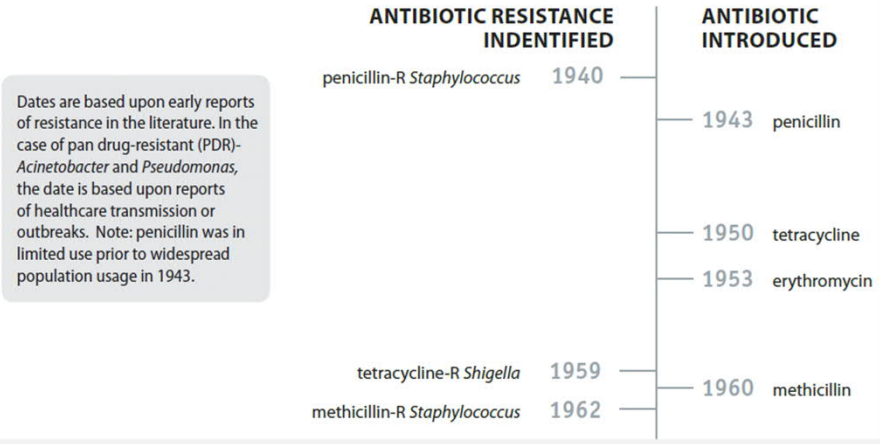
MRSA

- Community acquired or hospital acquired
- Seen mostly in hospitalized patients
- Vancomycin is first-line treatment
  - narrow spectrum antibiotic against gram positive bacteria only
  - may be combined with rifampicin or aminoglycoside if not effective alone
- Linezolid is an alternative treatment

67

Developing Resistance

Timeline of Key Antibiotic Resistance Events



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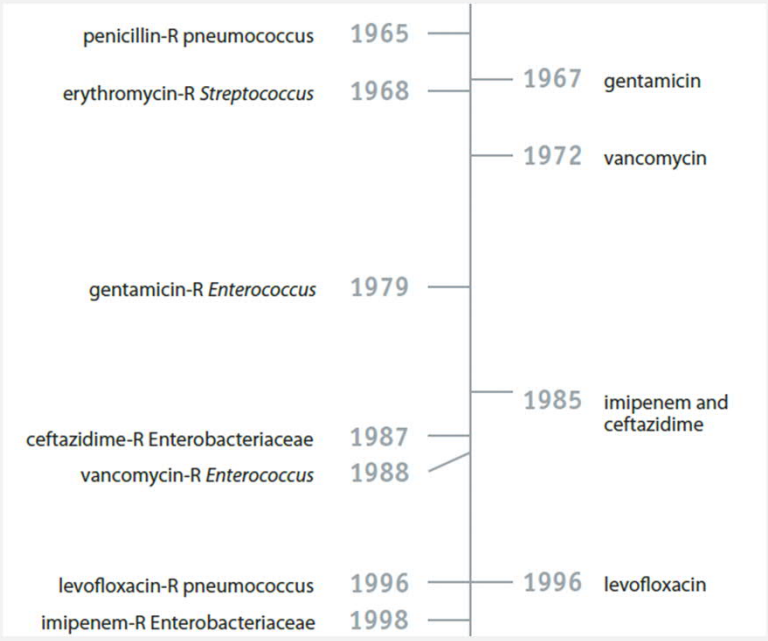

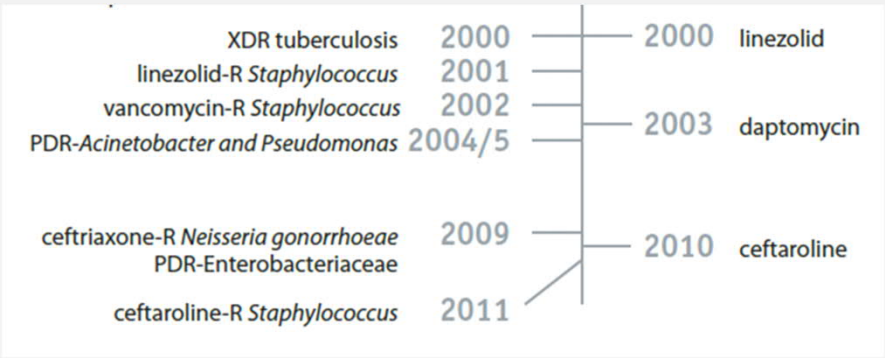

EARLY ONSET VAP

Second or third generation cephalosporin: e. g., ceftriaxone: 2 g daily;

cefuroxime: 1.5 g every 8 hours;

cefotaxime: 2 g every 8 hours

OR

Fluoroquinolones

e. g., levofloxacin: 750 mg daily;

moxifloxacin: 400 mg daily

OR

Aminopenicillin + beta-lactamase inhibitor e. g., ampicillin + sulbactam: 3 g every 8 hours

OR

Ertapenem

1 g daily

71

LATE ONSET VAP

Cephalosporin

e. g., cefepime: 1–2 g every 8 hours;

ceftazidime 2 g every 8 hours

OR

Carbepenem

e. g., imipenem + cilastin: 500 mg every 6 hours or 1 g every 8 hours;

meropenem: 1 g every 8 hours

OR

Beta-lactam/beta-lactamase inhibitor

e. g., piperacillin + tazobactam: 4.5 g every 6 hours

PLUS

Aminoglycoside

e. g., amikacin: 20 mg/kg/day;

gentamicin: 7 mg/kg/day;

tobramycin: 7 mg/kg/day

OR

Antipseudomonal fluoroquinolone

e. g., ciprofloxacin 400 mg every 8 hours;

levofloxacin 750 mg daily

PLUS

Coverage for MRSA

e. g., vancomycin: 15 mg/kg every 12 hours

OR

linezolid: 600 mg every 12 hours

72

Table 3 Recommended therapy for suspected or confirmed multidrug resistant organisms and fungal VAP [1], [34], [35], [41]

Pathogen	Treatment
Methicillin-resistant *Staphylococcus aureus* (MRSA)	See Table 2
Pseudomonas aeruginosa	Double coverage recommended. See Table 2
Acinetobacter species	Carbapenem e. g., imipenem + cilastin; 1 g every 8 hours; meropenem 1 g every 8 hours OR Beta-Lactam/beta-lactamase inhibitor e. g., ampicillin + sulbactam: 3 g every 8 hours OR Tigecycline: 100 mg loading dose, then 50 mg every 12 hours
Extended-spectrum beta-lactamase (ESBL) positive enterobacteriaceae	Carbepenem e. g., imipenem + cilastin: 1 g every 8 hours; meropenem: 1 g every 8 hours
Fungi	Fluconazole: 800 mg every 12 hours; caspofungin: 70 mg loading dose, then 50 mg daily; voriconazole (for aspergillus species): 4 mg/kg every 12 hours
Legionella	Macrolides (e. g., azithromycin) OR Fluoroquinolones (e. g., levofloxacin)

3

QUESTION 1

- All of the following antibiotics are β lactams EXCEPT:
  - A. CEPHALOSPORINS
  - B. MONBACTAM (Aztreonam)
  - C. CARBAPENAM
  - D. POLYMXIN

74

ANSWER 1

- D: Polymyxin
  - Polymyxin (colistin) is the only antibiotic listed that does not have β lactams
  - Acts only against gram negatives

75

QUESTION 2

A CLABSI (central line associated blood stream infection) is treated with antibiotics. Which infection requires the longest duration of treatment:

A. staph aureus
B. staph epidermis
C. candida
D. Gram negative

76

ANSWER 2

- C: candida
  - Candida needs antibiotics for 14 days after last positive blood cultures
  - All others range from 7 to 10 days

77

QUESTIONS?

78

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Surgical Critical Care Syllabus 2

Cardiac Physiology & Resuscitation

Surgical Critical Care

David Hampton MD, MEng, FACS
Original notes prepared by Ahmed Mahmoud, MD,
And Laura Crankshaw, MD

Adult Cardiac

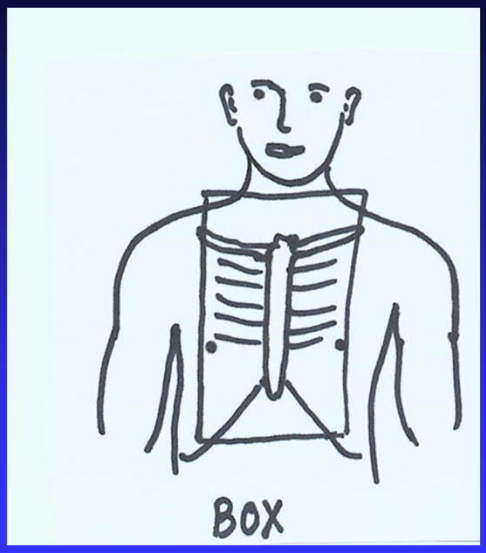
- **Tumors**
- **Metastatic** malignant tumors 20 times more common than primary
- **Primary tumors** → 75% benign, 25% malig
- **Myxoma** is the most common (40%)
- 75% in left atrium near fossa ovalis
- familial in young females, sporadic in old

Myxoma

- Mitral valve obstruction
- Embolization in 50% with acute ischemia in peripheral circulation
- Histology of the surgically removed embolus is diagnostic
- Echo is test of choice
- Treatment is excision

Cardiac Trauma

- 10% of penetrating chest trauma involves the heart
- Right ventricle is the most commonly injured in stab wounds
- **Diagnosis:** Suspect in any injury near the heart (the BOX) **(precordium-epigastrium-superior mediastinum)**



Cardiac Trauma

- Patient may present with hypotension, and shock, hemothorax or in tamponade)
- **Beck triad (Tamponade)**: distended neck veins- muffled heart sounds – hypotension
- **Shock with elevated CVP:**
  - Tamponade # tension PNX
  - Difference > Air way pressure

Cardiac Trauma (Diagnosis)

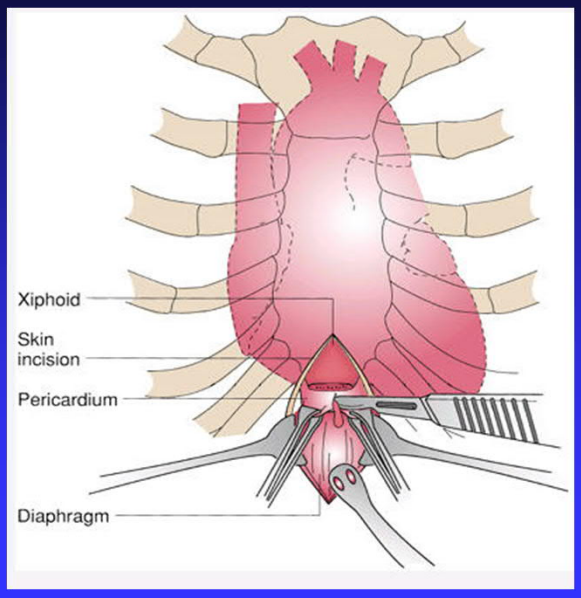
- **FAST**
  - Looks promising. Should be available in the ER > 90 % sensitivity and specificity
- **Pericardiocentesis**
  - Not a diagnostic test, only done for temporary relief of blood on the way to the OR. Blood is not clotted

Cardiac Trauma (Diagnosis)

Subxiphoidal exploration

- Definitive diagnostic test in stable patient
- Good test to rule out tamponade in patients who do not have tamponade
- Positive pericardiocentesis or window
  - Median sternotomy
- Most 95% cardiac injuries are repaired without Bypass

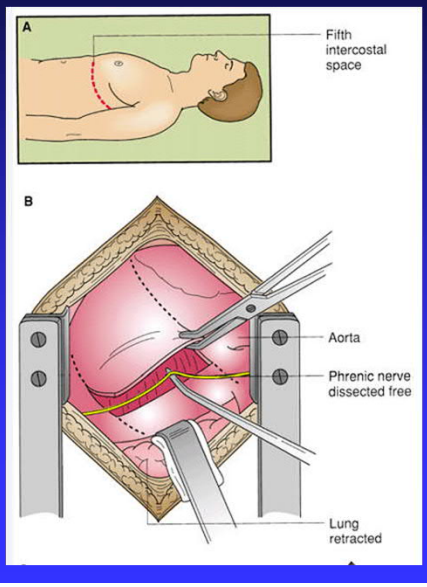
Subxiphoidal Pericardial Window



ER (Resuscitative Thoracotomy)

- **Indications**
  1) penetrating chest injuries with failure of aggressive resusc to raise BP >80mmhg (12% survival)
  2) penetrating chest or abd injuries who lost vitals during transfer
  3) blunt injuries who had vitals before arrival (3% survival)

ER Thoracotomy, Open Pericardium



ER THORACOTOMY (Steps)

- Left anterolateral thoracotomy down to the table
- Control obvious source of bleeding e.g. left hilum
- Open pericardium, watch phrenic nerve
- If no injury is seen, extend to the right chest
- Open cardiac massage. Clamp the Aorta

Blunt Cardiac Injury (Myocardial Contusion)

- Incidence: unknown. Most are asymptomatic
- May range from benign arrhythmia to fatal ventricular fibrillation. Myocardial rupture and death
- ST, T changes and sinus tachycardia are the most common EKG changes
- Tests of DX: Stable → EKG monitor for 12-24 h

- Unstable: + Echo → to look for pericardial fluid or other injuries e.g. valve rupture
- Treatment is supportive

Traumatic Rupture of Aorta

- Rapid deceleration injury with shearing force
- Most common site is just distal to the left subclavian 60%.
  - Followed by distal thoracic aorta 20%
- 60% die at the scene: 40 % make it to the hospital, third of which die during initial resuscitation

Traumatic Rupture Aorta

Clinical presentation

- Free rupture, death shortly after injury (60%)
- Contained rupture: detected on CXR (40%)
- Chronic rupture: (rare) chest pain, back pain, hoarseness of voice years later
- Examination shock: Look for associated injuries

Question

- 32 y o was brought to the ER after MVA. Evaluation: SBP 90, HR 130. CXR wide mediastinum. Abdomen tender. Bilateral pelvic fracture. Most appropriate action?
  - A. CT Abdomen pelvis
  - B. Aortography for wide mediastinum
  - C. Pelvic binder
  - D. DPL - FAST

Answer

- **D: FAST or DPL** would be the immediate safe step to take to determine cause of patient's haemodynamic instability. Is it from pelvis bleeding or bleeding related to spleen and other organs

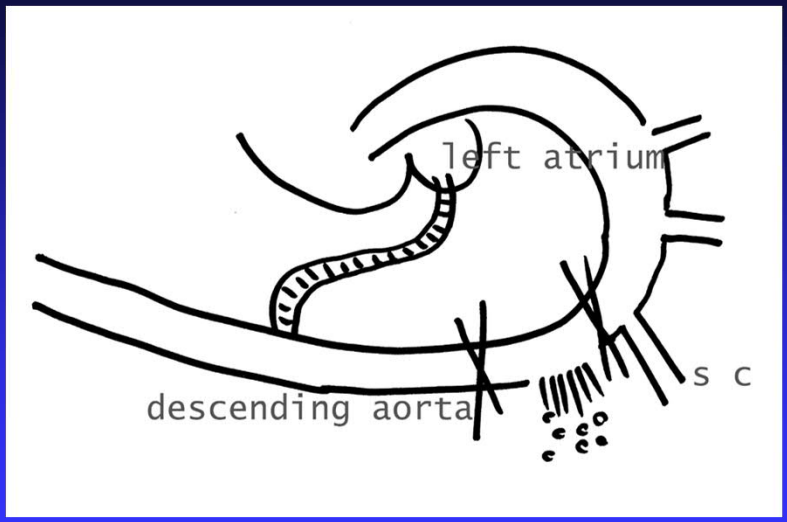
Diagnosis of Ruptured Aorta

- **CXR:** wide mediastinum > 8cm transverse
- Normal CXR does not r/o rupture
- Aortography: used to be the gold standard. Was always needed before surgery
- Helical CT / reconstruction views are now accepted (virtual angiogram)

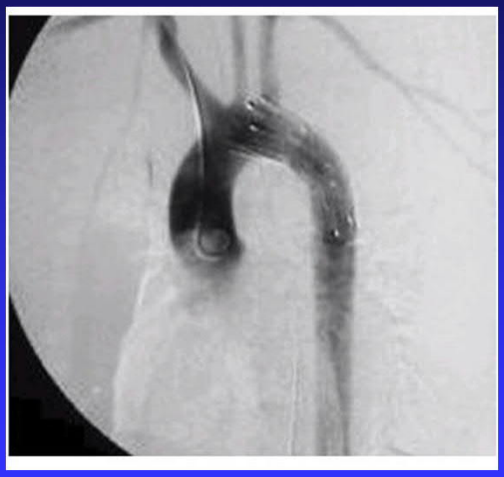
- **Transesophageal Echo (TEE):** Can be done in OR / ICU… only good for typical location. PNX may limit its value

Treatment of Aortic Rupture

- **Preoperative preparation**: A line, control the shearing force (BP /HR): Nipride.
  - If tachy --Esmolol
- ? Simple clamp and repair
- Partial By pass from left atrium to descending aorta or to Femoral artery → no need for oxygenator. No full heparin
- Paraplegia 10% regardless. Ant spinal art
- Clamp time >30 min



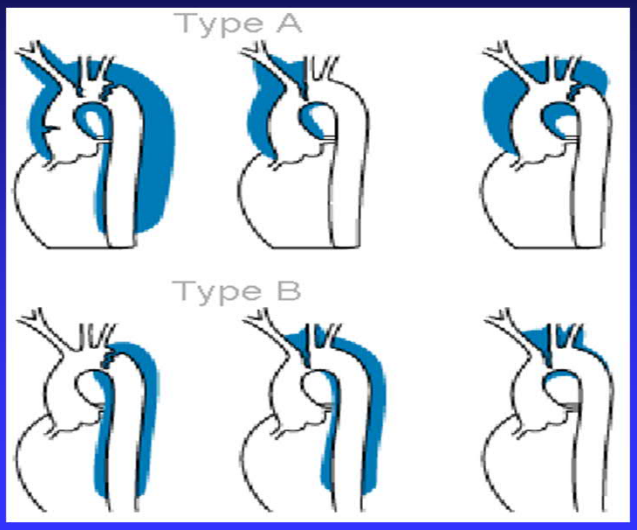
Stents in Thoracic Aorta



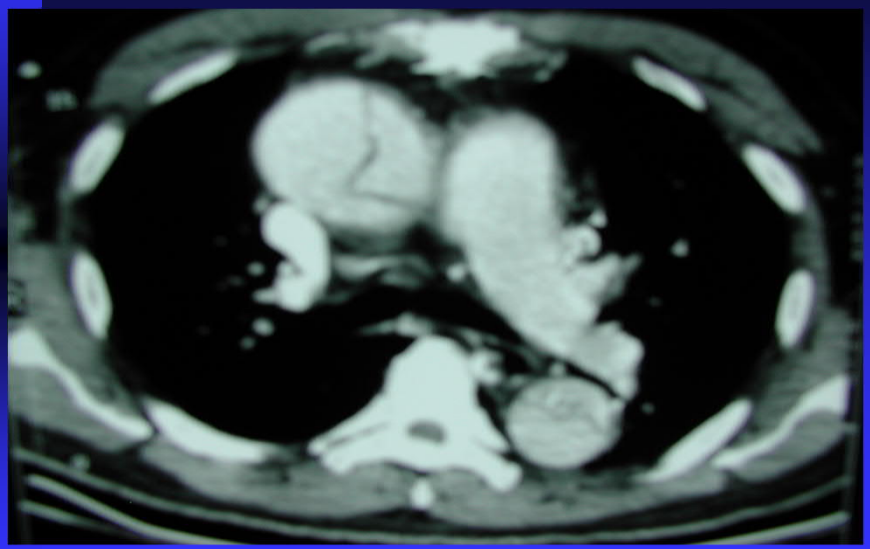
Acquired Cardiac Disease

- **Aortic Dissection**
- **Type A**: involves ascending aorta, usually has AI, **emergency** surgical treatment. If rupture → fatal pericardial tamponade
- **Type B**: Descending. Treatment is medical by aggressive control of hypertension

Classification - *Stanford*



Aortic dissection (false & true) lumen



- **Coronary artery disease**
- Myocardial revascul could be done by surgery (**CABG**) or by Angioplasty(**stent)**
- CABG is the correct option in the following
  1) **left main disease
  2) 3 vessel disease
  3) 2 vessel disease (one must be LAD)
  4) low EF +/- diabetes

Mechanical Complications of MI

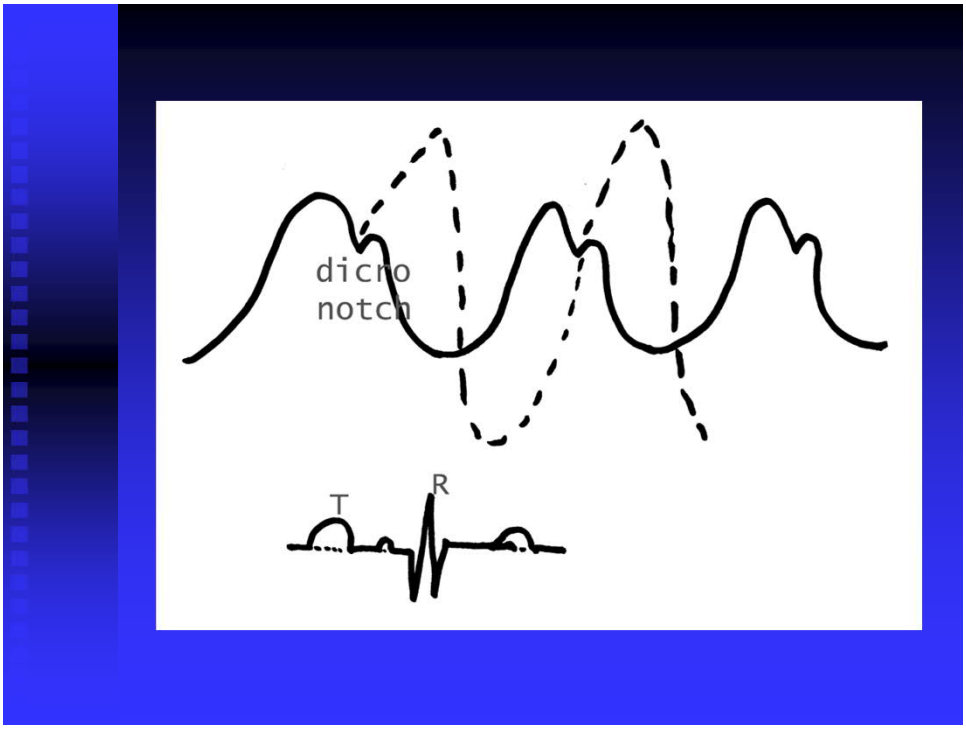
1) **Ventricular septic defect**: pansystolic murmur. 1 % of MI.
   - Differentiate from mitral regurgitation: by measuring the O2 sat step up at the right ventricle: if more than 10 → VSD
   - Treatment is initial medical support (if possible) until scar matures then surgical closure. Sometimes emergency closure

2. **Mitral regurgitation**
   - Worse prognosis than other causes of Mitral regurg- try medical support before surgery

2. **Ventricular Aneurysm**
   - If small (2-3) cm and asymptomatic=no ttt
   - Large/symptomatic or false needs surgery

Intra Aortic Balloon Pump

- Significant improvement in cardiac patients
- Balloon is placed at descending aorta, just distal to the left subclavian
- **Inflate at diastole** = peak of T wave = dicrotic notch = aortic valve closure
- **Deflate at systole** = before R wave



- Increase in coronary inflow by increase in diastolic blood flow
- Decrease in afterload
- Decrease in myocardial O 2 consumption
- Absolute contraindications for IABP is:
  - Aortic regurgitation

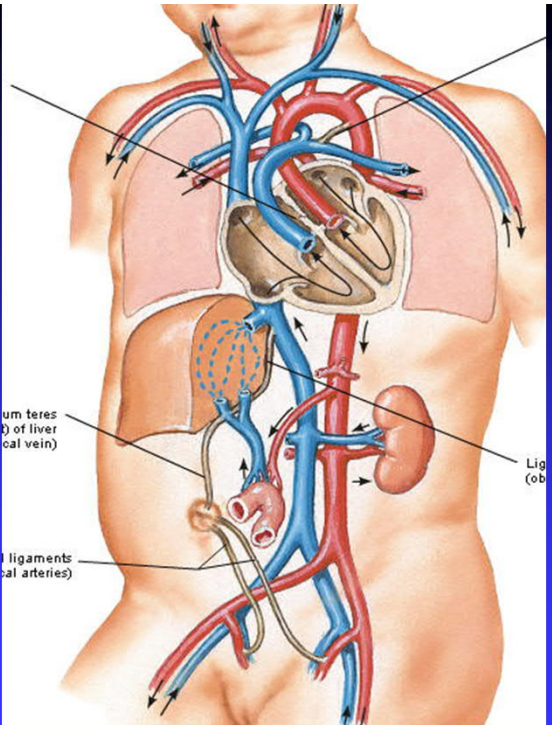
Valve Diseases

- Determination of valve stenosis is based on measurement of pressure and flow across the valve
- **Mitral stenosis is significant and requires surgery if:** the valve area is < 1.5 cm2 (normal is 5-7) and the pressure gradient (LAP-LVED) is 15mmhg (normally: none)

- **Aortic valve stenosis is significant and requires surgery if:**
  - The valve area is less than 1 cm (nl. 2-3)
  - The pressure gradient is > 50mm Hg (nl. none)
  - Aortic stenosis is the most serious of all and should be always corrected before any surgery

Congenital Cardiac

- The **fetal pulmonary vascular** resistance is high in utero, causing blood to shunt from right to left through the patent foramen ovale and also from the pulmonary artery into the descending aorta by the Patent ductus arteriosus
- After birth, the **PVR starts to fall** with the beginning of ventilation



- The infant **PVR becomes equal to the adult level by 4 weeks**
- **Persistent fetal circulation** (persistent pulmonary hypertension) may be caused by: hypoxia, acidosis or pulmonary hypoplasia

- **ACYANOTIC**
  - With increase in pulmonary flow: VSD-PDA-ASD
  - With normal pulm flow: PS-MS-AS-Coarctation
- **CYANOTIC *(all T)***
  - With increase in pulm flow TGA-TAPVR
  - With normal pulm flow: TOF- PS-ASD-Eisenmenger syndrome

Ventricular Septal Defect

- **Ventricular septic defect**
- **Most common anomaly**, usually at membranous septum, many will close spontaneously by age of 4, pansystolic murmur
- **Indications for surgery**: infants with severe CHF or respiratory infections in first 3 months – Shunt ratio > 2:1(pulmon to syst) – pulmonary vascular res up to 8 (> 10 is inoperable) – Aortic regurgitation

- Patent ductus arteriosus
  - Associated anomalies in 20%
  - Try antiprostaglandins first

- **Atrial septal defect (secundum defect)**
  - Located near the fossa ovalis
  - Fixed wide splitting of second heart sound
  - May be associated with anomalies in the pulmonary venous return
  - Closed by interventional cardiology

- **ASD (PRIMUM) = A V CANAL DEFECT**
  - Earlier presentation than secundum
  - Part of AV canal defects with valve lesions
  - CHF is more common

- **TGA**
  - Most common cause of cyanosis at birth
  - Patent VSD, ASD or PDA are essential for survival before surgery (switch)

- **Tetralogy of Fallot**
  - Most common cyanotic congenital heart disease (cyanosis usually occurs not at birth but shortly after)
  - VSD + PS + OVERRIDDING OF AORTA = RIGHT VENTRICLE HYPETROPHY
  - Cyanotic spells, relieved by squatting, no emergency surgery

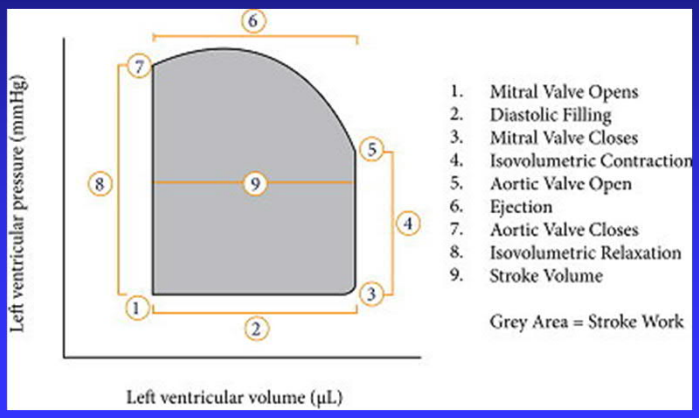
Coarctation

- Obstruction at the aorta 2 cm distal to left subclavian
- 2 types: infantile (usually severe CHF) and childhood
- Repair by a subclavian artery flap or extended resection and anastomosis
- Recurrence is repaired by Angioplasty

CARDIAC OUTPUT

- Cardiac Output = Heart Rate x Stroke Volume
- Stroke Volume
  - Preload
  - Myocardial Contractility
  - Afterload

CARDIAC CYCLE (P-V LOOP)



PRELOAD

- Etiology of Deficit
  - Hemorrhage: Therapy is blood products
  - Other Body Fluids – Therapy is:
    - Balanced Electrolyte Solutions
    - Colloid Solutions

MYOCARDIAL CONTRACTILITY

- Most common etiology is myocardial ischemia
- Acute Coronary Syndrome
  - Morphine, Oxygen, Nitroglycerin, Aspirin
  - Adjuncts: β-Blockers, Heparin, Antiplatelet agents
  - Within 24 hrs: ACE Inhibitors, Statin therapy
- Myocardial Ischemia (STEMI < 12 hrs)
  - Percutaneous Coronary Intervention: 90 minutes
  - Fibrinolysis: 30 minutes

LEFT VENTRICULAR FAILURE

- Goals
  - Preload Reduction
  - Inotropic Support
  - Afterload Reduction
- Preload Reduction
  - Diuretics: Furosemide, Thiazides, K-Sparing
  - Venodilators: Nitroglycerin
- Inotropic Support: Dobutamine, Milrinone, Isoproterenol, Epinephrine
- Afterload Reduction
  - Arterial Vasodilators
  - Nitroprusside, Nicardipine

RIGHT VENTRICULAR FAILURE

- Goals – In relation to RV
  - Preload Reduction
  - Inotropic Support
  - Afterload Reduction
- Preload Reduction: Diuretics
- Inotropic Support: Dobutamine, Epinephrine, Dopamine
- Afterload Reduction
  - Pulmonary Vasodilators
  - Milrinone, Inhaled NO

AFTERLOAD

- Diuretics
- ACE Inhibitors
- Nitrates
- Calcium Channel blockers
- Phosphodiesterase Inhibitors
- Intra-aortic balloon pump

OXYGEN DELIVERY

- DO_2 - Rate at which oxygen reaches the systemic capillaries.
- $DO_2 = CO \times CaO_2 \times 10$
- $CaO_2 = (1.34 \times Hb \times SaO_2) + .0031 \times PaO_2$
- Normal DO_2 = 900 – 1,100 mL/min

OXYGEN CONSUMPTION

- VO_2 – Rate at which oxygen leaves capillary blood and moves into tissues.
- Approximates metabolic rate
- Cannot clinically measure
- Different tissues = Different VO_2
- $VO_2 = CO \times (CaO_2 - CvO_2) \times 10$
- Normal = 200 – 270 mL/min
- Highest VO_2 Organ = Heart

OXYGEN – EXTRACTION RATIO

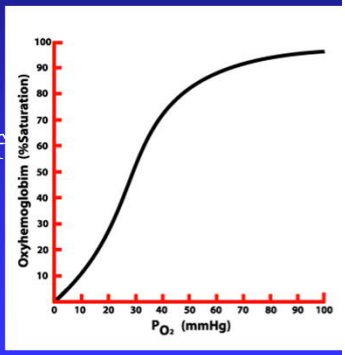
- O_2ER – Fraction of oxygen delivered to the capillaries that is taken up into the tissues.
- $O_2ER = VO_2 / DO_2$
- Normal O_2ER: 0.2 – 0.3
- High Extraction
  - Excessive consumption
  - Poor delivery
- Low Extraction
  - Lack of tissue consumption
  - Diffusion abnormalities

OXYGEN TRANSPORT

- Series of steps from lungs to cells
- There is NO storage of oxygen
- Oxygen Delivery must be continuous
- Process Involves
  - Pulmonary System
  - Circulatory System

OXYGEN – HOW MUCH?

- Decreased O_2 Affinity (Right shift)
  - Increased Temperature
  - Increased 2,3-DPG
  - Increased pCO_2
  - Decreased pH (acidosis)
- Increased O_2 Affinity (Left shif
  - Decreased Temperature
  - Decreased 2,3-DPG
  - Decreased pCO_2
  - Increased pH (Alkalosis)



OXYGEN – OFFLOADING

- Requires Hypoxemia
- Generates capillary pO_2 which becomes the O_2 diffusion driving force
- Increased only by:
  - Further decrease in tissue pO_2
  - Right shift in Oxyhemoglobin dissociation curve
- Due to Oxygen gradient between capillaries and cells

REVIEW

With all other factors remaining constant, a decreased oxygen extraction ratio in the tissues may be seen as:

a. Increased oxygen consumption.
b. Decreased oxygen delivery.
c. Increased mixed venous saturation.
d. Decreased cardiac output.

REVIEW

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OXYGEN DEBT

- When aerobic metabolism is insufficient
- Anerobic metabolism increases
  - Lactate production rises
  - H^+ production rises
- Oxygen debt = oxygen required to reverse the processes

OXYGEN DEBT

- Effects
  - Metabolism changes
  - Deterioration in cell structure/function
  - Pro-inflammatory mediator release – SIRS
  - Endothelial Cell
    - Vascular reactivity – maldistribution of capillary flow
    - Cell aggregation – Neutrophils/platelets/mediator release
    - Cell junction separation – Capillary leak
  - Compensatory responses occur

OXYGEN DELIVERY – DETERMINANTS

- Hemoglobin/Oxygen Binding
  - Anemia/decreased hemoglobin
  - Hemoglobin dysfunction
  - Atmospheric hypoxia
- Pulmonary
  - Primary/secondary lung disease
  - Hypoventilation
  - V/Q abnormality

OXYGEN DELIVERY – DETERMINANTS

- Cardiovascular
  - Decreased CO states
  - Peripheral vascular dysfunction
  - Some areas may be OK: some may be critical
- Oxygen Offloading – Rarely causes significant issues

OXYGEN DELIVERY – COMPENSATION

- Mechanisms
  - Usually work well in mild-moderate states
  - Acute vs Chronic Compensation
- **Acute Mechanisms**
  - CO Increase
  - Arterial flow redistribution
  - Local microcirculatory response
    - Result is to increase flow
    - Autonomics
    - Endothelial cell mediator release (e.g. vasodilators)
  - Increased offloading of oxygen from hemoglobin
  - Intracellular biochemical protective responses
    - Heat shock proteins
    - Glucose regulating proteins
- **Chronic Mechanisms**
  - Generally organ function changes
  - Primarily cardiovascular or pulmonary
  - Hemoglobin increase

REVIEW

Decreased oxygen affinity seen in the hemoglobin molecule may be the result of:

a. A decrease in temperature.
b. A decrease in pH.
c. The presence of fetal hemoglobin.
d. A decrease in pCO_2.

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SEPTIC SHOCK & THE HEART

- Mainstays of Therapy
  - Hypovolemia: Adequate volume resuscitation
  - Systemic vasodilation: Potent vasoconstrictors
  - Myocardial Depression: Inotropes
- Hypovolemia
  - Colloid solutions
  - Blood products

SEPTIC SHOCK & THE HEART

- Systemic Vasodilation
  - Norepinephrine
    - First line agent
    - Potent α-constrictor
  - Dopamine
    - Alternative first-line agent
    - More tachycardia than norepinephrine

SEPTIC SHOCK & THE HEART

- Myocardial Depression
  - Dobutamine
  - Milrinone
  - Epinephrine
    - Increased lactate
    - Possible myocardial ischemia and arrhythmias
    - Reduced splanchnic blood flow

SEPTIC SHOCK & THE HEART

- Vasopressin
  - Adequate fluid-resuscitated septic shock
  - Refractory to high doses of catecholamines
  - Rationale: Septic shock is relative vasopressin deficient state
  - High Doses: Coronary, splanchnic, and digital ischemia

VALVULAR HEART DISEASE

- Aortic Regurgitation
  - Etiology: Rheumatic fever, Marfan syndrome, aortic dissection
  - Up to 60% to 70% of SV may regurgitate
  - Prolonged course due to LV adaptation(dilation)
  - Dx: Widened pulse pressure, diastolic murmur
  - Therapy: Valve replacement for symptomatic patients

VALVULAR HEART DISEASE

- Mitral Stenosis
  - Etiology: Chronic rheumatic disease
  - Causes chronic pulmonary venous obstruction
  - Dx: Dyspnea, opening snap, diastolic rumble
  - Therapy: Valve repair/replacement for severe obstruction, AF onset, pulmonary hypertension, infective endocarditis

VALVULAR HEART DISEASE

- Aortic Stenosis
  - Etiology: Congenital or degenerative
  - Left ventricle hypertrophies
  - Most frequently fatal valvular lesion
  - Dx: Loud, harsh systolic murmur
  - Therapy: Vale replacement with angina, CHF, syncope, critical stenosis (< 0.7 cm^2)

VALVULAR HEART DISEASE

- Mitral Regurgitation
  - Etiology: Rheumatic heart disease, ischemia, infective endocarditis
  - Acute MR leads to rapid deterioration
  - Dx: Dyspnea, apical pansystolic murmur
  - Therapy: Valve replacement for symptomatic patients, pulmonary hypertension, new Afib.

REVIEW

Which of the following would produce a greater increase in oxygen delivery?

a. Saturation increase from 50% to 100%.
b. Hemoglobin increase from 8 to 12.
c. Cardiac output increase from 1.5 to 2.5.
d. PaO_2 increase from 100 to 200.

REVIEW

Which of the following would produce a greater increase in oxygen delivery?

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d. PaO_2 increase from 100 to 200.

THE UNRESPONSIVE PATIENT

- ADULT – No breathing/inadequate breathing
  - Activate EMS
  - Get AED
- Check Pulse
  - Time limit: 10 seconds
  - Yes: Maintain airway (1 breath/5-6 secs)
  - No: Begin CPR

THE UNRESPONSIVE PATIENT

- Cardiopulmonary Resuscitation (CPR)
  - 30 compressions
  - 2 breaths
- Assess Cardiac Rhythm – Shockable?
  - Yes: Shock, then CPR for 2 minutes
  - No: Continue CPR, reassess every 2 minutes

HIGH QUALITY CPR – ADULT

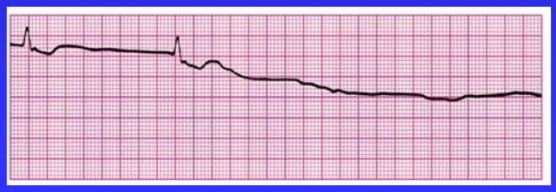
- Compression Rate: 100-120 per minute
- Compression Depth: At least 2 inches
- Allow for complete chest recoil
- Minimize interruptions
- Avoid hyperventilation

CARDIAC ARREST

- Activate EMS/Begin CPR/Analyze Rhythm
- Shockable – Yes
  - Defibrillate/Biphasic/360J
  - CPR (2 minutes)
  - Epinephrine (1 mg every 3-5 mins)
  - Re-analyze Rhythm – Shockable – Yes
  - Defibrillate/Biphasic/360J
  - CPR (2 minutes)
  - Amiodarone (1^{st}: 300 mg: 2^{nd}: 150 mg)
  - Re-analyze Rhythm – Shockable – Yes
  - REPEAT

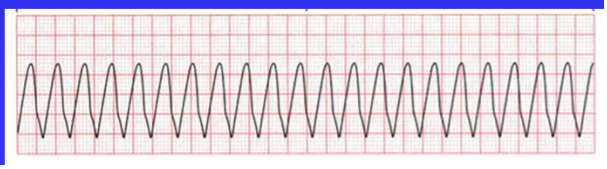
CARDIAC ARREST

- Shockable – No
  - PEA/Asystole
    - CPR (2 minutes)
    - Epinephrine (1 mg every 3-5 mins)
    - Review H's & T's



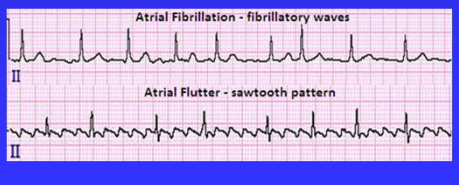
TACHYARRHYTHMIAS

- Ventricular Tachyarrhythmias
  - Ventricular Tachycardia/Ventricular Fibrillation
    - Increased mortality
    - Unstable: Unsynchronized Cardioversion
    - Stable Ventricular Tachycardia
      - Amiodarone
      - Procainamide
      - Sotalol



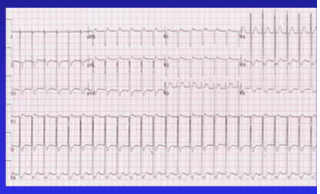
TACHYARRHYTHMIAS

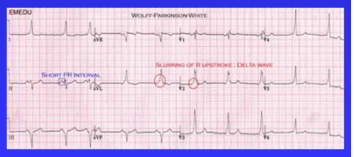
- Supraventricular Tachyarrhythmias
  - Atrial Fibrillation/Atrial Flutter
    - MOST significant SVTs in early postoperative period
    - Unstable: Immediate synchronized cardioversion
    - Stable: Amiodarone = SINGLE most effective agent: β-Blockers
    - Cardioselective β-Blockers: Useful in setting of reactive airway disease (esmolol, metoprolol, atenolol).



TACHYARRHYTHMIAS

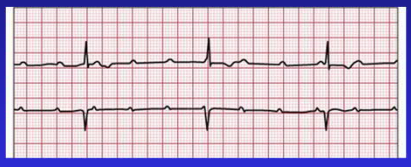
- Supraventricular Tachyarrhythmias
  - Paroxysmal SVT
    - Unstable: Synchronized Cardioversion
    - Stable
      - Wide: Adenosine, Amiodarone
      - Narrow: Vagal maneuvers, Adenosine, β-Blockers, Calcium Channel Blockers
    - Wolfe-Parkinson White (WPW)
      - Blocking AV node worsens
      - Delta Wave: Short PR
      - Amiodarone is preferred





BRADYARRHYTHMIAS

- Symptomatic Bradycardia
  - Atropine, then Transcutaneous Pacing
  - Dopamine or Epinephrine infusion
- High-Degree AV Nodal Block
  - Types:
    - Type II Second Degree
    - Third Degree
  - Therapy
    - Stable: As symptomatic bradycardia
    - Unstable: Pacemaker (External or Transvenous)
- Consider H's & T's



H's & T's

- Hypoxia
- Hypovolemia
- Hydrogen ion (Acidosis)
- Hypo/Hyperkalemia
- Hypothermia
- Toxins
- Tamponade (Cardiac)
- Tension Pneumothorax
- Thrombosis (coronary and pulmonary)
- Trauma

ACUTE CORONARY SYNDROME

- Oxygen
- Without contraindications: Aspirin, Nitroglycerin, Morphine
- Obtain 12-lead ECG
- STEMI vs Non-Stemi vs Non-cardiac

ACS – NSTEMI

- Unstable Angina/Non-ST Elevation
- Nitroglycerin
- Heparin
- β-Blocker
- Clopidogrel
- Glycoprotein IIb/IIIa inhibitor
- Consider Invasive therapies

ACS – STEMI

- ECG: STEMI or New LBBB
- Adjunctive Therapies: Nitroglycerin, Heparin, β-Blocker, etc.
- Symptom Onset ≤ 12 hours
  - No: Medical Management
  - Yes
    - Balloon PCI within 90 minutes (of arrival)
    - Fibrinolytic therapy
      - Within 30 minutes (of arrival)
      - No contraindications

VASOACTIVE AGENTS

Medication	Mechanism	Indications
Norepinephrine	α1, α2	Vasoconstrictor
Epinephrine	α1, α2, β1, β2	Inotrope, Vasoconstrictor
Dopamine	α1, α2, β1, β2, Dopamine	Inotrope, Vasoconstrictor
Dobutamine	β1, β2	Inotrope
Phenylephrine	α1	Vasoconstrictor
Isoproterenol	β1, β2	Inotrope
Milrinone	Phosphodiesterase inhibitor	Inotrope, Vasodilator
Vasopressin	V1, V2 receptor	Vasoconstrictor

REVIEW

A 72 y/o female is immediately post operative from a sigmoid colectomy for diverticular disease. She complains that her heart is beating fast. Your evaluation reveals a female in mild distress with a heart rate of 130 bpm, blood pressure of 135/75, and saturating 99% on room air. An ECG is shown. Your first choice of action would be:

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c. Administer IV adenosine.
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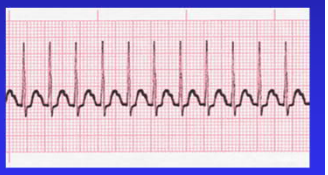

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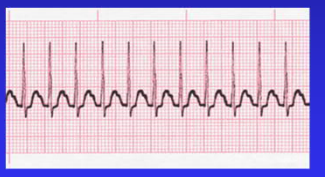

QUESTIONS

?

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BURN & INHALATION INJURY
For The General Surgery Board Exam

David Hampton M.D., M.Eng, FACS

Original notes prepared by Gregory York, MD and Laura Crankshaw, MD

Why this topic is important

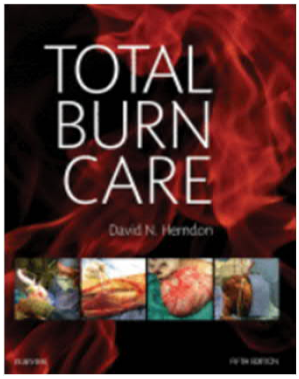
- Burn is a *Core Disease/Condition*
  - Initial Assessment and Management of Trauma
  - Frostbite, Hypothermia
  - Electrical injuries
- Smoke Inhalation Injury and CO Poisoning is an *Advanced Disease/Condition*

SCORE *Surgicalcore.org*

Resources for this Lecture

American Burn Association

Advanced Burn Life Support Course
PROVIDER MANUAL
2018 UPDATE



Important Topics in Acute Burn Care Management

- Inhalation Injury
- Hypermetabolic Response to Trauma
- Fluid Resuscitation
- Early Excision
- Skin Grafting
- Topical Control of Infection
- Nutritional Support

Important Topics in ABLS

Chapter 1	Introduction
Chapter 2	Initial Assessment and Management
Chapter 3	Airway Management and Smoke Inhalation Injury
Chapter 4	Shock and Fluid Resuscitation
Chapter 5	Burn Wound Management
Chapter 6	Electrical Injury
Chapter 7	Chemical Burns
Chapter 8	Pediatric Burn Injuries
Chapter 9	Stabilization, Transfer and Transport
Chapter 10	Burn Disaster Management

EPIDEMIOLOGY & BURN BASICS

CDC WISQARS™ Injury Data
WISQARS™ — Web-based Injury Statistics Query and Reporting System

American Burn Association

- United States
  - 450,000 people receive medical attention for burns annually
  - 45,000 hospitalizations for burn injuries
  - 4,000 annual deaths, primarily from residential fires (3,500)
  - 75% deaths occur at the scene or during initial transport
  - Leading cause of fire death in US is from fires due to smoking materials, especially cigarettes
  - Inhlalation injury remains major determinant of mortality in severe burns

EPIDEMIOLOGY & BURN BASICS
TREND

- 1942-1952: shock, sepsis and multi-organ failure => 50% mortality rate in children with 50% TBSA burn
- In burned children a 98% TBSA burn now has a 50% survival rate

INITIAL ASSESSMENT & MANAGEMENT

Every Burn Patient is a Trauma Patient

INITIAL ASSESSMENT & MANAGEMENT
PRIMARY SURVEY: ABCs

- **Airway:**
  - ALL burn patients get supplemental O_2
  - Intubate if: GCS ≤ 8, CO ≥ 20%, TBSA ≥ 30%
  - Inhalation injury: carbonaceous sputum, hoarseness, stridor
- **Breathing:**
  - 100% O_2 if inhalation injury suspect
  - Circumferential full thickness burns of trunk & neck
- **Circulation:**
  - Burn shock = hypovolemic shock
  - Cardiac arrhythmias may be due to electrical injury
  - Fluid resuscitation: start LR 500cc/hr
  - Circumferential full thickness burns of extremity

INITIAL ASSESSMENT & MANAGEMENT
PRIMARY SURVEY

- **Disability (GCS)**:
  - CO poisoning -> ***100% FiO_2***
  - Cyanide poisoning (elevated lactate, persistent acidosis, unexplained hypoxemia) → ***hydroxycobalamin***
- **Exposure / Environment**:
  - Stop the burning process, remove all clothing and jewelry
  - Maintain core body temp (37 °C)

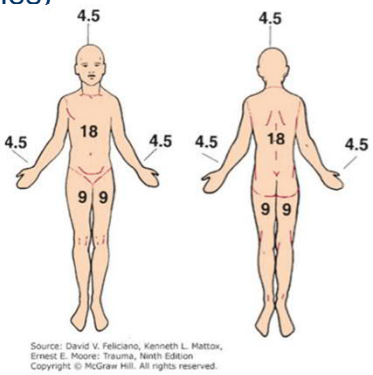
INITIAL ASSESSMENT & MANAGEMENT
SECONDARY SURVEY

- Secondary Survey
  - Determine TBSA (Rule of Nines)
  - Determine need for transfer

Box 7.1 Criteria for Transfer of a Burn Patient to a Burn Center

- Second-degree burns >10% total body surface area (TBSA)
- Third-degree burns
- Burns that involve the face, hands, feet, genitalia, perineum, and major joints
- Chemical burns
- Electrical burns including lightning injuries
- Any burn with concomitant trauma in which the burn injuries pose the greatest risk to the patient
- Inhalation injury
- Patients with pre-existing medical disorders that could complicate management, prolong recovery, or affect mortality
- Hospitals without qualified personnel or equipment for the care of critically burned children

From American Burn Association. Advanced Burn Life Supporters Manual. *Chicago, IL: American Burn Association; 2011.*



AIRWAY MANAGEMENT & SMOKE INHALATION INJURY

Major determinant of mortality in a patient with severe burns is inhalation injury

Types of Inhalation Injury

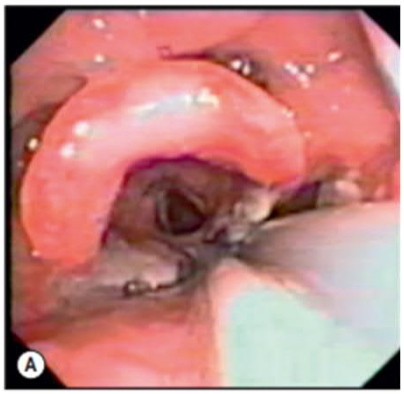
- **Exposure to toxic gases** (carbon monoxide, cyanide)
- **Supraglottic injury** (oropharynx): thermal injury, causes severe mucosal edema.
- **Subglottic injury** (tracheobronchial): chemical injury, causes airway inflammation and edema

Treatment for Specific Types of Inhalation Injury

- **Exposure to toxic gases**
  - **Carbon monoxide** -> 100% FiO_2 until COHb wnl
  - **Cyanide**: hydroxycobalamin based on RF:
    - Exposed to fire with smoke
    - Decreased GCS
    - Carbonaceous sputum
    - Dyspnea, convulsions in presence of persistent metabolic acidosis
    - No response to treatment with 100% O_2 & resuscitation

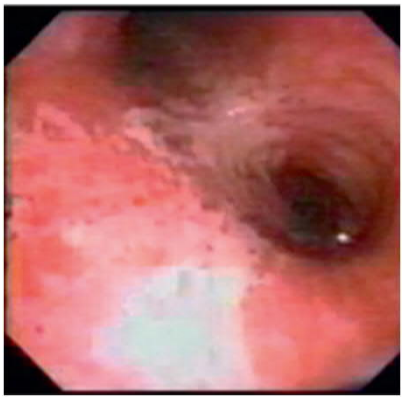
Treatment for Specific Types of Inhalation Injury

- **Supraglottic:**
  - Due to direct heat
  - Upper airway obstruction can progress rapidly
  - Look for pharyngeal edema, hoarseness, stridor
  - ABG & pulse ox not useful to determine need for intubation
  - Treatment is intubation
    - Elevate HOB, assess for cuff leak to monitor edema



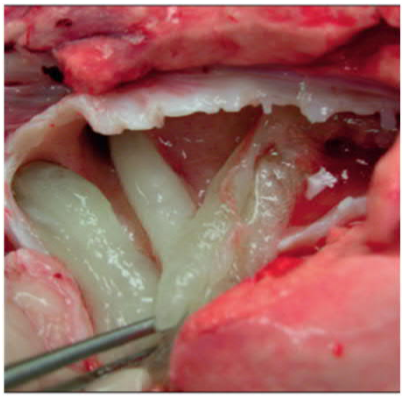
Treatment for Specific Types of Inhalation Injury

- **Subglottic / tracheobronchial injury**:
  - Thick bronchial secretions & wheezing
  - Impairs respiratory gas exchange
    - Impairment may be delayed
    - First sign is decreased PaO_2 (vs abnl CXR)
  - Treatment is intubation to clear secretions, relieve dyspnea, and ensure adequate oxygenation and ventilation



SUBGLOTTIC INHALATION INJURY
PATHOPHYSIOLOGY OF OBSTRUCTION

- **Airway Exudate**
  - Separation of ciliated epithelium
  - Exudate formation within airways
  - Consists of proteins from lung lymph
  - Fibrin cast development from coalesced proteins
  - "Ball Valve" Effect
    - Inspiration: Airway diameter increases and air flows past cast
    - Exhalation: Airway diameter decreases, fibrin cast traps occludes airway
    - Increased volume leads to barotrauma, pneumothorax, and decreased compliance



INHALATION INJURY
DIAGNOSIS

- Diagnosis
  - History: Smoke exposure in closed space
  - Exam
    - Hoarseness, Wheezing
    - Carbonaceous sputum
    - Large burns or facial burns
  - Bronchoscopy
    - Establishes the diagnosis, grades the injury
    - Erythema, edema, ulceration, mucosal sloughing, prominent vasculature

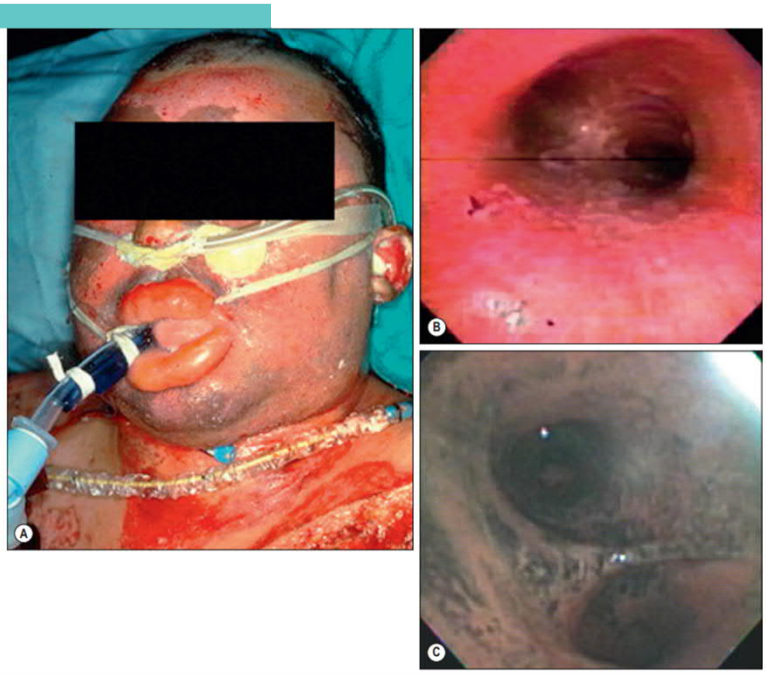


Fig. 16.3 Facial and airway injury after burn and smoke inhalation. **(A)** A facial burn is often associated with thermal injury to the upper airway. **(B)** Hyperemia of airway epithelium. **(C)** Formation of airway obstructive cast. (A is from Cancio LC. Airway management and smoke inhalation injury in the burn patient. *Clin Plast Surg*. 2009;36(4):555–567.)

INHALATION INJURY
TREATMENT

- Maintain open airway, maximize gas exchange
- Prophylactic intubation
- Principles of mechanical ventilation
  - Avoid large tidal volumes
  - PEEP 5-8mmHg to prevent alveolar collapse
  - HFPV in severe cases
- Clear secretions
  - Pulmonary hygiene w/ chest physiotherapy/suctioning
  - Therapeutic Bronchoscopy
- Pharmacologic Triad
  - Bronchodilators: treats bronchospasm
  - Nebulized Heparin: reduces cast formation
  - N-Acetylcysteine: mucolytic (loosens mucous plugs)

SHOCK & FLUID RESUSCITATION

Burn Shock = Hypovolemic Shock
Principles of Resuscitation in First 24 hours

PATHOPHSIOLOGY
BURN SHOCK & BURN EDEMA

- Inflammatory Mediators of Burn Injury
  - Etiology
    - Mast Cell, Platelets, WBCs
    - Mediators: Histamine, Prostaglandins, Thromboxane A_2, Kinins, Serotonin, Catecholamines, Reactive O_2 species, Nitric Oxide, platelet aggregation factor, Angiotensin II & Vasopressin
  - Hemodynamic Effects
    - ↑ vascular permeability → intravascular hypovolemia
      - Massive fluid shifts from capillary leak causing tissue edema
    - ↓ CO + ↑ SVR is early manifestation
    - Vasoconstriction, Vasodilation → combined hypovolemic and distributive shock

PATHOPHSIOLOGY
HYPERCATABOLISM

- Hormonally Mediated
  - ↑ Catecholemines, Cortisol, Glucagon
- ↑ Gluconeogenesis
- ↑ O_2 consumption, ↑ CO_2 production
- ↑ Core body temperature
- ↑ Protein Catabolism
- ↑ Lipolysis
- Nitrogen Loss

PATHOPHSIOLOGY
ORGAN FUNCTION

- Decreased plasma volume
- Increased peripheral vascular resistance
- Decreased cardiac output
- Decreased pulmonary static compliance
- Decreased renal blood flow
- Increased resting energy expenditure
- Depressed immunoglobulin production
- Depressed bactericidal activity

INITIAL CARE & RESUSCITATION
FLUID MANAGEMENT – First 24 Hours

- Who?
  - Adult patient with burn ≥ 20% TBSA (2nd & 3rd degree only)
- Type of Fluid
  - Lactated Ringers
- How Much?
  - Too much → compartment syndromes (extremity, orbital, abdominal)
  - Not enough → shock & organ failure

RESUSCITATION FORMULAS & CONSENSUS STATEMENT

- Parkland Formula: 4 mL/kg/%TBSA / 24hrs
- Modified Brooke Formula: 2 mL/kg/%TBSA / 24hrs

- Half of calculated 24hr volume given in first 8 hrs post-burn, calculated from time of injury
- Volume of subsequent fluid is based on urinary output and clinical response

INITIAL CARE & RESUSCITATION
FLUID MANAGEMENT – First 24 Hours

- Goal of resuscitation – UOP
  - **Adults: 0.5 ml/kg/hour (or 30-50 ml/hour)**
  - Children (≤ 30kg): 1 ml/kg/hour
- If UOP too low/high – adjust fluid rate by 20%
- Challenging patients to resuscitate:
  - Polytrauma, electrical injury, inhalation injury
  - *Adult patients with high voltage electrical injuries with evidence of myoglobinuria: 75 – 100 ml/hour until urine clears.*

BURN WOUND MANAGEMENT

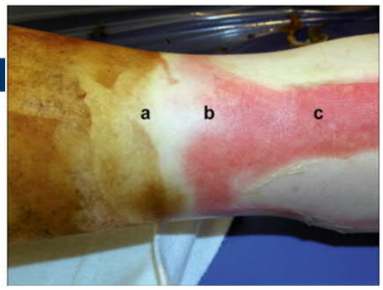
Assessing Burn Depth
Topical Treatments
Early Excision & Grafting

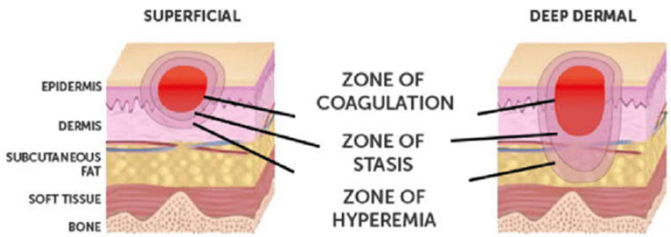
BURN WOUND MANAGEMENT
BURN CATEGORIES

- Flash & Flame ~40% admissions
  - Duration of exposure to intense heat
- Scald (hot water, grease, oil)
  - Depth of injury depends on water temp, skin thickness, and duration of contact
- Contact (hot metals, plastic, glass)
  - Small but deep
- Chemical
- Electrical

BURN WOUND MANAGEMENT
ZONES OF INJURY

- (A) Coagulation
- (B) Stasis
- (C) Hyperemia



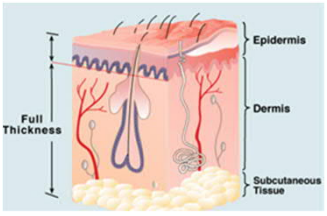


BURN WOUND MANAGEMENT
DEPTH OF BURN

- First Degree
  - Confined to epidermis
  - Painful, erythematous, blanches
  - Sunburn, minor scald, flash burn
  - Heals in 3-6 days
  - No scarring
  - Treatment aimed at comfort

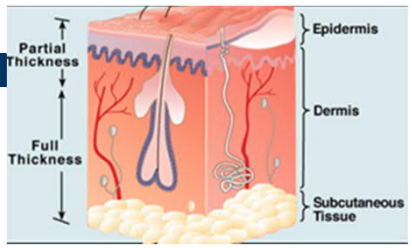
BURN WOUND MANAGEMENT
DEPTH OF BURN

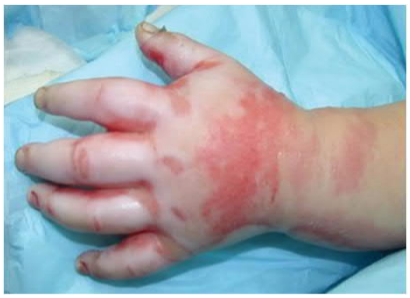
- Second Degree (Superficial)
  - Erythematous, painful, may blanch
  - Blisters
  - Scald injuries, flash flame burns
  - Re-epithelializes from retained epidermal structures in rete ridges, hair follicles, and sweat glands (7-14 days)
  - Long term shows slight skin discoloration



BURN WOUND MANAGEMENT
DEPTH OF BURN

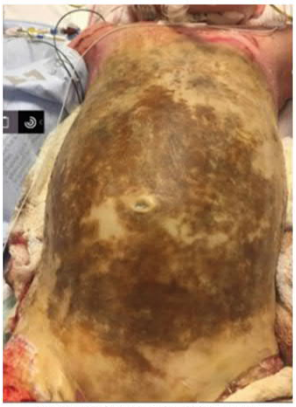
- Second Degree (Deep)
  - Into reticular dermis
  - Pale, mottled, does not blanch
  - Painful to pinprick
  - Heals by re-epithelialization from hair follicles and keratinocytes in sweat glands (21 – 28 days)
  - Timely healing requires excision and grafting
  - Severe scarring due to loss of most of dermis





BURN WOUND MANAGEMENT
DEPTH OF BURN

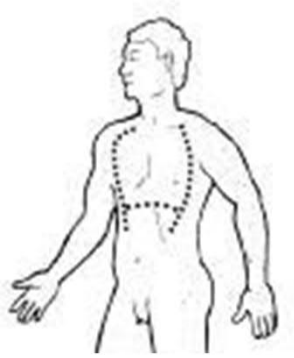
- Third Degree
  - Full thickness through dermis
  - Firm, leathery eschar, painless
  - Heals by re-epithelialization from wound edges by contraction
  - Timely healing requires excision and grafting



Source: David V. Feliciano, Kenneth L. Mattox, Ernest E. Moore: Trauma, Ninth Edition

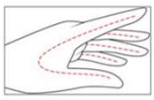
CIRCUMFERENTIAL BURNS
TRUNK

- Trunk
  - Limits chest wall excursion
  - ↓ ventilation
  - ↑ pCO_2
  - ↑ peak airway pressures
  - Desaturation
  - Treatment: Truncal Escharotomy



CIRCUMFERENTIAL BURNS
EXTREMITY

- Extremity
  - Etiology: Deep 2nd/3rd degree burns encompassing the extremity
  - Generalized edema
    - Develops beneath eschar
    - Impedes venous outflow
    - Eventually affects arterial inflow
    - Compromised tissue perfusion
  - Signs/Symptoms
    - Extremity numbness/tingling
    - Increased pain in the digits
    - Decreased Doppler signals/capillary refill
  - Treatment
    - Escharotomy: Releases eschar component
    - Fasciotomy: Compromised musculofascial compartments



BURN WOUND MANAGEMENT
GENERAL PRINCIPLES

- Assessment
  - Extent & Depth of wound
  - Clean & Debride wound
- Wound Management
  - Early Excision
  - Skin Grafting
  - Topical Control of Infection
- Adjunctive therapy
  - Tetanus, nutritional support, oxandrolone
  - DVT/GI prophylaxis (TBSA > 25%)

BURN WOUND MANAGEMENT
WOUND COVERAGE

- Functions of wound coverage
  - Protect & splint damaged epithelium
  - Reduce evaporative heat loss & minimize cold stress
  - Pain control
- Coverage by type
  - 1st Degree: Topical ointment
  - Partial thickness (2nd degree)
    - Superficial: topical antimicrobial & coverage
    - Deep: early excision and autografting
  - Full thickness (3rd degree): early excision & autografting

BURN WOUND MANAGEMENT
TOPICAL ANTIMICROBIALS

Topical Agent	Advantages	Disadvantages
Silver Sulfadiazine	Broad Spectrum (GPC, GNR, Fungus) Painless application Large burns	Some Pseudomonas resistance Transient Leukopenia and/or Thrombocytopenia Poor eschar penetration
Silver Nitrate	Excellent Spectrum (GPC, GNR, Fungus) Painless application Large burns	Staining Electrolyte abnormalities (hyponatremia) Poor eschar penetration Methemoglobinemia
Mafenide acetate (Sulfamylon)	Broad Spectrum (GPC, GNR) Good eschar penetration Small burns	Painful Application Metabolic Acidosis Not effective against fungus

BURN WOUND MANAGEMENT
EXCISION & GRAFTING

- Considerations
  - Accomplish ASAP after excision (1-7 days)
  - Autograft is best
  - Cadaver allograft: Temporary for large burns
- Types of Coverage
  - Autograft
  - Cadaver Allograft
  - Xenograft (Porcine)
  - Synthetic: Integra, Biobrane, Transcyte

ELECTRICAL INJURY

Compartment Syndrome
Cardiac Dysfunction
Myoglobinuria

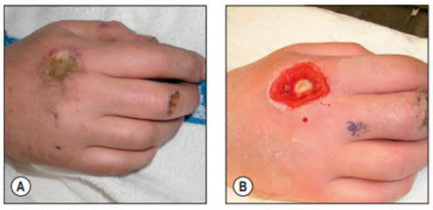


Fig. 10.6 High-voltage contact point on a hand: **(A)** prior to, and **(B)** after débridement. Once wound is debrided, much deeper injury to tendon and bone is revealed.

ELETRICAL BURN INJURIES

- Characteristics
  - Injury is MOSTLY internal
  - Current follows path of LEAST resistance: nerves, blood vessels, muscle
  - High index of suspicion for:
    - Compartment syndrome
    - Myoglobinuria
- Treatment
  - Vigorous resuscitation (watch for myoglobinuria)
  - UOP greater than 1 mL/kg/hr
  - Monitor for arrhythmias for 24 to 48 hours
  - Regular burn wound care

ELETRICAL BURN INJURIES
MYOGLOBINURIA

- Characteristics
  - Associated with high voltage injuries
  - Myoglobin precipitates in and damages renal tubules
- Diagnosis
  - Dark red urine
  - U/A positive for hemoglobin (no cells)
  - Elevated serum myoglobin
- Treatment
  - Hydration
  - Maintain UOP 1 – 2 mL/kg/hr
  - Alkalinization
    - Use of sodium bicarbonate
    - Decreases hemochromogen precipitation in renal tubules (not proven)
  - Mannitol to induce diuresis
  - Monitor serum potassium

OTHER BURNS

Chemical Burns
Frostbite
Hypothermia

CHEMICAL BURNS

- Pathophysiology
  - Acid: Coagulative necrosis; confined
  - Base: Liquefaction necrosis; extends into tissues
- Treatment
  - Initial Therapy: Copious irrigation with saline
  - Neutralization Attempts
    - Result in heat production
    - Extend injury
  - Once controlled, treat as regular burn injury
- Hydrofluoric Acid
  - Severe burns/fluoride ion precipitates calcium
  - Systemic hypocalcemia – even with small burns
  - Copious irrigation
  - Calcium gluconate (2.5%) gel to the affected skin
  - Monitor EKG changes: QT interval, torsade de pointes, VF

FROSTBITE
LOCAL COLD INJURY

- “flash freeze” / cold contact pathophysiology
- Signs/Symptoms:
  - cold, clumsy, insensate extremity
  - appears pale or mottled blue
- Treatment
  - Pad, splint and elevate affected extremity
  - immersion in gently circulating water at 40–42°C for 15 to 30 minutes
  - Tetanus, pain control

HYPOTHERMIA

- Accounts for ~500 deaths per year
- Primary vs. secondary hyperthermia
- Symptoms non-specific

Table 1. Findings in Hypothermia

Hypothermia Class	CoreTemperature	Characteristics
Mild	32°C–35°C (90°F–95°F)	Vasoconstriction, shivering, cold sensations, coagulopathy
Moderate	28°C–32°C (82.4°F-90°F)	Bradycardia, confusion or agitation, metabolic acidosis, cold-induced diuresis
Severe	20°C-28°C (68°F-82.3°F)	Coma, respiratory depression, profound hypovolemia
Profound	Below 20°C (Below 68°F)	Apnea, asystolic arrest

HYPOTHERMIA TREATMENT

- All patients: rewarming, fluid resuscitation, correction of electrolytes
- Mild-Moderate hypothermia
  - PO hot liquids, external warming methods (e.g warm air via convective heating blankets)
- Severe hypothermia:
  - Active rewarming by immersion in water bath at 40°C (most rapid technique)
  - Wrap cold extremities in dry towels, rewarm only when until the core T 35° C

CRITERIA FOR TRANSFER TO BURN CENTER

- 2nd & 3rd Degree Burns > 10% TBSA
- 3rd Degree Burns any age group
- Face, hand, feet, eyes, ears, genitalia, perineum, or major joint involvement
- Electrical burns, including lightening injury
- Chemical burns
- Inhalation injury
- Patients with significant co-morbid conditions
- Concomitant trauma in which the burn poses the greatest mortality and morbidity
- Children in a hospital without qualified personnel or equipment to care for children
- Patients who will require special social, emotional, or long-term rehabilitation

PATIENT MANAGEMENT
PRIOR TO BURN UNIT TRANSFER

- Keep patient warm
- Cover with clean or sterile sheets
- Early application of topical antimicrobial in deep partial and full thickness burns
- Do NOT apply antimicrobial before communication with receiving burn unit

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QUESTIONS

?

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Pediatric Trauma & Critical Care

Surgical Critical Care

David Hampton MD, MEng, FACS

Original notes prepared by Mark Slidell, MD, MPH,
modified by Laura Crankshaw, MD

1

Pediatric trauma: Burden of disease

- Most common cause of death and disability in childhood
- Affects 1 in 3 children.
- Children account for ~25% of all traumatic injuries.
- Injury has been the leading cause of death for children 1 to 14 years of age for decades.

- Morrison, W., Wright, J. L., & Paidas, C. N. (2002). Pediatric trauma systems. Critical Care Medicine, 30 (11, Suppl), S448–S456.

Background
Trauma Surgery

- Rise of Trauma Surgery in the late 60's and 70's
- Optimal Resource Guidelines 1976.
- Rise of trauma surgery and pediatric surgery occurred somewhat in parallel.
- Pediatric trauma was not at the forefront.
  - Children approached as little adults unless evidence to the contrary
- Development of generalized trauma protocols
  - Applied to adults and children

Background
Pediatric Trauma Surgery

- Pediatric Trauma Score – (1987)
  - Predicts injury severity and mortality
- ACS-COT refines Optimal Resources Document to include Peds Level 1 and Level 2 designations.
  - Unintended consequences
    → Decr. investment in comprehensive PTCs.
- Currently ~136 "High level" PTCs in the U.S.
  - "High level" defined as L1 or L2 centers.
- Reduction in injury-related mortality and morbidity for children, but disparities in access to PTCs persist.

Types of trauma centers

- Adult Trauma Centers (ATC)
- Adult Trauma Centers w/ Additional Qualifications (ATC-AQ)
  - Mixed Trauma Centers (MTC)
- Pediatric Trauma Centers (PTC)
- Designation criteria vary from state to state
- Most State guidelines are either the same or similar to those of the ACS-COT

PTC and Pediatric outcomes

- Differences between adults and children?
- Can children be treated with adult trauma protocols?
  "Children are just little adults"
- Can the pediatric-specific protocols be applied to adults as well?

  "Adults are just big kids"

Resources to Address Injured Children's Unique Needs

- **Specialized care** for unique:
  - Anatomical characteristics
  - Physiological characteristics
  - Psychological characteristics
- **Specialized resources**
  - Equipment (specially sized)
  - Personnel (pediatric certification)
  - Medication dose adjustments

Epidemiology

Causes and Mechanism of Injury

(Varies by age and urban vs rural)

1. Falls
2. Motor Vehicle Accidents
3. Pedestrian Injury
4. Bicycle Injury

Unique Aspects of Children

Psychological Status

- Emotional instability can result in regressive psychologic behavior
- Child's interaction with unfamiliar health care workers may be limited

Unique Aspects of Children

Long Term Effects

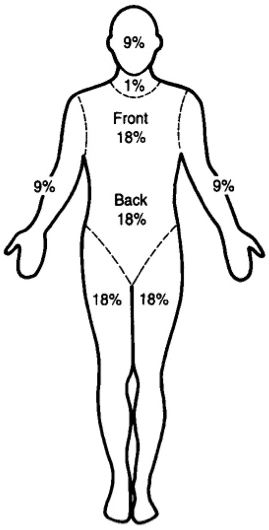
- Injury may affect growth and development
- 60% of children with severe multi-system trauma have residual personality problems
- 50% of these patients have physical and cognitive handicaps

Anatomic and physiologic differences between children and adults

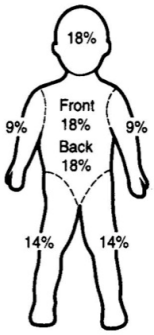
- Implications for specific injury patterns
  - Head injuries
  - TBSA
  - Spine injuries
  - Thoracic injuries
  - Abdominal and pelvic injuries
  - Burns
  - Non-Accidental Trauma

Anatomic and Physiologic Differences – Head

Adult body	% of total
Part	BSA
Arm	9%
Head	9%
Neck	1%
Leg	18%
Anterior trunk	18%
Posterior trunk	18%



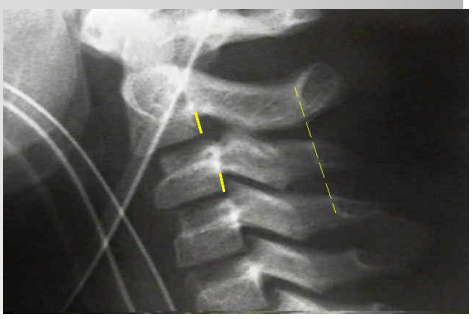
Head Trauma
Open fontanelles (infants)
Subdural Hematomas



Child body	% of total
Part	BSA
Arm	9%
Head and neck	18%
Leg	14%
Anterior trunk	18%
Posterior trunk	18%

Anatomic and Physiologic Differences: Spine

- Predisposition to cervical spine injuries
  - Large head → angular momentum → increased risk of cervical spine injury
    - May not be difficult to appreciate on radiographic evaluation.
    - SCIWORA – Spinal Cord Injury Without Radiologic Abnormality
- Flexible spinal ligaments
- Anteriorly wedged vertebrae
- Flat facet joints
- Pseudosubluxation



Anatomic and Physiologic Differences: Thorax

- Chest wall is soft and pliable
  - Less protection from rigid chest wall→ pulmonary contusions
  - Implication that a child with a rib fractures was subjected to significant force
- Ribs aligned in a more horizontal orientation. Underdeveloped intercostal muscles
- Pliable chest wall; Increased tension physiology during a tension PTX secondary to a mobile mediastinum

Anatomic and Physiologic Differences: Abdomen/Pelvis

- Softer, thinner, abdominal wall
- Underdeveloped muscles
- Thinner skin
- Lower-riding liver, spleen is more exposed to blunt trauma
- In young children the bladder is partly an intra-abdominal organ
- Smaller the child, the easier it is to access the entire abdomen/pelvis through small incisions

Pediatric Abdominal Trauma Organs Injured:

▪Spleen	▪50%
▪Liver	▪20%
▪Kidney	▪10%
▪GI Tract	▪1-2%
▪Pancreas	▪1-2%

Pediatric Splenic Trauma
Evolution of practice patterns

- First splenectomy for closed trauma was in a child

1892 Sir William Arbuthnot Lane - Guy's Hospital in London
Two unsuccessful reports of splenectomy
15 y.o. boy fell off a brougham → splenectomy, but died 5 hrs later
4 y.o. boy struck by pole of a carriage → splenectomy, but died

1893 Oskar Riegner - All Saint's Hosp. in Wrocław, Poland (Breslau)
14 y.o. laborer who fell two stories from scaffolding.
Hypodermoclysis into thigh led to gangrene of LLE → amputation.
Discharged after 5 months.

Nonoperative Management of Splenic Trauma in Children: A Report of Six Consecutive Cases

Daniel Z. Aronson, M.D., Arnold W. Scherz, M.D., Arnold H. Einhorn, M.D., Jerrold M. Becker, M.D., *and* Keith M. Schneider, M.D.

From the Department of Pediatrics and the Division of Pediatric Surgery, Bronx Municipal Hospital Center, Albert Einstein College of Medicine, Bronx, New York

ABSTRACT. Postsplenectomy sepsis, although infrequent, is real and hazardous. We have taken a nonoperative approach to the child with a ruptured spleen who is in stable condition. Six consecutive patients with a diagnosis confirmed by angiography or scan were treated without surgery. The results suggest the usefulness of this approach in the child who is not bleeding massively. *Pediatrics* 60:482-485, 1977, SPLENECTOMY, POSTSPLENECTOMY SEPSIS, NONOPERATIVE MANAGEMENT.

Aronson DZ, Nonoperative Management of Splenic Trauma in Children: A Report of Six Consecutive Cases. Pediatrics, Oct. 1977, Vol. 60/4

Pediatric Splenic Trauma Evolution of practice patterns

- Wesson (1981) "Ruptured spleen--when to operate?"
- 63 patients with splenic injuries 5-yr period from 1974-1979 Toronto, ON
- Stable on admission or after initial resuscitation → treated non-operatively.
  - strict bed rest, NGT, IVFs, blood products prn
  - 40 patients were treated nonoperatively.
- Results: No other morbidity and no mortality following non-operative treatment

" we believe that where adequate facilities exist, non-operative treatment of splenic injuries is both safe and effective. "

Wesson DE, et. al, Ruptured spleen--when to operate? Pediatr Surg. 1981 Jun;16(3):324-6.

Evidence-Based Guidelines for Resource Utilization in Children With Isolated Spleen or Liver Injury

By Steven Stylianos and the APSA Trauma Committee
New York, New York

- By 2000, the non-operative mgmt. pediatric splenic injuries was standard of care
- APSA Committee on Trauma, organized multicenter study
  - 856 children who were treated at 32 centers
  - Outcome for non-operative mgmt. of solid organ injuries in children
  - Conclusions:
    - Non-operative management of the hemodynamically stable child with a liver or spleen injury is safe and feasible
    - Resource utilization increased with injury severity

- Stylianos S. Evidence-based guidelines for resource utilization in children with isolated spleen or liver injury. The APSA Trauma Committee. J Pediatr Surg. 2000;35(2):164Y167.

Solid Organ Injuries: Pediatric trauma vs. Adult trauma

- Solid organ injuries in adults
  - ~ 70% of adults are successfully managed non-operatively
  - 90% of liver injuries in children
  - >95% of splenic injuries.
- Differences in response to injury
- Different comorbidities

Pediatric Solid Organ Injuries: Current protocols

Updated APSA Blunt Liver/Spleen Injury Guidelines 2019

Admission

- **ICU Admission Indicators**
  - Abnormal vital signs after initial volume resuscitation
- **ICU**
  - Activity - Bedrest until vitals normal
  - Labs – q6hour CBC until vitals normal
  - Diet – NPO until vital signs normal and hemoglobin stable
- **Ward**
  - Activity - No restrictions
  - Labs - CBC on admission and/or 6 hours after injury
  - Diet – Regular diet

Procedures

- **Transfusion**
  - Unstable vitals after 20 cc/kg bolus of isotonic IVF
  - Hemoglobin < 7
  - Signs of ongoing or recent bleeding
- **Angioembolization**
  - Signs of ongoing bleeding despite pRBC transfusion
  - Not indicated for contrast blush on admission CT without unstable vitals
- **Operative exploration with Control of Bleeding**
  - Unstable vitals despite pRBC transfusion
  - Consider massive transfusion protocol

Set Free

- Based on clinical condition **NOT** injury severity (grade)
- Tolerating a diet
- Minimal abdominal pain
- Normal vital signs

Aftercare

- **Activity Restriction**
  - Restricting activity to grade plus 2 weeks is safe
  - Shorter restrictions may be safe but there is inadequate data to support decreasing these recommendations
- **Follow up Imaging**
  - Risk of delayed complications following spleen and liver injuries is low
  - Consider imaging for ***symptomatic*** patients with prior high grade injuries

- High costal margin
- Abdomen = nipple down
- Small pliable rib cage and undeveloped muscles provide little protection
- Solid organs commonly injured
- Nonoperative management
- Evaluation--CT/FAST

Abdominal Injuries

- Duodenal hematomas quite common after blunt trauma
- Mechanisms: MVA, bicycle handlebars, soccer balls, etc
- Signs: abdominal distension, bilious emesis
- Work-up: CT then UGI
- Treatment: NPO, NG, TPN (2-3 weeks)

Pediatric Abdominal Trauma
Pancreas/Duodenum

- Pancreatic contusion/hematoma most common-- causes “pancreatitis”
- Treatment: CT, NPO, NG, TPN. Follow serial amylase/lipase levels. Usually get follow-up CT to document recovery, rule out pseudocyst.
- Pancreatic duct disruption: rare, requires distal pancreatectomy

Pediatric Abdominal Trauma
Pancreas/Duodenum

- Uncommon (1% of 1488 children who had CT at Children’s National Med Ctr 1994)
- Small intestine > large intestine
- Difficult to diagnose
- Diagnosis made by: physical exam (peritonitis), DPL, CT

Pediatric Abdominal Trauma
Intestinal Injury

- Most common cause of death/disability
- Recover more fully and more frequently than similarly injured adult
- Principal determinant of outcome
- GCS < 8 = ICP monitor
- Seizures more common
- Large fontanelle in young child

CNS Injury

- C Spine injury uncommon
- Usually involves 1st two vertebrae
- Compression fractures
- 40% pseudosubluxation C2-C4
- Flexion-distraction (Chance) fracture of L spine
  - Lap seat misuse

CNS Injury

- SCIWORA (spinal cord injury without radiological abnormality)
  - Incompletely calcified vertebral column
  - Stretching of cord/roots w/ no evidence injury
  - Neurologic deficit changed/resolved by time child in ED
  - MRI may resolve

CNS Injury

- Most common cause head injury < 2 yr
- Retinal hemorrhage
- SDH/SAH
- Little evidence external trauma
- Shearing of cervical tissue
  - Edema may take several hours to develop (interval CT)

"Shaken-baby" Syndrome

Ionizing Radiation (ALARA)

TABLE 1 Estimated Medical Radiation Doses for a 5-Year Old Child

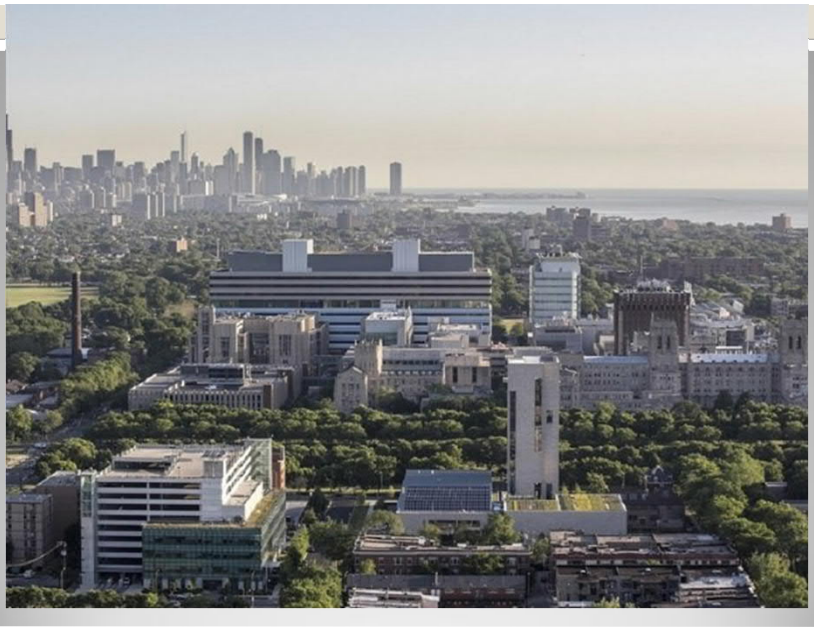
	mSv	CXR equiv
3-view ankle	0.0015	1/14th
2-view chest	0.02	1
Tx 99m radionuclide gastric emptying	0.06	3
Natural background (Denver)	3.5	175
Head CT	4	200
Chest CT	3	150
Abdomen CT	5	250

- Measurements of radiation exposure
  - Gray (Gy)—absorption of 1 joule radiation energy by 1 kg matter
  - Sievert (Sv)—accounts for the biologic effects of radiation
- Children more sensitive to radiation effects than adults
  - Growing organs
  - Long latent period of oncogenic effect (varies with type of cancer)
  - For CT, any given exposure results in a dose that is relatively higher since kid's have a smaller cross-sectional area
  - **Imaging equipment carefully calibrated for children**

Brody, *Pediatrics*, 2007; Rice, *JPS*, 2007

Summary of some observed differences between: PTC and ATC

- Mortality rates for <u>some</u> patients
- Non-operative treatment successfully employed more often in PTCs, e.g. Splenic preservation rates usually higher
- Transfusion rates much lower in PTCs
- Judicious use of radiation in imaging [ALARA "As Low As (is) Reasonably Achievable"]
- Neurosurgery
  - ↑ Decompressive Craniectomy → improved outcomes, ↓LOS and ↓ costs
  - Data controversial for many reasons…
- Femur fractures
  - ↑ Early traction or operative fixation → improved outcomes, ↓LOS and ↓ costs



Thank You!

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EVIDENCED BASED MEDICINE, ETHICS, & PROFESSIONALISM

Paras Khandhar MD, FAAP, FAMIA

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THE AMERICAN BOARD OF SURGERY

SURGICAL CRITICAL CARE

CONTENT OUTLINE FOR THE CERTIFYING EXAMINATION (CE) AND CONTINUOUS CERTIFICATION (CC) READMISSIBILITY EXAMINATION

J. Trauma	10%
K. Thermal Injury	4%
L. Monitoring, Bioengineering, and Biostatistics	6%
M. Life-Threatening Pediatric Conditions	2%
N. Principles and Techniques of Administration and Management	3%
O. Pharmacology, Pharmacokinetics, and Drug Metabolism in Critical Illness	2%
P. Ethical and Legal Aspects in Surgical Critical Care Medicine	2%

EVIDENCED BASED MEDICINE

Goals

- Address this evolving process in the context of the Board Exam
- Will not really discuss how to implement
  - Although this is a critical skill!
- Focus on terms and…
- Epidemiologic aspects

Rationale

- Most physicians practice without the best current evidence
- An individual is limited by memory, cognition, & judgment
- Individual physicians have a wide variation in treatment approaches for the same problem

Rationale

- Look for the best available evidence
- Gain comfort with assessing studies and their value
  - A "well-designed" study should generally carry more weight
- Recognize difference between disease outcomes and patient outcomes

5 Steps to Practice EBM

- Ask an answerable question
- Search for the best evidence
- Critically appraise the evidence
- Integrate the evidence with clinical expertise and the patient's biology and values
- Evaluate performance (outcomes)

WHAT MIGHT YOU SEE ON THE BOARDS?

Systematic Review

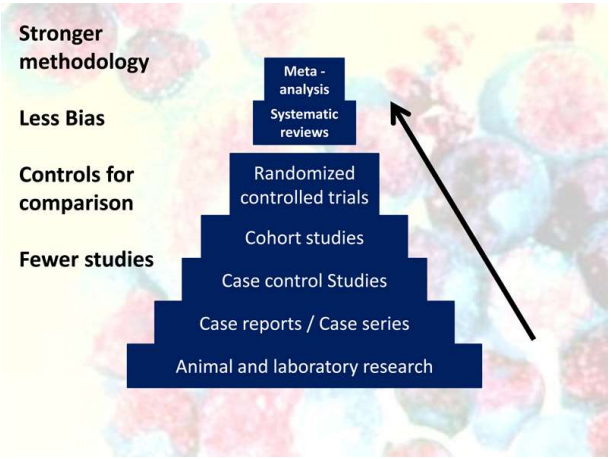
Well defined approach to locate and summarize articles related to a clinical question.

Requires:

- Specific clinical question
- Thorough search for studies
- Clear explanation for which studies were and were not considered
- Descriptive results section summarizing the findings

Levels of Evidence

- From Lowest to Highest (Kinda)



Meta-Analysis

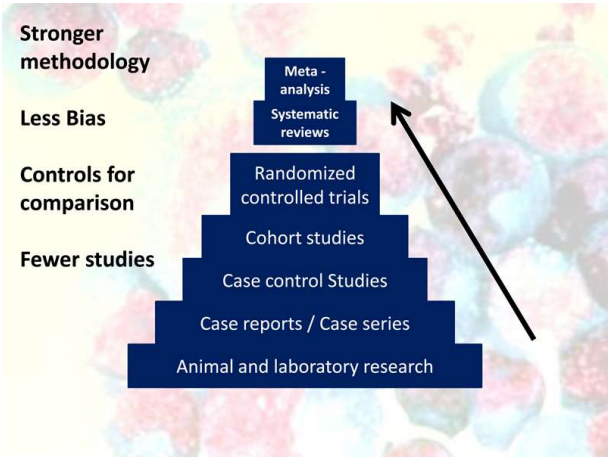
Use published information from other studies and combine the results to reach an overall conclusion

Meta-analyses

- Includes quantitative assessment & summary of findings
- Reports whether combining statistics was even possible
- Especially useful when studies have small sample sizes or come to different conclusions

Levels of Evidence

- From Lowest to Highest (Kinda)



Internal Validity

Study Design	Strengths	Weaknesses
Randomized controlled trials	• High internal validity • Reduced risk of confounding variables	• Reduced external validity • Expensive, time-consuming
Cohort studies	• Useful for sequential events • Can study multiple outcomes • *Retrospective:* less expensive	• Requires large sample size • Risk of confounding variables • Difficult to study rare outcomes • *Prospective:* Expensive
Case-control studies	• Useful for rare outcomes • Can study several exposures • Inexpensive	• Risk of confounding variables
Cross-sectional studies	• Can study multiple outcomes and exposures	• Cannot infer causality • Risk of confounding variables • Less useful for rare exposures or outcomes
Case studies	• Useful for rare outcomes • Convenient, inexpensive	• Risk of confounding variables • Lack of a comparison group • Cannot infer causality

Reprinted with permission from Perry-Parrish C, Dodge R. Validity hierarchy for study design and study type. *Pediatr Rev.* 2010;31(1):28.

Randomized Control Trials

Parachute use to prevent death and major trauma related to gravitational challenge: systematic review of randomised controlled trials

Gordon C S Smith, Jill P Pell

Abstract

Objectives To determine whether parachutes are effective in preventing major trauma related to gravitational challenge.
Design Systematic review of randomised controlled trials.
Data sources: Medline, Web of Science, Embase, and the Cochrane Library databases; appropriate internet sites and citation lists.
Study selection: Studies showing the effects of using a parachute during free fall.
Main outcome measure Death or major trauma, defined as an injury severity score > 15.
Results We were unable to identify any randomised controlled trials of parachute intervention.
Conclusions As with many interventions intended to prevent ill health, the effectiveness of parachutes has not been subjected to rigorous evaluation by using randomised controlled trials. Advocates of evidence based medicine have criticised the adoption of interventions evaluated by using only observational data. We think that everyone might benefit if the most radical protagonists of evidence based medicine organised and participated in a double blind, randomised, placebo controlled, crossover trial of the parachute.

accepted intervention was a fabric device, secured by strings to a harness worn by the participant and released (either automatically or manually) during free fall with the purpose of limiting the rate of descent. We excluded studies that had no control group.

Definition of outcomes

The major outcomes studied were death or major trauma, defined as an injury severity score greater than 15.[9]

Meta-analysis

Our statistical apprach was to assess outcomes in parachute and control groups by odds ratios and quantified the precision of estimates by 95% confidence intervals. We chose the Mantel-Haenszel test to assess heterogeneity, and sensitivity and subgroup analyses and fixed effects weighted regression techniques to explore causes of heterogeneity. We selected a funnel plot to assess publication bias visually and Egger's and Begg's tests to test it quantitatively. Stata software, version 7.0, was the tool for all statistical analyses.

Results

Our search strategy did not find any randomised controlled trials of the parachute.

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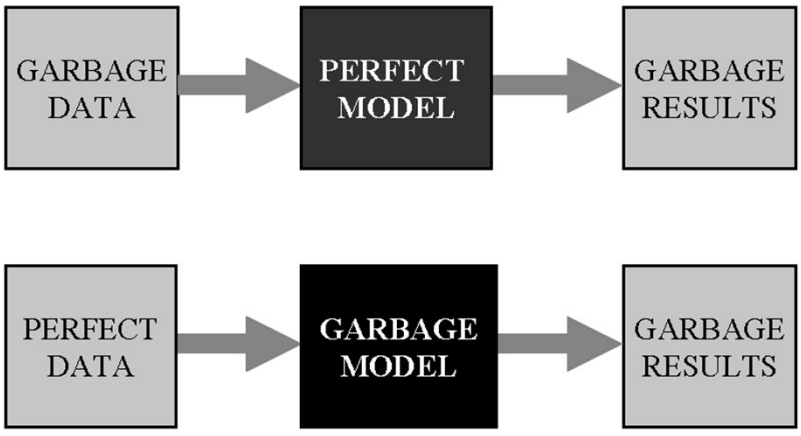
Gordon C S Smith *professor*
Department of Public Health, Greater Glasgow NHS Board, Glasgow G3 8YU
Jill P Pell *consultant*
Correspondence to: G C S Smith
gcss2@cam.ac.uk

BMJ 2003;327:1459–61

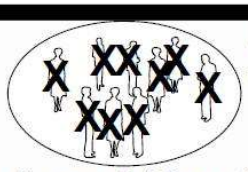
MODEL CALCULATIONS

"Garbage In-garbage Out" Paradigm



TYPES OF STUDY DESIGNS

Is there just one group?



8 people with bird flu

Did all subjects have the disease? (**Case Series**)

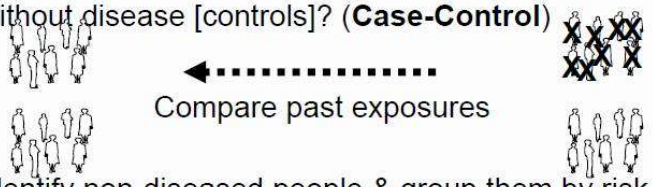
Did they evaluate presence of disease and risk factors at the same point in time?

(**Cross-sectional Survey**

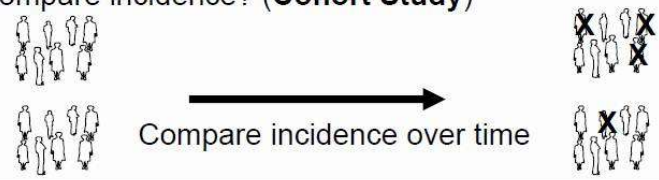
Do you have heart disease?
Are you active?

Two or more groups being compared?

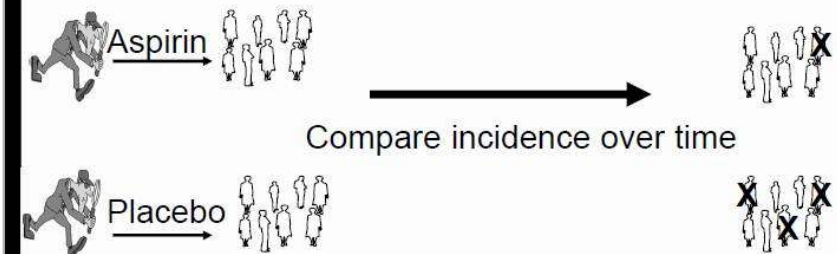
- How were they selected? Did they find people with disease [cases] and then find a comparison group without disease [controls]? (**Case-Control**)

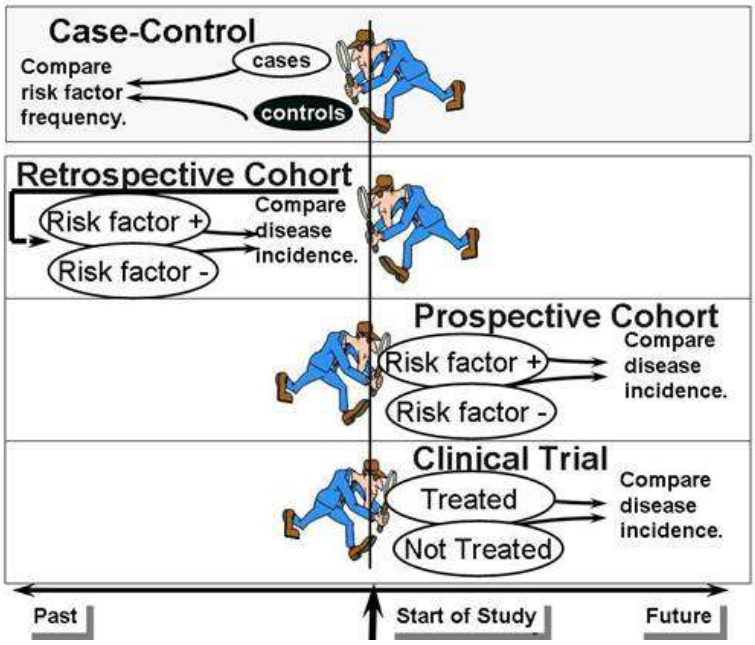


- Identify non-diseased people & group them by risk factor status? Then follow them longitudinally to compare incidence? (**Cohort Study**)



Did the investigators <u>assign</u> subjects to a treatment or intervention and follow them to compare outcomes? **(Clinical Trial)**





Question	Step 1 (Level 1*)	Step 2 (Level 2*)	Step 3 (Level 3*)	Step 4 (Level 4*)	Step 5 (Level 5)
How common is the problem?	Local and current random sample surveys (or censuses)	Systematic review of surveys that allow matching to local circumstances**	Local non-random sample**	Case-series**	n/a
Is this diagnostic or monitoring test accurate? (Diagnosis)	Systematic review of cross sectional studies with consistently applied reference standard and blinding	Individual cross sectional studies with consistently applied reference standard and blinding	Non-consecutive studies, or studies without consistently applied reference standards**	Case-control studies, or "poor or non-independent reference standard**	Mechanism-based reasoning
What will happen if we do not add a therapy? (Prognosis)	Systematic review of inception cohort studies	Inception cohort studies	Cohort study or control arm of randomized trial*	Case-series or case-control studies, or poor quality prognostic cohort study**	n/a
Does this intervention help? (Treatment Benefits)	Systematic review of randomized trials or *n*-of-1 trials	Randomized trial or observational study with dramatic effect	Non-randomized controlled cohort/follow-up study**	Case-series, case-control studies, or historically controlled studies**	Mechanism-based reasoning
What are the COMMON harms? (Treatment Harms)	Systematic review of randomized trials, systematic review of nested case-control studies, *n*-of-1 trial with the patient you are raising the question about, or observational study with dramatic effect	Individual randomized trial or (exceptionally) observational study with dramatic effect	Non-randomized controlled cohort/follow-up study (post-marketing surveillance) provided there are sufficient numbers to rule out a common harm. (For long-term harms the duration of follow-up must be sufficient.)**	Case-series, case-control, or historically controlled studies**	Mechanism-based reasoning
What are the RARE harms? (Treatment Harms)	Systematic review of randomized trials or *n*-of-1 trial	Randomized trial or (exceptionally) observational study with dramatic effect			
Is this (early detection) test worthwhile? (Screening)	Systematic review of randomized trials	Randomized trial	Non -randomized controlled cohort/follow-up study**	Case-series, case-control, or historically controlled studies**	Mechanism-based reasoning

*** Level may be graded down on the basis of study quality, imprecision, indirectness (study PICO does not match questions PICO), because of inconsistency between studies, or because the absolute effect size is very small; Level may be graded up if there is a large or very large effect size.**

**** As always, a systematic review is generally better than an individual study.**

Cheat Sheet to Studies

- RCT → Optimal way to evaluate the effect of 2 different treatments for a disease
  - Most likely to yield valid information about the benefits and/or harms of an intervention.
- Case-control studies → Useful to study individuals with a disease compared to individuals without the disease to evaluate risk factors and outcomes for the disease
- Cohort studies → Follows a population of patients with a disease over time. This approach is likely to yield important information about the natural history of a disease

EBM TERMINOLOGY

Bias

- Flaw in studies that leads to erroneous results
  - Should be minimized in study design
  - Some forms can attempt to be accounted for in statistical analysis

Bias

- Flaw in studies that leads to erroneous results
  - Should be minimized in study design
  - Some forms can attempt to be accounted for in statistical analysis

- Many Types:
  - **Selection Bias: selection of patients creates a difference between the treatment and control groups**
    - **Prevented by randomization and blinding**
  - Recall Bias
  - Funding Bias
  - Attrition Bias
  - Confounding Bias
  - Lead-Time bias

Bias

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- Many Types:
  - Selection Bias
  - **Recall Bias: systematic error caused by differences in the accuracy or completeness of the recollections retrieved ("recalled") by study participants regarding events or experiences from the past.**
  - Funding Bias
  - Attrition Bias
  - Confounding Bias
  - Lead-Time bias
    - Especially true with chronic diseases

Bias

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  - Some forms can attempt to be accounted for in statistical analysis
- Many Types:
  - Selection Bias
  - Recall Bias
  - **Funding Bias: Groups may be treated differently based on the funding sources**
  - Attrition Bias
  - Confounding Bias
  - Lead-Time bias

Bias

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  - Should be minimized in study design
  - Some forms can attempt to be accounted for in statistical analysis

- Many Types:
  - Selection Bias
  - Recall Bias
  - Funding Bias
  - **Attrition Bias: Different rate of loss to follow-up between 2 groups**
    - **May happen when the outcome in question limits a patient's ability to come in for follow-up appointments**
  - Confounding Bias
  - Lead-Time bias

Bias

- Flaw in studies that leads to erroneous results
  - Should be minimized in study design
  - Some forms can attempt to be accounted for in statistical analysis

- Many Types:
  - Selection Bias
  - Recall Bias
  - Funding Bias
  - Attrition Bias
  - **Confounding Bias: Associated risk factor in one of the study groups is related to the outcome**
    - **Can be controlled in study design or statistical analysis**
      - **Groups matched for particular confounders**
      - **Exclude subjects with the confounding factor**
      - **Use of regression models**
  - Lead-Time bias

Bias

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  - Should be minimized in study design
  - Some forms can attempt to be accounted for in statistical analysis

- Many Types:
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  - **Lead-Time bias**
    - **Especially true with chronic diseases**

Sample Question

Research published in a well-respected medical journal studied screening for lung cancer using a new method. The researchers reported that patients who were screened and had lung cancer detected lived longer after diagnosis than people who were not screened.

Which is true?

A) This shows that screening is effective at prolonging survival

B) This may be an example of lead time bias

C) This may be an example of recall bias

D) Well-respected medical journals are always right

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Which is true?

A) This shows that screening is effective at prolonging survival

B) **This may be an example of lead time bias**

C) This may be an example of recall bias

D) Well-respected medical journals are always right

Lead Time Bias

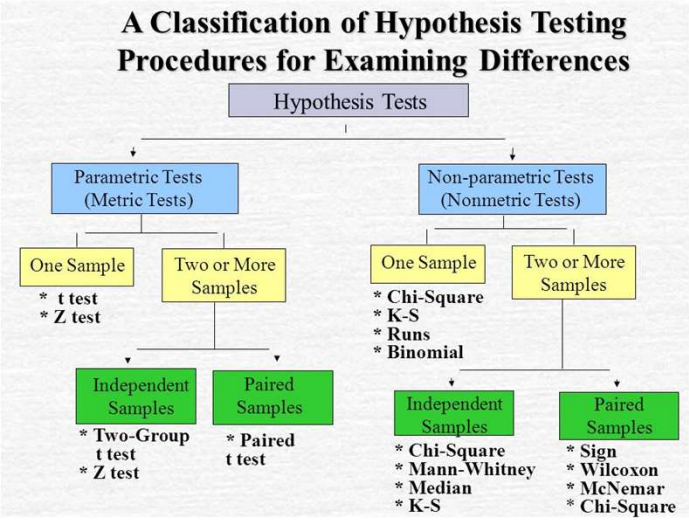
Lead time: length of time between the detection of a disease and its usual clinical presentation and diagnosis.

Lead time bias occurs if testing increases the perceived survival time without affecting the course of the disease.

Lead time versus Length time bias

Lead time bias: earlier detection of disease based on new technology/methods/etc,	• Cologuard or liquid biopsies via blood for colon cancer at one point in time
Length time bias: overestimation of disease numbers based on the indolent nature of the disease state,	• Continual colonoscopies for colon cancer and doing it at regular intervals

Type of Test



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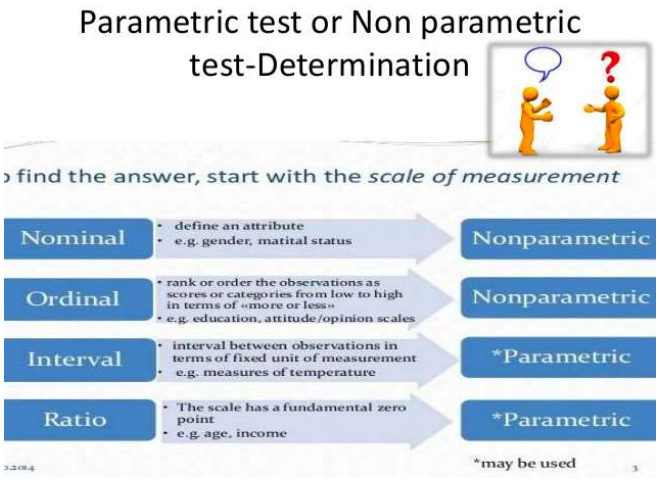

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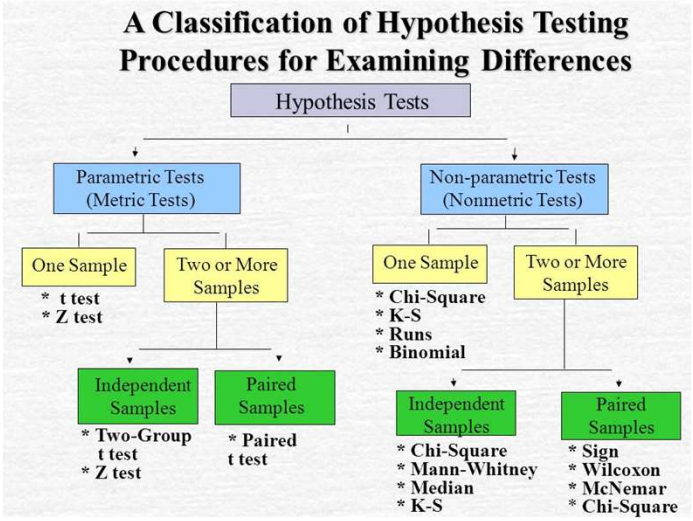
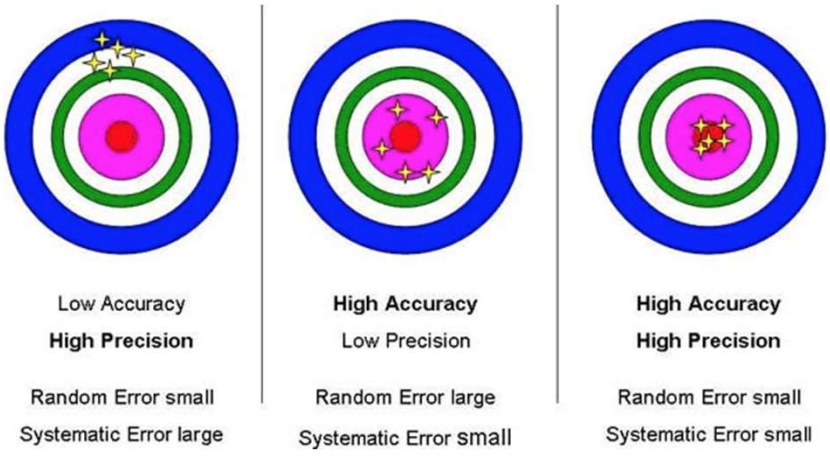


Accuracy (Validity) & Precision (Reliability)

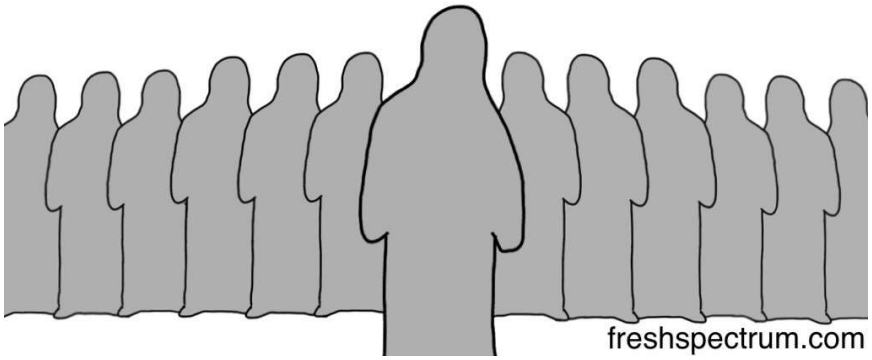
Accuracy (Validity): Ability of a tool to measure what was intended

Precision (Reliability): Ability of a test to give consistent results on repeated trials

Accuracy (Validity) & Precision (Reliability)



I am what is
The default, the status quo
I am already accepted, can only be rejected
The burden of proof is on the alternative
I am the null hypothesis



NH

- **Null Hypothesis:**
  - **Assumption the NO DIFFERENCE exists between 2 groups**
  - **Experiment is trying to REJECT null hypothesis**

- Type I Error (α error): Incorrectly reject null hypothesis
  - Saying there is a difference when there really isn't one
  - α is the probability of making Type I error
    - Set at 0.05, accept 1/20 risk that findings are due to chance

- Type II Error (β error): Incorrectly accepting the null hypothesis
  - Saying there is no difference when one exists
  - β is the likelihood of making Type II error
  - Type **II** error is **too** few patients

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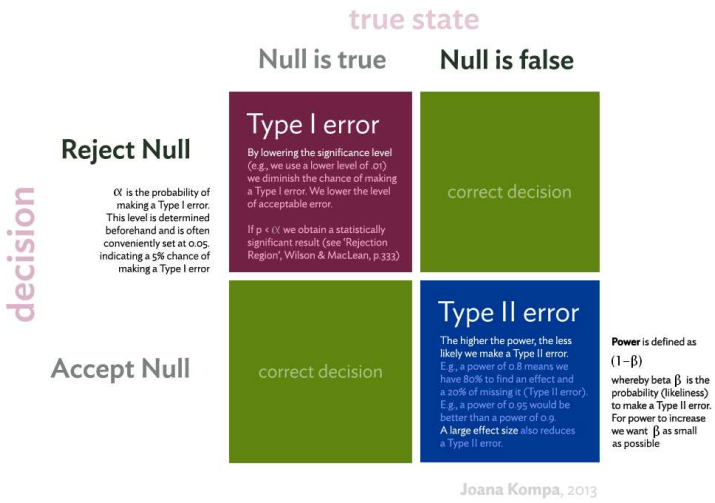
  - **Type II error is too few patients**

NH



P-value

- Risk of false positive result due to chance
  - P-value < 0.01 = highly statistically significant
  - P-value ≤ 0.05 = statistically significant
  - P-value > 0.05 = statistically insignificant
- It does not describe the size of effect, only the strength of the result
- If you have a choice to choose between 0.01 or 0.05, choose 0.01
- Statistical significance does NOT equal clinical significance, regardless of how small the P-value

Sample Question

As part of a quality control study, the HbA1C values of patients with diabetes at two clinics are compared. In a study of 4000 patients, it is found that the mean HbA1C value in group 1 is 7.4% and the mean HbA1C in group 2 is 7.6%. The authors did the correct statistical test and found a p-value of 0.04 for this comparison.

Based on this information, you conclude:

A) Group 1 is significantly different from group 2, reject the null hypothesis
B) Group 1 is not significantly different from group 2, don't reject the null hypothesis
C) Group 1 is not significantly different from group 2, reject the null hypothesis
D) Group 1 is not significantly different from group 2, don't reject the null hypothesis

Sample Question

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P-value

Previous Question: HbA1C 7.4 vs 7.6 with p-value of 0.04

Statistical significance does NOT equal clinical significance, regardless of how small the P-value

Statistical Power

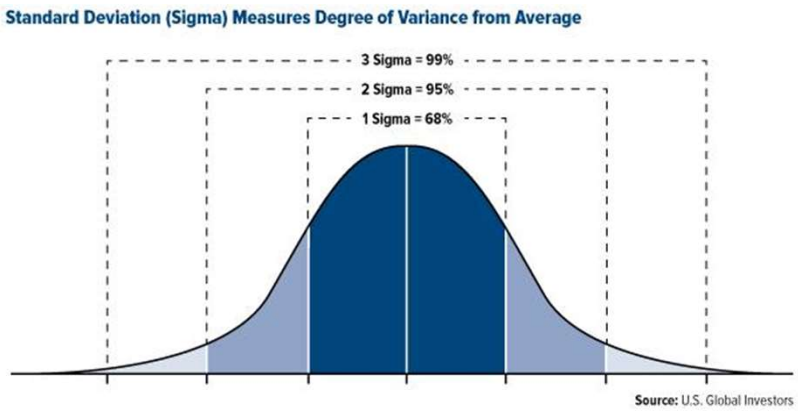
- Likelihood a study correctly disproves the null hypothesis
  - Likelihood of finding a difference between two groups
- Power = 1 - Beta
- Depends on:
  - The effect size (i.e. the amount of difference investigator desires to detect between groups)
  - The level of significance desired (p value)
  - The sample size

Intention To Treat Analysis

Includes all patients randomized to either treatment arm regardless of whether or not they completed the study

Prevents the loss of statistical power that may be encountered with a failure to complete study protocols (dropout) or noncompliance

Standard Deviation



Confidence Interval

Range within which magnitude of effect lies

If alpha 0.05, then results reported as 95% CI

95% of time 'true' result will fall within this range

Statistically significant if:

CI does not include or span the "no effect point" of **ONE or ZERO** for risk ratios like relative risk ratio and odds ratio

Numbers do not **OVERLAP**

Sample Question

In a pharmaceutical study, Group A is the placebo group and Group B is the group that received the actual new drug. Data were gathered on Groups A and B and confidence intervals were calculated.

Which of the following group comparisons are statistically significantly different?

A) Group A CI 30-46% and Group B 44-48%
B) Group A CI 10-30% and Group B 44-88%
C) Group A CI 0.1-0.3% and Group B 0.2-0.4%
D) Group A CI 88-90% and Group B 88-90%
E) None of the above is statistically different

Sample Question

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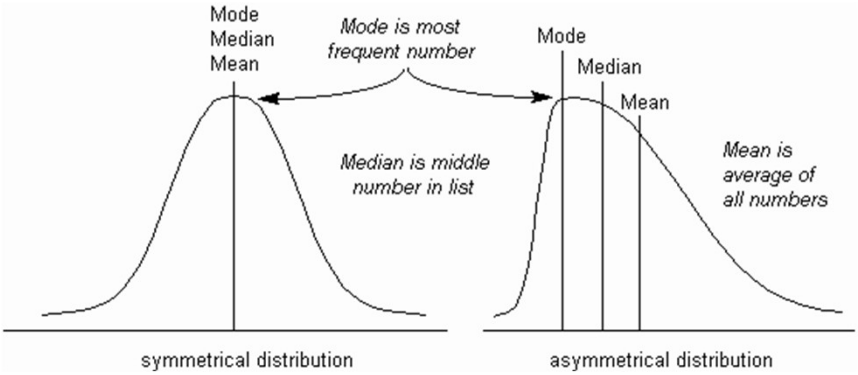
STATISTICS

MMM



MMM

For example: 3 4 7 8 8

Mean =

Median =

Mode =

MMM

For example: 3 4 7 8 8

Mean = (3+4+7+8+8)/5 = 6

Median = 7

Mode = 8

Definitions

DICHOTOMOUS TESTS

A test which gives either a positive or a negative result is called a "dichotomous test." The probabilities involved in such a test may be summarized in the form of a table which is sort of a combination of a Punnett square and Bayesian probability.

	Actual presence of *disease*	**Actual absence of *disease***		
Positivity of *test*	A = true positives	B = false positives	A+B = total number of positive tests	$\frac{A}{A+B}$ = predictive value of positive test
Negativity of *test*	C = false negatives	D = true negatives	C+D = total number of negative tests	$\frac{D}{D+C}$ = predictive value of negative test
	A+C = total number of people who actually have the disease	B+D = total number of people who actually do not have the disease	A+B+C+D = total	
	$\frac{A}{A+C}$ = sensitivity	$\frac{D}{D+B}$ = specificity		

Analysis of the probabilities involves the concepts of sensitivity, specificity and predictive value.

Definitions

DICHOTOMOUS TESTS

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	A+C = total number of people who actually have the disease	B+D = total number of people who actually do not have the disease	A+B+C+D = total	
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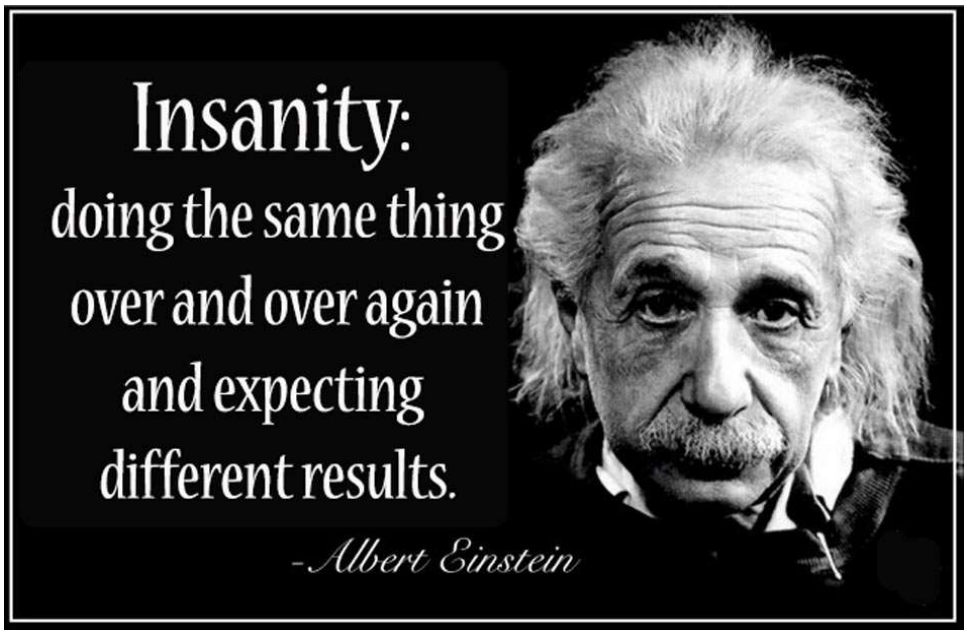


	Sensitivity	Specificity
Definition	Proportion of patients with a disease who test positive	Proportion of patients without the disease who test negative
100% (1.0) Means	The test correctly identify every person who has the target disorder	The test correctly identify every person who does not have the target disorder
Statistical Outcome	True Positive	True Negative
Ideal Test Result	Negative Test Result	Positive Test Result
Test Interpretation	They are definitely not positive → They DON'T have it	They are definitely not negative → They DO have it
The Rule	Rule Out (SnOut)	Rule In (SpIn)

Predictive value of positive test $= \frac{A}{A+B} = \frac{\text{number of true positive tests}}{\text{total number of positive tests}}$

The **predictive value of a positive test** is the percent of positive test results that are true positives (i.e. the number of true positives divided by the total number of positives).

Knowing the **predictive value of a positive test** answers the following question: Given a positive test result, what is the probability that it is correct (i.e. that the positive result is a true positive)?

The lower the prevalence, the lower the predictive value of a positive test.

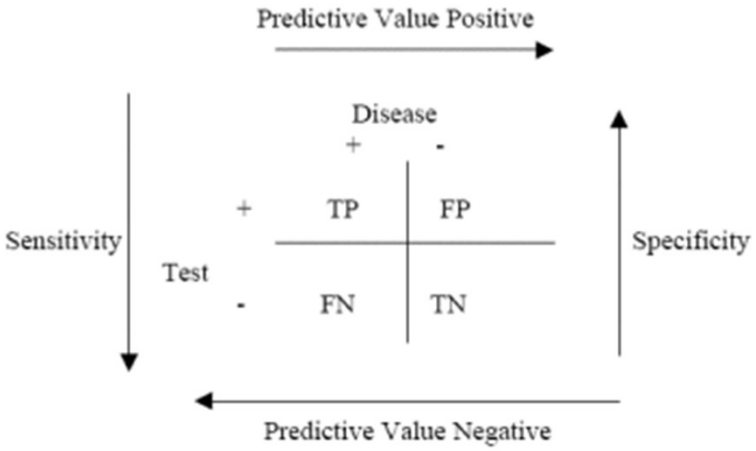
In class when the professors speak of "predictive value," they're usually speaking of the "predictive value of positive tests."

Predictive value of negative test $= \frac{D}{D+C} = \frac{\text{number of true negative tests}}{\text{total number of negative tests}}$

The **predictive value of a negative test** is the percent of negative test results that are true negatives (i.e. the number of true negatives divided by the total number of negatives).

Knowing the **predictive value of a negative test** answers the following question: Given a negative test result, what is the probability that it is correct (i.e. that the negative result is a true negative)?

The lower the prevalence, the higher the predictive value of a negative test.

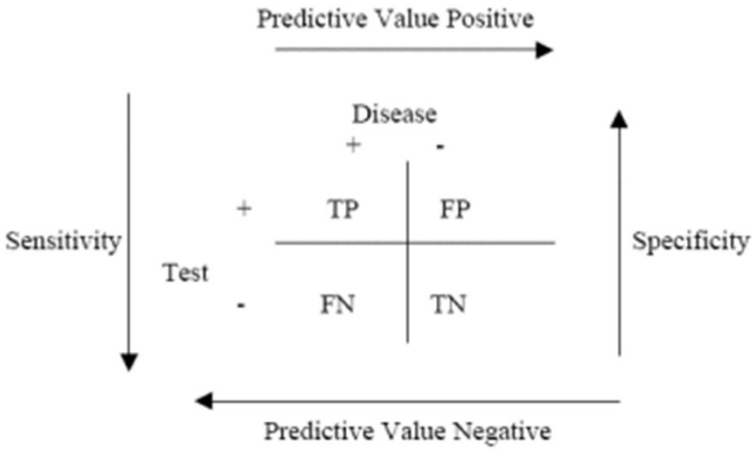


Sample Question

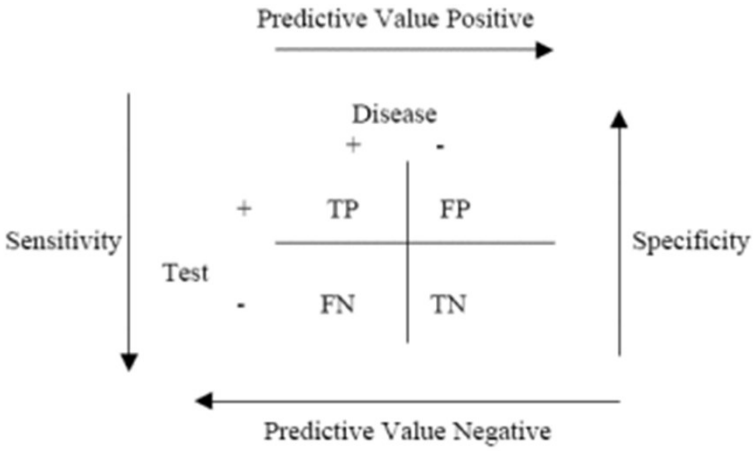
Below are some hypothetical results from women who had a mammogram and the incidence with breast cancer.

- What is the sensitivity of the mammogram?
- What is the specificity of the mammogram?

	Breast Cancer (Disease)			
Mammogram (Test)		(+)	(-)	
	(+)	70	180	
	(-)	30	720	



	Breast Cancer			
Mammogram		(+)	(-)	
	(+)	70	180	PPV = 70/250 = 28 %
	(-)	30	720	NPV =720/750 = 96%
	Total	100	900	Prevalence = 100/1000 = 10%
		Sensitivity = 70/100 = 70%	Specificity = 720/900 = 80%	



Prevalence/Incidence

- Prevalence = # of EXISTING cases
- Incidence = # of NEW cases
- Example: In a Medicaid Insurance population of 15,000 children, 200 were diagnosed with autism, bringing the total of children with autism to 500.
  - Incidence = ?
  - Prevalence = ?

Prevalence/Incidence

- Prevalence = # of EXISTING cases
- Incidence = # of NEW cases
- Example: In a Medicaid Insurance population of 15,000 children, 200 were diagnosed with autism, bringing the total of children with autism to 500.
  - Incidence = 200/15000 = 1.3%
  - Prevalence = 500/15000 = 3.3%

Sample Question

Cervical cancer is a disease in which early detection can make a great difference in halting disease progression. One screening procedure for this disease is the Pap smear. To assess the competency of technicians who read the Pap smear slides, a local lab checked their technicians work against patient records.

A total of 1000 Pap smears were read. Of these, 100 patients had cervical abnormalities based on biopsy (gold standard). Of this group, 75 had abnormal (positive) Pap smears and 25 had negative pap smears. There were 900 women without disease. Of these 900 women, 200 had positive Pap smears and 700 negative Pap smears.

Using the data above, which of the following is true about this survey of Pap smear technicians?

A) NPV is 20%
B) PPV is 15%
C) Sensitivity of Pap test is 75%
D) Specificity of Pap test is 98%
E) Prevalence of cervical cancer in this sample is 7.5%

	Disease			
TEST		(+)	(-)	
	(+)			
	(-)			

	Disease			
TEST		(+)	(-)	
	(+)	75	200	
	(-)	25	700	
	Total	100	900	

	Disease			
TEST		(+)	(-)	
	(+)	75	200	PPV = 75/275 = 27%
	(-)	25	700	NPV =700/725 = 97%
	Total	100	900	Prevalence = 100/1000 = 10%
		Sensitivity = 75/100 = 75%	Specificity = 700/900 = 78%	

Sample Question

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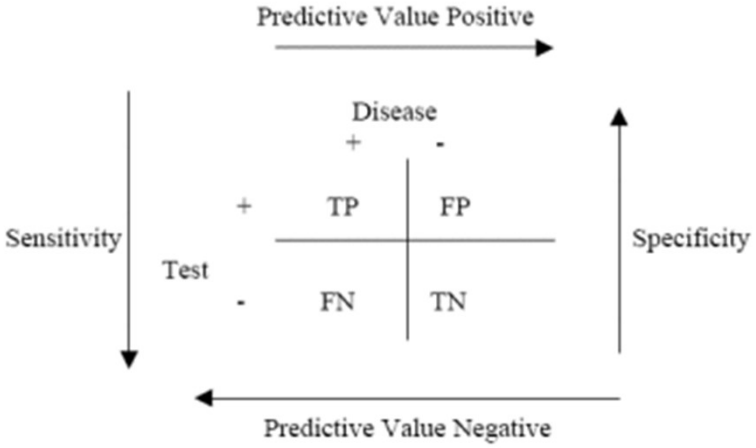


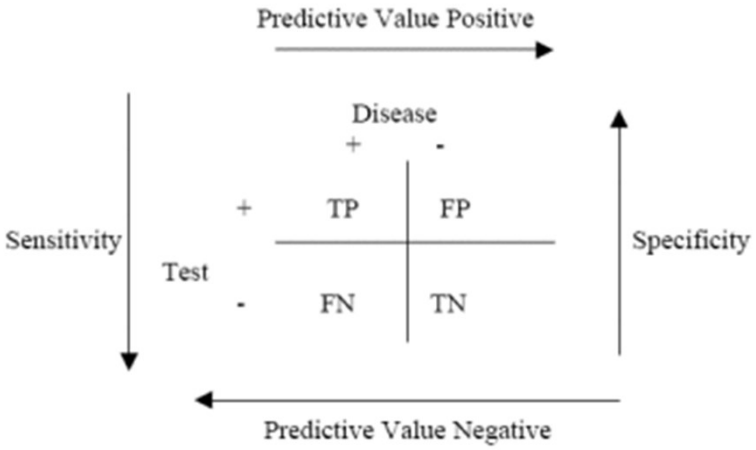
Sample Question

To evaluate the performance of a new diagnostic test, the developer checks it out on 100 known cases of the disease for which the test was designed, and on 200 controls known to be free of the disease. 90 of the cases yield positive tests, as do 30 of the controls. Based on these data, what is the specificity?

A. 10%
B. 15%
C. 25%
D. 85%
E. 90%

	Disease			
TEST		(+)	(-)	
	(+)			
	(-)			

	Disease			
TEST		(+)	(-)	
	(+)	90	30	
	(-)	10	170	
		100	200	



	Disease			
TEST		(+)	(-)	
	(+)	90	30	PPV = 90/120 = 75%
	(-)	10	170	NPV =170/180 =94.4%
	Total	100	200	Prevalence = 100/300 = 33.3%
		Sensitivity = 90/100 = 90%	Specificity = 170/200 = 85%	

Sample Question

To evaluate the performance of a new diagnostic test, the developer checks it out on 100 known cases of the disease for which the test was designed, and on 200 controls known to be free of the disease. 90 of the cases yield positive tests, as do 30 of the controls. Based on these data, what is the specificity?

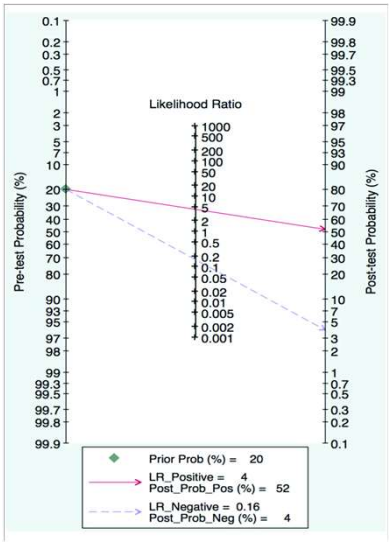
A. 10%
B. 15%
C. 25%
D. 85%
E. 90%

Sample Question

To evaluate the performance of a new diagnostic test, the developer checks it out on 100 known cases of the disease for which the test was designed, and on 200 controls known to be free of the disease. Ninety of the cases yield positive tests, as do 30 of the controls. Based on these data, what is the specificity?

A. 10%
B. 15%
C. 25%
D. 85%
E. 90%

Pre/Post Test Probability (Fagan Nomogram)



- Likelihood ratios provide information about diagnostic tests by combining sensitivity and specificity of the test into a single measure.
- Can be calculated as: sensitivity/(1-specificity)

Risk Reduction (Relative vs. Absolute)

Relative risk reduction (RRR) = (1-RR)/RR

- (Risk in control group) – (risk in treatment group)
  - /(Risk in control group)
- Range from 0 (no reduction) to 100% (full reduction)

Absolute risk reduction (ARR)

- (Risk in control group) – (risk in treatment group)
- Can be expressed as a proportion or a percentage
- Statistically significant if does not include zero

Mortality Trials

Trial	Placebo	Treatment	RRR	ARR
A	50%	5%	(50-5/50): 90%	50% - 5% = 45%
B	5%	0.5%	(5-0.5/5): 90%	5 – 0.5 = 4.5%

Which is a better treatment?
A or B?
Is there a better way to know which one is better?

Number Needed to Treat (NNT)

# of patients who would have to receive treatment for 1 of them to benefit	Calculated as 100/ARR
If treatment group has more negative results, than the result is Number Needed to Harm (NNH)	Same Calculation
http://www.thennt.com/thennt-explained/	

Number Needed to Treat (NNT)

- If they ask you about NNT:

1. BIG NUMBER – SMALL NUMBER= X
2. DIVIDE 100 by X

Sample Question

One common and debilitating complication of diabetes is neuropathy. In a study of patients with diabetes, one group had routine therapy and an experimental group had intensive therapy. The first group, routine therapy, had 10% of patients develop neuropathy. The second group, had 2% of patients develop neuropathy.

Using the data above, how many patients with diabetes need to be treated with intensive therapy to prevent the development of one case of neuropathy?

A) 10
B) 11
C) 8
D) 12.5
E) 25.5

Use of Formula

BIG NUMBER – SMALL NUMBER= X

- 10 – 2 = 8

DIVIDE 100 by X

- 100/8 = 12.5

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Using the data above, how many patients with diabetes need to be treated with intensive therapy to prevent the development of one case of neuropathy?

A) 10
B) 11
C) 8
D) 12.5
E) 25.5

ARR = 10% - 2% = 8%
NNT = 100/ARR = 100/8 = 12.5

Sample Question

The anticlotting properties of aspirin are well studied. In a trial studying the long-term outcome of stroke patients, 1% of patients on long term aspirin therapy developed new onset of strokes and 50% of patients without aspirin therapy developed new strokes.

Using the data above, how many stroke patients need to be treated with aspirin therapy to prevent one new stroke?

A) 2
B) 8
C) 10
D) 12
E) 25

Use of Formula

BIG NUMBER – SMALL NUMBER= X

- 50 – 1 = 49

DIVIDE 100 by X

- 100/49 ~ 2

Sample Question

The anticlotting properties of aspirin are well studied. In a trial studying the long-term outcome of stroke patients, 1% of patients on long term aspirin therapy developed new onset of strokes and 50% of patients without aspirin therapy developed new strokes.

Using the data above, how many stroke patients need to be treated with aspirin therapy to prevent one new stroke?

A) **2**
B) 8
C) 10
D) 12
E) 25

ARR = 50 – 1 = 49
NNT = 100/ARR = 100/ 49 = ~2

Mortality Trials Revisited

Trial	Placebo	Treatment	RRR	ARR
A	50%	5%	(50-5/50): 90%	50% - 5% = 45%
B	5%	0.5%	(5-0.5/5): 90%	5 – 0.5 = 4.5%

Will NNT Help?

Mortality Trials Revisited

Trial	Placebo	Treatment	RRR	ARR	NNT
A	50%	5%	(50-5/50): 90%	50% - 5% = 45%	100/45: ~2
B	5%	0.5%	(5-0.5/5): 90%	5 – 0.5 = 4.5%	100/4.5: ~22

Treatment A: For every 2 patients on therapy, 1 life would be saved
Treatment B: For every 22 patients on therapy, 1 life would be saved

Which would you choose?

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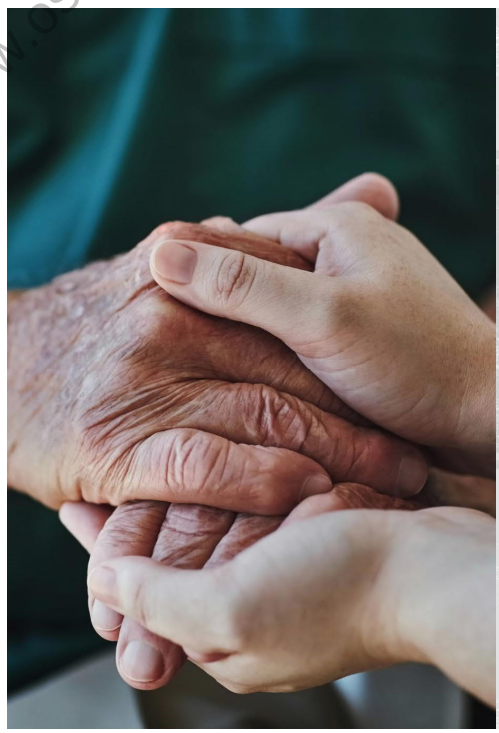

PATIENT SAFETY, QUALITY IMPROVEMENT, ETHICS, PROFESSIONALISM, AND CREDENTIALING

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THE AMERICAN BOARD OF SURGERY

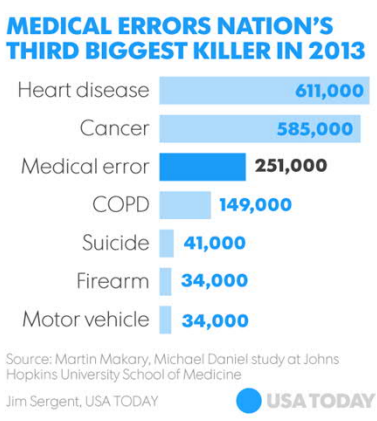
SURGICAL CRITICAL CARE

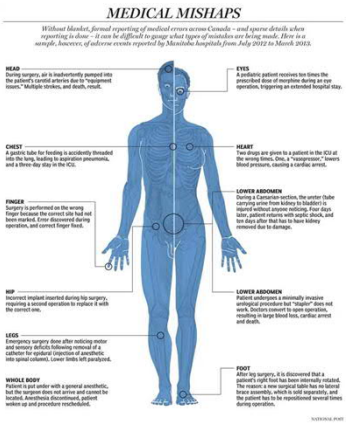
CONTENT OUTLINE FOR THE CERTIFYING EXAMINATION (CE) AND CONTINUOUS CERTIFICATION (CC) READMISSIBILITY EXAMINATION

J. Trauma	10%
K. Thermal Injury	4%
L. Monitoring, Bioengineering, and Biostatistics	6%
M. Life-Threatening Pediatric Conditions	2%
N. Principles and Techniques of Administration and Management	3%
O. Pharmacology, Pharmacokinetics, and Drug Metabolism in Critical Illness	2%
P. Ethical and Legal Aspects in Surgical Critical Care Medicine	2%

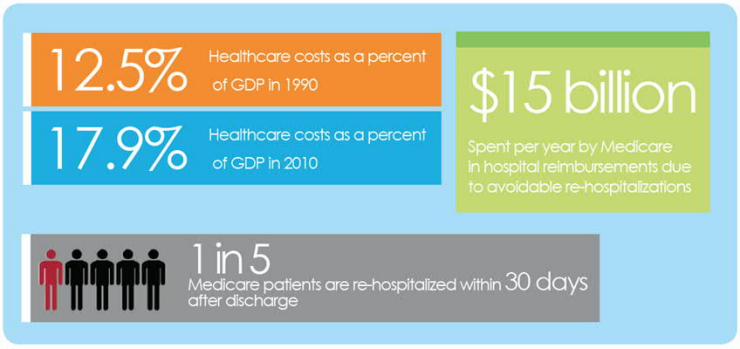
PATIENT SAFETY & QUALITY IMPROVEMENT

WHY IS THIS IMPORTANT



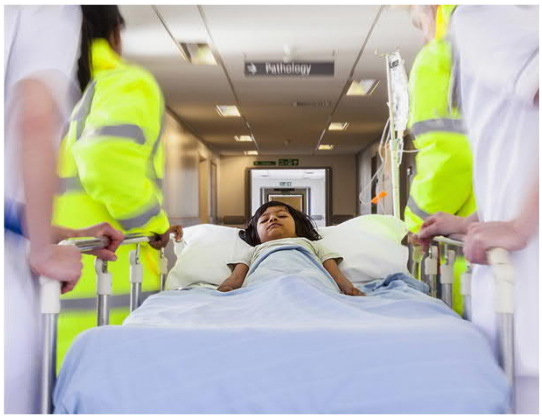


WHY IS THIS IMPORTANT



Growing trend for decreased reimbursement based on rehospitalizations or for hospital acquired conditions.

WHY IS THIS IMPORTANT

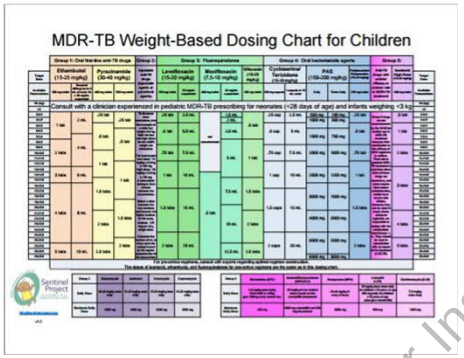


- Adverse events occur in 1% of pediatric hospitalizations
  - 60% of these are preventable
- Adverse event rates are increasing every year but could be an example of lead-time bias
  - Increased ability to detect rather than an actual increase in incidence of events

CHILDREN ARE NOT LITTLE ADULTS

- Risk for adverse drug events is 3x higher in hospitalized children than adults
  - Weight based dosing and miscalculations
  - Dosing errors involving compounding adult prepackaged medications

Children's & Junior Strength ACETAMINOPHEN				
Tempra®, Tylenol®, Panadol®				
		Infant and Children's Suspension Liquid and Elixir	Children's Chewable Tablets	Junior Strength Chewable Tablets/Caplets
DOSE		160mg/5ml	80mg each	160mg each
WEIGHT		Syringe	Tablet	Tablet/Caplet
Pounds	Kilos			
6 -11 lbs.	2.7 - 5.4	1.25ml		
12-17 lbs.	5.5 - 7.9	2.5ml		
18-23 lbs.	8.0 - 10.9	3.75ml		
24-35 lbs.	11 - 15.9	5ml	2	
36-47 lbs.	16 - 21.9	7.5ml	3	
48-59 lbs.	22 - 26.9	10ml	4	2
60-71 lbs.	27 - 31.9	12.5ml	5	2 1/2
72-95 lbs.	32 - 43.9	15ml	6	3
96 lbs & over	44.0 & over			4



CHILDREN ARE NOT LITTLE ADULTS

Special issues for children- relevant to medication safety

1. Weight based dosing (and weights change frequently)
2. Organ system development is variable, affecting metabolizing and excretion
3. Meds mixed by pharmacists or nurses at time of use
4. Pediatric meds often need to be diluted from adult formulations
5. Many pediatric medications come in multiple formulations
6. Children less likely to recognize/communicate an error or harm

Premature infants are particularly complex; they have immature functioning of the hepatic and renal systems that increases the complexity of drug dosing, as well as extended lengths of stay and continually changing weights. These factors, among others, make the neonatal intensive care unit a particularly error-prone setting.

DEFINITIONS

MEDICAL ERROR

Failure to complete a planned action as intended, or the use of a wrong plan to achieve an aim

Medication Error → Most common type

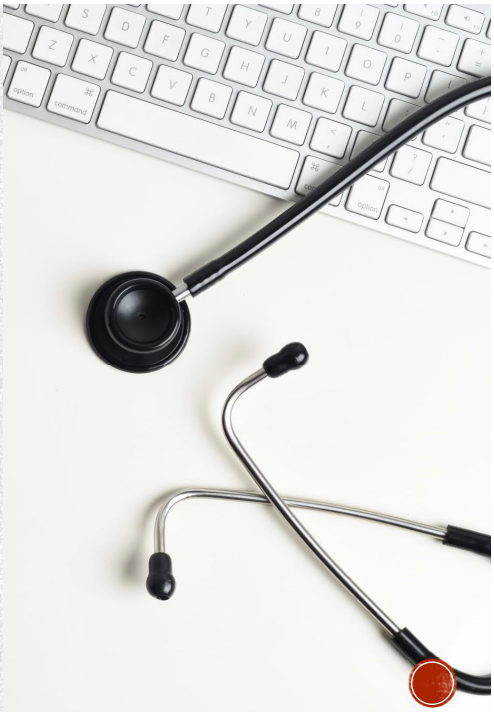
Errors in:
- Ordering
- Transmissions of E-scripts
- Preparing, labeling, or administering the medication

MEDICATION ERROR

- Any error in the process of:
  - Ordering
  - Transcribing
  - Dispensing
  - Administering
  - Monitoring a medication
- Caveats: Medication errors are not defined by outcome and may not result in an actual adverse drug event

ADVERSE EVENTS

- Injury caused by medical management rather than the underlying disease or condition
- Adverse events causes harm to the patient
  - Most medical errors do not lead to the adverse events

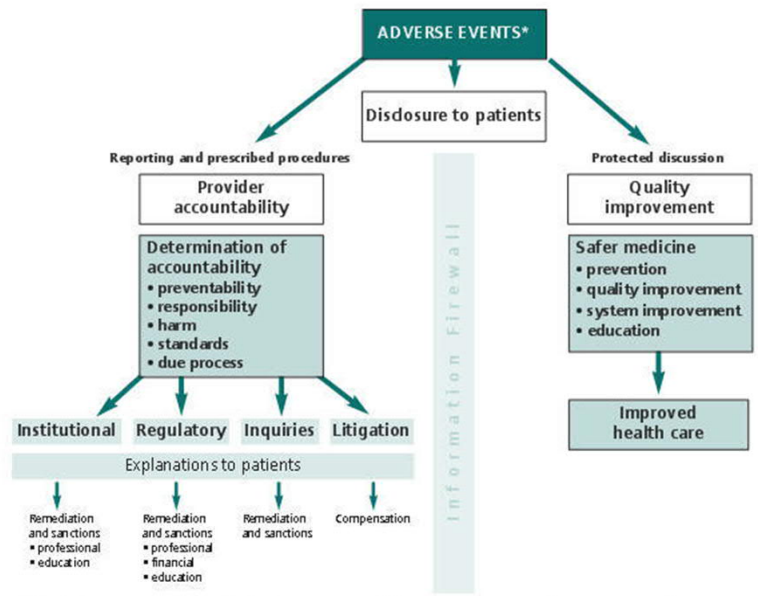


ADVERSE DRUG EVENT

Injury resulting from the use of drug

May result from medication error (i.e. unforeseen drug reaction)

Diagram A — The response to adverse events



* While all adverse events need to be disclosed to patients, not all adverse events require further review and analysis.

NEAR MISS EVENT

- Medical error places a patient at risk for injury without actually resulting an injury
- 2 types:
  - Intercepted
  - Non-intercepted: error reaches the patient but causes no harm

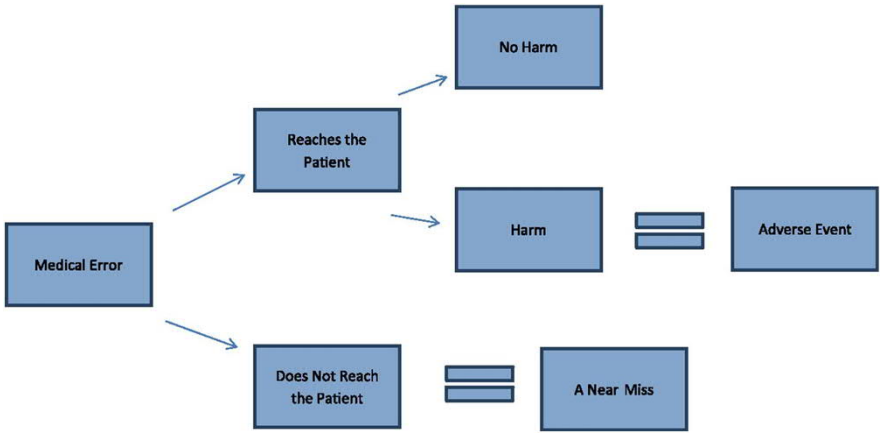
SENTINEL EVENT

Actual or potential death or serious injury as a result of medical error

SENTINEL EVENTS – ROOT CAUSES

- Communications/orientation/training
- Staffing/organization culture
- Availability of information
- Competency/credentialing
- Environment safety/security
- Leadership/continuum of care/care planning

PUTTING IT ALL TOGETHER



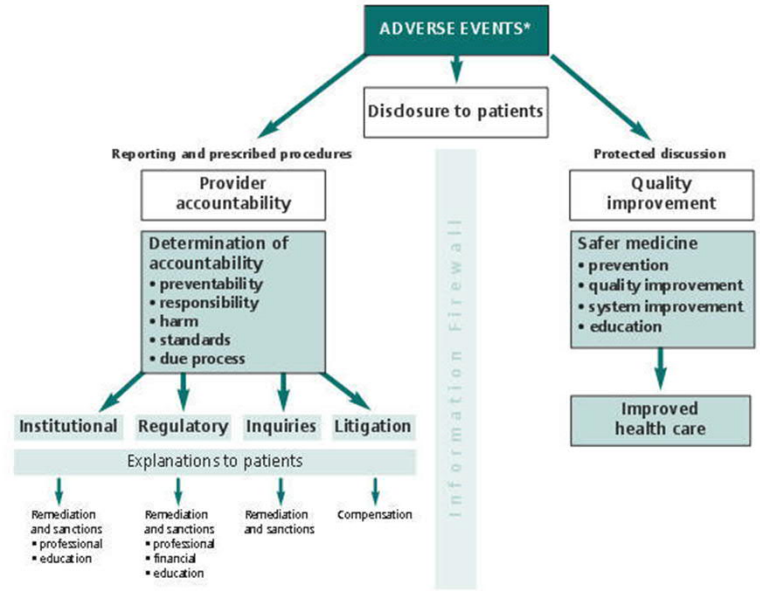
Medscape® www.medscape.com

Term	Definition
Medical error	Failure of a planned action to be completed as intended or the use of a wrong plan to achieve an aim.
Serious medical error	A medical error that causes harm (or injury) or has the potential to cause harm. Includes preventable adverse events, intercepted serious errors, and nonintercepted serious errors. Does not include trivial errors with little or no potential for harm or nonpreventable adverse events.
Intercepted serious error	A serious medical error that is caught before reaching the patient.
Nonintercepted serious error	A serious medical error that is not caught and therefore reaches the patient but because of good fortune or because the patient had sufficient reserves to buffer the error, it did not cause clinically detectable harm.
Adverse event	Any injury due to medical management, rather than the underlying disease. Examples of an injury would be a rash caused by an antibiotic, deep vein thrombosis following accidental omission to continue prophylactic subcutaneous heparin orders on transfer to the critical care unit, and ventricular tachycardia due to placement of a central venous catheter tip in the right ventricle.
Nonpreventable adverse event	Unavoidable injury due to appropriate medical care.
Preventable adverse event	Injury due to a nonintercepted serious error in medical care.

Source: Crit Care Med © 2005 Lippincott Williams & Wilkins

PROVIDER ACCOUNTABILITY

Diagram A — The response to adverse events



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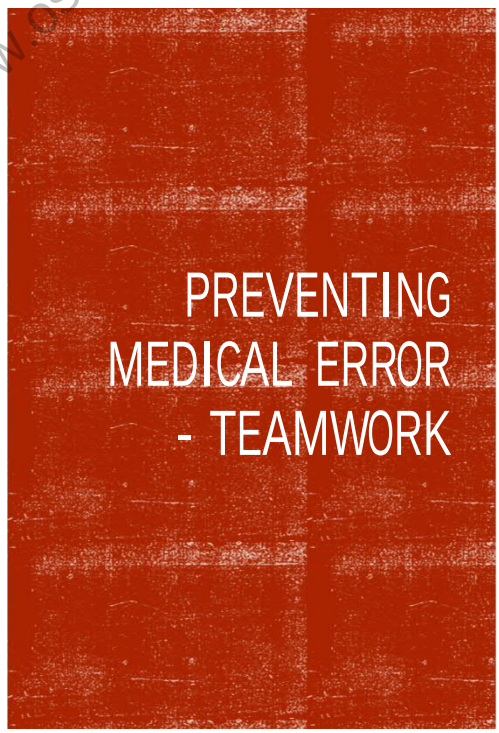
WHY DO MEDICAL ERRORS HAPPEN?

- Error is likely in certain fields because of task complexity and routinely working under risky conditions
- All humans make errors: there is baseline rate of errors
- There should be multiple safeguards or barriers to prevent harm-high reliability organizations (HROs)
- Alignment of a series of mishaps at different levels – Swiss cheese model
- Aim is to build a better system of checks and balances to limit harm

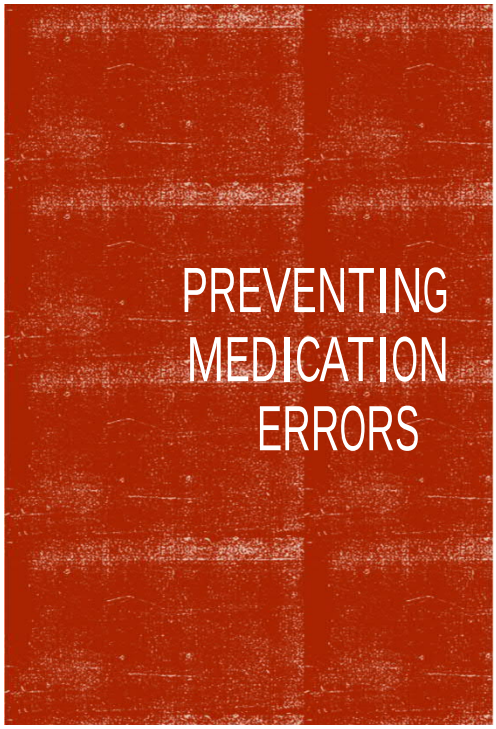
WHY DO MEDICAL ERRORS HAPPEN

- Workload fluctuations
- Interruptions
- Fatigue
- Multi-tasking
- Failure to follow-up
- Poor handoffs
- Communication
- Hidden agendas
- Complacency
- Task fixation
- High risk phase
- Break from protocol



Patient safety requires that all team members, the patient, and family members are informed, attentive, and communicate openly with one another.

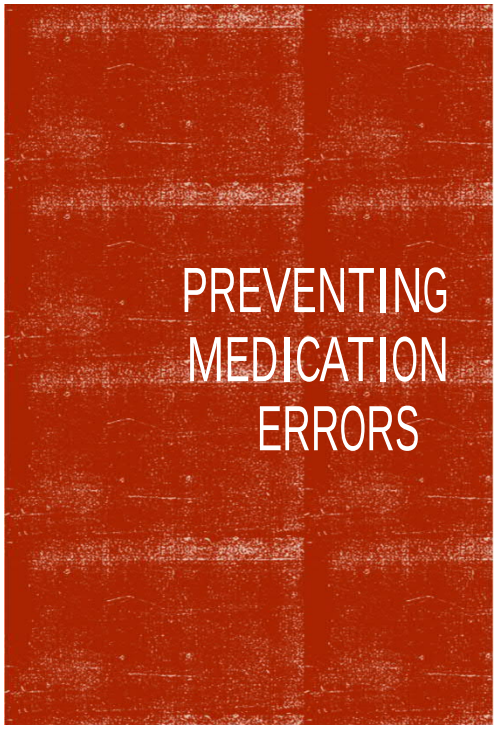
- Improves patient & process outcomes
- Increase patient & staff satisfaction
- Reduce malpractice claims



- Identifying current medications including OTC and herbal medications
- Make sure to stay away from medications on allergy list
- Use of EMR → can update medications and decrease written orders
- Avoid obscure and confusing abbreviations

DO NOT USE LIST OF ABBREVIATIONS

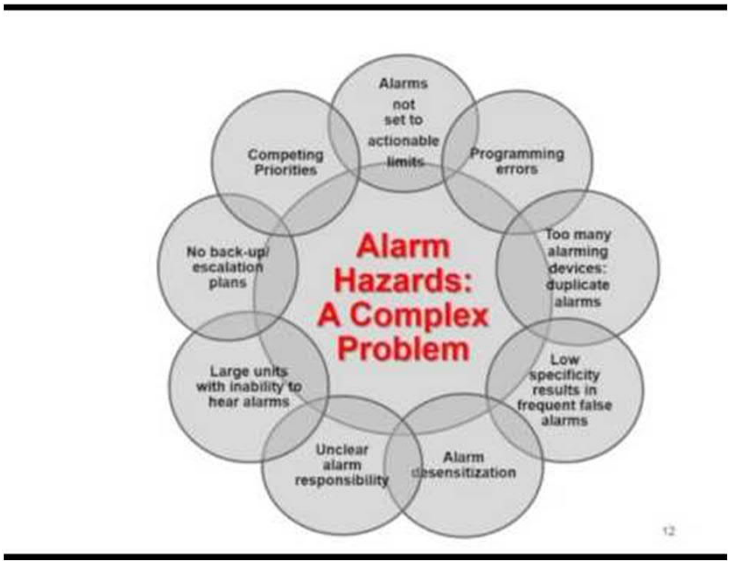
Abbreviation	Problem
MS	Can mean magnesium sulfate or morphine sulfate
MSO_4, $MgSO_4$	Can be confused
Q.D., q.d., qd	Can be confused
Q.O.D. q.o.d, qod, Q.I.D., q.i.d, qid	
IU	Can be mistaken for IV or 10
u	Can be mistaken for zero or 4
Lack of leading zero .X	Decimal point can be missed
Trailing zero X.0	Decimal point can be missed



- Weight based dosing for pediatric patients
- Decimals: 0's precede and never follows
  - i.e., 0.5 but not 5.0 (can be confused with 50)
- For medications that sound alike → diagnosis should be included for prescriptions and verbal orders
  - i.e., methadone vs methylphenidate, Iodine (NSAID) vs codeine, clonidine vs Klonopin

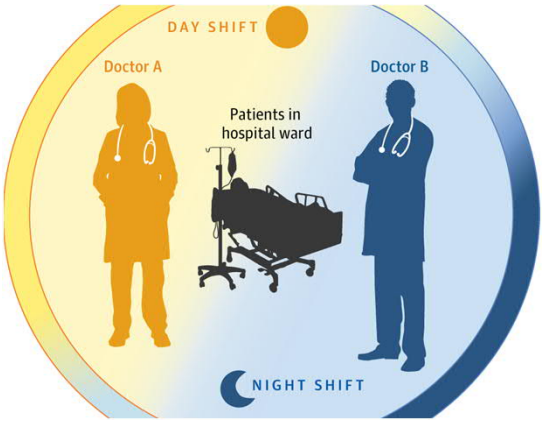
PREVENTING MEDICATION ERRORS: FOLLOW THE RIGHTS OF MEDICATION ADMINISTRATION

- Right patient
- Right drug
- Right dose
- Right time
- Right route
- Right recording
- Right assessment
- Right education
- Right evaluation
- Right to refuse medication



ALARM FATIGUE

- Occurs when a high frequency of alerts results in desensitization to the alarms
- Mitigated by avoiding alarm thresholds that are not set to actionable items



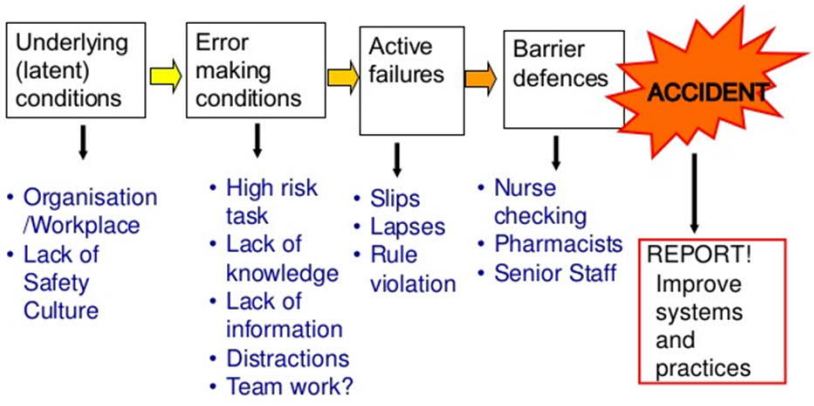
PATIENT HANDOFFS

- Resident duty hour have increased the frequency of patient handoffs
- Standardization of handoffs decreases the risk that important information will be omitted
- Elements that improve handoff quality:
  - Face-to-face communication
  - Using a mnemonic to ensure that all important elements are included,
  - Having the receiving team summarize information provided by the team handing over care.
- Printed patient summary documents can help with the transfer of information during the handoff process; however, these documents generally remain accurate for only a short period.

EMR

- By and large, EMR decreases medication errors
  - Eliminates handwriting issues
  - Available pediatric calculators for weight-based dosing
  - Alerts for drug-drug interactions and allergies
- However, that is not the end-all, be all
  - Pharmacist on rounds or reviewing orders reduced pediatric medication errors
  - Family centered rounds
  - Patient ID bands

Why Medication Errors Happen?



"Swiss Cheese Model" – sometimes the errors get through gaps in processes

Vincent et al, (1998), BMJ 316: p 1154-1157

DETECTING AND REPORTING EVENTS

able 1. Barriers to Disclosure

• Fear of retribution from the patient
• Fear of retribution from colleagues or peers
• Fear of financial judgment
• Fear of conducting the conversation poorly
• Fear of having to handle the patient's emotions
• Belief that the disclosure is unnecessary
• Belief that disclosure is primarily a factual conversation and not a complex interpersonal conversation
• Belief that the outcome is not related to action on the part of the discloser
• Belief that the eventual outcome would have likely occurred witho the error

dapted from ref 10

- Nobody wants to admit or be called out for a medical error
  - But we should make decisions based one what's best for the patient

HUMAN ERROR

- Human error viewed in two ways: person & system approach
- Traditional approach human error focused on the person committing unsafe acts and more punitive
- System approach → humans are fallible, and errors are expected
  - Implementing protections that reduce unwanted variability → change work conditions

Focus on people
- Nurses
- Physicians
- Pharmacists

Views unsafe acts arising from aberrant mental processes
- Forgetfulness
- Inattention
- Poor motivation
- Carelessness
- Negligence
- Reckless

Countermeasures:
- Posters
- Fear
- More procedures
- Discipline
- Threat of litigation
- Retraining
- Naming
- Blaming and shaming

PEOPLE APPROACH

Humans are fallible and errors are expected (even the best organizations)

Errors are consequences not causes

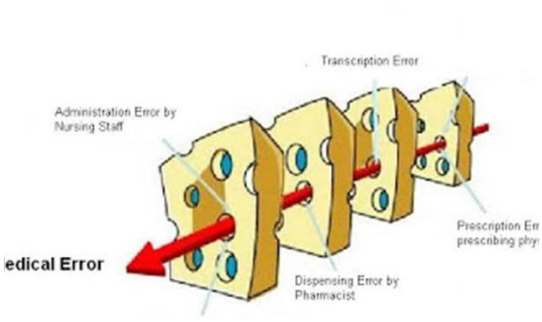
Origin in upstream systemic factors

Countermeasures directed at changing the conditions that humans work under

Central idea is system defenses (HROs)

SYSTEM APPROACH

SWISS CHEESE MODEL



Picture : Swiss - Cheese Model

- Health care system is viewed as a series of slices of Swiss cheese, each with strengths (areas of intact cheese) and weaknesses (holes)
- Each part of the health care system contributes differently to the risk of error.
- When a system is designed to prevent errors, the "holes" will not line up.
- Each layer of care has a unique role in identifying and averting medical errors.

SAMPLE QUESTION

A 3-month-old has been admitted to the hospital with a newly diagnosed ventricular septal defect. She is in early congestive heart failure and digoxin is indicated. After discussing the proper dose with the attending physician, you write an order for the drug. Thirty minutes later the baby vomits and then has a cardiac arrest and dies. You discover that in writing the digoxin order you misplaced the decimal point and the child got 10 times too much digoxin.

What is your duty here?

A. Inform the parents about the mistake and say you are sorry.
B. Do not inform the parents.
C. Do not say sorry.
D. Let the liability insurance company handle everything, because a malpractice lawsuit may follow.

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C. Do not say sorry
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SAMPLE QUESTION - ANSWER

- The correct answer to this question is A. Unfortunately, there was a medical error that caused a bad outcome in the form of a mortality. When you discover the error that has happened, it is the ethical thing to do to disclose the medical error to the parents even if there will be consequences down the road. In this discussion: make sure to describe what happened in facts and not opinions, tell the consequences of the event, tell steps that are being taken to manage the event, tell steps that are being taken to prevent recurrence, and express your sorrow and regret.

Diagram A — The response to adverse events

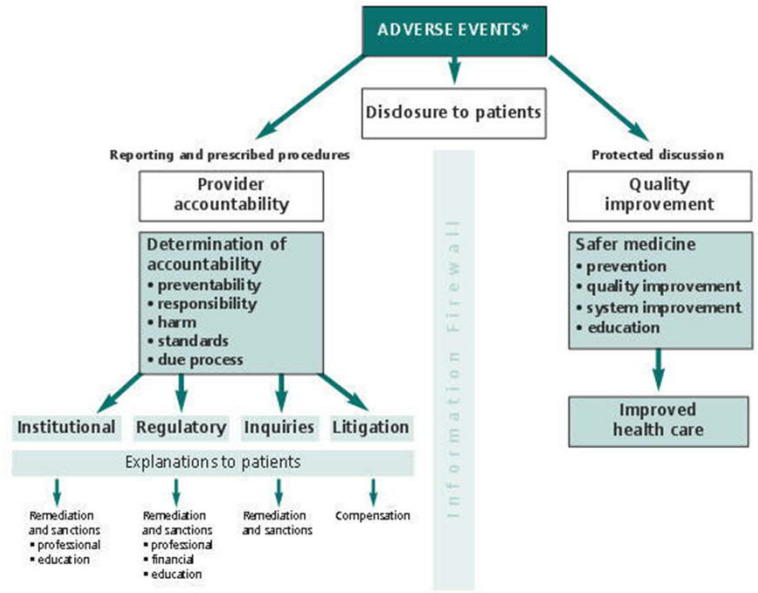

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TABLE 2 Discussing medical errors with a patient

1. Learn all the facts of the case.
2. Have another physician or a nurse present.
3. Choose a quiet place for the discussion where you will not be interrupted.
4. Briefly review the initial assessment of the situation, the plan of care, and the unanticipated change.
5. Express empathy for the patient and family.
6. Ask the patient and family about their emotions, particularly their anger, and listen carefully to them.
7. Review the patient's and family's understanding of the sequence of events as you related it to them, providing explanations or correcting misunderstandings as needed.
8. Afterward, document details of the meeting, as well as plans for future meetings with the patient and family.

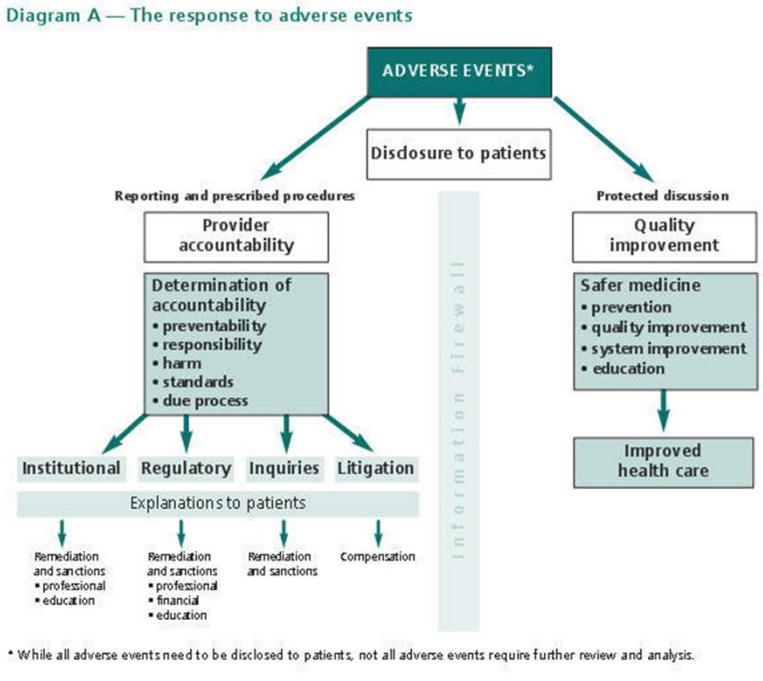
TABLE 1 Resources on managing medical errors

Licensure and accreditation

- (Video) Disclosing Medical Errors to Patients: Recent Developments, Future Directions (March 4, 2009) http://www.youtube.com/watch?v=DtjCGyKawMc.
- University of Washington School of Medicine Tough Talk: A Toolbox for Medical Educators http://depts.washington.edu/toolbox/errors.html.
- U.S. Department of Health and Human Services Agency for Healthcare Research and Quality Health Care Innovations Exchange Innovation Profile: Full Disclosure of Medical Errors Reduces Malpractice Claims and Claim Costs for Health System. http://www.innovations.ahrq.gov/content.aspx?id=2673.
- Cook AF and Hoas H. Ethics Conflicts in Rural Communities: Recognizing and Disclosing Medical Errors. In: Nelson WA. *Handbook for Rural Health Care Ethics: A Practical Guide for Professionals*. Lebanon, NH: Dartmouth College Press, published by the University Press of New England; 2009. http://geiselmed.dartmouth.edu/cfm/resources/ethics/chapter-12.pdf.
- Institute of Medicine. *To Err Is Human: Building a Safer Health Care System*. Washington, DC: National Academies Press; 1999.
- Institute of Medicine. *Preventing Medication Errors*. Washington, DC: National Academies Press; 2006.

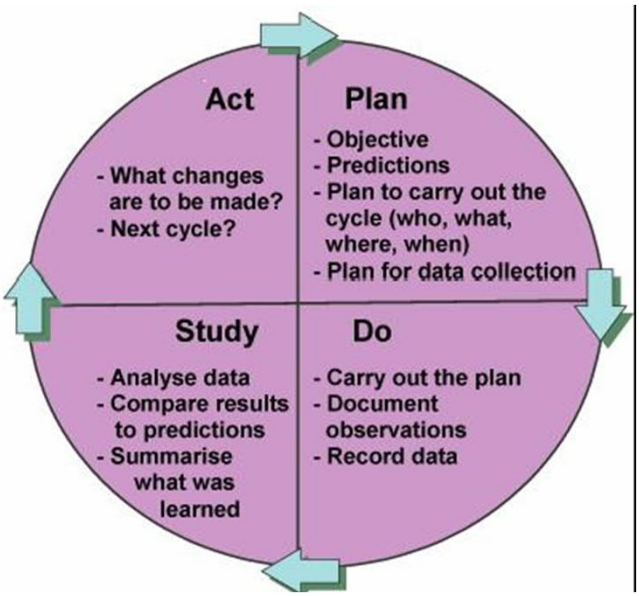
ADVERSE EVENTS - PROVIDERS

- Immediately after any adverse event resulting in harm to a patient, counseling services should be made available to the providers involved
- Providers involved in an adverse event resulting in harm to a patient may become second victims.
- Counseling services should be offered to health-care providers after an adverse event to ensure healthy coping.
- Debriefing sessions may offer a safe space in which members of the medical team can begin to process their feelings after an adverse event.

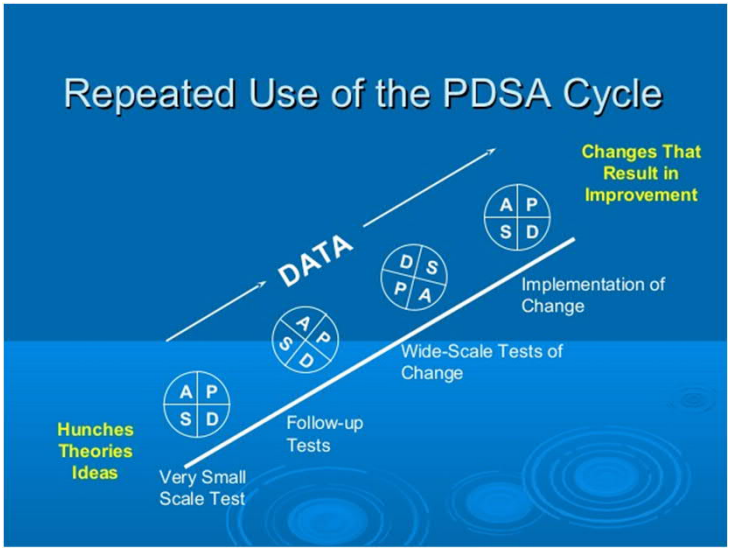


ELEMENTS OF SUCCESSFUL PSQI

- Peer review in hospital settings has traditionally been a retrospective process.
  - Generally prompted by an adverse outcome
  - Peer reviewer was usually a colleague or competitor practicing in the same institution
  - Problems with lack of appropriate expertise, reluctance to criticize, fear of social or legal repercussions
- Measuring Performance
  - Selecting quality indicators: a measurable dimension (medical event, procedure, diagnosis, or outcome)
- Establishing standards
  - Can be rate based and numerical



IMPLEMENTING A CHANGE



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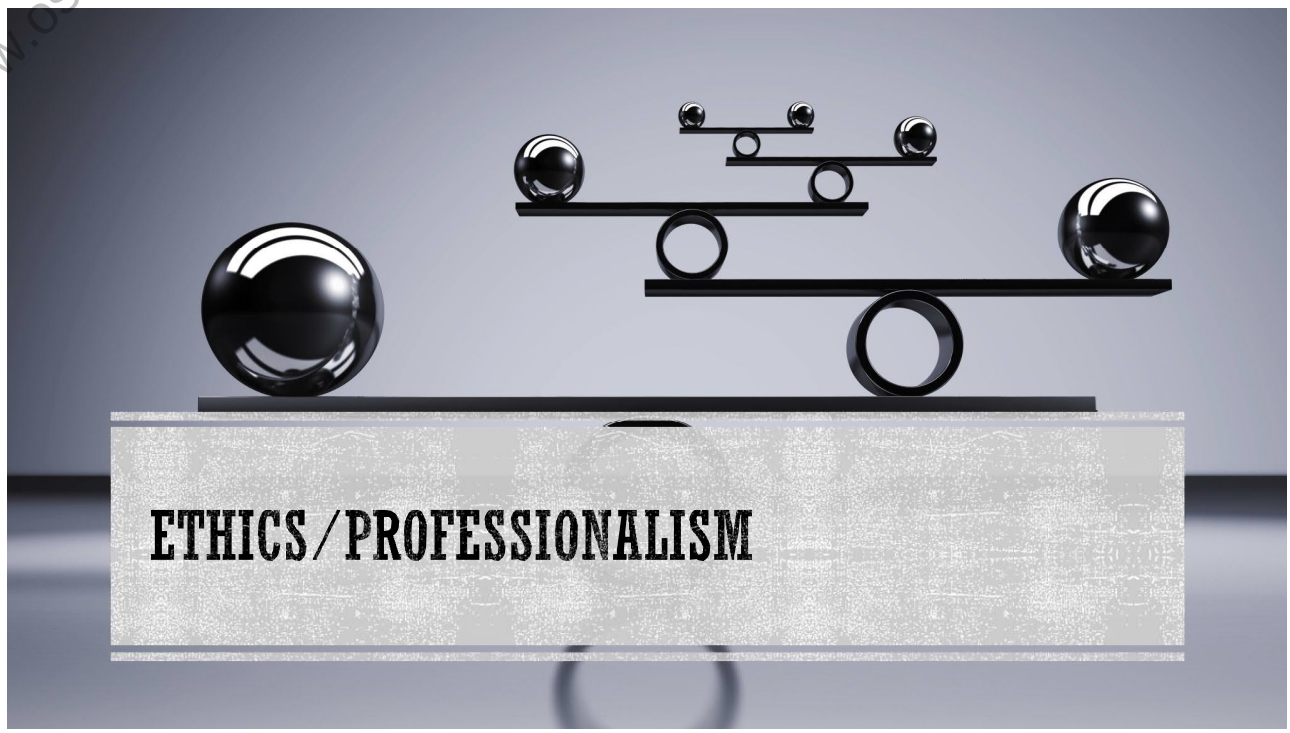

SAMPLE QUESTION

Which of the following is NOT an underlying goal of ethics committees?

A. To promote the rights of patients
B. To promote shared decision making between patients (or their surrogates if decisionally incapacitated) and their clinicians
C. To promote fair policies and procedures that maximize the likelihood of achieving good, patient-centered outcomes
D. To improve the public perception of health care professionals and health care institutions.

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C. To promote fair policies and procedures that maximize the likelihood of achieving good, patient-centered outcomes; and
D. To improve the public perception of health care professionals and health care institutions.

SAMPLE QUESTION - ANSWER

- The correct answer to this question is D. The purpose of an ethics committee is to protect human subjects and therefore have respect for persons, promote beneficence, and promote justice. Therefore, the underlying goal of ethics committee is to promote the rights of patients, promote shared decision-making between patients and their clinicians, enter promote fair policies and procedures that maximize the likelihood of achieving good outcomes. While improving the public perception of healthcare professionals and healthcare institutions is something that would be nice to do additionally, it is not a primary underlying goal of the ethics committee.

THE NATIONAL COMMISSION (BELMONT REPORT)

- All institutions receiving federal funding MUST establish Institutional Review Board (IRB).
- The IRB, a research oversight committee, has measures in place to overcome the issue of limited autonomy and ensure minimal risk while respecting the dignity of the patients involved in a research study.
  - Purpose is to PROTECT HUMAN SUBJECTS
    - Respect for Persons
    - Beneficence/Non-maleficence
    - Justice

RESPECT FOR PERSONS

Respect for autonomy

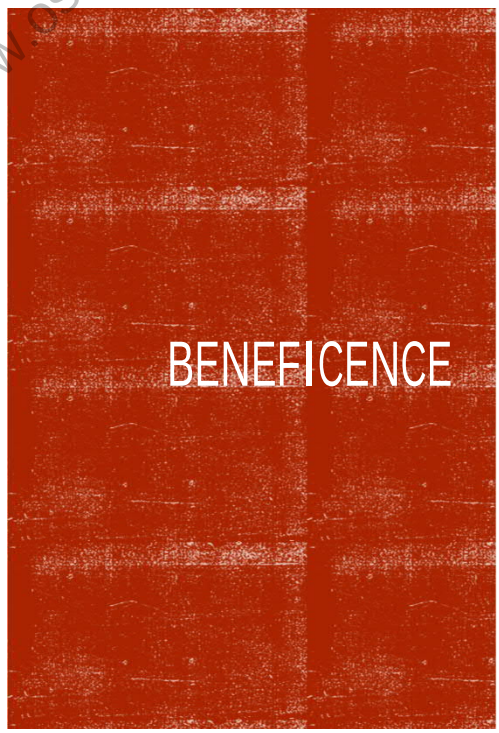
Respect the choices and wishes of competent individuals

Protection of vulnerable individuals

Those not capable of making an informed decision must have their interest protected

Is parent's decision in best interest of child?

- If not → Must be challenged



- Asserts an obligation to help others (BENEFIT) further their important and legitimate interests

NON-MALEFICENCE

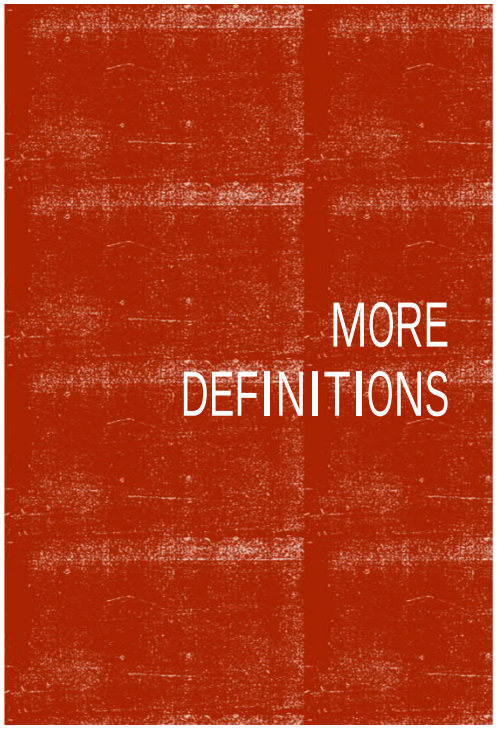
Above all, DO NO HARM

Avoid needless risk

When risk is inevitable, minimize the risk

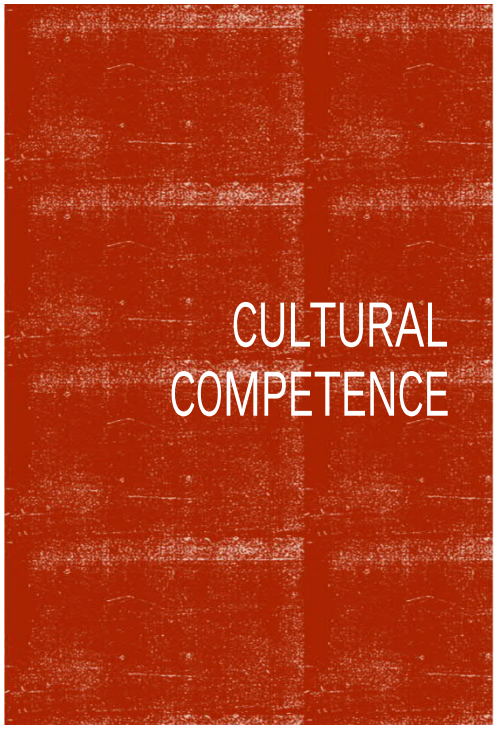
Ethical Principle	Definition	Daily Practice Examples
Autonomy	In its basic form, personal autonomy involves the ability of a patient to act without any outside influences, with clear understanding, and with the capacity to understand the material presented. As this concept relates to pediatric patients, the parents/guardians are granted the ideals of this principle as it relates to providing care and guidance for children.	Respect the choice(s) of parents to refuse treatment or care. Respect that parents will make decisions based on religious and personal views. Allow for assent—allowing the child to provide input in an appropriate plan of care. Provide parents with information to allow for informed decision-making; do not lie or withhold information. Protect the confidentiality and privacy of patients.
Beneficence	The promotion of patient well-being; as this concept relates to pediatric patients, choices should be made that promote the highest possible good outcome.	Respect the parents' ability to make medical decisions based on the best interest of the child. Consider and compare the risks and burdens associated with medical treatment—for example, vaccinations. Encourage patients and families to eat healthy foods. Promote weight loss in overweight, obese patients.
Nonmaleficence	Actions should not cause, create, or result in harm to others; *primum non nocere* (first, do no harm). As this concept relates to pediatric patients, the ideas of physical and emotional harm must be considered and avoided to honor this principle.	Physicians should step in to avert the decisions made by parents or court order/guardianship. Refuse to provide medically unnecessary treatment. Be mindful of medication interactions.
Justice	In its basic form, justice promotes equal treatment to all patients. As this concept relates to pediatric patients, this principle allows for a more ethical and transparent approach to meeting the health care needs of vulnerable populations.	Consider the legal and moral obligations to all patients. Avoid the exploitation of children, acknowledging their status as a vulnerable patient population

Reprinted with permission from Ransom H, Olsson J. Allocation of health care resources: principles for decision-making. *Pediatr Rev*. 2019;38:322.

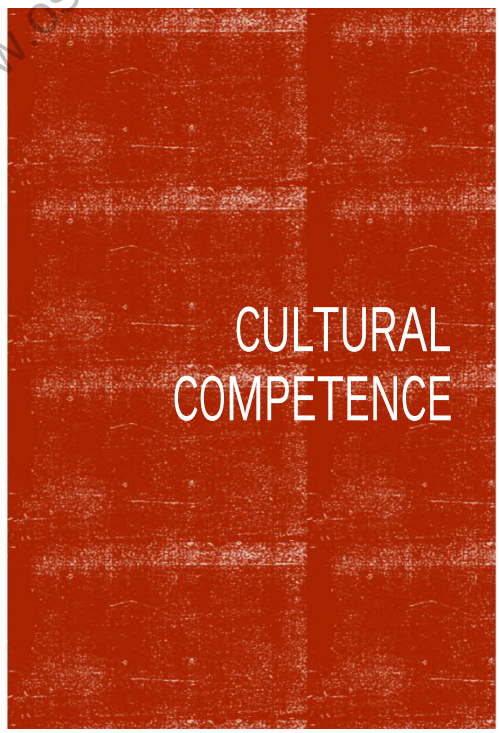


- Utilitarian theory → Greatest good for the greatest number of people
  - i.e., Reporting child abuse can lessen doctor-patient trust, but a greater good is served by protecting the children
- Paternalism → Doctors know what is best for the patient and should decide on treatments
  - Opinion and guidelines of family prevail over your opinion as a physician unless it is a conflict against the best interest of the child
- Autonomy → Competent adults have the right to make their own treatment decisions
  - Guiding principle in answering ethics questions

- Do the benefits of a study outweigh the risks to the subjects?
- Did the subject give genuine informed consent to being in the study (versus merely agreeing to sign the consent form)?
- Does the patient know that they may be getting a placebo and is this ethical if they have a condition which would benefit from getting the real drug instead?
- Is there a conflict of interest, such as financial gain, for a particular result?
  - E.g. A new drug looks more effective by excluding severely ill patients from the clinical trial.
- Ethical considerations include the use of "vulnerable populations"
  - E.g. Incarcerated and institutionalized populations

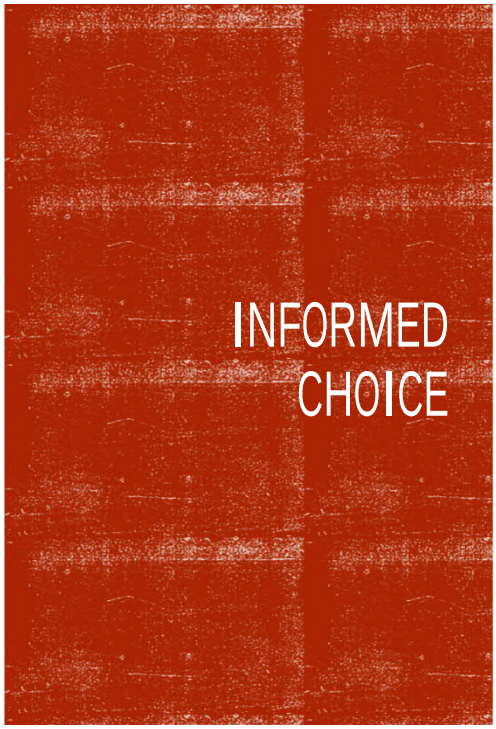


- Ability to effectively interact with people of different cultures and meet their social, cultural, and linguistic needs
- Being a culturally competent provider requires the consideration of immigration status, education level, socioeconomic status, beliefs, and family structure

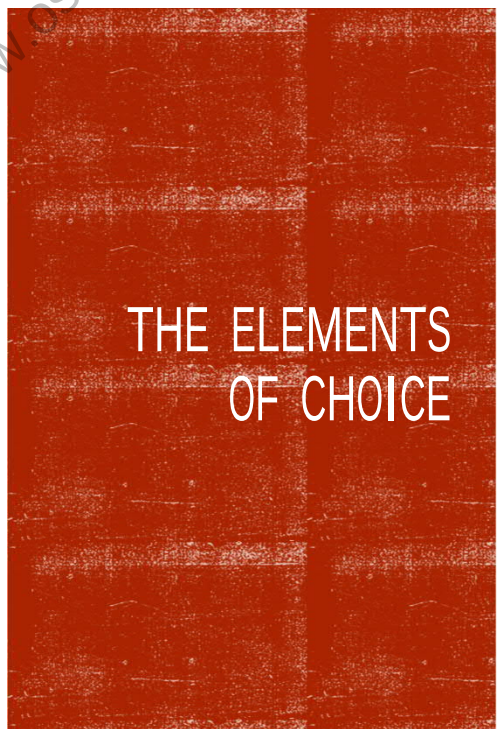


To provide culturally competent care:

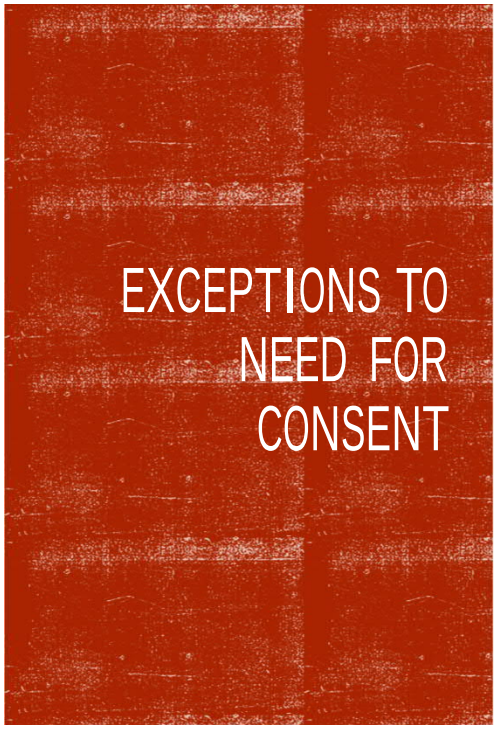
- Describe the medical condition and treatment in terms the patient and family can understand and in their native language with a medical interpreter
- Prioritize treatment options.
- Determine the patient and parent priorities.
- Describe the plan of care.
- Determine if the patient and parents accept the plan of care.
- If the patient and parent are in conflict about the devised treatment plan, provide more information and renegotiate.



- Informed Choice is a process of information exchange with:
  - A competent (understands nature/consequences of actions) un-coerced patient who understands the procedure, its risks, benefits and alternatives then makes a free informed choice.
- Process of arriving at a decision is a conversation.
- Once a decision is made:
  - Documented on an "Informed Consent" form
  - The form is the outcome and not a substitute for the process of information exchange.

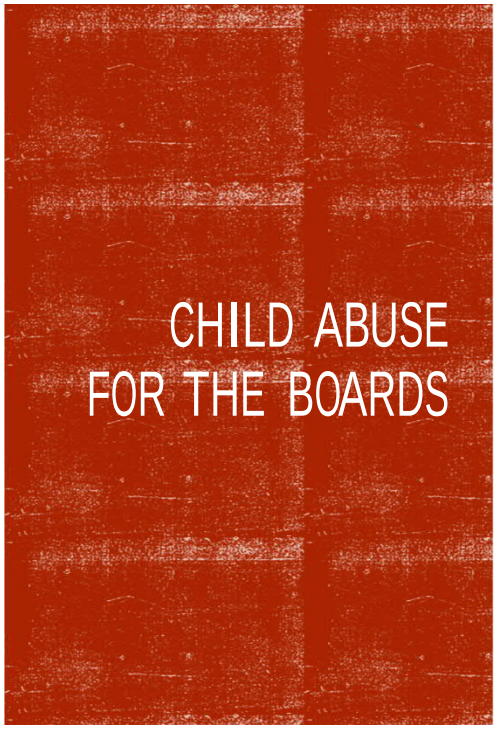


- Nature of the procedure
- Risks and possible benefits
- Alternatives to the procedure
- Competent patient, engaged professional
- No coercion or duress
- A full discussion and documentation.



- Emergency
- Unforeseen developments in surgical procedure
  - No need to wake patient up for permission if immediate medically necessary action is required

CHILD ABUSE



- Any bruising on a nonmobile infant should raise concern for child physical abuse.
- A skeletal survey is indicated in any child less than 2 years of age when physical abuse of any kind is suspected.
- Pediatric clinicians with expertise in child abuse and child protection teams at tertiary centers can be valuable resources when a clinician is uncertain is suspicious for abuse.

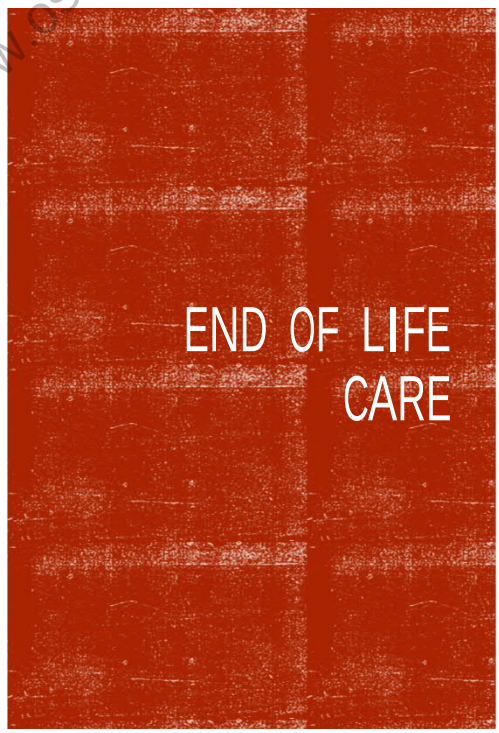
REPORTING TO CHILD PROTECTIVE SERVICES

- Reporting to CPS is mandated by law whenever you have reasonable suspicion that a child is a victim of abuse
  - Failure to report can result in further injury to the patient involved and civil or criminal penalties for the physician
- Important to discuss the concern and requirement to report child abuse to CPS with the child's parents
- Any injury to a young non-ambulatory infant, including bruises, mouth injuries, and intracranial/abdominal injury are suggestive of abuse and should be reported
- Transferring the child to another physician or facility for further care does not release them from the requirement to report suspected abuse

END OF LIFE CARE

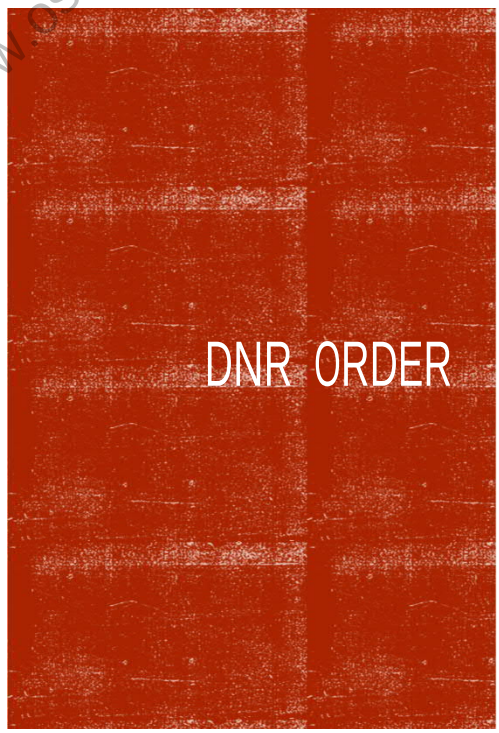
- Emotionally difficult for the patient, family, and health care providers
- 1st responsibility: ensure that the needs of the affected patient remains primary focus
  - Patient's comfort is maintained to the greatest degree possible
- Expected outcome
- Potential risks, benefits, side effects of treatment
- Option of providing comfort care or withdrawal of care

Until a DNR order has been established, all attempts at CPR must be made.

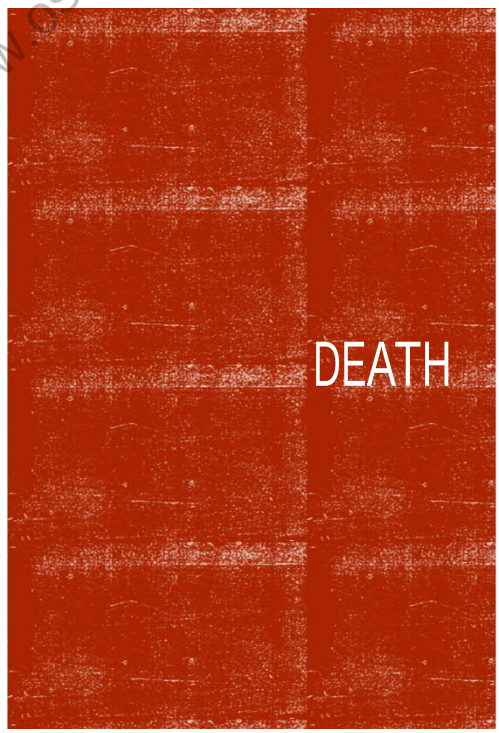


Doctrine of Double Effect

- Rarely, appropriate interventions may hasten death; however, if the intent was to provide symptomatic relief, the intervention may be ethically justified under the doctrine of double effect
- The components that make the unintended consequence justifiable include the following:
  - The act itself (treating suffering) must be inherently good
  - The agent intends the good effect (treating suffering) rather than the bad effect (hastening death)
  - The good effect must outweigh the bad effect (e.g., hastening death by many years for mild pain would be unacceptable)
- The use of a medication for the sole intent of hastening death (such as high-dose potassium chloride) is medically and ethically inappropriate



- Appropriate to withhold CPR:
  - Competent patient requests it
  - Surrogate agent (usually family member) requests this for an incompetent patient
  - Attempting CPR is futile



- Modern technology has led to various definitions of death
- Traditionally 2 types are described
  1. Cardiac Death → Cessation of cardiac and respiratory activity
  2. Brain Death → Irreversible cessation of all functions of the entire brain, including the brainstem
- Uniform Anatomical Gift Act provides the legal foundation on which human organs and tissues can be donated for transplantation.

BRAIN DEATH

Reversible conditions or conditions that can interfere with the neurologic examination must be excluded prior to brain death testing.
See text for discussion

1. Coma. The patient must exhibit complete loss of consciousness, vocalization and volitional activity.
- Patients must lack all evidence of responsiveness. Eye opening or eye movement to noxious stimuli is absent.
- Noxious stimuli should not produce a motor response other than spinally mediated reflexes. The clinical differentiation of spinal responses from retained motor responses associated with brain activity requires expertise.

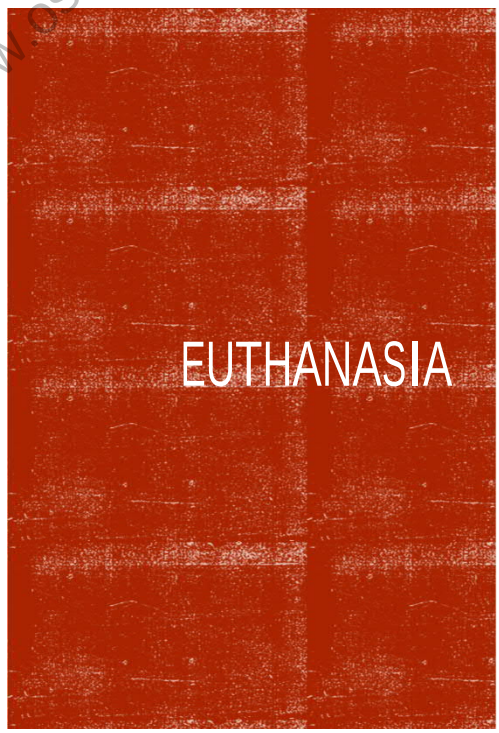
2. Loss of all brain stem reflexes including:
Midposition or fully dilated pupils which do not respond to light.
Absence of pupillary response to a bright light is documented in both eyes. Usually the pupils are fixed in a midsize or dilated position (4–9 mm). When uncertainty exists, a magnifying glass should be used.
Absence of movement of bulbar musculature including facial and oropharyngeal muscles.
Deep pressure on the condyles at the level of the temporomandibular joints and deep pressure at the supraorbital ridge should produce no grimacing or facial muscle movement.
Absent gag, cough, sucking, and rooting reflex
The pharyngeal or gag reflex is tested after stimulation of the posterior pharynx with a tongue blade or suction device. The tracheal reflex is most reliably tested by examining the cough response to tracheal suctioning. The catheter should be inserted into the trachea and advanced to the level of the carina followed by 1 or 2 suctioning passes.
Absent corneal reflexes
Absent corneal reflex is demonstrated by touching the cornea with a piece of tissue paper, a cotton swab, or squirts of water. No eyelid movement should be seen. Care should be taken not to damage the cornea during testing.
Absent oculovestibular reflexes
The oculovestibular reflex is tested by irrigating each ear with ice water (caloric testing) after the patency of the external auditory canal is confirmed. The head is elevated to 30 degrees. Each external auditory canal is irrigated (1 ear at a time) with ~10 to 50 mL of ice water. Movement of the eyes should be absent during 1 minute of observation. Both sides are tested, with an interval of several minutes.

3. Apnea. The patient must have the complete absence of documented respiratory effort (if feasible) by formal apnea testing demonstrating a $Paco_2$ ≥ 60 mm Hg and ≥ 20 mm Hg increase above baseline.
- Normalization of the pH and $Paco_2$, measured by arterial blood gas analysis, maintenance of core temperature > 33°C, normalization of blood pressure appropriate for the age of the child, and correcting for factors that could affect respiratory effort are a prerequisite to testing.
- The patient should be preoxygenated using 100% oxygen for 5–10 minutes prior to initiating this test.
- Intermittent mandatory mechanical ventilation should be discontinued once the patient is well oxygenated and a normal $Paco_2$ has been achieved.
- The patient's heart rate, blood pressure, and oxygen saturation should be continuously monitored while observing for spontaneous respiratory effort throughout the entire procedure.
- Follow up blood gases should be obtained to monitor the rise in $Paco_2$ while the patient remains disconnected from mechanical ventilation.
- If no respiratory effort is observed from the initiation of the apnea test to the time the measured $Paco_2$ ≥ 60 mm Hg and ≥ 20 mm Hg above the baseline level, the apnea test is consistent with brain death.
- The patient should be placed back on mechanical ventilator support and medical management should continue until the second neurologic examination and apnea test confirming brain death is completed.
- If oxygen saturations fall below 85%, hemodynamic instability limits completion of apnea testing, or a $Paco_2$ level of ≥ 60 mm Hg cannot be achieved, the infant or child should be placed back on ventilator support with appropriate treatment to restore normal oxygen saturations, normocarbia, and hemodynamic parameters. Another attempt to test for apnea may be performed at a later time or an ancillary study may be pursued to assist with determination of brain death.
- Evidence of any respiratory effort is inconsistent with brain death and the apnea test should be terminated.

4. Flaccid tone and absence of spontaneous or induced movements, excluding spinal cord events such as reflex withdrawal or spinal myoclonus.
- The patient's extremities should be examined to evaluate tone by passive range of motion assuming that there are no limitations to performing such an examination (eg, previous trauma, etc) and the patient observed for any spontaneous or induced movements.
- If abnormal movements are present, clinical assessment to determine whether or not these are spinal cord reflexes should be done.

Clinical examinations done by 2 physicians separated by age-dependent observation periods, 12 or 24 hours apart depending on the child's age, each including a clinical neurologic examination and apnea testing

Ancillary testing, such as EEG or cerebral blood flow scanning, should be undertaken to help determine brain death, only if the clinical examination is equivocal or cannot be reliably performed.



- Passive euthanasia → Common and involves "letting" people die (Palliation)
- Active euthanasia → Hastening the dying process
- Generally speaking, active euthanasia is illegal

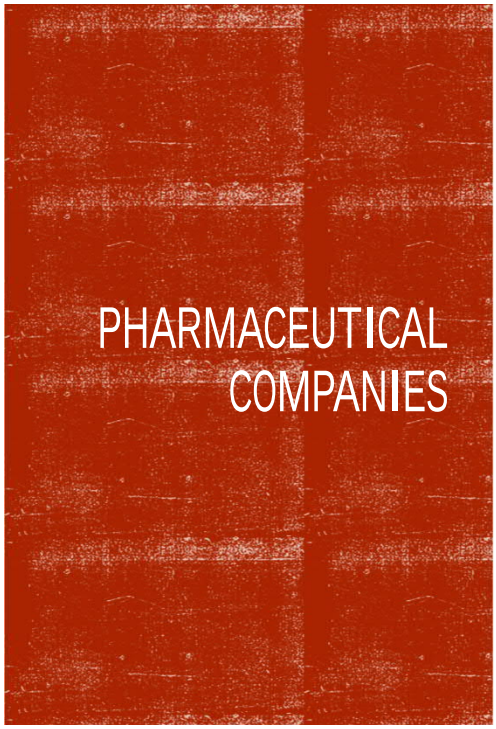
TABLE 1 Rules of Thumb to Distinguish Appropriate Palliation From Euthanasia

Palliation	Euthanasia
The primary goal is to relieve suffering without considering life expectancy. These goals are clear to the health care team and the family	The primary goal is to end life as a way of ending suffering
Medications to treat suffering are escalated by using well-defined measures of treatment effectiveness to reach goal of comfort. Medication escalation is stopped once suffering is adequately controlled	Medications are escalated regardless of physical symptoms
The safest and least harmful medications are chosen first before escalating to medications that are more likely to cause adverse effects of respiratory depression. Medications that hasten death without treating symptoms are avoided	Medications are chosen based on efficacy of achieving death quickly
Use of medications and the process for decision-making are open and documented in the patient medical record	Tendency to keep knowledge about the use of specific medications secret

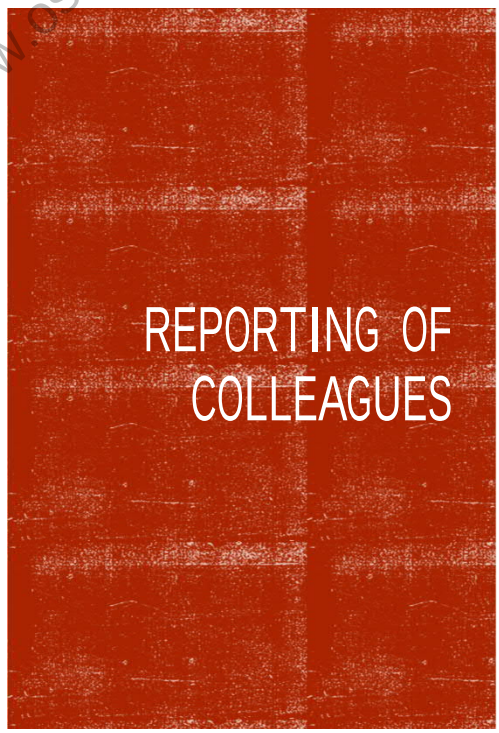
eprinted with permission from Morrison W, Kang T. Judging the quality of mercy: drawing a line between alliation and euthanasia. *Pediatrics.* 2014;133:S31-S36.

CONFLICT OF INTEREST

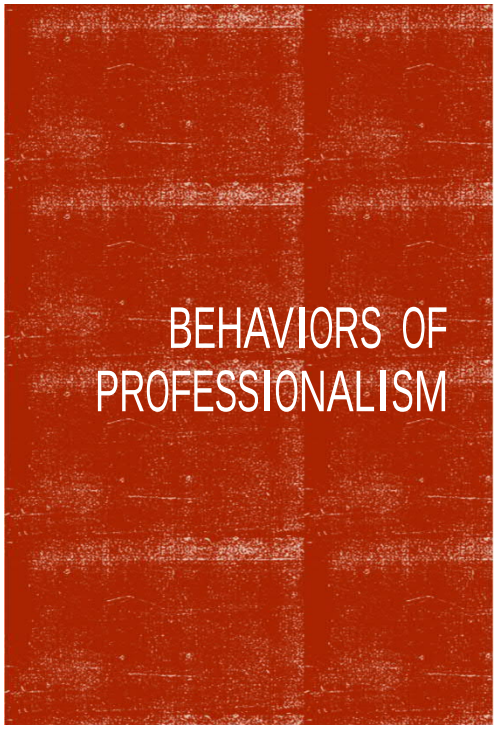
- Institute of Medicine: circumstances that create a risk that professional judgements or actions regarding a primary interest will be unduly influenced by a secondary interest
- Can occur in research education and clinical practice
- Disclosure of any potential conflicts of interest is an important step in mitigating them



- Growing concern in organized medicine that drug companies are "buying influence" with gifts to the doctors
- Ethical doubts arise when companies pay for extravagant items and not of primary educational value
- "Quid Pro Quo" arrangements are virtually always seen as unethical
  - i.e., Getting a check for writing prescriptions or an honorarium contingent upon saying favorable things about a drug
  - Dilemma: Drug companies take over more of the medical education funding that was once provided by the government



- It is an ethical responsibility of physicians to ensure that impaired colleagues are not practicing patient care
- We have a duty to report impaired colleague to some higher authority
- All 50 states have physician health programs to assist in the rehabilitation and monitoring of physicians with substance use disorders



- Altruism
- Responsibility and Accountability
- Leadership
- Caring, Compassion, and Communication
- Excellence and Scholarship
- Respect
- Honor and Integrity

BOUNDARIES OF PROFESSIONALISM

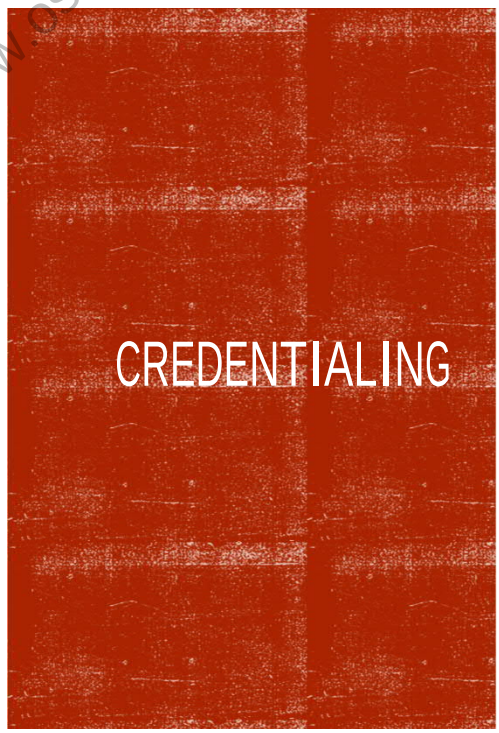
- Romantic and Sexual Relationship
  - Don't do it
- Gifts and other expressions of gratitude
  - Potential slippery slope→ Expectation of preferential treatment
  - Board Answer: Politely decline gifts

MALPRACTICE AND RISK MANAGEMENT

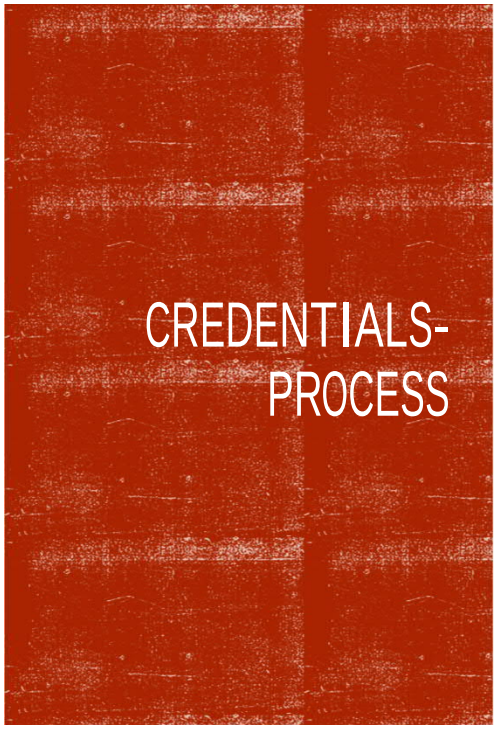
- 4 D's of malpractice: Dereliction (negligence) of Duty that Directly leads to Damages
- Deviation from standard practice which needs expert witness input
  - Expert witness must be thorough, fair, objective, and impartial
- Disclosing errors to families is legally and ethically mandated

MALPRACTICE AND RISK MANAGEMENT

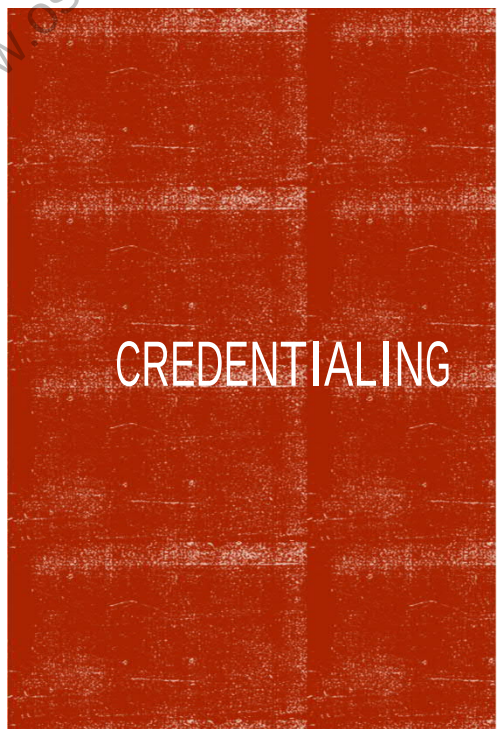
- Can be lessened with good communication and good charting
- Explain issues to your patients and try to lessen any surprises
- Remember to listen to patients
- Return phone calls
- Never alter a medical record in a way that might be construed as deceptive. Note explicitly when you make changes (date/time/initials besides the correction)



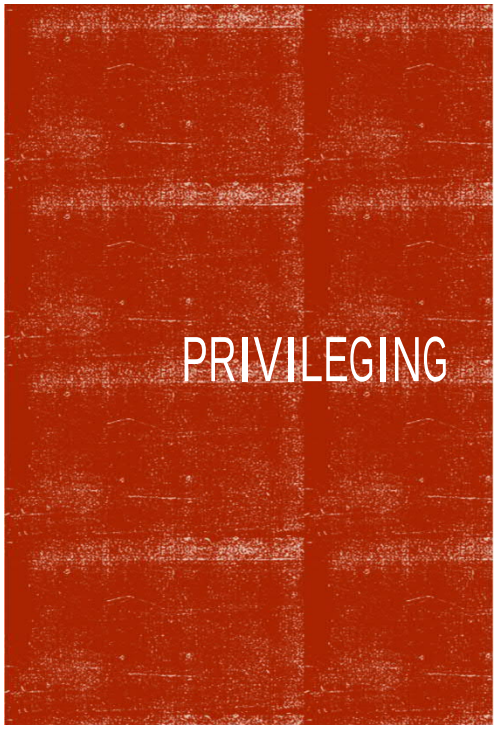
The process of obtaining, verifying, and assessing the qualifications of a health care practitioner to provide patient care services in or for a health care organization



Current license
Education
Training
Experience
Competence
Professional judgment



- Varies from hospital to hospital
- Awards privileges that allow you to practice
- Approved by the Hospital's Credentialing Committee



Determines what procedures a credentialed practitioner is permitted to perform at the facility

Based on

- Training
- Experience
- Clinical competence

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HEAD & NECK TRAUMA
Surgical Critical Care

Gregory B. York, MD, FACS
Trauma/Critical Care

EPIDEMIOLOGY – TBI

- #1 Cause of Traumatic Death (30%)
  - 2,800,000 TBI per year (50K+ deaths)
    - Mild: 75%
    - Moderate: 15%
    - Severe: 10%
  - Distribution: Ages 0-4 & 15-19 & > 65.
  - Etiology
    - Falls: 47%
    - Motor Vehicle Accidents: 14%
    - Pedestrian Impact: 15%
    - Assault: 9%
    - Unknown/Other: 15%

EPIDEMIOLOGY – TBI

- Overall Mortality
  - Mild: 0%
  - Moderate: 7-10%
  - Severe: 30%
- Overall Disability
  - Mild: 10%
  - Moderate: 50-67%
  - Severe: > 95%
- Penetrating GSW to Head
  - Vegetative or Severely Disabled: 10%
  - Moderate Disability: 20%
  - Good Outcome: 20%
  - The Others: Dead

PATHOPHYSIOLOGY – TBI

- Basic Injury
  - Primary: Forces imparted at the time of impact
  - Secondary: Occur after initial impact
    - Often due to loss of autoregulation or normal homeostasis
    - Swelling and release of chemicals that promote cell injury or death
    - Include hypoxemia, ischemia, initial hyperemia, cerebral edema, expansion of hemorrhage
- Mechanisms: Blunt or Penetrating
- Severity: Mild, Moderate, Severe

GLASCOW COMA SCALE

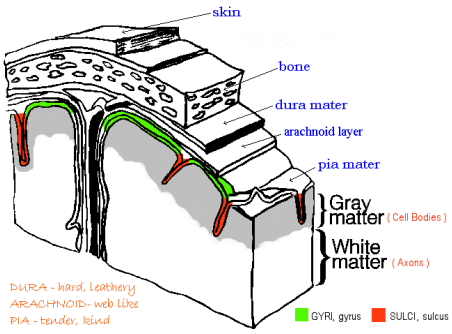
- TBI Measurement of Severity
  - 3 Parameters: Best score in each
    - Motor Function
    - Verbalization
    - Eye Movement
  - Maximum: 15
  - Minimum: 3
  - Intubated: Score of 1 for verbal; annotate with "T"
- TBI Severity
  - Mild: GCS 14 – 15
  - Moderate: GCS 9 – 13
  - Severe: GCS 3 - 8

GLASGOW COMA SCALE (GCS)

EYE OPENING (E)	Spontaneous	4
	To Speech	3
	To Pain	2
	None	1
BEST MOTOR RESPONSE (M)	Obeys Commands	6
	Localizes Pain	5
	Normal Flexion (Withdraws)	4
	Abnormal Flexion (Decorticate)	3
	Extension (Decerebrate)	2
	None	1
VERBAL RESPONSE (V)	Oriented	5
	Confused Conversation	4
	Inappropriate Words	3
	Incomprehensible Sounds	2
	None	1

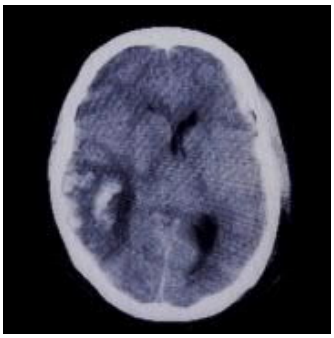
ANATOMY

- Pia Mater
- Arachnoid Layer
- Dura Mater



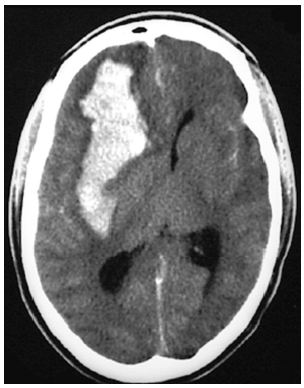
CEREBRAL CONTUSION

- Injury (“bruise”) to superficial gray matter
- Classic – “Coup”/ “Countercoup” lesions
- Location
  - Temporal Lobe: 50%
  - Frontal Lobes: 33%
  - Multiple/Bilateral: 90%
- Violates BBB
- May enlarge in first 12 hrs
- Edema may worsen over first several days



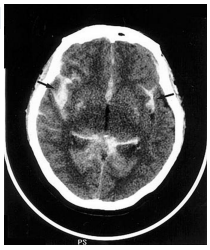
INTRAPARENCHYMAL HEMORRHAGE

- Similar to Cerebral Contusions
- May increase over time
- May produce increasing mass effect/neurologic deterioration



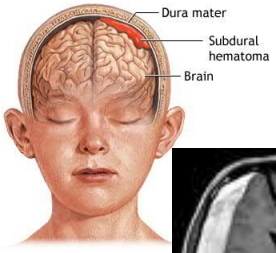
SUBARACHNOID HEMORRHAGE

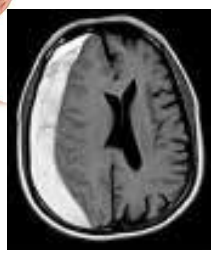
- Location: Between pial and arachnoid membranes
- Etiology: Venous tears in subarachnoid space
- Vasospasm
  - May occur in 2 – 41% of traumatic SAH
  - As early as 2 – 3 days
  - Heightened incidence: 3 – 14 days
  - May last up to 3 weeks



SUBDURAL HEMATOMA

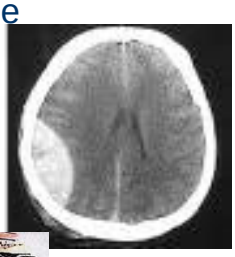
- Location: Between arachnoid and dura
- Etiology: Stretching/tearing of Cortical Bridging Veins
- Classification
  - Hyperacute: < 6 hrs
  - Acute: 6 hrs – 3 days
  - Subacute: 3 days to 3 wks
  - Chronic: 3 wks to 3 mos
- Concave (crescent) shaped
- Mortality: Higher than similar EDH
  - Greater force
  - Slower onset

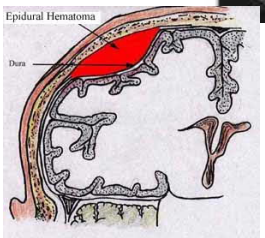




EPIDURAL HEMATOMA

- Location: Between dura and inner table
- Etiology
  - Arterial (90%): Middle Meningeal Artery
  - Venous (10%): Venous Sinus
- Classic presentation
  - Brief LOC
  - Lucid interval
  - Obtundation
  - Contralateral Hemiparesis
  - Ipsilateral pupil dilation
- Convex shaped
- Side of Impact: 70%





DIFFUSE AXONAL INJURY (DAI)

- Traumatic axonal stretch injury
  - Diffuse cellular injury to the brain from rapid rotational movement
  - Axonal structural failure due to mechanical forces along axons
  - Culmination in physical separation of axon into proximal and distal segments
- Number of axonal disruptions = Amount of deficit

REVIEW

A 45 y/o female suffered a severe blunt head trauma when she jumped from a moving vehicle. On arrival in the emergency department her airway is patent. Breathing and circulation are intact. Vitals signs are stable. Her eyes are closed, and she opens them only to painful stimuli. She moans unintelligible sounds and with noxious stimuli, there is decorticate posturing. Pupils are equal and reactive bilaterally. What is her GCS?

a. 11
b. 9
c. 7
d. 5

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a. 11
b. 9
c. 7 (E – 2, V – 2, M – 3)
d. 5

EVALUATION

- First Priority: Airway, Breathing, Circulation
- Disability
  - Pupillary Exam: Size & Reaction to Light
  - Calculate GCS
- Resuscitation
  - Standard Techniques
  - DO NOT postpone treating hypotension & hypoxia
  - Preferred Crystalloid: Hypertonic Saline or Isotonic Saline
  - Blood

EVALUATION

- Secondary Survey
  - Assess for other injuries
  - Complete neurological assessment
- Radiologic Evaluation
  - Hemodynamically stable
  - CT Scan of Head: Gold Standard

INITIAL MANAGEMENT

- Treat Primary Injury
- Prevent Secondary Injury
- Ischemic Penumbra
  - Zone around nonviable tissue
  - Electrically silent; marginally viable
  - May recover
  - Avoid hypotension and hypoxia

INITIAL MANAGEMENT

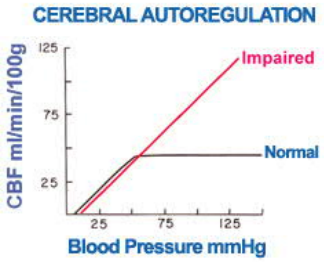
- ATLS: Primary & Secondary Surveys
- Resuscitation
- HOB 30^{o} – 45^{o}
- Oxygenation
- GI Prophylaxis (Cushing's Ulcer)
- Anti-seizure Prophylaxis
- Temperature (Trauma Pt)
- Surgical Intervention vs Medical Management?
- Intracranial Pressure Measurement?

EMERGENT SURGICAL INTERVENTION

- Criteria
  - Significant mass effect – lesion dependent
    - IPH > 50 cm^3
    - EDH > 30 cm^3
    - SDH thickness > 1 cm
  - Midline shift $\geq$ 5 mm
  - GCS dependent
- Procedures
  - Bifrontal DC is NOT recommended to improve outcomes in severe TBI pts with diffuse injury and ICP > 20 mm Hg for 15 mins (w/l 1-hr period) refractory to 1^{st} tier therapies
  - Large frontotemporoparietal DC is recommended over small for reduced mortality and improved neurologic outcomes
- Penetrating injuries may need simple debridement

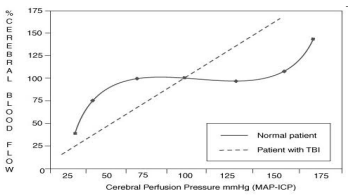
CEREBRAL PERFUSION

- Autoregulation
  - Maintains constant CBF over range of MAP
  - Depends on intact BBB
  - Regulated by: pCO_2, pH, Blood Pressure
- Cerebral Blood Flow (CBF)
  - Essential for cerebral oxygenation
  - Difficult to measure



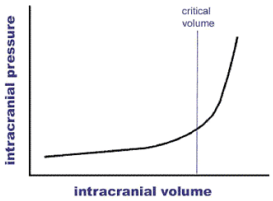
CEREBRAL PERFUSION

- Cerebral Perfusion Pressure (CPP)
  - Linear relation to CBF
  - Indirect measurement of CBF
  - CPP = MAP - ICP
- CPP Monitoring
  - Recommended to decrease 2-week mortality
  - Goal: 60 – 70 mm Hg
  - Avoid CPP > 70 mm Hg with fluids and pressors due to risk of respiratory complications/worse outcomes



MONRO-KELLIE MODEL

- Under normal conditions – Fixed Volumes
  - Brain Parenchyma
  - Cerebrospinal Fluid
  - Cerebral Blood Volume
- If any volumes increase, compensation must occur to maintain normal ICP
  - Displacement of CSF
  - Displacement of venous blood



INTRACRANIAL PRESSURE (ICP)

- Normal
  - Adult: < 10 – 15 mm Hg
  - Children: 3 – 7 mm Hg
  - Infants: 1.5 – 6 mm Hg
- Threshold: 22 mm Hg
  - Increased Morbidity
  - Increased Mortality
- Cushing Reflex (seen with rapid increase in ICP)
  - Hypertension
  - Bradycardia
  - Respiratory Irregularities

INITIAL MEDICAL MANAGEMENT

- Blood Pressure (Hypotension)
  - Increased mortality
  - Worse outcomes
  - INDEPENDENT predictor of outcome
  - Age-related recommendations
    - Age 50 – 69: Maintain SBP $\geq$ 100 mm Hg
    - Age 15 – 49: Maintain SBP $\geq$ 110 mm Hg
    - Age > 70: Maintain SBP $\geq$ 110 mm Hg

INITIAL MEDICAL MANAGEMENT

- Oxygenation
  - Hypoxemia: PaO_2 < 60 mm Hg or Sats < 90%
    - Increased Mortality
    - Single episode = Independent predictor of worse outcome
  - Keep Sats > 98%
- Tissue Oxygenation
  - Maintain $S_{jv}O_2 \geq 50\%$
  - Jugular bulb monitoring of $AVDO_2$ (calculated from $S_{jv}O_2$) may be considered to reduce mortality and improve outcomes(3 & 6 mo post-injury)

INITIAL MEDICAL MANAGEMENT

- Anti-seizure Prophylaxis
  - Seizure Effects
    - Increases metabolic demand
    - Increases ICP
  - Post Traumatic (PTS) Seizure Prophylaxis
    - Late-Post TBI Seizure (> 7 days)
      - No Benefit shown
      - Not Recommended beyond 7 days
    - Early-Post TBI Seizure
      - Recommended to decrease incidence of early PTS
      - Prophylaxis for first 7 days following TBI
      - Although, early PTS has not been associated with worse outcomes

INITIAL MEDICAL MANAGEMENT

- Nutrition
  - Attain basal caloric replacement
  - At least by 5th day, not later than 7th day post injury
  - Decreased mortality
  - Transgastric jejunal feeding: Reduced VAP incidence
- Early Tracheostomy
  - 3 – 6 days
  - Reduces mechanical ventilation days
  - No effect on mortality or rate of nosocomial pneumonia

INTRACRANIAL PRESSURE

- Management: Keep Less than 22 mm Hg
- Recommendation: Use of information from ICP monitoring to reduce in-hospital and 2-week post injury mortality
- Who Needs Monitor? (3rd Ed)
  - Severe TBI + Abnormal Head CT (hematomas, contusions, swelling, herniation, or compressed basal cisterns)
  - Severe TBI + Normal Head CT + 2 of:
    - Age > 40
    - SBP < 90 mm Hg
    - Unilateral or Bilateral motor posturing

ICP MANAGEMENT STRATEGIES

- Positioning: HOB 30º – 45º
- Hyperventilation
  - Decreases ICP
  - Lowers CBF
  - Targets
    - First 24 hours: $PaCO_2$ of 35 – 45 mm Hg
    - After 24 hours & IF necessary: Short term $PaCO_2$ 30 – 35 mm Hg
  - Prophylactic Hyperventilation ($PaCO_2$ < 35 mm Hg)
    - Avoid in first 24 hours
    - In severe TBI, CBF in first 24 hours is reduced by at least 50%
  - Prolonged Prophylactic Hyperventilation ($PaCO_2 \leq 25$ mm Hg): Not Recommended

ICP MANAGEMENT STRATEGIES

- Analgesia & Sedation
  - Pain and agitation increase ICP
  - Short-acting, reversible, continuous infusion
  - Fentanyl and its related derivatives
  - Propofol
    - Neuroprotective Effect (reduces cerebral metabolism)
    - Avoid high doses/prolonged (> 48 hrs) use
  - Neither shown to improve neurologic outcome/mortality
- Paralytics: NOT INDICATED
- Steroids: CONTRAINDICATED (associated with increased mortality)

ICP MANAGEMENT STRATEGIES

- Mannitol
  - Plasma Expander: Reduced Hct/blood viscosity/increased CBF
  - Osmotic Effect: Decreases cerebral edema
  - Assess volume status of trauma patient before administration; arterial hypotension (SBP < 90 mm Hg) should be avoided
  - Disadvantages
    - Opens BBB; may cross the BBB
    - High doses/infusions may lead to increased cerebral edema
- Hypertonic Saline
  - Oncotic pressure gradient mobilizes water from cerebral tissue
  - Volume expander that improves RBC rheology and CBF
  - Given as bolus or infusion: 3% or 7.5% NaCl
  - Serum sodium goal: 145 – 160 mEq/L
  - May lower ICP; insufficient evidence on clinical outcomes

ICP MANAGEMENT STRATEGIES

- Ventriculostomy
  - Allows ICP measurement
  - Serves as therapeutic measure to lower ICP
- Second Tier Therapy
  - High Dose Barbiturate Therapy
    - Decrease cerebral metabolic demand; lowers ICPs
    - Immunosuppressive; hypotension
    - Recommended for ICPs refractory to max medical & surgical mgmt.
    - Prophylactic use NOT recommended.
  - Decompressive Craniectomy/Temporal Lobectomy
  - Hypothermia
    - Early: Within 2.5 hrs
    - Short Term: 48 hours/32º – 33º C.
    - Risks: Coagulopathy, Immunosuppression, Cardiac Dysrhythmias
    - NOT recommended at this time

REVIEW

A 28 y/o male involved in a motor vehicle collision arrives in the emergency department in stable condition, intubated, with a GCS of 7T. Workup reveals an isolated subarachnoid hemorrhage. The neurosurgeons place an ICP monitor and initial pressures are 35 mm Hg. Which of the following is an appropriate maneuver?

a. Begin IV hydrocortisone 200 mg QD.
b. Adjust respiratory rate to achieve a pCO_2 of 35-40.
c. Paralysis with IV cisatricurium.
d. Pentobarbital coma.

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FACIAL INJURIES – ISSUES

- Airway
  - Obstruction/Compromised Ventilation
  - Delayed edema of tongue/pharyngeal tissues
  - Early recognition/Controlled intubation
  - Nasotracheal Intubation: Contraindicated
  - Cricothyroidotomy/Tracheostomy in:
    - Severe maxillary and mandibular fractures
    - Massive soft-tissue damage
    - Massive edema

FACIAL INJURIES – ISSUES

- Bleeding
  - Robust vascularity of head and neck
  - Pressure Dressings
  - Posterior Nasal Packing
  - Anterior Nasal Packing
  - Rapid Fx Reduction (MMF)
  - Selective Angiographic Embolization
  - Direct Arterial Ligation: Int Maxillary, Ext Carotid, Superficial Temporal

FACIAL INJURIES – EVALUATION

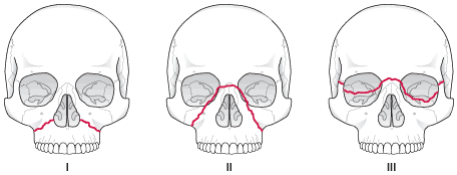
- Clinical Exam
  - Bruises, lacerations, tenderness, etc.
  - Visual Acuity – Important
  - CSF Rhinorrhea: Cribiform plate/Ethmoid fx
  - Cranial Nerves
  - Inside the mouth!!
- Radiographic: CT Face
- Therapeutic Timing: Rarely addressed immediately
- Associated Injuries
  - TBI: 20%
  - C-Spine: 2 – 4%

NASAL FRACTURE

- MOST common facial fracture
- Persistent nasal bleeding: Controlled primarily with intranasal packing
- Septal Hematoma: Must be identified/ drained; prevents ischemia/avascular necrosis
- Repair: Typically 5-7 days; allows edema to resolve

MAXILLARY FRACTURE

- Le Fort Classification
  - I: Horizontal fx separating maxilla from upper midface
  - II: Pyramidal fx separating central, pyramidal-shaped nasomaxillary segment from upper midface
  - III: Craniofacial dysjunction with midface fx through upper portion of orbits

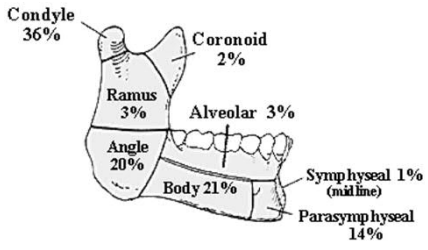


MAXILLARY FRACTURE

- Diagnosis
  - Malocclusion; elongated face
  - Mobile maxilla
  - Nasopharyngeal bleeding
  - CT Face
- Treatment
  - Restoring preinjury habitual occlusal relationship
  - MMF
  - ORIF

MANDIBULAR FRACTURE

- Features
  - 2nd to Nasal Fracture
  - Multiple Fractures: 50%
  - Considered "Open" Fracture: Abx prophylaxis
- Diagnosis
  - Malocclusion: Most sensitive indicator
  - Drooling
  - Decreased V_3 sensation
  - CT Face, Panorex plain film
- Therapy
  - Restore occlusal relationship
  - MMF
  - ORIF



REVIEW

A 20 y/o male sustains a severe closed head injury in a motor vehicle crash. On arrival his GCS is 6T after intubation. He is hemodynamically stable and head CT shows compression of the ventricles along with SAH. The most appropriate management is:

a. Ventriculostomy.
b. Steroid protocol.
c. Hyperventilation to $pCO_2 \leq 30$ mm Hg.
d. Fluid restriction.

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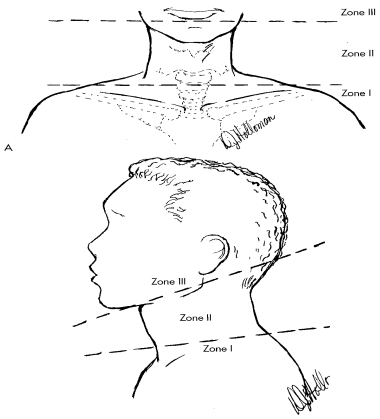
NECK INJURIES – PENETRATING

- Frequency – Penetrating Neck Wounds
  - Laryngotracheal: 10-15%
  - Pharyngeal/Esophageal: 7%
  - Major Vascular Injury: 15%
- Carotid: Most common vascular injury
- The ZONES
  - I: Highest Mortality
  - II: Most Common; Lowest Mortality

NECK ANATOMY – THE "Zones"

- I: Clavicles to Cricoid
  - Great Vessels
  - Upper Lung
  - Esophagus
  - Trachea
  - Thoracic Duct
- II: Cricoid to Angle of Mandible
  - Carotid/Vertebral Arteries
  - Larynx
  - Trachea
  - Esophgus
- III: Angle of Mandible to Base of Skull
  - Nasopharynx
  - Distal Carotid/Vertebrals
  - Parotid
  - Cranial Nerves



PHYSICAL EXAMINATION

- "Hard" Vascular Signs
  - Active Bleeding
  - Large, pulsatile, and/or expanding hematoma
  - Cervical bruit or thrill
  - Central neurological deficit
- "Hard" Aerodigestive Signs
  - Odynophagia
  - Subcutaneous air
  - Air bubbling from wound
  - Hemoptysis
  - Dyspnea or Stridor
  - Hoarseness
  - Pneumomediastinum

MANAGEMENT

- General
  - Secure airway
  - Stop Bleeding
  - Do NOT Probe
  - Associated Injuries
- Unstable Patients
  - Immediate Operative Exploration

MANAGEMENT – STABLE PATIENT

- Zone I
  - Digital Subtraction Arteriography or CT Angiogram
  - Bronchoscopy
  - Esophagography/Esophagoscopy
  - Hard Signs: Angio helps to localize injury/plan incision
- Zone 3
  - Digital Subtraction Arteriography or CT Angiogram
  - Laryngoscopy
  - Esophagography/Esophagoscopy
  - Exposure may require help from facial trauma subspecialist

MANAGEMENT – STABLE PATIENT

- Zone 2
  - Vascular Hard Signs
    - Present: Operative Exploration
    - Absent: Digital Subtraction Arteriography or CT Angiogram
  - Laryngotracheal Hard Signs
    - Obvious: Massive subcutaneous air, Air from wound – Operative Exploration
    - Less Obvious: Hoarseness, Hemoptysis, Pneumomediastinum, etc.) – Contrast studies/bronchoscopy/endoscopy

OPERATIVE MANAGEMENT

- Incision – Unstable Patient
  - Zone 1: Median Sternotomy
  - Zones 2 & 3: Anterior Neck/Cervical
- Vascular Injury
  - Carotid
    - Patch Angioplasty, Primary Repair, Interposition Graft, etc.
    - Coma: Repair, unless complete occlusion
  - Internal Jugular: Repair if possible; may ligate

OPERATIVE MANAGEMENT

- Vascular Injury (continued)
  - Incisions
    - Median Sternotomy: Prox R Subclavian, Innominate, Prox L Carotid
    - Clavicular: Mid/Distal R Subclavian, Distal L Subclavian
    - L Anterolateral Thoracotomy: Prox L Subclavian
  - Subclavian Arteries
    - Repair primarily/interposition graft
    - May be ligated/repaired later/carotid-subclavian bypass
    - Ligate distal to Vertebral artery
  - Subclavian Veins: May ligate

OPERATIVE MANAGEMENT

- Aerodigestive Tract
  - Dx: Esophagography + Endoscopy = 98.5 – 100% Sensitivity
  - Anterior Neck/Cervical Incision
  - Esophageal Repair
    - Primary Repair Possible: Two layers and Drain
    - Primary Repair Not Possible: Debride, Divert, and Drain
- Laryngotracheal Injury
  - Dx: Laryngoscopy and/or Bronchoscopy
  - Anterior Neck/Cervical Incision
  - Single layer closure
  - Tracheostomy when necessary; distal to injury
  - Tracheal/Esophageal Injury: Buttress tissue between repairs

REVIEW

In severe, uncontrolled posterior nasal bleeding, it may be necessary to perform surgical ligation or radiological embolization to stop the bleeding. The vessel that provides the most specific control of posterior nasal bleeding, i.e. the one that should be ligated or embolized is:

a. Anterior Ethmoidal.
b. Posterior Ethmoidal.
c. Internal Maxillary.
d. External Carotid.

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NECK INJURIES - BLUNT

- Rare compared to Penetrating
- 0.6 – 1.0 % of Blunt Trauma Admissions
- Presentation
  - Lower Grades: Subtle, if any
  - Higher Grades: Neurologic
- Diagnosis
  - Digital Subtraction Angiography
  - CT Angiogram

RISK FACTORS

- LeFort II or II Fracture
- Mandible Fracture
- Complex skull fracture/basilar skull fracture/occipital condyle fracture
- CHI consistent with DAI and GCS < 6
- Cervical Spine Fracture Patterns
  - Subluxation or Ligamentous Injury
  - Transverse Foramen Fracture
  - Any body fracture
  - Fractures of C1 – C3
- Near hanging with anoxic brain injury
- Clothesline type injury or seatbelt abrasion
- TBI with thoracic injuries
- Scalp degloving
- Thoracic vascular injuries
- Blunt cardiac rupture

BLUNT CEREBROVASCULAR INJURY

Grade	Description of Injury
I	Irregularity of the vessel wall or a dissection/intramural hematoma with less than 25% luminal stenosis
II	Intraluminal thrombus or raised intimal flap is visualized, or dissection/intramural hematoma with 25% or more luminal narrowing
III	Pseudoaneurysm
IV	Vessel Occlusion
V	Vessel Transection with extravasation

MANAGEMENT

- Consider
  - Grade
  - Location
  - Symptomatology
- Grade I-IV: Antithrombotic or Antiplatelet therapy (if heparin contraindicated)
- Grade II, III, IV, V – If surgically accessible
  - Operative Repair
  - Endovascular Intervention – Not recommended at this time
- Follow-Up
  - CTA in 7 – 10 days
  - If CTA shows resolution, discontinue Antithrombotics
  - If no resolution, continue for 3 – 6 months, then re-image

REFERENCES

- Mattox KL, Moore EE, Feliciano DV. Trauma. 7th Edition. McGraw-Hill. New York. 2013.
- Cameron JL, Cameron AM. Current Surgical Therapy. 10th Edition. Elsevier Saunders. Philadelphia. 2011

QUESTIONS

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THORACIC TRAUMA
Surgical Critical Care

Gregory B. York, MD, FACS
Trauma/Critical Care

EPIDEMIOLOGY

- Mortality
  - Chest Trauma
    - 20-25% of Traumatic Deaths
    - 3rd most common cause of traumatic death
  - Pre-Hospital Mortality
    - Early Death: Within 30 mins to 3 hrs of injury
    - Cardiac Tamponade, Airway Obstruction, Aspiration
    - 1/3 of patients
  - In-Hospital Mortality
    - Isolated Thoracic: 4-8%
    - Multiple Trauma: 35%

EPIDEMIOLOGY

- Etiology – Thoracic Component
  - Civilian (30% of injuries)
    - Blunt: 70%
    - Penetrating: 30%
  - Military (15% of injuries)
    - Blunt: 10%
    - Penetrating: 90%

EPIDEMIOLOGY

- Surgical Intervention
  - Blunt: Less than 10%
  - Penetrating: 15-30%
- Management: Chest Tube or Less = 85%
- Early survival depends on:
  - Initial resuscitation
  - Timeliness & correct sequence of diagnostic investigations
- Late survival depends on:
  - Post-traumatic complications

INITIAL EVALUATION

- Primary Survey
  - ABCs
  - Airway Control!!!!!
  - Cardiac monitor and pulse oximeter
  - FAST
  - Concurrent resuscitation
  - Emergent procedures as needed (e.g. chest tubes, etc.)
- Secondary survey
  - Physical Exam
  - CXR
  - Labs

SENTINEL INJURIES

- First Rib Fx: Heart/Vessels 15%, Head/Neck 50%
- Scapula Fx: CNS 55%, Brachial Plexus 13%, Vessels 13%
- Sternal Fx: Cardiac Contusion 80%, Vessels 7%
- Lower Rib Fx: Spleen 15%, Liver 15%

REVIEW

A 33 y/o female is seen in the Emergency Department for an isolated stab wound to the right chest. She is hemodynamically stable and her FAST exam is negative. The vast majority of penetrating injuries to the chest are treated by:

a. Endovascular therapy.
b. Thoracoscopy.
c. Observation and supportive care.
d. Tube thoracostomy.

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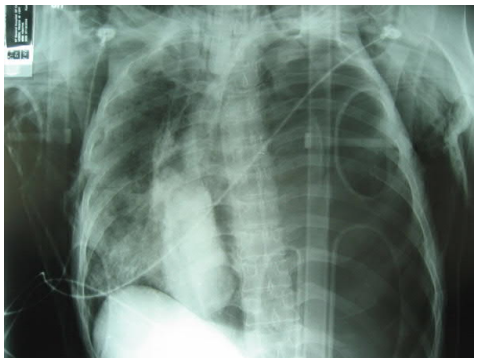
LIFE-THREATENING INJURIES

- Tension Pneumothorax
- Massive Hemothorax
- Cardiac Tamponade
- Open Pneumothorax
- Flail Chest

TENSION PNEUMOTHORAX

- Characteristics
  - Clinical diagnosis
  - "One-way" valve traps air
  - Lung collapse
  - Mediastinal shift to contralateral side
  - Decreased venous return
  - Obstructive Shock
- Signs & Symptoms
  - Respiratory distress
  - Unilateral breath sounds
  - Distended neck veins
  - Hypotension
  - Tachycardia
  - Cyanosis



TENSION PNEUMOTHORAX

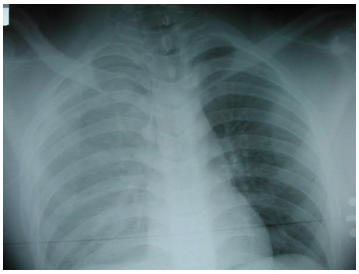
- Most common cause: Mechanical ventilation with positive-pressure ventilation in pts with visceral pleural injury
- Diagnosis: Clinical
- Treatment: Immediate Decompression
  - Needle: 2nd IC space/midclavicular line
    - 5 cm: Reaches pleural space > 50% of time
    - 8 cm: Reaches pleural space > 90% of time
  - Tube Thoracostomy: 5th IC space/ant-mid axillary line

MASSIVE HEMOTHORAX

- Characteristics
  - Rapid accumulation of greater than 1500 cc or 1/3 blood volume in chest cavity
- Signs & Symptoms
  - Hypotension
  - Unilateral breath sounds
  - Dullness to percussion
  - Hypoxia

MASSIVE HEMOTHORAX

- Diagnosis
  - CXR
  - Tube thoracostomy
- Treatment
  - Tube Thoracostomy
  - Thoracotomy/Thoracoscopy
    - Greater than 1500 cc
    - ≥ 200 cc/hr for 2 – 4 hrs
    - On-going hemorrhage
    - Patient physiologic status

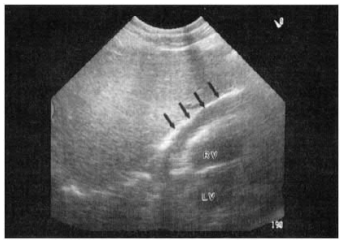


THORACOTOMY/THORACOSCOPY

- Indications
  - Excessive or persistent chest tube drainage
  - Persistent hypotension despite resuscitation
  - Lack of lung re-expansion
  - Increasing hemothorax (CXR or CT) despite chest tube output

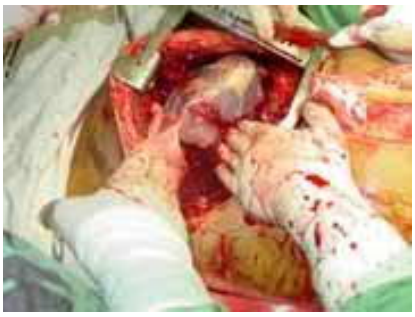
CARDIAC TAMPONADE

- Characteristics
  - Penetrating >> blunt trauma
  - High index of suspicion
- Pathophysiology
  - Fluid in pericardium
  - Compression of R Heart
  - Obstruction of venous return
  - Cardiovascular collapse
- Signs & Symptoms
  - Beck's Triad: Distended neck veins, hypotension, muffled heart tones
  - Respiratory distress
  - Bilateral breath sounds



CARDIAC TAMPONADE

- Diagnosis
  - FAST
  - Pericardial Window
  - Pericardiocentesis
    - Diagnostic/Therapeutic
    - Not Definitive Treatment
- Treatment
  - Stable patient: Median Sternotomy or Thoracotomy
  - Unstable patient: Emergent Thoracotomy
  - Anesthetic Concerns

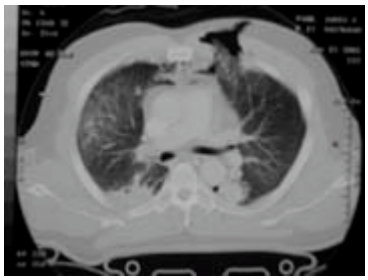


OPEN PNEUMOTHORAX

- Characteristics
  - "Sucking" chest wound
  - Air follows least resistance
- Signs & Symptoms
  - Respiratory distress
  - Unilateral breath sounds
  - Open thoracic wound
  - Hypoxia & Hypercarbia

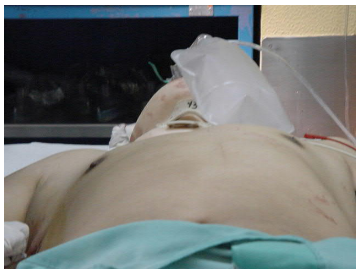
OPEN PNEUMOTHORAX

- Diagnosis: Clinical
- Treatment
  - Flutter-Valve Dressing: Taped on 3-sides
  - Tube Thoracostomy: At remote site
  - Operative Intervention
  - Taped on 4-sides = Tension pneumothorax



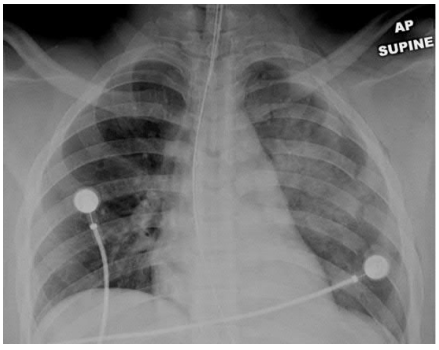
FLAIL CHEST

- Characteristics
  - Pulmonary contusion always; main cause of hypoxia
  - 2 or more adjacent ribs fractured in two or more places
  - Floating chest wall segment
- Signs & Symptoms
  - Respiratory distress
  - Crepitus
  - Paradoxical chest wall motion
    - Inspiration: Inward
    - Expiration: Outward
  - Visible deformity



FLAIL CHEST

- Diagnosis
  - Clinical signs
  - CXR: Multiple rib fractures
- Treatment
  - Resuscitation
  - Adequate Oxygenation
  - Improve Ventilation
  - Analgesia
    - Narcotics
    - Regional
    - Epidural
  - Pulmonary Toilet

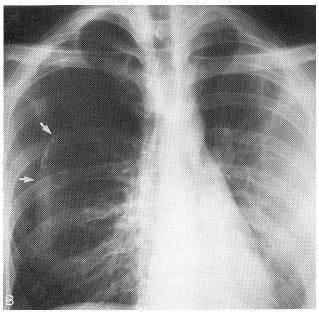


POTENTIALLY LIFE-THREATENING INJURIES

- Simple Pneumothorax
- Hemothorax
- Tracheobronchial Tree Injury
- Blunt Cardiac Injury
- Traumatic Aortic Disruption
- Diaphragmatic Rupture
- Esophageal Injury

SIMPLE PNEUMOTHORAX

- Characteristics
  - Most common intrathoracic injury
  - Air enters potential space between visceral and parietal pleura
  - Most commonly from lung laceration
- Signs & Symptoms
  - Unilateral breath sounds
  - Respiratory distress



SIMPLE PNEUMOTHORAX

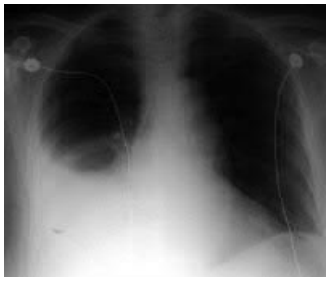
- Diagnosis: CXR
- Treatment
  - Observation
    - Size less than 20% on CXR
    - Seen ONLY on Chest CT
  - High Oxygen Concentration: Enhances air absorption
  - Simple aspiration of air: 50 – 80% success
  - Tube Thoracostomy
- Tension Pneumothorax prophylaxis

HEMOTHORAX

- Characteristics
  - Second most common intrathoracic injury
  - Bleeding
    - Blunt: Rib Fractures, Intercostal Vessels
    - Penetrating: Int Mammary vessels, Intercostal vessels, Lung Parenchyma
    - Most stop with lung re-expansion
- Signs & Symptoms
  - Decreased breath sounds
  - Respiratory distress

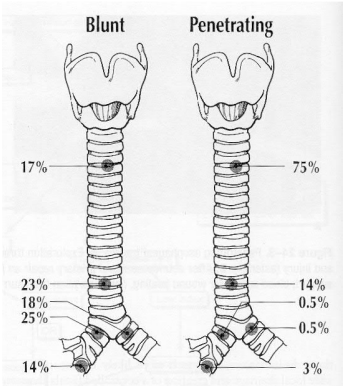
HEMOTHORAX

- Diagnosis
  - Clinical suspicion
  - CXR confirmation
- Treatment
  - Tube Thoracostomy
  - Thoracotomy: Control of hemorrhage
- Complications
  - Empyema
    - Most common risk factor is concurrent injury to intraabdominal organs
    - Retained Hemothorax: Early/aggressive prevention protocols w/VATS
  - Fibrothorax



TRACHEOBRONCHIAL TREE INJURY

- Distribution
  - Penetrating vs Blunt
  - Overall: Rare (0.03%)
  - Within 2.5 cm of Carina: 80%
- Signs & Symptoms
  - Subcutaneous emphysema
  - Hemoptysis
  - After tube thoracostomy:
    - Persistent air leak
    - Lung does not properly inflate



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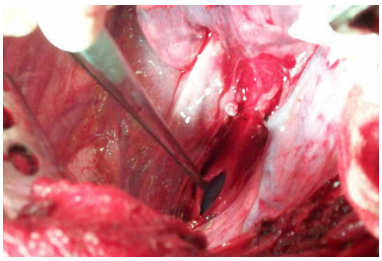
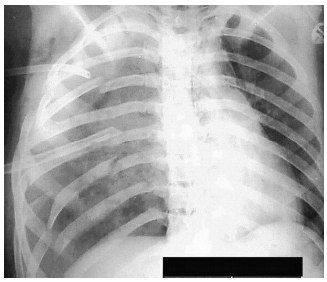


- Diagnosis
  - May see on CT scan (e.g. pneumomediastiunum, etc.)
  - Confirm with Bronchoscopy
- Treatment
  - Operative Intervention
    - Proximal: Collar Incision
    - Distal Mid Trachea/Right Mainstem: Right Posterolateral Thoracotomy
    - Left Mainstem: Left Posterolateral Thoracotomy
  - Observation
    - < 2 cm
    - Nontransmural tears

REVIEW

A 42 y/o male involved in a fall from 10 feet arrives hemodynamically stable in the Emergency Department. Exam reveals chest contusions with paradoxical motion. His chest x-ray is below. Therapeutic efforts should be directed at:

a. The underlying pulmonary parenchymal injury.
b. The associated pneumothorax.
c. Stabilization of the injured segment.
d. The underlying myocardial contusion.



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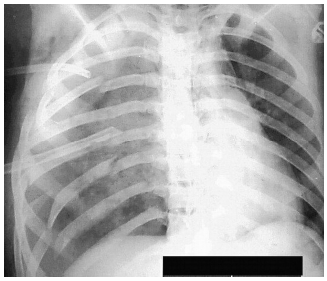


BLUNT CARDIAC INJURY

- Characteristics
  - Right Heart: Most common side
  - Mortality: 0 – 25%
  - Clinically Significant: 13%
- Signs & Symptoms
  - Most are asymptomatic
  - Symptomatic: Cardiac tamponade, hemorrhage, or severe cardiac dysfunction
  - Abnormal EKG in first 24 hours
  - Arrhythmias
  - Hypotension

BLUNT CARDIAC INJURY

- Diagnosis
  - No Gold Standard
  - EKG
  - Cardiac Enzymes
    - Elevation – Inconclusive
    - Negative Predictive Value
  - Echocardiogram – If EKG abnormal
- Treatment
  - Asymptomatic
    - No EKG Changes: Observation/Serial EKGs
    - EKG Changes: Telemetry/24hrs Observation
  - Symptomatic/Abnormal Telemetry
    - Echocardiogram
    - Supportive Care
    - ICU Admission
  - Definitive Repair of Injuries

TRAUMATIC AORTIC DISRUPTION

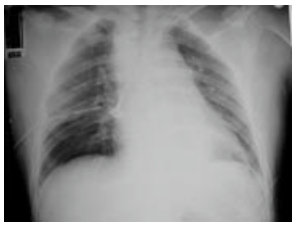
- Characteristics
  - Blunt Mechanism: Acceleration/Deceleration injury
  - Location: Mortality/Cause/Place of Death
    - Scene: 85%; Hemorrhage; Scene
    - Unstable transport: 96%; Multi-system Trauma; EMS/ED
    - Stable transport: 5-30%; CNS Injury; ICU
    - Within 24 hrs of Admission: 50%
- Signs & Symptoms
  - Mechanism
  - Thoracic trauma
  - Hypotension
  - Unequal BPs/Pulses
  - Sternal/Thoracic Spine/Rib fractures
  - High index of suspicion

TRAUMATIC AORTIC DISRUPTION

- Location of Injury
  - Prox Desc Thoracic Aorta just distal to Ligamentum Arteriosum: 90%
  - Above Aortic Valve: 4%
  - Desc Thoracic Aorta: 2%
  - Multiple Sites: 3%
- At Risk
  - High Speed MVC
  - Falls > 15 ft
  - MV – Ped Thrown > 10 ft

TRAUMATIC AORTIC DISRUPTION

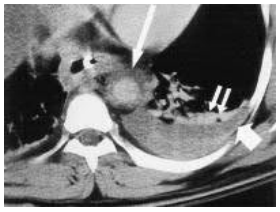
- Diagnosis
  - Chest X-ray
    - Wide Mediastinum (>8 cm) 85%
    - Indistinct aortic knob 24%
    - Left pleural effusion 19%
    - 1st or 2nd rib fracture 13%
    - Tracheal deviation 12%
    - NG Tube deviation 11%
    - Negative findings 7%
    - Depressed left bronchus 5%

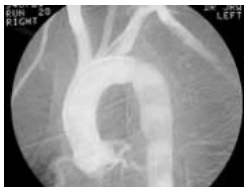


  - Transesophageal Echocardiography

TRAUMATIC AORTIC DISRUPTION

- Diagnosis
  - Helical CT Angiogram
    - Sensitivity: 90-100%
    - Specificity: 83-100%
    - NPV: 99-100%
  - Catheter Angiography
    - Sensitivity: 92-100%
    - Specificity: 95-99%
    - NPV: 97-100%





TRAUMATIC AORTIC DISRUPTION

- Medical Management
  - Minimize arterial pressure impulse (dP/dT)
  - Systolic BP $\leq$ 100 – 140 mm Hg
  - HR < 100
  - Begin with β-blocker; add nitroprusside

TRAUMATIC AORTIC DISRUPTION

- Operative Management
  - Endovascular Therapy
    - Dominant repair strategy
    - Mortality: 3.8%
    - Paraplegia: < 1%
    - Complications: L Subclavian Artery occlusion, graft compression, entry site problems
  - Direct Repair: Clamp and Sew
    - Mortality: 13%
    - Paraplegia: 10%
    - Complications: ARDS, CNS problems, neurologic

DIAPHRAGM RUPTURE

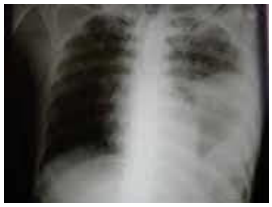
- Characteristics
  - Most occur on Left (75%)
    - Blunt: Left >>> Right
    - Penetrating: Left > Right
  - Blunt = Large tears (> 10 cm)
  - Penetrating = Small perforations (< 2 cm)
- Signs & Symptoms
  - Abdominal Pain
  - Respiratory distress
  - High index of suspicion
  - Mechanism
  - None: 20 – 45%

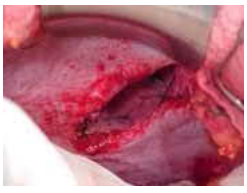
DIAPHRAGM RUPTURE

- Associated Injuries
  - Penetrating: 50% have another injury
  - Blunt: 80% have another injury
    - Pelvic Fracture: 40%
    - Both hepatic and splenic: 25%
    - Thoracic aorta: 5%
    - CHI: 50 – 75%
    - Kidney, Pancreas, SB: 10 – 12%
    - Lung: 10 – 20%

DIAPHRAGM RUPTURE

- Diagnosis
  - CXR/NGT
    - Elevated hemidiaphragm
    - Effusion
    - Intrathoracic contents
  - GI Contrast Study
  - Helical CT: Sens 71 – 100%, Spec 75 – 100%
  - Laparoscopy, Thoracoscopy, Laparotomy
- Treatment
  - Operative Repair
  - Laparotomy recommended due to high rate of associated abdominal injuries



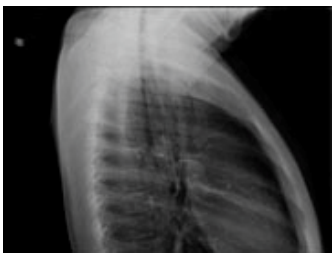


ESOPHAGEAL INJURIES

- Characteristics
  - Penetrating >> Blunt
  - Location
    - Penetrating: Cervical Esophagus
    - Blunt: Thoracic Esophagus
  - Blunt Mechanisms
    - Neck: Neck injury; hyperextension against vertebra
    - Thoracic: Severe blow to epigastrium
  - High index of suspicion based on mechanism
  - Diagnostic delay = significant morbidity/mortality
    - < 24 hrs: 10 – 20% (mortality)
    - > 24 hrs: 40 – 66% (mortality)

ESOPHAGEAL INJURIES

- Signs & Symptoms
  - Fever, Tachycardia, Leukocytosis, Shock
  - Pain out of proportion
  - Cervical or Mediastinal emphysema
  - Peritonitis
- Diagnosis
  - CXR
    - Effusion or pneumo/hemothorax
    - Mediastinal air
    - Particulate matter in chest tube
  - GI Contrast Study
    - Gastrografin then Barium
    - + Esophagoscopy = 100% Sensitivity



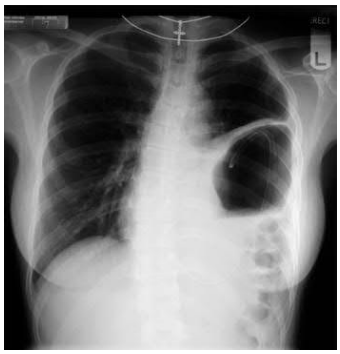
ESOPHAGEAL INJURIES

- Treatment
  - Operative Repair
    - Primary Repair Possible: Two layers & Drain
    - Primary Repair Not Possible: Debride, Divert, & Drain
  - Two layer – tension free
  - Upper 2/3 thoracic esophagus: Right thoracotomy
  - Lower 1/3 thoracic esophagus: Left thoracotomy
  - Thoracic: Reinforcement/buttress
  - External Drainage
  - Feeding Access

REVIEW

A 30 y/o female involved in a motor vehicle collision arrives in the Emergency Department. Her BP is 125/75 mm Hg and HR is 85 bpm. Her exam reveals contusions to her chest wall bilaterally. Chest x-ray is shown below. Which of the following would most likely NOT be part of her management?

a. Tube Thoracostomy.
b. Exploratory Laparotomy.
c. Observation; oxygen therapy.
d. CT Scan Abdomen



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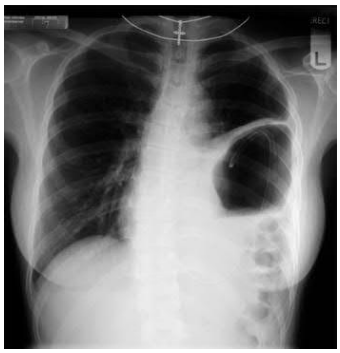


MISCELLANEOUS

- Rib Fractures
- Pulmonary Contusion
- Tube Thoracostomy
- Resuscitative Thoracotomy

RIB FRACTURES

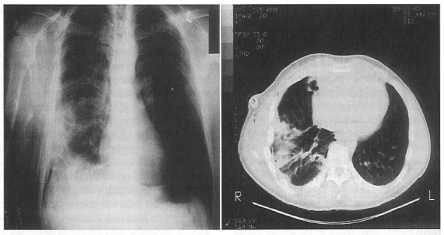
- Most common finding in chest trauma
- Pathway: Pain – Hypoventilation – Atelectasis – Excessive Secretions – Hypoxia – Pneumonia
- Visceral Involvement
  - Right: Liver = 19 – 56%
  - Left: Spleen = 22 – 28%
- Treatment
  - Analgesia
  - Pulmonary Toilet

PULMONARY CONTUSION

- Pathophysiology
  - Can occur without rib fractures or flail chest
  - Lung bruising; parenchymal injury; no laceration
  - Microhemorrhage
  - Interstitial edema
  - Alveolar collapse
  - Hypoxia/Ventilatory Failure: 24 – 48 hours

PULMONARY CONTUSION

- Diagnostics
  - CXR: Seen over 4 – 6 hours
  - CT Chest: Viewed almost immediately
- Treatment
  - Analgesia
  - Avoid fluid overload
  - Pulmonary Toilet
  - No Abx
  - Intubation (significant hypoxia; PaO_2 < 65 mm Hg)



TUBE THORACOSTOMY

- Complications
  - Pain: Most Common
  - Empyema: 2 – 10%
  - Improper Insertion/Dislodgement: 1 – 7%
  - Non-Functioning

RESUSCITATIVE THORACOTOMY

- Therapeutic Utility
  - Release Pericardial Tamponade/Repair Cardiac Injuries
  - Control exsanguinating intrathoracic hemorrhage
- Adjunctive Abilities
  - Open Cardiac Massage
  - Aortic Cross-Clamping

RESUSCITATIVE THORACOTOMY

- Indications
  - Postinjury Cardiac Arrest – Witnessed
    - Penetrating Thoracic: < 15 mins prehospital CPR
    - Penetrating Non-Thoracic: < 5 min prehospital CPR
    - Blunt: < 10 min prehospital CPR
  - Postinjury Hypotension – Persistent (SBP < 60 mm Hg)
    - Cardiac Tamponade
    - Hemorrhage – Intrathoracic, intra-abdominal, extremity, cervical
    - Air Embolism
- Survival
  - Blunt: 1%
  - Penetrating: 10%
  - Stab Wounds (17%) > GSW (4%)

REVIEW

A 53 y/o male sustained a GSW to the right chest and presents a day later with significant chest pain. Workup reveals an injury to the mid-thoracic esophagus. On exploration, there is a 2 cm segmental defect with severe mediastinitis. Among the options below, the best treatment at this time would be:

a. Antibiotics and parenteral nutrition with no attempt at surgical intervention.
b. Primary surgical repair with mediastinal drainage and feeding catheter jejunostomy.
c. Esophogeal exclusion, wide mediastinal drainage, temporary closure of the GE junction, gastrostomy, and jejunostomy.
d. Primary surgical repair with buttressing of the suture line with pleura or muscle, wide mediastinal drainage and feeding catheter jejunostomy.

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REFERENCES

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- Cameron JL, Cameron AM. Current Surgical Therapy. 10th Edition. Elsevier Saunders. Philadelphia. 2011

QUESTIONS

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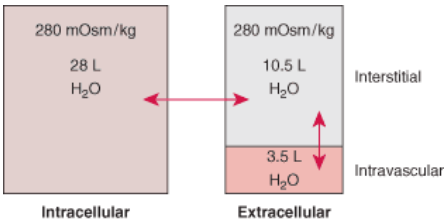
FLUIDS, ELECTROLYTES & ACID-BASE

Surgical Critical Care

Gregory B. York, MD, FACS
Trauma/Critical Care

BODY WATER DISTRIBUTION

- Percent of Body Weight
  - Male: 55-60%
  - Female: 50-55%
- Fluid Compartments
  - ICF: 2/3 of Body Water
  - ECF: 1/3 of Body Water
- Extracellular Fluid (ECF)
  - Plasma: 25% of ECF
  - Interstitial Fluid: 75% of ECF



GI SECRETIONS

Source	Volume (mL/Day)	$[Na^+]$ (mEq/L)	$[K^+]$ (mEq/L)	$[Cl^-]$ (mEq/L)	$[HCO_3^-]$ (mEq/L)
Saliva	500-750	60	20	16	50
Gastric	1000-1500	59	9.3	89	0-1
Upper SB		105	5.6	99	10
Lower SB		112	5.0	106	15-20
Bile	600 - 1000	145	5.2	100	50
Pancreas	500 - 1200	142	4.6	77	70

OSMOLALITY

- Definition
  - Measure of solute (osmoles) per KG of water
  - Only accounts for solute which contributes to the solution's osmotic pressure
- Serum Osmolality
  - Formula: (2 X [Na]) + [Glucose]/18 + [BUN]/2.8
  - Normal: 278 – 300 mOsm/kg

ELECTROLYTE DISTURBANCES

- Sodium
- Potassium
- Calcium
- Phosphorus
- Magnesium

PSEUDOHYPONATREMIA

- Low serum sodium can occur as a result of volume displacement by:
  - Hyperlipidemia
  - Hyperproteinemia
  - Hyperglycemia
- Correction Factor
  - mEq/L dec in Na = Triglycerides (g/L) x 0.002
  - mEq/L dec in Na = (Plasma protein – 8) x 0.025
  - mEq/L dec in Na = (Serum glucose – 100) x 0.016

HYPONATREMIA – HYPOVOLEMIC

- U_{Na} < 20 (Extrarenal)
  - Vomiting: Hypokalemia, Metabolic Alkalosis
  - Diarrhea: Metabolic Acidosis
  - GI Fistula: Metabolic Acidosis
- U_{Na} > 20 (Renal)
  - Diuretics: Hypokalemia, Metabolic Alkalosis
  - Hypoaldosteronism: Hyperkalemia, Metabolic Acidosis
  - "Salt Losing"
- Treatment
  - Resuscitation with Isotonic Saline
  - If hyperchloremic, use isotonic sodium acetate solution

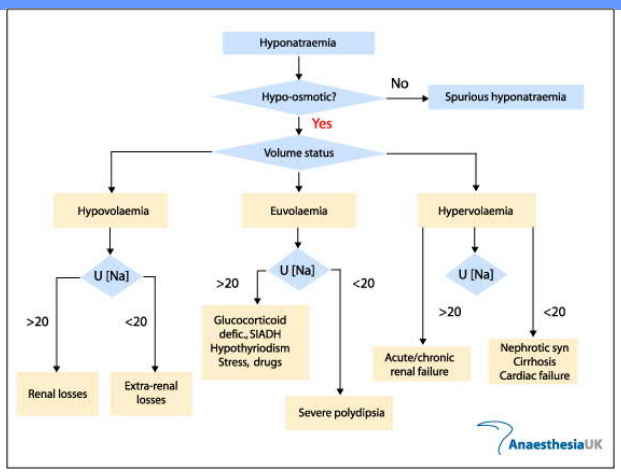
HYPONATREMIA – EUVOLEMIC

- U_{OSM} < 100
  - Polydipsia: Hypokalemia, U_{Na} < 30
  - Malnutrition: U_{Na} < 30
- U_{OSM} > 100
  - SIADH: Hypokalemia, U_{Na} > 30
  - Hypothyroidism: U_{Na} > 30
  - Hypocortisolism: Hyperkalemia, U_{Na} > 30
- Treatment: Water Restriction

HYPONATREMIA – HYPERVOLEMIC

- U_{Na} < 20 (except Renal Failure)
- U_{OSM} > 300 (except Renal Failure)
- CHF, Cirrhosis/Ascites, Nephrosis, Renal Failure
- Renal Failure: U_{Na} > 20
- Treatment: Water & Salt Restriction

HYPONATREMIA - SUMMARY



HYPERNATREMIA – HYPOVOLEMIC

- GI Losses: U_{Na} < 20, U_{OSM} > 400
  - Vomiting: Hypokalemia, Metabolic Alkalosis
  - Diarrhea, Fistula: Hypokalemia
  - Lactulose
- Renal Loses: U_{Na} > 30, U_{OSM} 300-800
  - Hyperglycemia
  - Mannitol
  - High Protein Feedings
  - Postobstructive Diuresis: Hypokalemia
- Treatment: Isotonic Saline, then Water

HYPERNATREMIA – EUVOLEMIC

- U_{OSM} > 400
  - Sweating, Hypodipsia: U_{Na} < 20
- U_{OSM} < 300
  - Central DI: After Vasopressin, U_{OSM} 300 - 800
  - Nephrogenic DI: No Vasopressin response
- Treatment: Water

HYPERNATREMIA – HYPERVOLEMIC

- U_{OSM} > 400
- U_{Na} > 30
- Hypertonic Saline, Hypertonic $NaHCO_3$, Sea water ingestion
- Treatment: Water

TREATMENT ISSUES

- Hyponatremia
  - Na Deficit = (Desired Na – Current Na) x TBW
- Hypernatremia
  - Water Deficit (L) = ((Na/140) – 1) x .6 x Wt (kg)
- Rate
  - Correct by 0.5 to 1.0 mEq/L per Hour
  - Not more than 12 mEq/L in 24 hours
  - Replace over 48 – 72 hours
- Hypertonic Saline: Symptomatic Hyponatremia

HYPERKALEMIA - ETIOLOGY

- Excess Intake
  - Potassium Supplements
  - Blood
- Intracellular – Extracellular Shift
  - β Blockers
  - Hypoaldosteronism
  - Insulin Deficiency
  - Succinylcholine
  - Cell Lysis
  - Acidosis
- Decreased Renal Excretion
  - Decreased GFR
  - Hypoaldosteronism
  - Nephritis
  - Drugs: Spironolactone, Cyclosporine, Tacrolimus, NSAIDS, etc

HYPERKALEMIA – TREATMENT

Serum K	EKG Changes	Treatment
< 6	None	Avoid NSAID Restrict K
> 6	Peaked T Prolonged PR	ECV Expansion Loop Diuretic IV Kayexalate PO
> 7	Widened QRS	Glucose/Insulin Albuterol (Inhaled) $NaHCO_3$ Dialysis
> 8	Sine Wave	Calcium Dialysis

HYPOKALEMIA – ETIOLOGY

- GI Losses: Diarrhea, Fistula, Villous Adenoma
- Extracellular – Intracellular Shift
  - Insulin
  - Beta Agonists
  - Periodic Paralysis
  - Refeeding
  - Alkalosis
- Renal Losses
  - Hyperaldosteronism: Adrenal Adenoma, Cushing's, Vomiting, Renal Artery Stenosis, Steroids
  - Nonabsorbable Anions: Ticarcillin, Piperacillin, Ketones
  - Increased Urine Flow: Diuretics, RTA
  - Magnesium Depletion

HYPOKALEMIA – TREATMENT

- Cardiac Findings
  - U Waves
  - Flat T Waves
  - AV Block
- Treatment: Potassium Replacement

REVIEW

A 32 y/o 70-kg female sustains a closed head injury in a motor vehicle accident. She remains intubated with a GCS of 5. On hospital day 4, her morning labs are normal except her Na has risen to 165 meq/L. What is this patient's free water deficit?

a. 7.5 Liters.
b. 20 Liters.
c. 1 Liter.
d. 12.5 Liters.

REVIEW

A 32 y/o 70-kg female sustains a closed head injury in a motor vehicle accident. She remains intubated with a GCS of 5. On hospital day 4, her morning labs are normal except her Na has risen to 165 meq/L. What is this patient's free water deficit?

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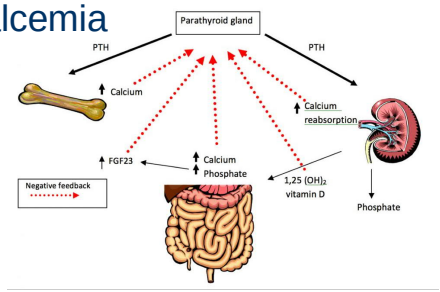
CALCIUM – PARATHYROID HORMONE

- PTH Actions
  - Stimulates osteoclast resorption of bone
  - Increases Renal reabsorption of Calcium
  - Stimulates production of Calcitriol
- PTH Secretion: Hypocalcemia
- PTH Suppression
  - Hypercalcemia
  - Hypomagnesemia



CALCIUM – SOURCES

- Diet/GI
  - Vitamin D controls absorption
- Renal
  - PTH controls reabsorption
  - Vitamin D hydroxylated by Kidney
- Bone
  - PTH & Vitamin D act on bone to release Calcium

PSEUDOHYPERCALCEMIA

- Albumin effects on Total Calcium
  - Δ Albumin 1 g/dL = Δ Total Calcium 0.8 g/dL
- Elevated Albumin = Elevated Total Calcium
- Low Albumin = Low Total Calcium
- NO effect on Ionized Calcium

HYPERCALCEMIA

- Etiology
  - GI Absorption: Vit D intoxication, Ectopic Vitamin D (TB, Lymphoma, Sarcoidosis)
  - Increased Bone Resorption: Primary HyperPTH, Ectopic PTH, Multiple Myeloma, Immobilization
  - Increased Renal Reabsorption: HyperPTH, Thiazide diuretics
- Signs/Symptoms
  - GI: Constipation, Nausea, Vomiting, Abdominal Pain
  - CV: Hypotension, Shortened QT, Bradycardia
  - Renal: Polyuria, Nephrocalcinosis
  - Neuro: Weakness, confusion, lethargy, obtundation

HYPERCALCEMIA

- Treatment
  - Volume Hydration
  - Restrict Calcium Intake
  - Furosemide Diuresis
  - Corticosteroids (if due to Vit D)
  - Calcitonin, Mithramycin, Pamidronate
  - Dialysis

HYPOCALCEMIA

- Etiology
  - GI: Vitamin D deficiency, Malabsorption
  - Bone: HypoPTH, PTH resistance (hypomagnesemia)
  - Intravascular/Tissue Chelation: Citrate, Albumin, Hyperphosphatemia, Alkalosis
- Signs/Symptoms: Peri-oral paresthesia, muscle spasms, tetany, Chvostek's sign, prolonged QT
- Treatment: Calcium Replacement, Correct Hypomagnesemia

REVIEW

Which of the following is associated with hypokalemia?

a. ACE Inhibitor Therapy.
b. Metabolic Acidosis.
c. Hypomagnesemia.
d. β-Blockade therapy.

REVIEW

Which of the following is associated with hypokalemia?

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HYPERPHOSPHATEMIA

- Etiology
  - Renal Insufficiency
  - Widespread cell necrosis
- Manifestations
  - Not well documented
  - Soft tissue insoluble calcium phosphate complexes
- Treatment
  - GI binding of phosphate (sucralfate or aluminum)
  - Dialysis

HYPOPHOSPHATEMIA

- Etiology
  - Intracellular Shift
    - Glucose Loading
    - Respiratory Alkalosis
    - β Agonists
  - Renal Excretion: DKA
  - GI Tract Malabsorption
    - Antacids with Aluminum
    - Aluminum Compounds
  - Refeeding Syndrome

HYPOPHOSPHATEMIA

- Manifestations
  - Low Cardiac Output
  - Anemia
  - Oxyhemoglobin Curve: Shifts left; decreased 2,3-DPG
  - Impaired ATP Production
- Treatment: PO_4^{-3} Replacement

MAGNESIUM

- Facts
  - Primarily intracellular; stored in bone, skeletal muscle
  - No hormones control magnesium balance
  - Regulates movement of Ca into smooth muscle cells
  - Actively involved in PTH release

HYPERMAGNESEMIA

- Etiology
  - Renal Insufficiency
  - Excess IV Magnesium
- Signs/Symptoms
  - 4-6 meq/L: Nausea, flushing, headache, lethargy, hyporeflexia
  - 6-10 meq/L: Respiratory depression, bradycardia, hypotension, hypocalcemia
  - > 10 meq/L: Paralysis, heart block, cardiac arrest
- Treatment
  - Volume Expansion
  - IV Calcium
  - Dialysis

HYPOMAGNESEMIA

- Etiology
  - GI Losses: Diarrhea, Gastric suction, Malabsorption
  - Renal Losses: ATN, Diuretics, Aminoglycosides, Ketoacidosis, RTA
  - Increased Cellular Uptake: Refeeding, Insulin, Pancreatitis, Rhabdomyolysis
- Signs/Symptoms
  - < 1.6: Weakness, anorexia, hypokalemia, hypocalcemia
  - < 1.2: Tetany, wide QRS, peaked T, Chovestek sign
  - < 0.8: Convulsions, ventricular arrhythmia, Torsades
- Treatment
  - Correct etiology
  - Replace Magnesium

REVIEW

Which of the following is NOT a cause of hypercalcemia?

a. Hydrochlorothiazide Therapy.
b. Sarcoidosis.
c. Pulmonary Malignancy.
d. Secondary Hyperparathyroidism.

REVIEW

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a. Hydrochlorothiazide Therapy.
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ACID-BASE PHYSIOLOGY

$$H^+ + HCO_3^-$$

$$=$$

$$H_2CO_3$$

$$=$$

$$CO_2 + H_2O$$

PHYSIOLOGY

- Buffers
  - Minimize pH changes
  - H^+/HCO_3^-; NH_3/NH_4^+, Organic Acids
  - Henderson-Hasselbach

$$pH = pK + \log \{[HCO_3^-] / [CO_2]\}$$
$$pH = 6.1 + \log \{[HCO_3^-] / [.03 \times p_aCO_2]\}$$
$$pH = 6.1 + \log \{24 / (40 \times .03)\}$$
$$pH = 6.1 + \log 20 = 6.1 + 1.3 = 7.40$$

COMPENSATORY MECHANISMS

- Metabolic Disorders
  - Acidosis: $pCO_2 = (1.5 \times [HCO_3^-]) + 8 (\pm 2)$
  - Alkalosis: $pCO_2 = (0.7 \times [HCO_3^-]) + 21 (\pm 1.5)$
- Respiratory Disorders
  - Alkalosis
    - Acute: $HCO_3^- = \{(pCO_2 - 40)/10\} + 24$
    - Chronic: $HCO_3^- = \{(pCO_2 - 40)/3\} + 24$
  - Acidosis
    - Acute: $HCO_3^- = \{(40 - pCO_2)/5\} + 24$
    - Chronic: $HCO_3^- = \{(40 - pCO_2)/2\} + 24$

ANION GAP ACIDOSIS

- Anion Gap (AG)

  $AG = [Na] - \{[Cl] + [HCO_3^-]\}$

- Normal AG = 3 – 11
- Hypoalbuminemia
  - Can significantly lower the Anion Gap
  - May disguise an Anion gap Acidosis as Non-gap

ANION GAP ACIDOSIS

- Specific Etiologies
  - Ketoacidosis (Diabetic, Alcoholic, Starvation)
  - Lactic Acidosis
  - Alcoholic Ketoacidosis
  - Toxins (Salicylate, methanol, ethylene glycol)
  - Renal Failure/Uremia

NON-ANION GAP ACIDOSIS

- Cause
  - Loss of HCO_3^- or addition of H^+ and Cl^-
  - AG is not widened secondary to matching by Cl^-
- Specific Etiologies
  - GI Loss of Bicarbonate
    - Diarrhea
    - Urinary Diversion
    - Fistula (Small Bowel, Pancreatic, Biliary)
  - Renal Loss of Bicarbonate
    - RTA
    - Renal Insufficiency
    - Recovery phase of DKA
  - Acidifying Substances: HCl, NH_4Cl, Sulfur, etc

DELTA GAP

- Identifies mixed metabolic abnormalities
- Delta Gap = AG_{Calc} - AG_{Normal}
- Delta Gap ~ Actual Change in [HCO_3^-]
- Interpretation
  - If [HCO_3^-] decreased more than expected; accompanying non-anion gap acidosis
  - If [HCO_3^-] increased more than expected; accompanying metabolic alkalosis

DELTA GAP

- Example
  - Na = 140 K = 4.0 Cl = 113
  - ABG: 7.20/17/115/6
- Solution
  - AG = 21
  - Delta Gap = 21 - 10 = 11
  - Actual [HCO_3^-] Change = 24 – 6 = 18
- Diagnosis:
  - [HCO_3^-] should have only gone down by 11 to 13, but it actually decreased more (by 18) down to 6, SO:
  - The diagnosis is a Mixed Anion Gap Metabolic Acidosis and Non-Anion Gap Metabolic Acidosis

METABOLIC ALKALOSIS

- Cause: Addition of alkali or loss of H^+
- Role of Chloride: Determines etiology
- Adverse Effects
  - Neurologic: Can lead to Seizures
  - Hypoventilation: Compensatory; results in Cerebral Vasoconstriction
  - Tissue oxygenation
    - Ionized calcium: Decreases; result is impaired myocardial contractility
    - Oxyhemoglobin curve: Shifts left; decreasing hemoglobin ability to release oxygen in tissues
    - Glycolysis: Stimulates; increases tissue oxygen requirements

CHLORIDE-RESPONSIVE METABOLIC ALKALOSIS

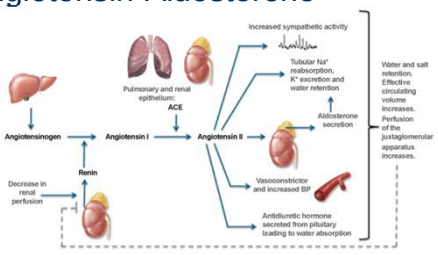
- Characteristics
  - Urine Chloride < 20
  - Volume depletion responsive to fluid and chloride replacement
- Specific Etiologies
  - Loss of gastric acid (i.e. emesis, NG suction)
  - Diuretic therapy
  - Volume depletion
  - Posthypercapnia
  - Villous adenoma

CHLORIDE-RESISTANT METABOLIC ALKALOSIS

- Characteristics
  - Urine Chloride > 30
  - Volume expansion
- Specific Etiologies
  - Mineralocorticoid Excess (e.g. Conn's syndrome, Cushing's syndrome, drugs, renal artery stenosis)
  - Potassium depletion

CONTRACTION ALKALOSIS – HOW?

- Volume Depletion PROMOTES Metabolic Alkalosis
  - Loss free water concentrates HCO_3^- stores and raises $[HCO_3^-]$
  - Stimulation of Renin-Angiotensin-Aldosterone Axis
    - Loss of H^+
    - Loss of K^+



RESPIRATORY ACIDOSIS

- Etiology
  - Ventilatory Failure
  - Disorder central control of ventilation
- Compensation
  - Acute: $\Delta pH = .008 \times (pCO_2 - 40)$
  - Chronic: $\Delta pH = .003 \times (pCO_2 - 40)$
- Treatment
  - Underlying Disorder
  - Permissive Hypercapnia: Well tolerated

RESPIRATORY ALKALOSIS

- Etiology
  - Hypoxia
  - Increased CNS Respiratory Drive
  - Pulmonary Disorders
- Compensation
  - Acute: $\Delta pH = .008 \times (40 - pCO_2)$
  - Chronic: $\Delta pH = .017 \times (40 - pCO_2)$
- Treatment
  - Underlying Disorder

REVIEW

A 65 y/o insulin-dependent diabetic female is brought to the ER confused, ill, and vomiting for several days. She is on no other medications and her laboratory panel is below. What is her acid-base profile?

Na 132 meq/L K 2.8 meq/L Cl 80 meq/L HCO_3^- 16 meq/L Glucose 450 mg/dL
pH 7.39 pO_2 133 mm Hg pCO_2 32 mm Hg

a. Nonanion Gap Metabolic Acidosis.
b. Anion Gap Metabolic Acidosis with a concurrent Metabolic Alkalosis.
c. Anion Gap Metabolic Acidosis with a concurrent Nonanion Gap Metabolic Acidosis.
d. Anion Gap Metabolic Acidosis with a concurrent Respiratory Alkalosis.

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- Parrillo JE, Dellinger RP. Critical Care Medicine. 2nd Edition. Mosby. St Louis 2002.

QUESTIONS

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RENAL PHYSIOLOGY & RENAL FAILURE (Acute & Chronic)

Surgical Critical Care

Gregory B. York, MD, FACS
Trauma/Critical Care

PHYSIOLOGY

- Preservation of Intravascular Volume
- Maintenance of Acid-Base Status
- Removal of Metabolic Products
- Cardiovascular/Endocrine Functions

FILTRATION - EXCRETION - SECRETION

GLOMERULAR FILTRATION

- First Process in Urine Formation
- Results in a filtrate that is:
  - Low in protein
  - Contains solutes not bound to non-filterable proteins
- Normal (adult) GFR: 100-125 ml/min

TUBULAR REABSORPTION

- Occurs in renal tubule
- From the filtrate - reabsorbs approximately 98% of:
  - Water
  - Solutes
- Most occurs in proximal tubule – 67%-88%
- Mechanisms: Active & Passive

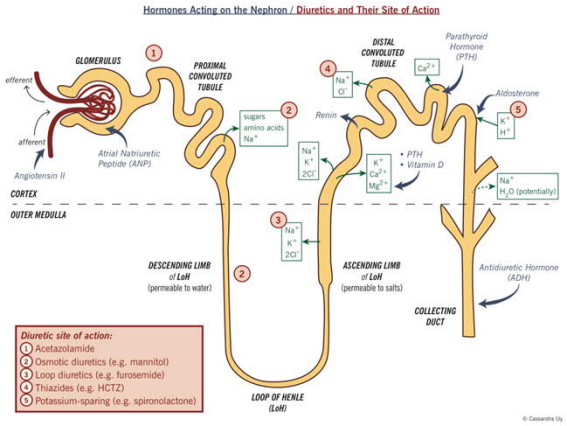
TUBULAR REABSORPTION

- Passive
  - Water, urea
  - So . . . . Vascular volume DEPENDS on Na absorption.
- Active
  - Glucose, phosphate, sulfate, amino acids
  - Sodium

TUBULAR REABSORPTION

- Distal Nephron
  - Much less water & solute reabsorption
  - Responds MORE to local or systemic alterations
    - Mineralocorticoids
    - Diuretics

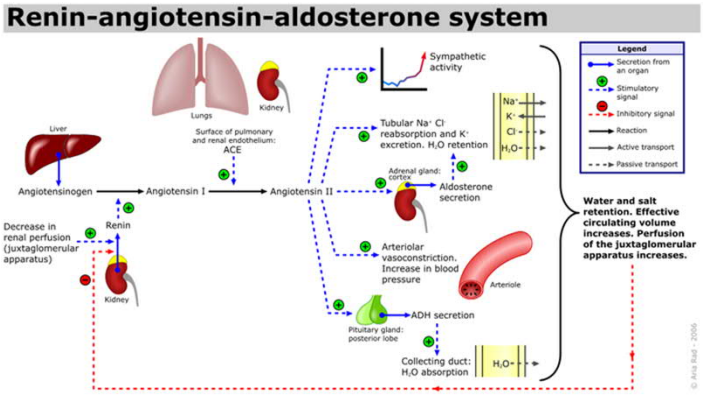
PHYSIOLOGY – SUMMARY



REABSORPTION OF SODIUM

- Major active transport mechanism of entire nephron
- Three Determinants
  - GFR: High GFR = lower reabsorption (time available)
  - Aldosterone: Augments Na reabsorption in distal tubule
  - Atrial Natriuretic Factor (ANF): Lower reabsorption

RENIN-ANGIOTENSIN-ALDOSTERONE



FILTRATION – EXCRETION – REABSORPTION
WATER – ELECTROLYTES - SOLUTES

Substance	Measure	Filtered	Excreted	Reabsorbed	Filtered Load Reabsorbed (%)
Water	L/day	180	1.5	178.5	99.2
Na	mEq/day	25,200	150	25,050	99.4
K	mEq/day	720	100	620	86.1
Ca	mEq/day	540	10	530	98.2
HCO_3	mEq/day	4320	2	4318	99.9+
Cl	mEq/day	18,000	150	17,850	99.2
Glucose	mmol/day	800	0.5	799.5	99.9+
Urea	g/day	56	28	28	50.0

ACUTE RENAL FAILURE (ARF)

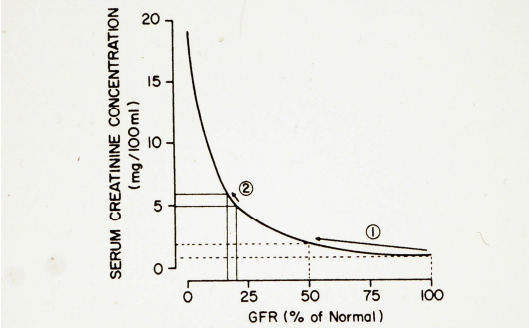
- Deterioration of renal function resulting in:
  - Accumulation of waste products
  - Imbalance in homeostasis
- Associated Factors
  - Pre-existing renal conditions
  - Systemic medical problems
  - Age
- Categories
  - Pre-renal: $FE_{Na} < 1\%$; $U_{Na} < 20$ mEq/L; $U_{OsM} > 500$
  - Post Renal
  - Renal (Intrinsic): $FE_{Na} > 1\%$; $U_{Na} > 40$ mEq/L; $U_{OsM} < 350$
- $FE_{Na} = [(U_{Na} \times P_{Cr}) / (U_{Cr} \times P_{Na})] \times 100$

ACUTE RENAL FAILURE

- Risk Factors – Acute
  - Volume depletion
  - Aminoglycosides
  - Radiocontrast dye
  - Sepsis
  - Dehydration
  - Hypotension
  - Pigments
- Risk Factors – Chronic
  - Pre-existing renal disease
  - Hypertension
  - CHF
  - DM

ACUTE RENAL FAILURE

- Function declines prior to laboratory changes
- GFR vs Creatinine



PRERENAL ARF

- Decreased renal perfusion
  - Intravascular volume depletion
  - Decreased effective blood volume
    - Congestive Heart Failure
    - Cirrhosis
    - Nephrotic syndrome
  - Renal vasoconstriction
    - Hepatorenal syndrome
    - Hypercalcemia
    - NSAIDs
    - Drugs: Tacrolimus, Cyclosporin A

POST RENAL ARF

- Lower tract obstruction
  - BPH
  - Bladder cancer/stones
  - Blood clot
  - Neurogenic bladder
  - Prostate cancer
  - Urethral stricture
- Upper tract obstruction (must be bilateral; unless only one kidney)
  - Aortic aneurysm
  - Blood clot
  - Kidney stone
  - Pelvic malignancy
  - Renal papillary necrosis
  - Retroperitoneal fibrosis/tumor
  - Transitional cell carcinoma

INTRINSIC ARF

- Acute Interstitial Nephritis
- Acute Glomerulonephritis
- Intratubular Obstruction
- Acute Vascular Syndromes
- Acute Tubular Necrosis (ATN)

ACUTE INTERSTITIAL NEPHRITIS

- Acute Interstitial Nephritis
  - Inflammation of renal interstitium & tubules
  - Eosinophilic infiltrate
  - Clinical triad: Fever, rash, eosinophilia
  - Drug hypersensitivity: Penicillins, cephalosporins, sulfa drugs, diuretics, and anticonvulsants.

ACUTE GLOMERULONEPHRITIS

- Acute (over hours) or Subacute (over days/weeks)
- Goodpasture's
  - Anti-basement membrane Ab
  - Associated with pulmonary hemorrhage
- Hematuria and RBC casts
- Diagnosis: Renal Bx

INTRATUBULAR OBSTRUCTION

- Crystal deposition
- Paraproteins
- Tumor Lysis Syndrome
  - Hyperkalemia
  - Hyperphosphatemia
  - Severe hypeurecemia
  - Many uric acid crystals
- Multiple Myeloma: Immunoglobulin light chains, hypercalcemia, hyperuricemia

ACUTE VASCULAR SYNDROMES

- Renal artery thromboembolism
- Malignant hypertension (arteriolar spasm)
- Scleroderma
- Atheroembolic disease
- Renal vein thrombosis (rare cause)

ACUTE TUBULAR NECROSIS (ATN)

- Pathophysiology
  - Oxidative injury to renal tubular epithelial cells
  - Sloughing of cells into the lumen
  - Sloughed cells create obstruction
  - Tubular pressure increases
  - Net filtration across glomerular capillaries decreases
  - GFR decreases

ACUTE TUBULAR NECROSIS (ATN)

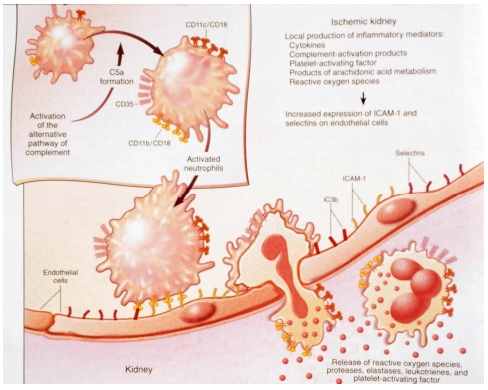
- Causes more than 50% of ICU oliguria
- Etiology
  - Ischemic
    - Circulatory shock
    - Sepsis
    - Hypotension (any cause)
    - Drugs causing hypoperfusion (ie. ACE inhibitors, NSAIDS)
  - Nephrotoxic
    - Drugs (ie. Aminoglycosides, Amphotericin B, Cisplatin, etc)
    - Radiocontrast dye
    - Myoglobin

ACUTE TUBULAR NECROSIS (ATN)

- Ischemic
  - Most common pathophysiologic factor
  - May have no cardiovascular symptoms
  - Hypotension present < 50% of cases
  - Renal medulla susceptible (Loops of Henle)
  - Systemic hypoperfusion vs renal response to cytokines (vasoconstriction & vasodilation)

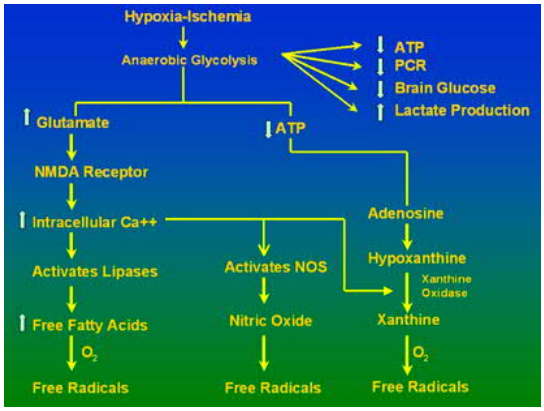
ACUTE TUBULAR NECROSIS (ATN)

- Ischemia/Reperfusion Injury



ACUTE TUBULAR NECROSIS (ATN)

- Mechanisms of Cell Death



ACUTE TUBULAR NECROSIS (ATN)

- Nephrotoxic
  - Direct tubular cell injury
  - Etiology
    - Intrinsic
      - Pigments: Hemoglobin, Myoglobin
      - Crystals: Uric acid, Calcium, Oxalate
      - Malignancy: Tumor lysis, Myeloma
    - Extrinsic
      - Antibiotics
      - Heavy metals
      - Chemicals/Poisons
      - Contrast media
  - Damage regulated by multiple factors

ACUTE TUBULAR NECROSIS (ATN)

- Gradual Onset (days)
- Initial rise in BUN and/or Creatinine
- Other abnormal parameters
  - FeNa
  - CrCl
- Urine Output
  - Variable
  - Oliguric vs Non-oliguric

ACUTE TUBULAR NECROSIS (ATN)

- Oliguria
  - Defined as < 400 ml/day
- Non-Olioguric
  - Normal to as high as 8 L/day
  - More often due to Nephrotoxins
  - Fewer complications
  - Mortality lower
    - Oliguric: 60% - 90%
    - Non-oliguric: 10% - 20%

ACUTE TUBULAR NECROSIS (ATN)

- Effects
  - Decreased GFR, Reabsorption, & Clearance
    - Increased nitrogenous wastes
    - Effects other clearances
  - Homeostasis Imbalance
    - Body water content/electrolytes
    - Acid-Base
    - Coagulopathy
  - Metabolic
    - Hypercatabolic state

ACUTE TUBULAR NECROSIS (ATN)

- Complications
  - Hyperkalemia
  - Metabolic Acidosis
  - Electrolyte Imbalance
  - Excess total body water
  - Malnutrition
  - Abnormal drug metabolism
  - Uremia

ACUTE TUBULAR NECROSIS (ATN)

- Characterics/Lab Values
  - Oliguric or Non-oliguric
  - Urine concentrating ability is lost
  - Urine diluting ability is lost
  - Osmolality is 300 mOsm/kg
  - Specific Gravity is 1.01
- Sodium
  - Reabsorption is impaired.
  - U_{Na} > 40 mEq/L
  - FE_{Na} > 1%.

ACUTE TUBULAR NECROSIS (ATN)

- Prevention
  - Correct/maintain volume/perfusion
  - Limit/eliminate nephrotoxins
  - Diuretics (ie. Furosemide)
    - Little/no chance it will reach site of action
    - If used – should be given in continuous infusion
  - Dopamine
    - No evidence of benefit in patients with acute oliguric renal failure
    - Negative: Hemodynamics, immune function, endocrine function (dec TSH)

ACUTE TUBULAR NECROSIS (ATN)

- Management
  - Reverse the cause
  - Optimize renal perfusion
  - Remove renal toxins
  - Adjust drugs for renal failure
  - Serum electrolytes
    - Potassium, Phosphate, Magnesium
    - Acid-Base Status
  - Nutrition: Protein 1.0-1.5 gm/kg/day

RENAL REPLACEMENT THERAPY

- Indications
  - Uremia
  - Volume overload
  - Metabolic Acidosis
  - Hyperkalemia
- Transport Principles
  - Fluid: Ultrafiltration
  - Solute: Convection vs Diffusion
- CRRT vs IHD
  - Renal Recovery: Some studies show better with CRRT
  - Mortality: No difference

RENAL REPLACEMENT THERAPY

- Ultrafiltration
  - Movement of fluid across semipermeable membrane secondary to pressure gradient
- Diffusion
  - Solute transport across semipermeable membrane
  - Movement based on concentration gradient
  - Best for small molecule clearance; < 300 daltons
- Convection
  - Solute transport across semipermeable membrane
  - Solute moves WITH solvent in response to transmembrane pressure
  - Removes broad spectrum of solute sizes

HEMODIALYSIS

- Diffusion: Driven by concentration gradient of solute across membrane
- Countercurrent Exchange
- Blood/dialysis fluid move in opposite directions
- Advantages
  - Rapid clearance of solutes
  - Only a few hours are needed
- Disadvantages
  - Need at least 300 mL/min blood flow
  - Hypotension

HEMOFILTRATION

- Convection: Solute-containing fluid moves across permeable membrane due to pressure difference
- Solvent Drag
- Must be performed continuously
- Advantages
  - High flow rates not needed
  - Less risk hypotension
  - Gradual removal of solutes/more physiological
- Disadvantages
  - Slower process
  - Requires anticoagulation

CONTINUOUS RRT

- CVVH (or CAVH)
  - Achieve desired volume through ultrafiltration (rates up to 24L/day)
  - Clears solutes through convection
  - Requires replacement fluid to avoid volume depletion/large shifts; solutes small to larger size > 300 daltons
  - May clear midrange molecules (ie. sepsis) vs CAVHD
- CVVHD (or CAVHD)
  - Achieves desired volume through ultrafiltration (rates up to 8L/day)
  - Clears solutes through countercurrent dialysis
  - Requires dialysate; no replacement fluid; small solutes < 300 daltons
  - Safer/simpler than CAVH

REFERENCES

- Marino PL. The ICU Book. 3rd Edition. Lippincott Williams & Wilkins. Philadelphia. 2007.
- Parrillo JE, Dellinger RP. Critical Care Medicine. 2nd Edition. Mosby. St Louis 2002.

QUESTIONS

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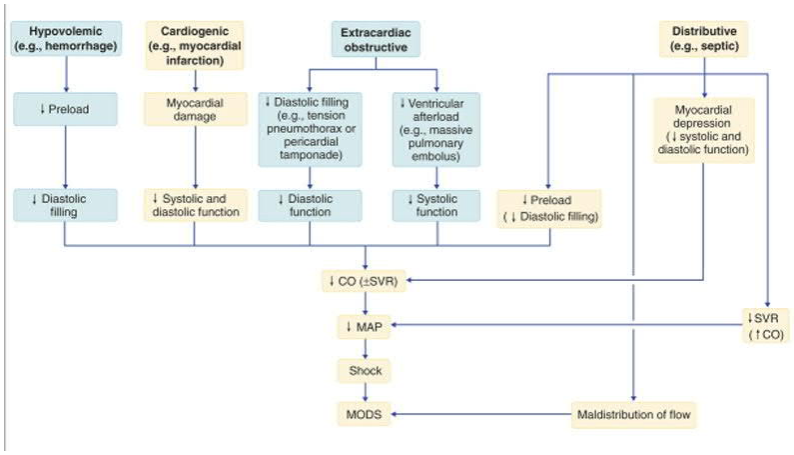
SIRS, MODS & SEPSIS
STANDARDS OF CARE
Surgical Critical Care

Gregory B. York, MD, FACS
Trauma/Critical Care

SHOCK

State in which the delivery of oxygen and metabolic substrates to tissues and cells is insufficient to maintain normal aerobic metabolism and leads first to reversible, and then, if prolonged, to irreversible cellular injury

CLASSIFICATION OF SHOCK



DEFINITIONS

- Obstructive: Impaired cardiac output caused by physical obstruction of blood flow
- Cardiogenic: Inadequate tissue perfusion resulting from myocardial dysfunction
- Hypovolemic: Inadequate tissue perfusion resulting from an absolute deficiency of intravascular blood volume
- Distributive: Inappropriate distribution of blood volume with inadequate organ and tissue perfusion

PATHOPHYSIOLOGY – OVERVIEW

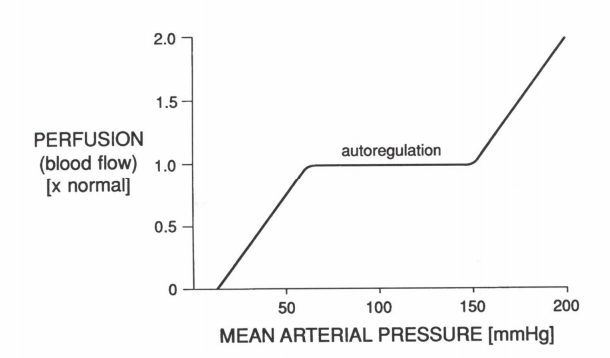
- Hemodynamic Basis of Shock
  - Mean Arterial Pressure
  - Cardiac Output
  - Starling Curves
  - Venous Return in Shock
- Microvascular Function in Shock
  - Vasomotor Dysfunction
  - Endothelial Cell Dysfunction
- Mechanism of Cellular Injury

MEAN ARTERIAL PRESSURE

- CO = [MAP-CVP]/SVR
- MAP DEPENDS on:
  - CO
  - SVR
- End-organ vascular supply DEPENDS on:
  - Maintenance of normal BP
  - Within a defined range
  - i.e. AUTOREGULATION

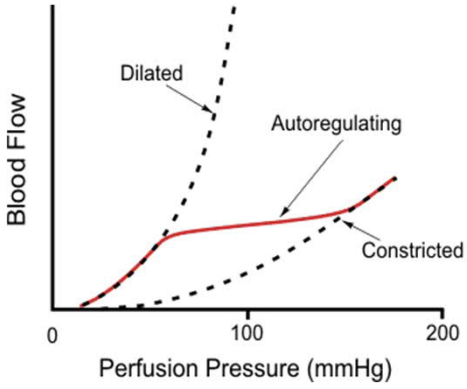
AUTOREGULATION

- Within range, end-organ perfusion is relatively constant



FAILURE OF AUTOREGULATION

- Outside the range, end-organ perfusion is a function of:
  - MAP
  - Passive mechanical properties of the blood vessel



AUTOREGULATION

Failure to maintain minimum MAP for autoregulation is indicative of a severe reduction in cardiac output

CARDIAC OUTPUT

- CO = HR x SV
- SV DEPENDS on:
  - Preload
    - Degree of myocardial fiber stretch before contraction
    - Physiologic parameter: EDV (End Diastolic Volume)
  - Afterload
    - Total sum forces that oppose ejection of blood from the ventricle
    - Physiologic parameter: SVR (Systemic Vascular Resistance)
  - Contractility
    - Intrinsic ability of myocardial fibers to shorten under given loading conditions
    - Physiologic parameter: PSP/ESV (Peak Systolic Pressure/ End Systolic Volume)

CARDIAC OUTPUT

- Preload
  - Decreased: Hypovolemic, Distributive
  - Increased: Cardiogenic, Obstructive
- Afterload
  - Decreased: Distributive
  - Increased: Hypovolemic, Cardiogenic, Obstructive
- Contractility
  - Decreased: Hypovolemic, Cardiogenic, Obstructive
  - Increased: Distributive

REVIEW

The central feature, common to all types of shock is:

a. Low systemic vascular resistance.
b. Low cardiac output.
c. Cellular ischemia.
d. Diminished tissue oxygen delivery.

REVIEW

The central feature, common to all types of shock is:

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b. Low cardiac output.
c. Cellular ischemia.
d. Diminished tissue oxygen delivery.

HEMODYNAMIC PROFILE

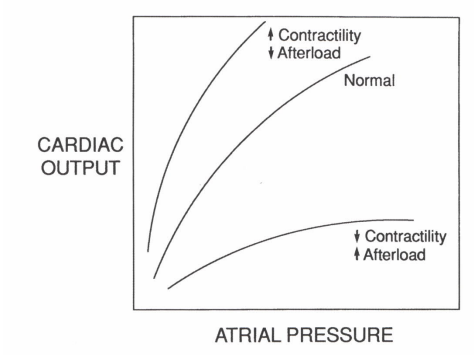
- A Hemodynamic Profile consists of:
  - Cardiac Function Curve
  - Venous Return Curve
- Cardiac Function and Venous Return:
  - Can be independently regulated
  - Are INTERDEPENDENT
- Changes in either are balanced via the Frank-Starling Mechanism

FRANK-STARLING MECHANISM

- The ability of the heart to change its force of contraction & therefore stroke volume in response to changes in venous return
- Independent of neural and humoral influences

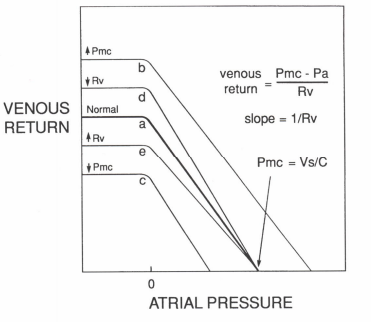
STARLING CURVE

- Ventricle
  - Does NOT operate on one curve
  - Operates on a family of curves defined by:
    - Inotropic State
    - Afterload
- "Old Heart"
  - Inc Afterload
  - Dec Inotropy
  - Down & Right
- "Athletic Heart"
  - Dec Afterload
  - Inc Inotropy
  - Up & Left



VENOUS RETURN

- Flow of blood back to the heart
- Components
  - Pmc = Peripheral venous pressure
  - Pa = Arterial pressure
  - Rv = Venous Resistance
- As resistance decreases, venous return increases



MICROVASCULAR FUNCTION

- Vasomotor Dysfunction
- Endothelial Cell Dysfunction

MICROVASCULAR FUNCTION
VASOMOTOR DYSFUNCTION

- End-organ beds are affected by:
  - Vasodilating substances: TNF-α, IL-1
  - Vasoconstrictive substances: Catecholamines
- Autonomics >>> Autoregulation
- Adaptive mechanisms
  - Preserve: Heart, Brain
  - Sacrifice: Liver, Kidneys, Splanchnic, Skin

MICROVASCULAR FUNCTION
ENDOTHELIAL CELL DYSFUNCTION

- "Capillary Leak" as a result of:
  - Free radicals – "activated" neutrophils (IL-6)
  - Vasoactive mediators – Histamine, Bradykinin, PAF, Leukotrienes, TNF-α, IL-1
- Plasma Protein Loss leads to:
  - Hypovolemia
  - Interstitial edema
  - ARDS

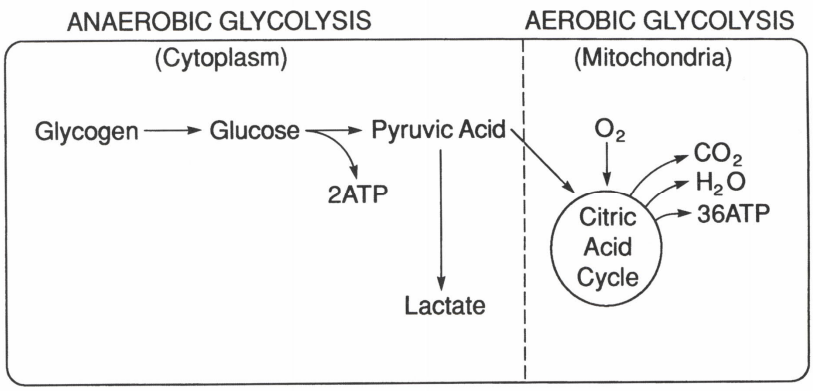
CELLULAR INJURY/ORGAN FAILURE

- Cellular Ischemia
- Inflammatory Mediators
- Free Radical Injury

CELLULAR INJURY/ORGAN FAILURE
CELLULAR ISCHEMIA

- Hypoperfusion/Ineffective perfusion
- Anaerobic Metabolism
- ATP Demands > > > ATP Supply
- Na/K Pump Failure
- Na/Water influx
- Organelle swelling/rupture
- Cell Death

CELLULAR INJURY/ORGAN FAILURE
CELLULAR ISCHEMIA



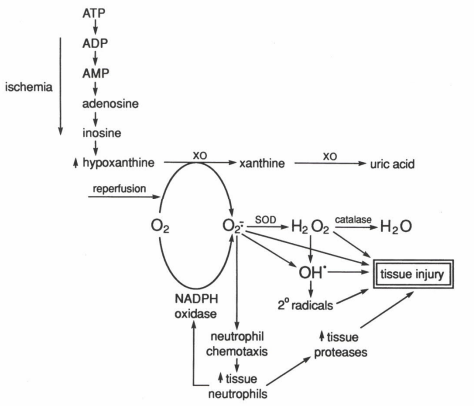
CELLULAR INJURY/ORGAN FAILURE
INFLAMMATORY MEDIATORS

- Circulating or Local: TNF-α, IL-1, IL-6
- Myocardial Depression
  - Seen in Sepsis
  - Mediated by TNF-α and IL-1β
- Nitric Oxide
  - Inducible NO synthetase
  - Results in large amounts of NO production
  - Systemic vasodilation

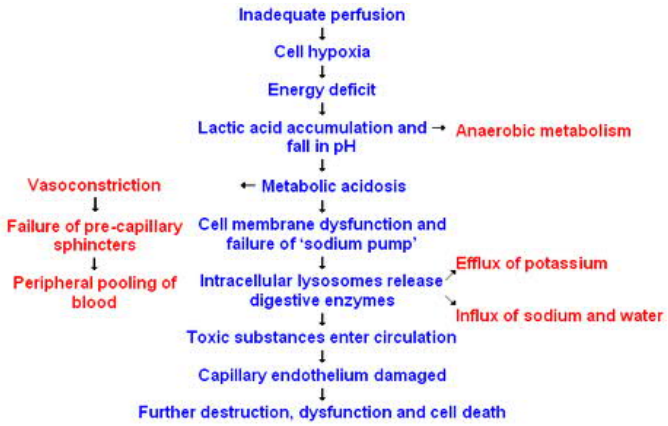
CELLULAR INJURY/ORGAN FAILURE
FREE RADICAL INJURY

- Sources
  - Reperfusion/washout
  - Neutrophil activation (IL-6, IL-8)
- Chain: Superoxide anion to Hydrogen Peroxide to Hydroxyl Radical
- Hydroxyl Radical destroys lipid membranes
- Cell rupture/death

CELLULAR INJURY/ORGAN FAILURE
FREE RADICAL INJURY



PATHOPHYSIOLOGY – SUMMARY



EXTRACARDIAC OBSTRUCTIVE SHOCK

- Characteristics
- Etiology
- Presentation
- Clinical Management

EXTRACARDIAC OBSTRUCTIVE SHOCK
CHARACTERISTICS

- Obstruction to flow within the cardiac circuit
- Either impairment of diastolic filling or excessive afterload

EXTRACARDIAC OBSTRUCTIVE SHOCK
ETIOLOGY

- Impaired Diastolic Filling
  - Direct venous obstruction
  - Increased intrathoracic pressure
    - Tension pneumothorax
    - Mechanical ventilation (e.g. PEEP)
    - Asthma (with auto-PEEP)
  - Decreased cardiac compliance
    - Constrictive pericarditis
    - Cardiac tamponade
- Impaired Systolic Contraction
  - Right ventricle
    - Pulmonary embolus
    - Pulmonary hypertension
  - Left ventricle
    - Saddle embolus
    - Aortic Dissection

EXTRACARDIAC OBSTRUCTIVE SHOCK
PRESENTATION

- History – Usually indicative of etiology
- Signs: Cardiac failure usually prevails secondary to inciting event
- Examination: Tachycardia, tachypnea, hypotension, altered mental status

EXTRACARDIAC OBSTRUCTIVE SHOCK
MANAGEMENT

- ABCs of resuscitation
- Volume therapy
- Emergency intervention for life-threatening etiologies
- Hemodynamic monitoring
- Use of PAC
- Use of vasopressors/inotropes

HYPOVOLEMIC SHOCK

- Characteristics
- Etiology
  - Hemorrhagic
  - Fluid Depletion (non-hemorrhagic)
  - Increased Vascular Capacitance
- Pathophysiology – Starling Curve
- Presentation
- Stages of Hemorrhagic Shock
- Management

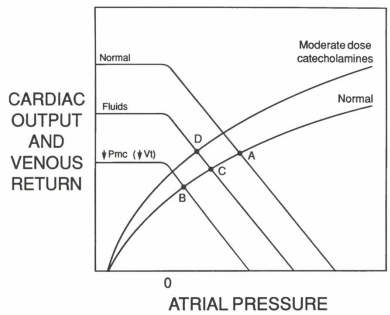
HYPOVOLEMIC SHOCK
CHARACTERISTICS

- Decreased circulating blood volume in relation to the total vascular capacity
- Reduction of diastolic filling pressures & volumes

HYPOVOLEMIC SHOCK
ETIOLOGY

- Hemorrhagic – most common
  - Trauma
  - GI
  - Retroperitoneal
- Fluid Depletion (non-hemorrhagic)
  - External loss: Dehydration, vomiting, diarrhea, polyurea
  - Internal redistribution: Thermal injury, trauma, anaphylaxis
- Increased Vascular Capacitance
  - Sepsis
  - Anaphylaxis
  - Toxins/Drugs

HYPOVOLEMIC SHOCK
STARLING CURVE



A – B: Loss of volume
B – C: Volume therapy
C – D: Sympathetic and catecholamine augmentation of cardiac output

HYPOVOLEMIC SHOCK
PRESENTATION

- History: May be indicative (e.g. trauma, etc.)
- Signs are apparent (e.g. hemorrhage, etc.)
- Examination
  - Physical: Cool, clammy skin; altered mental status; anxiety/agitation.
  - Vitals: Tachycardia; decreased BP, low urine output

HEMORRHAGIC SHOCK
STAGES

	I	II	III	IV
Blood Loss (%) (mL)	≤ 15% ≤ 750	15-30 750-1500	30-40 1500-2000	≥ 40 ≥ 2000
HR	< 100	100-120	120-140	> 140
BP	Normal	Normal	↓	↓↓
RR	14-20	20-30	30-40	> 35
UOP (mL/hr)	> 30	20-30	5-15	Negligible
MS	Slightly Anxious	Mildly Anxious	Anxiety & Confusion	Confusion & Lethargy

HYPOVOLEMIC SHOCK
MANAGEMENT

- Initial: "ABCs of resuscitation"
- Fluid: Crystalloid followed by packed RBCs
- Continuous hemodynamic monitoring
- Surgical intervention as needed
- Use of PAC
- Vasopressors/Inotropes

REVIEW

A 65 y/o male is involved in a head-on collision with a telephone pole. The medics found empty bottles of beer in his car. His airway is clear and respiratory rate is 28 breaths/min with equal breath sounds bilaterally, but with poor air entry. His BP is 80/40 mm Hg, HR 125 bpm, and GCS 15. There is no movement below the C-5 spinal level and priapism is noted. Which one of the following types of shock is the patient most likely experiencing?

a. Cardiogenic Shock.
b. Hypovolemic Shock.
c. Neurogenic Shock.
d. Septic Shock.

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CARDIOGENIC SHOCK

- Characteristics
- Etiology
- Pathophysiology – Starling Curve
- Presentation
- Cardiogenic Shock “Death Spiral”
- Management

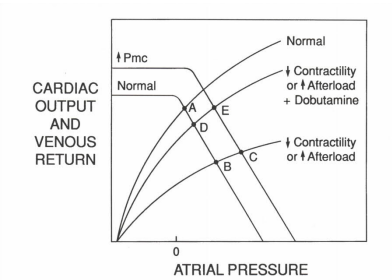
CARDIOGENIC SHOCK
CHARACTERISTICS

- Cardiac pump failure secondary to:
  - Loss of myocardial contractility/functional myocardium or,
  - Structural/mechanical failure of cardiac anatomy
- Elevation of diastolic filling pressures/volumes

CARDIOGENIC SHOCK
ETIOLOGY

- Myopathic
  - MI
  - Myocardial Contusion
  - Myocarditis
  - Cardiomyopathy
  - Pharmacologic
  - Septic depression
- Mechanical
  - Valvular failure
  - Hypertrophic cardiomyopathy
  - VSD
- Arrhythmic
  - Bradycardia
    - Sinus
    - AV Block
  - Tachycardia
    - SVT
    - Ventricular

CARDIOGENIC SHOCK
STARLING CURVE

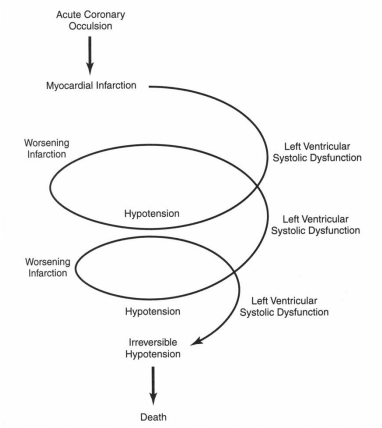


A – B: Decreased contractility or increased afterload
B – C: Volume therapy only
B – D: Dobutamine only
B – E: Volume therapy and Dobutamine

CARDIOGENIC SHOCK
PRESENTATION

- Cardiac History: May be indicative
- Examination
  - Tachycardia; hypo/hypertension; tachypnea; peripheral edema
  - Signs of pulmonary congestion
- CXR: Fluid overload

CARDIOGENIC SHOCK
DEATH SPIRAL



CARDIOGENIC SHOCK
MANAGEMENT

- Initial: “ABCs of resuscitation”
- Treat underlying cause (e.g. MI, etc.)
- Correct hypovolemia, hypoxemia, and acidosis
- Continuous hemodynamic monitoring
- Avoidance of negative inotropes
- May need PAC guidance
- Use of vasodilators/afterload reducers & inotropes

DISTRIBUTIVE SHOCK

- Characteristics
- Etiology
- SIRS to Septic Shock
- Pathophysiology – Starling Curve
- Presentation
- Management
- Goal Directed Therapy

DISTRIBUTIVE SHOCK
CHARACTERISTICS

- Loss of vasomotor control resulting in arteriolar and venular dilation
- Increased CO with decreased SVR (after fluid resuscitation)

DISTRIBUTIVE SHOCK
ETIOLOGY

- Septic
- Anaphylactic
- Neurogenic
- Endocrinologic
- Toxic

SIRS

- Presence of ≥ 2 of the following:
  - Temperature < 36.8ºC or > 38.8ºC
  - Heart Rate > 90 bpm
  - Respiratory Rate > 20 breaths/min or pCO_2 < 32 mm Hg
  - Elevated (or depressed) WBC
    - > 12,000 cells/μL OR
    - < 4,000 cells/μL OR
    - > 10% immature forms
- Clinical syndrome that is a form of dysregulated inflammation
- Excluded as part of Sepsis definition in 2016, since SIRS is not always caused by infection

SEPSIS

- 2016 SCCM/EISCM Consensus
- Life-threatening organ dysfunction caused by a dysregulated host response to infection
- Exists on a continuum of severity
  - Infection & bacteremia to sepsis & septic shock
  - Can lead to MODs & death
- Organ dysfunction: An increase of two or more points in the SOFA score.
- Severe Sepsis: Term no longer used.

SOFA

- Sequential (sepsis-related) Organ Failure Assessment
- Organ Dysfunction: Increase in score by $\geq$ 2 points

Points	1	2	3	4
Neurological (GCS)	13-14	10-12	6-9	<6
Respiratory (P/f ratio)	<400	<300	<200 (+ resp support)	<100 (+resp support)
Cardiovascular (Heart Rate, bpm)	<70	dopamine ≤5 or dobutamine (any dose)	dopamine >5 or EPI ≤0.1 or NOREPI ≤0.1	dopamine >15 or EPI >0.1 or NOREPI >0.1
Renal (Cr or UOP)	110-170	171-299	300-440 (or < 500 ml/day)	>440 (or < 200 ml /day)
Hematologic (Platelet Ct)	<150	<100	<50	<20
Liver (Bilirubin)	20-32	33-101	102-204	>204

MEWS

- Modified Early Warning Score
- Sepsis: MEWS ≥ 5 + Infection (Medium risk 2-4)

Points	0	1	2	3
SBP (mm Hg)	101-199	81-100	71-80 ≥ 200	≤ 70
HR (bpm)	51-100	41-50 101-110	< 40 111-129	≥ 130
RR (bpm)	9-14	15-20	21-29 ≤ 9	≥ 30
Temp (ºC/ºF)	35-38.4 / 95-101.1		< 35 / 95 > 38.5 / 101.3	
AVPU	Alert	Reacts to voice	Reacts to pain	Unresponsive

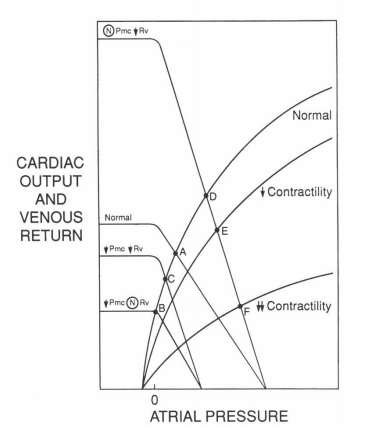
SEPTIC SHOCK

Sepsis + Circulatory, Cellular, And Metabolic Abnormalities that are associated with a greater risk of mortality than Sepsis Alone.

SEPSIS

- Risk Factors
  - ICU Admission
  - Bacteremia
  - Advanced age (≥ 65 yrs)
  - Immunosuppression
  - Diabetes and Cancer
  - Community acquired pneumonia
  - Previous hospitalization
  - Genetic factors

SEPTIC SHOCK
STARLING CURVE



A – B: Overall volume deficit (e.g. vasodilation, third-spacing, insensible loss)
B – C: Augmentation of venous return by decreased venous resistance
C – D: Volume therapy allows hyperdynamic state to manifest
D – E: Myocardial depression seen in septic shock
E – F: Severe myocardial depression seen in 20% of patients after resuscitation

SEPTIC SHOCK
PRESENTATION

- "Warm" Shock: Hot, dry, flushed skin
- "Cold" Shock: Cold, clammy skin; lethargy; hypotension
- PE: Hx/evidence of infection/source (e.g. fever, increased WBC, infectious etiology)

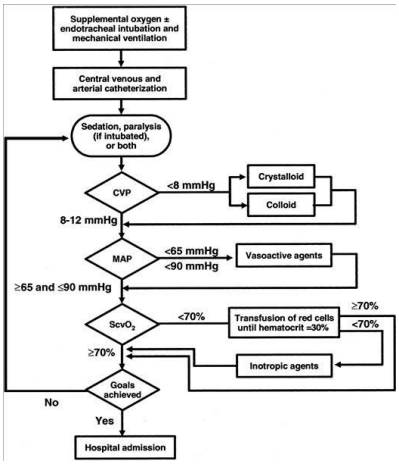
SEPTIC SHOCK
MANAGEMENT

- Initial: "ABCs of resuscitation"
- Crystalloid (balanced) fluid (30 mL/kg) w/i first 3 hours
- Continuous hemodynamic monitoring
- May need PAC guidance
- Use of vasopressors and/or inotropes
- Therapy for infectious etiology

SEPTIC SHOCK
EARLY GOAL-DIRECTED THERAPY

- Initial Resuscitation
  - Crystalloid/Colloid/Blood Products
  - End Points: Lactate, CVP, MAP, UOP, SvO_2, DO_2I
- Vasopressors
  - Hypotension + Adequate Volume
  - Norepinephrine > Dopamine
  - Vasopressin: Refractory Hypotension on Norepinephrine
  - Epinephrine: Refractory on Norepinephrine and Vasopressin
  - Cardiac Dysfunction: Dobutamine
- Empiric Antimicrobial Therapy
  - Gm-Negative Rods
  - MRSA
- Steroids: Septic Shock + Vasopressor Requirement

SEPTIC SHOCK
EARLY GOAL-DIRECTED THERAPY



NEUROGENIC SHOCK

- Etiology
- Presentation
- Management

NEUROGENIC SHOCK
ETIOLOGY

- Trauma
- Closed head injury/intracranial pathology
- Spinal cord injury/pathology

NEUROGENIC SHOCK
PRESENTATION

- Etiology may be present with history
- Warm, dry skin; bradycardia; hypotension; altered mental status

NEUROGENIC SHOCK
MANAGEMENT

- Initial: "ABCs of resuscitation"
- Correct hypovolemia
- Continuous hemodynamic monitoring
- Central venous monitoring
- May need PAC guidance
- May need vasopressor support

SPINAL SHOCK
WHAT IS IT?

- Spinal cord dysfunction
- Secondary to physiologic reasons
- Not from structural damage
- Can result in neurologic deficits
- Usually lasts 24 – 48 hrs
- Resolution comes with return of distal reflex arc (bulbocavernous reflex)

REVIEW

A 42 y/o male sustains a GSW to the abdomen. Five days following laparotomy and resection of injured small bowel, the patient develops a fever to 103º F. Overnight the patient becomes tachycardic and hypotensive. IV fluid therapy is begun, with minimal response. A pulmonary artery catheter is placed and the following readings are obtained:

Vitals: HR 125 BP 90/45		
CI 5.5 L/min/m^2	Na 141	ABG 7.35/35/140/19
SVO_2 70%	K 3.9	
SVRI 450 dyne-s-cm^5/m^2	Cl 105	Hgb/Hct 9.2/28.0
PAWP 6 mm Hg	CO_2 19	

What is the next appropriate step in the patient's management?

a. Begin IV Norepinephrine.
b. Provide a 30 cc/kg bolus of Lactated Ringers.
c. Start IV Hydrocortisone.
d. Transfuse 2U packed red blood cells.

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SHOCK
HEMODYNAMIC PROFILES

	CO	CVP	PAW	SVR	SVO_2
Cardiogenic	↓↓	↑↑	↑↑	↑	↓
Hypovolemic	↓↓	↓↓	↓↓	↑	↓
Distributive	↑↑	↓	↓	↓	↑↑
Obstructive	↓↓	↑↑	↑↑	↑	↓

SHOCK
RESUSCITATION ENDPOINTS

HEMODYNAMIC	MAP > 60-65 mm Hg
	PAW = 12 – 18
	CI > 2.1 L/min/m^2 (Cardiogenic/Obstructive)
	CI > 3.0 – 3.5 L/min/m^2 (Hypovolemic/Distributive)
OXYGEN DELIVERY	Hgb > 10 g/dL
	SaO_2 > 92%
	SvO_2 > 65%
	DO_2I > 600 mL/min/m^2
	Normalization of serum lactate (< 2.2 mEq/L)
ORGAN DYSFUNCTION	Reverse encephalopathy
	UOP > 0.5 mg/kg/hr

MULTIPLE ORGAN DYSFUNCTION SYNDROME (MODS)

- Modern Syndrome made possible by:
  - Better Comprehension
  - Better Treatment
- Common Etiologies
  - Hemorrhagic Shock
  - Severe Sepsis
  - Crush Syndrome
  - Abdominal Compartment Syndrome
  - Other Insults previously causing early death

MODS

- Early MODS
  - Less than 3 days post-injury/insult
  - Associated with higher incidence of heart failure
  - Mainly inflammatory; shock indices more frequently trigger MODS
  - Mortality: 44%
- Late MODS
  - Greater than 72 hrs post-injury/insult
  - Higher incidence of liver failure
  - Mainly sepsis; major infections more frequently trigger MODS
  - Mortality: 30%

MODS
RISK FACTORS

- Early MODS
  - Injury Severity Score (ISS) > 24
  - SBP < 90 mm Hg (in Emergency Department)
  - RBC Transfusion > 6 units (first 12 hrs)
  - Lactate > 2.5 (12 – 24 hrs postinjury)
- Late MODS
  - Age over 55 years
  - RBC Transfusion > 6 units (first 12 hrs)
  - Base Deficit > 8 mEq/L) (first 12 hrs)
  - Lactate > 2.5 (12 – 24 hrs postinjury)

MODS
PATHOPHYSIOLOGY

- Predominant Mechanism: Balance between the proinflammatory and counterinflammatory states
- Relevant Mechanisms – First Hit
  - Complement System Activation
  - Cytokines: From diverse cell types (injury site & systemic)
  - Activated Neutrophils
    - Marginate to end organs 6 – 12 hrs postinjury
    - Release NO, reactive oxygen species, IL-6, IL-8, and TNF-α
  - Reactive Oxygen Intermediates vs Antioxidant Defenses
  - Gut-derived inflammatory mediators/neutrophils
  - Resuscitation to mild or moderate systemic inflammation (primed)
- Relevant Mechanisms – Second Hit
  - Abdominal Compartment Syndrome
  - Blood Transfusion
  - Infection
  - Massive response (first hit) may eliminate need for second hit
- No second hit/massive response = resolution without incident

MULTI-ORGAN FAILURE
INTERVENTIONS

- Resuscitation Technique: Crystalloid vs Colloid
- Blood Transfusion: Judicious Use
- Fracture Management: Damage Control Ortho vs Early Total Care
- Protective Lung Ventilation
- Adrenal Insufficiency/Cortisol Replacement
- Insulin and Glycemic Control
- Immunonutrition
  - Early Enteral Nutrition (w/i 72 hrs)
  - Supplementing antioxidants (Se, Zn) and glutamine
- Continuous Renal Replacement Therapy

MULTI-ORGAN FAILURE

- Mortality
  - One Organ: 18%
  - Two Organs: 30%
  - Three Organs: 51%
  - ≥ 4 Organs: 65%

REVIEW

Systemic vascular resistance is commonly elevated in all of the following EXCEPT:

a. Hemorrhagic Shock.
b. Cardiogenic Shock.
c. Septic Shock.
d. Cardiac Tamponade.

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QUESTIONS

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BASIC IMMUNOLOGY AND ORGAN TRANSPLANTATION

September 2025

Updated by Arya Zarinsefat, MD

(Adopted from Dr. Melissa Coleman)

BASIC IMMUNOLOGY

MAJOR HISTOCOMPATIBILITY COMPLEX (MHC)

- Molecules found on the surface of cells that present antigen fragments (pieces of proteins) to T cells
- Allows immune system to recognize and respond to pathogens
- HLA is the HUMAN form of MHC

HLA CLASSES

- Class I
  - Found on all nucleated cells
  - Presents to CD8+ T cells
  - Presents endogenous proteins (e.g. viral proteins)
  - HLA A, B, C molecules
- Class II
  - Found on professional APCs (dendritic cells, B cells, macrophages)
  - Presents to CD4+ (helper) T cells
  - Presents exogenous antigens (e.g. bacteria)
  - HLA DP, DQ, DR molecules

T CELLS

- Helper T Cells (CD4+)
- Cytotoxic T Cells (CD8+)
- T cells involved in cell-mediated immunity
- Cell-mediated immunity does not require antibodies

HELPER T CELLS (CD4+)

- Release IL-2 → maturation of CD8+ T cells
- Release IL-4 → B cell maturation into plasma cells
- Release interferon-gamma which activates macrophages

CYTOTOXIC T CELLS (CD8+)

- Recognize and attack non–self-antigens attached to MHC class I receptors (eg viral gene products)

B CELLS

- IL-4 from helper T cells stimulates B cells to become plasma cells (antibody secreting)
- 10% become memory B cells which can be reactivated
- IgG (as opposed to IgM) is secreted with reinfection

B CELLS

- Unlike T cells, do not need MHC to recognize antigen
- BCR (includes membrane-bound Ab) directly bind antigen
- BCR-antigen binding triggers plasma cell formation
- Plasma cells secrete soluble Ab's that are identical
- IL-4 from helper T cells stimulates B cells to become plasma cells

PLASMA CELLS

- Ab's released by plasma cells bind to antigen to neutralize pathogens
- Ab binding results in opsonization
- Ab binding activates complement system to lyse pathogen

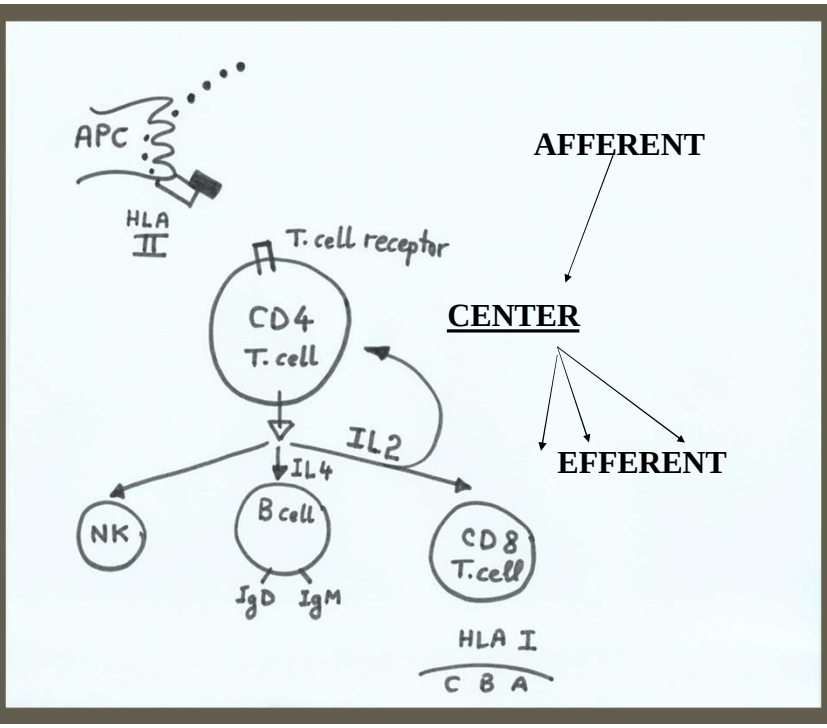
NK CELLS

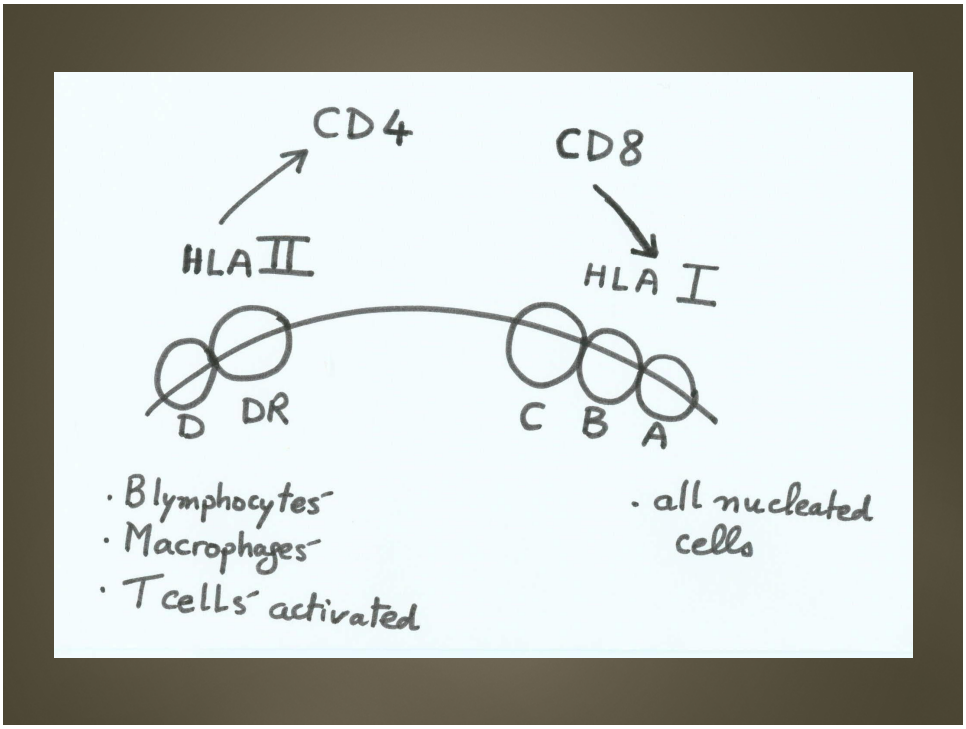
- Not considered T or B cells
- Not restricted by MHC, do not require previous exposure, or antigen presentation
- Recognize cells that lack self-MHC
- Part of the body's natural immunosurveillance for cancer

ANTIGEN PRESENTING CELLS

- B cells, macrophages, dendritic cells
- Ingest antigens, process them, then express them on their surface for TCR, in association with MHC II (HLA II)

AFFERENT	*CENTER*	EFFERENT
Antigen presenting cell	*CD4*	-CD8 -B Lymphocytes -NK





TRANSPLANTATION

HLA CROSSMATCHING

- HLA crossmatching is a laboratory test performed before organ transplantation to check whether the recipient has pre-existing antibodies against the donor's HLA antigens
- Necessary before kidney transplant but not liver transplant

HLA CROSSMATCHING

1. Donor lymphocytes mixed with recipient serum
2. If recipient has Ab's to donor, they will bind to donor cells
3. Detection method, e.g. flow cytometry
4. Positive crossmatch → Ab's present → rejection risk
5. Negative crossmatch → no Ab's → lower rejection risk

HLA CROSSMATCHING

- HLA-A, B, and DR most important
- Don't always need PHYSICAL crossmatch
- Can do VIRTUAL crossmatch
  - Compare recipient's anti-HLA antibodies that know are present, to donor's HLA type

IMMUNOSUPPRESSION

- Induction
  - Initial heavy load of immunosuppression at the time of transplant
- Maintenance
  - Long-term maintenance regimen

INDUCTION IMMUNOSUPPRESSION

- Thymoglobulin
  - Polyclonal Ab preparation
  - Targets multiple T cell surface antigens
  - Depletes T cells by complement-mediated lysis and opsonization
  - Robust response; higher risk of infections
- Basiliximab (Simulect)
  - Ab to CD25 → Blocks iL2-receptor → prevents T cell activation
  - Non-depleting inhibition of T cells
  - Milder induction

MAINTENANCE IMMUNOSUPPRESSION

1. Calcineurin inhibitor (CNI)
2. Anti-metabolite (Mycophenolate)
3. Steroids (Prednisone)

CNI

- Mechanism of action
  - Bind calcineurin → prevent NFAT activation → No IL-2 produced → T cells unable to proliferate/mount response
- Tacrolimus
  - Most prevalent
  - Side effects: nephrotoxic, neurotoxic, diabetes
- Cyclosporine
  - Side effects: nephrotoxic, neurotoxic, diabetes

ANTI-METABOLITE

- Mycophenolate (Cellcept)
  - Most prevalent
  - Inhibits de novo purine synthesis; inhibits T cell growth
  - Side effects: GI intolerance; myelosuppression
- Azathioprine
  - Similar mechanism

STEROIDS

- Solumedrol/Prednisone
  - Inhibit genes for cytokine synthesis (IL-2)
  - Typically steroid taper post-transplant
  - Commonly used solo or with other meds for treatment of rejection

QUESTION

- A 58-year-old woman develops a tremor, hypertension, and worsening renal function after liver transplantation. Which immunosuppressant is most likely responsible?
- A. Mycophenolate mofetil
  B. Prednisone
  C. Tacrolimus
  D. Basiliximab
  E. Azathioprine

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COMPLICATIONS OF TRANSPLANTATION

HYPERACUTE REJECTION

- Preformed antibodies against graft; complement cascade activated
- Occurs immediately after unclamping
- Vessel thrombosis occurs; irreversible
- Preventable by crossmatch; this should not happen anymore

ACUTE CELL-MEDIATED REJECTION

- T cell-mediated (cytotoxic and helper cells)
- Most common, especially early on post-transplant
- Treatment
  - Depends on severity and type of organ
  - Increase immunosuppression vs. steroids vs. thymoglobulin

ACUTE ANTIBODY-MEDIATED REJECTION

- Caused by recipient Ab's against donor HLA antigens on graft endothelium
- Ab's can be pre-formed or de novo
- Ab's bind to endothelium → activate complement cascade → endothelial damage
- Treatment
  - Depends on severity and type of organ
  - Steroids vs. Ab therapy vs. plasmapheresis

CHRONIC REJECTION

- Persistent, low-grade injury involving Ab and cellular immune mechanisms
- Most prominent driver: chronic antibody-mediated
- Donor-specific antibodies target endothelium
- Leading cause of graft loss in kidney transplant

INFECTIONS AND PROPHYLAXIS

- Viral
  - CMV: valganciclovir
- Bacterial
  - Pneumocystis pneumonia: Bactrim
  - UTI: Bactrim
- Fungal
  - Candida: Fluconazole
  - Aspergillus: No routine prophylaxis

CANCER

- PTLD
  - Group of lymphoproliferative disorders post-tx; can be lymphoma
  - Thymo use increases risk
  - Incidence 1-5%
  - Treatment: REDUCE immunosuppression, Rituximab, chemo

CANCER (CONT'D)

- Skin Cancer
  - SCC most common
  - Up to 75% of recipient develop SCC by 20 years post-tx
  - Yearly dermatology follow-up

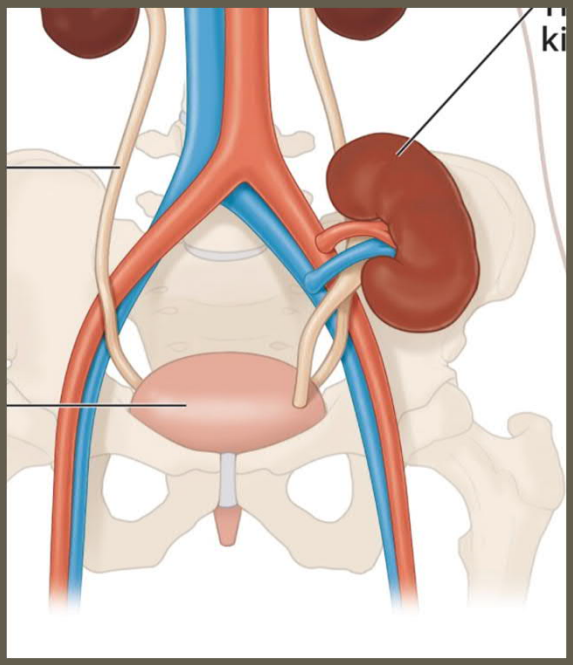
KIDNEY TRANSPLANT

KIDNEY TRANSPLANT

- Most common indication: T2DM
- CIT ideally ~ 24 hours
- Mean graft survival
  - Deceased donor: 11.7 years
  - Living donor: 19 years
- Substantial survival benefit over dialysis
  - Average gain of 2–3 years at 10 years
  - Up to 14 years over a lifetime, and a relative reduction in mortality risk of approximately 55%

STEPS

1. Right/left lower quadrant "Gibson" incision
2. Access retroperitoneum and EIA/EIV
3. Anastomose renal vein to EIV (end-to-side)
4. Anastomose renal artery to EIA (end-to-side)
5. Unclamp vessels
6. Anastomose ureter to dome of bladder



Adapted from NIDDK

QUESTION

The most common cause of mortality within the first year after Renal Transplant is

A. Malignant tumors
B. Cardiovascular disease
C. Sepsis

QUESTION

The most common cause of mortality within the first year after Renal Transplant is

A. Malignant tumors
B. Cardiovascular disease
C. Sepsis (close second)

COMPLICATIONS

- Arterial/venous thrombosis (rare)
- Ureter leak or stenosis (1-2%)
- Lymphocele (2-8%)
- ATN (up to 1/3)
- DGF (50%)

ULTRASOUND

- Best first test for post-op issues; always w/Duplex
- Arterial issues
- Venous issues
- Fluid collections/hematomas
- Resistive index

QUESTION

5 weeks after cadaveric renal transplant from a 60 year old healthy donor, a 50 year old patient shows gradual rise in Creatinine to 4. Renal US normal. Renal Bx shows dilated tubules, flat tubular epithelium. The most likely explanation is:

A. Hyperacute rejection
B. Acute rejection
C. Acute tubular necrosis

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LIVER TRANSPLANT

LIVER TRANSPLANT

- Most common indication: EtOH, MASH
- Waitlist based on acuity
  - Status 1 (acute liver failure, HAT or PNF re-transplant)
  - MELD score
- MELD 3.0: Tbili, INR, Cr, Albumin, Na, Gender
- CIT < 10 hours ideal
- No crossmatch needed

STEPS

1. Recipient hepatectomy
2. IVC (various techniques)
3. PV (end-to-end)
4. Hepatic artery (various way but typically end-to-end)
5. Bile duct (end-to-end vs RXY HJ)

COMPLICATIONS

- HAT (~4%)
  - Relist as Status 1
- PNF (2-5%)
  - Relist as Status 1
- Hematoma requiring washout (20%)
- Biliary leak/stricture (20-30%)

QUESTION

A POD#5 patient develops bilious drainage from surgical drain, mild abdominal tenderness, and rising bilirubin. Ultrasound shows a small fluid collection near the biliary anastomosis. Best initial management?

A. High-dose steroids
B. Percutaneous drainage ± ERCP with stent
C. Retransplantation
D. Increase tacrolimus
E. Observation

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QUESTIONS

A patient 1 week post-OLT is suspected of HAT. Best initial diagnostic test?

A. CT abdomen without contrast
B. Liver biopsy
C. ERCP
D. Doppler ultrasound
E. MRCP

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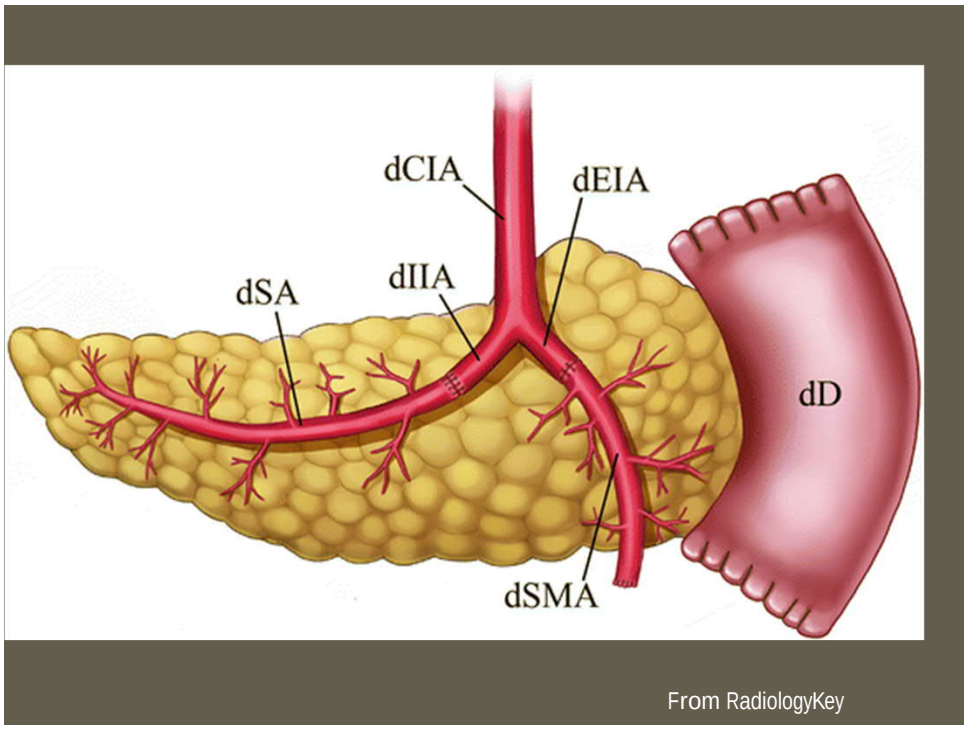
PANCREAS TRANSPLANT

PANCREAS TRANSPLANT

- Indication: T1DM with renal failure
- Typically do simultaneous pancreas/kidney
- Pancreas procured en bloc with cuff of duodenum

STEPS

1. Midline laparotomy
2. Kidney transplant on left iliacs
3. Pancreas Y-graft (donor iliac anastomosed to pancreas splenic artery and SMA) to iliac artery
4. Pancreas PV to iliac vein
5. Donor duodenum to recipient ileum



From RadiologyKey

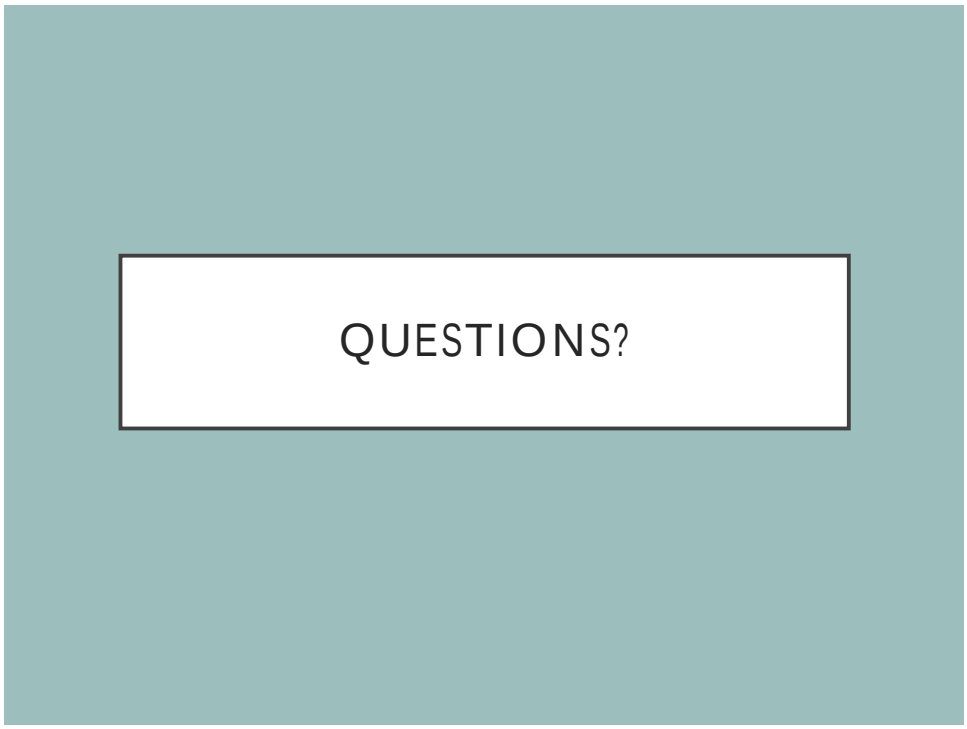
HEART/LUNG TRANSPLANT

HEART TRANSPLANT

- Routine RV biopsies (to check for rejection) are performed at set intervals
- Acute rejection – perivascular lymphocytic infiltrate w/myocyte inflammation and necrosis
- Chronic allograft vasculopathy (progressive diffuse coronary atherosclerosis) – MCC of late death and death overall following heart tx

LUNG TRANSPLANT

- #1 cause of early mortality – reperfusion injury (manage like ARDS)
- Acute rejection – perivascular lymphocytosis
- Chronic rejection – bronchiolitis obliterans; MCC of late death and death overall following lung tx



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- Adapted from NIDDK