CERTIFIED EMERGENCY NURSE

Review Manual & Test Preparation Guide

Inculdes a Full 150 Question "CEN-Like" Practice Exam With Rationales
CEN
(Certified Emergency Nurse)
Review Manual & Course Material

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Mark W Boswell
MSN, FNP-BC, CEN, CFRN, CTRN, TCRN, CPEN, SCRN, NREMT-P, EMT-T, W-EMT
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ORTHOPEDIC, INTEGUMENTARY & SELECTED TRAUMA

SELECTED CONDITIONS
- Abrasion
- Amputation
- Avulsion
- Bite/Sting
- Carpal Tunnel Syndrome
- Compartment Syndrome
- Contusion
- Costochondritis
- Foreign Bodies
- Fracture/Dislocation

ABRASIONS
Injury d/t dermis/epidermis
Falls, scrapes
Good wound care essential to avoid “tattooing effect” from debris
Should be cleaned in 6-8 hours
Diabetics, Immune compromised at risk
Wound cultures?
Clean, Debride; Betadine, H2O2, NS

Pain meds
Tetanus
Non stick dressing, porous

AMPUTATIONS
Avoid tunnel vision
ABCDs
Without cooling
Digits 12 hours
Others 6 hours

With cooling
Digits possible up 24 hours
Proximal to wrist 12 hours

Healing/reattachment points
Children
>50 y.o. - peripheral vascular disease
Functional vs hindrance
Can they complete rehab?

Important: note time of injury
Control bleeding first
Simple dsg up to tourniquet

Splint, elevate
Copolyous irrigation; NS only
Wrap stump with NS gauze
Amputation - cover dry sterile gauze
Place in bag
Place that bag into a bag of ice
### AVULSIONS
- Can’t approximate tissue edges
- Common in fingers
- Hemostasis sometimes difficult
- Small ones: delayed secondary healing
- Large: surgical

### Sterile NS dsg
- Vaseline/xeroform good
- Consider metal/protective splint

### BITES AND STINGS
- May cause: Punctures/lacerations, Anaphylaxis, infection, disease xmission

### Dog & Cat Bites
- Lacerations/puncture
- Possible crush injury (dog)
- 90% of ER bites are dogs
- Cat bites hi-risk infection (pasteurella)
- Dogs/cats - not likely rabid
- Consider rabid: bats, foxes, raccoons

### Known vs unknown animal
- Provoked?
- Gen appearance

### Thoroughly wash, irrigate
- Sutures +/-
- Pain meds/sedation
- Antibiotics/tetanus

### Rabies prophylaxis
- RIG (Rabies Immune Globulin) immediate protection

### Snakes
- Thousands annually
- Only 10-15 mortal
- Two families
- Viperidae (vipers, rattlesnakes)
- Elapidae (coral snake)

- Hands, feet, face injuries
- 20% “dry” bites
- Croatalide envenomation:
  - Rapid onset burning pain and edema
  - Ecchymosis ensues and spreads
  - Venom enzymes cause
  - Local tissue damage, edema
  - Hypotension, coagulopathy, shock, death
Elapidae envenom:
Little or no local reaction
Sx onset possible delayed up to 13 hours

Pt w/parasthesias, burning at local site at higher risk for neuromuscular blockage and resp compromise

Snake identification
Pit viper: triangular head, pit between eyes, elliptical pupils
Coral snakes: red, yellow, black bands; black head; “red on yellow kill a fellow”

Bite location/Time
S/Sx: weakness, parasthesia, diplopia, muscle pain, N/V/D, CP/SOB

Pit viper: fang marks
Coral snake: tiny punctures, “chew”
Both: edema, ecchymosis, vesicles

Coags, Fibrin degradation and split products (FDP, FSP), U/A-myoglobin

Elevate slightly, immobilize
Cardiac, SpO2

Routine wound care
Controversy over cooling

Antivenom: give within 4 hours of bite
Dose depends on degree of envenom
CroFab requires no skin testing

Insects (Bees, Wasps, Ants)
Concern: anaphylaxis
Oral facial edema, respiratory, urticaria

Hx Asthma?
Prior sting allergy/reaction?
Stridor, cough, hoarseness?
Stinger still in?

Remove stinger w/scraping motion
Ice, baking soda, aloe vera to area
Epi
Benadryl
Zantac
Steroids
Analgesics
Antibiotics?
Tetanus

Pt education
Epi-Pen
Prevention: insect repellent
**CARPAL TUNNEL SYNDROME**
Median nerve entrapment
Women: men 2:1
Pregnant women d/t fluid retention
Repetitive motions
Pain, paresthesias; median nerve area

Tinel’s Sign: percussion/tapping on median nerve
if tingling felt in hand = diagnostic

**Phalen’s Sign:** ________________

**X-rays r/o others**
Electro-myelography (EMG) - Gold Standard
Ice/Elevate/Non Steroidal anti-inflammatory (NSAIDS)
Wrist splint(s)
Orthopedics, Occupational Med Referral

**COMPARTMENT SYNDROME**
Trauma, Crush injuries, High pressure injections burns
Lower leg, forearm
Also d/t splints, casts, Pneumatic Anti-Shock Garments (PASG), Military Anti-Shock Trousers (MAST)
First affects low-flow system
Later arterial flow and nerves affected
5 P's: Pain, Pallor, Paresthesias, Paralysis, Pulse

PMHx: Hemophilia?
U/A myoglobin
Serum MB, CPK, CBC, Coags
Doppler U/S?

Compartment pressure monitoring
10 mmhg or less: normal/OK
30-40 mmhg: possible fasciotomy
Elevate slightly, don’t use Ice
Pain meds

**CONTUSIONS/BRUISES/HEMATOMAS**
A closed wound with ruptured blood vessels
Sx: swelling, discoloration, tenderness
PMHx: check for bleeding disorders or meds that prolong bleeding
Consider labs

Possible x-rays
Ice/elevate/splint

~ 4 ~ CEN Review Course
**COSTOCHONDritis**
Inflammatory chest wall pain
Possibly r/t exertion/strain
Pain similar to AMI or rib Fx's
Pain: sharp, pleuritic

MUST: consider Coronary disease/Acute MI risk factors and r/o as indicated
Don't forget PE, Pneumonia
Tx: NSAIDs, Muscle relaxers, Narcotics, Heat

**Sprains/strains**
Sprain: Ligament
Strain: Muscle/tendon
Risk factors:
“weekend warriors”
Rheumatoid Arthritis (RA), Steroid use/injections
Diagnostics: Ottawa ankle, knee, foot rules
Achilles rupture: “hit in back of leg” - classic

R.I.C.E.: Rest, Ice, Compression, Elevation
Immobilization
Wt bearing limitations
NSAIDs, Narcs

**Fractures**
Open Fx: hi-risk infection
Crush injuries: complicated Fx’s
Impacted
Spiral
Transverse
Oblique
Complete
Comminuted

BEWARE: Tunnel vision!
Splint in position found
Ice/elevate
Pain meds
Possible sedation/reduction

Open Fx's:
Cover w/sterile NS dsgs
Clean/irrigate only w/NS
Wound mgmt, possible cultures
Antibiotics/tetanus

Traction Splints:
Used to stabilize a mid-femur fraction along it's long axis using counter-traction
Without counter-traction it is possible for the spasming muscles or deformity to compromise the distal neuro-vascular function.
Indications:
For use in mid-femur fracture
Contraindications
Concurrent knee, hip or pelvis injury
Lower leg or ankle injury

Application:
Stabilize fracture site, manual traction
Secure splint proximally (ischial tuberosity)
Secure splint distally (ankle)
Apply mechanical traction
Re-assess distal neuro-vascular

DISLOCATIONS
Considered an EMERGENCY d/t potential nerve damage, blood vessel damage or tissue ischemia
KNEE: (not patella) immediate reduction
ANKLE: usually needs surgery; prompt reduction
SHOULDER: needs prompt reduction
Possible avascular necrosis (AVN)

RADIAL HEAD DISLOCATION
Nursemaid’s elbow
d/t sudden jerk or pulling
Refuses to use arm
Can flex and extend elbow, won’t pronate/supinate
Possibly no deformity and no significant pain
May be recurrent up to 5 years old

BURSITIS
Inflammation of bursal sac
Acute (trauma/infection) or chronic
Must r/o infection
Common: shoulder, elbow, knee
Red, warm, swollen, tender
Aspiration

X-rays, CBC, Sed Rate (ESR), C-reactive protein (CRP), Basic metabolic
Tx: NSAIDs, Narcs
Ice/elevation
Splinting/compressive dsgs
**TENDONITIS**
Inflammation of tendons or tendon-muscle attachments
Usually excessive/repetitive stresses
Common: Shoulder, elbow, knee, heel
Point tenderness

Look for: swelling, redness, heat
May need to r/o infection
Tx: NSAIDs, Narcs
Splinting/bracing/ortho-phys therapy referral

**GOUT**
Crystal induced arthropathy
Crystals trapped in joint space
D/T inc production or dec. elimination of uric acid
Thiazide diuretics and purine rich foods
Also assoc with many autoimmune and metabolic diseases
Male: Female = 9:1

Rapid onset (overnight)
No Hx trauma
Mod-severe pain

Hot, red, swollen, EXQUISITELY tender
Must consider infection also
Poss. IM/IV pain meds

X-rays
CBC, BMP, ESR, CRP, Uric Acid, Aspiration?
Educ: Hydration, Wt Loss
Meds: NSAIDs, Colchicine, Narcs

**JOINT EFFUSION**
Collection of fluid in joint space
Most common: knee
Assess for trauma/infection
Hx Substance Abuse: septic arthritis
Hx: Sexually transmitted diseases (STD’s), bleeding disorders
Consider cellulitis, gout
X-rays r/o Fx’s

Swelling, tenderness, fluctuance, ballotment

Check ROM
Labs: CBC, BMP, ESR, CRP, Uric Acid, Rapid Plasma Reagen (RPR - for syphilis), Hepatitis profile

Joint aspiration
Immobilization, Ice
Ortho referral
NSAIDs, Narcs, Steroid injection
OSTEOMYELITIS
Infection of the bone and tissues
D/T open wounds, puncture wounds, surgery
Exogenous vs hematogenous sources
Hematogenous: skin abscess, Otitis Media (OM), UTI, Pneumonia, abscessed teeth

Staph Aureus
Pain, fever, malaise

Swelling, redness, warmth
Possible Sx of Sepsis

PMHx: Diabetes, sickle cell, Immune compromised
Hx of surgical procedure, fixation
IVDU (Intravenous Drug Use)

CBC, ESR, CRP, BMP, Blood cultures
Bone Scan
Parenteral IV antibiotics long term
Transmission precautions
Pain meds
Orthopedics f/u

PUNCTURE WOUNDS
Commonly
Stepping on nails, tacks, needles, broken glass
Usually minimal bleeding and seal off - increasing infection risk
Near joints esp. hi-risk
Plantar surface through shoe inc risk of Pseudomonas d/t foreign body

CBC, BMP, X-rays
Remove foreign body if present
Assist with opening, debriding, irrigating and packing if contaminated
Antibiotics, tetanus, pain meds
Elevate, limit use

Home care: warm soapy water soaks
Should have wound check follow up made

FOREIGN BODIES
Many things
Wood, metal splinters, glass, clothing, (Gun shot wound) GSW fragments, fishhooks etc.
Vegetative (organic) items more likely to cause infection (thorns, wood), need to be removed
Diagnostic imaging
Routine wound care externally
***Don’t soak if wood embedded***
Home care: soaks, elevate, pain meds, antibiotics

MISSILE INJURIES
Penetrating: GSW, stabs and others
High velocity, may cause bony, neurovascular and other injuries remote from the path
Forensic considerations; evidence
Hi-pressure injection wounds might require debridement and extensive irrigation under anesthesia
X-rays usually indicated
Control local bleeding, elevation
Routine external wound care
Assist with debriding, exploring, irrigation

LACERATIONS
Wound mgmt goals:
Restore function
Repair tissue integrity
Minimize risk of infection
Epithelial cell growth begins as early as 6 hours post injury
Concern about areas of flexion/extension
Delayed closure issues
Assess distal CMS/NVS
Assess ROM, esp. flex/extension
Consider x-rays
Control bleeding
Clean/irrigate with NS or antiseptic soap
Debridement
Wound closure
Sutures, staples, Dermabond, Steri-strips
May need splinting too
Wound care instructions
Elevate x24-48 hours
Ice
Dressing on x48 hours
2-3 day wound check, first dsg change
Sunblock for 6mo after sutures
BEST prevention for infection is good, thorough wound cleansing, including hi-pressure NS irrigation
Betadine?
SUTURE REMOVAL TIMES:
Face 3-5
Scalp 5-8
Upper extremities
Non joint: 7-10
Joint: 10-12

Lower extremities
Thigh 7-10
Knee 12-14
Lower leg/foot 7-10

LOW BACK PAIN (LBP)
Usually benign, affecting 60-80% of everyone
Consider 3 groups

1) Non-specific LBP = 85% cases
2) LBP potentially assoc with spinal pathology
   (stenosis, sciatica, compression Fx)
3) LBP potentially assoc with another condition
   (cancer)

Most common causes:
Intra-vertebral disk disease
Disk herniation

Avg age of onset: 30-40 y.o.

Risk factors:
Obesity, body mechanics, lifting, prolonged
sitting, poor office furniture, hard floor
surfaces
Pain: localizes or radiates

Assoc sx: paresthesia, impaired bowel,
   bladder function, impaired sexual
   function
Assess: gait, posture, point tenderness or diffuse,
   spasm

Sensory exam
Front of thighs: L2
Middle/sides lower leg: L4-L5
Little toes: S1

Deep Tendon Reflexes (DTRs):
Grade:
0: none,
1+ below normal,
2+ average,
3+ stronger than avg,
4+ intense
**RED FLAGS:**
Age under 20 or over 55  
Non-mechanical pain  
Cancer (CA), steroids HIV or other significant past history  
Unwell, wt loss  
Widespread neurological e.g. bilateral leg signs  
Structural deformity  
Saddle anesthesia/sphincter disturbance  

X-rays, not routinely unless you suspect Fx  

NSAIDs, Steroids, Muscle Relaxers, Narcotics  

Evidence supports a rapid return to function.  
AndIE: bed rest for 1 day only, then gradual return to normal activity in 2-4 days
<table>
<thead>
<tr>
<th>Quick Review Questions</th>
<th>Answer</th>
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<tr>
<td>1. Name oneFx commonly assoc with compartment syndrome?</td>
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<td>2. What are late signs of compartment syndrome?</td>
<td></td>
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<td>3. What is initial nursing intervention for open wound near a Fx?</td>
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<td>4. What is the name of inflammation of the synovial cavity surrounding a joint?</td>
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<td>5. Onset of Gout - how long?</td>
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<td>6. What two physical examination assessments are done Dx carpal tunnel?</td>
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<td>7. Which gives immediate protection tetanus toxoid or tetanus immune globulin?</td>
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<td>8. What patients need immediate tetanus protection?</td>
<td>ANS:</td>
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<td>9. What labs test might help most to diagnose compartment syndrome?</td>
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<td>10. What are some important “rule-out” conditions to do for costochondritis?</td>
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<tr>
<td>11. What age range is a possible “red flag” for lower back pain (LBP)?</td>
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<td>Overdose Emergencies/Quick Fill-in-the-blanks</td>
<td>Antidotes</td>
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<tr>
<td>Acetaminophen</td>
<td>__________</td>
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<tr>
<td>Benzodiazepines</td>
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<tr>
<td>Carbon Monoxide</td>
<td>__________</td>
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<td>Cyanide</td>
<td>__________</td>
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<td>Iron</td>
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<td>Salicylates</td>
<td>__________</td>
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<tr>
<td>Tricyclic antidepressants</td>
<td>__________</td>
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**ACETAMINOPHEN**

In many OTC products
Easily available
Intentional or unintentional
Rapidly absorbed in GI tract
Metabolized by liver

Toxicity causes:
- Coagulopathies
- Hepatic necrosis/failure
- Early sx: malaise, n/v
- Late sx: metabolic Acidosis, RUQ pain,
  decreased urine output, jaundice,
  hypoglycemia, coagulopathy, DIC, sepsis

**ASSESSMENT**

Acute vs chronic
Concurrent ETOH or drug use
Reason for exposure (intentional vs side effect)

**PMHX**
- Psych Hx
- ETOH Hx
- Liver disease

**EXAM**
- LOC
- Jaundice
- RUQ ttp
- Hepatomegaly
- GI bleeding

**DIAGNOSTICS**
- Serum APAP 4 hours post-ingestion
- Rumack-Matthew nomogram
CBC, Chemistries, LFT's, Coags
U/A, UPT (urine preg test)
Toxicology panel

INTERVENTIONS
ABCs, IV, Cardiac, Pulse Ox PRN
NG tube (NGT), lavage possible
Possible admission

MEDICATIONS
Activated charcoal
N-acetylcysteine (Mucomyst) PO, NGT
Acetadote IV (not as effective as PO)

BENZODIAZEPINES
CNS depressants
Main concern: resp. depression
High addiction potential
Used for sleep/anxiety
Benzos not as toxic as barbiturates

ASSESSMENT
Agent ingested, amounts
Any co-ingestions
Reason (intentional/accidental)

PMHX
Psych hx
Seizure hx

EXAM
LOC; drowsiness, ataxia, euphoria
Hypotension, hypothermia
Dysrhythmias
Pupillary changes

DIAGNOSTICS
CBC, Chemistries
U/A, UPT
Tox panel
ABGs

INTERVENTIONS
ABC's, IV, Monitor, Pulse Ox PRN
Possible RSI if obtunded
Possible IVF boluses
Airway: positioning, Naso-pharyngeal (NPA), Oro-pharyngeal (OPA), intubation
NGT prn
Admission, dialysis?

MEDICATIONS
Activated Charcoal
Romazicon (Flumazenil); possible seizures
Sodium Bicarb: to alkalize
Vasopressors
CARBON MONOXIDE (CO)
Colorless, odorless, tasteless gas
Product of organic combustion
200x the affinity for Hemoglobin (Hgb) than O₂
Displaces O₂
Resultant hypoxia
Duration of exposure r/t CO level
Symptoms rarely correlate with CO level
Death occurs d/t dysrhythmias

ASSESSMENT
Poss. Concurrent burn injury
Enclosed space
Time of exposure
Intentional/accidental?
Pregnancy (fetus more at risk)
Headache (most common sx)
N/V, dizziness, chest pain

PMHX
Cardiac dysrhythmias
Pulmonary disease
Psych Hx
Social: smoker?

EXAM
LOC; possibly depressed
Hypotension
Seizures at high Carboxyhemoglobin (COHb) levels
“Cherry red” skin and mucus membranes
   -more often terminal findings

Cardiac dysrhythmias: ST and T changes

DIAGNOSTICS
ABGs, COHb (half life 4-5 hours)
Cardiac markers, EKG
U/A: myoglobin, UPT, tox panel
CXR: pulmonary edema, ARDS

INTERVENTIONS
ABCs, IV, Monitor

100% O₂: decreases half-life COHb
Pulse ox – misleading!
NGT prn
Admission, possible (hyperbaric oxygen)
   HBO if available
RSI if indicated

EDUCATION
Sources of CO poisoning
Symptoms of CO poisoning
CO detectors
CYANIDE
Lethal poison
If inhaled; death possible in 2 min
Disrupts cellular respiration
High venous O2 saturation
Oral ingestion possible
Common: industrial exposure or result of
burning plastic, housefires
Medically: long term use nitroprusside

ASSESSMENT
Circumstances of exposure
Route: oral, inhaled (fires)
Time of exposure

PMHX
Psych Hx
Occupational hazards
Other concurrent diseases

EXAM
LOC; seizures, comatose
Breath: “bitter almonds”
Hypertension w/bradycardia,
-then hypotension w/tachycardia,
-then cardiac arrest
Critically ill appearing

DIAGNOSTICS
Cyanide, lactate levels, CBC, Chemistries
Venous or arterial blood gas
U/A: UPT, tox panel
EKG

INTERVENTIONS
ABCs, IV, Monitor, Pulse Ox, supplemental O2
BLS/ACLS prn
No mouth-to-mouth d/t exhaled vapors
Advanced airway management; RSI
NGT prn

MEDICATIONS
Antidote kit:
Amyl Nitrite: nasally or inside mask
Sodium Nitrite: IV
Sodium Thiosulfate: IV
Vasopressors prn
Benzodiazepines for seizures

IRON
Important in children
Mainly unintentional ingestion of prenatal or
children’s vitamins
Blister packaging helped reduce
Toxicity depends on amount of elemental iron in
the product
Causes GI hemorrhage and CV collapse
ASSESSMENT
Number and kind of tablets ingested
Type of iron in compound
Time of exposure and reason
Concurrent ingestions

PMHX
Anemia
Other concurrent conditions

EXAM
Possible hypotension, tachycardia
Abdominal tender-to-palpation (TTP)
Occult/frank blood in emesis or stool

Phases of toxicities
Phase 1:
0-6 hours post-ingestion
Corrosive effects
Vomiting, abd pain, bloody diarrhea

Phase 2:
6-12 hours post-ingestion
Recovery phase
Pt appears to get better

Phase 3:
12-48 hours post-ingestion
CV collapse, shock, met. Acidosis, GI bleeding
Coagulopathy, hepatic damage, sepsis, coma

Phase 4:
>48 hours post-ingestion
If they survive
Intestinal obstructions, pyloric strictures

DIAGNOSTICS
CBC, Chemistries
Serum Fe: peaks in 3-5 hours post-ingestion
TIBC
Occult blood studies
Liver panel, coags
Type and cross
ABGs

INTERVENTIONS
ABCs, IV, Monitor, Pulse Ox, O2 prn
BLS/ACLS as indicated
Advanced airway control, RSI
NGT; lavage questionable usefulness
Whole bowel irrigation poss
### MEDICATIONS
- **Activated charcoal:** contraindicated, doesn't bind with Iron
- **Deferoxamine (Desferal):** chelator agent -causes pink or red urine
- **Whole bowel irrigation:** GoLYTELY

### SALICYLATE POISONING
- Commonly/readily available
- Many OTCs
- Possible short or long term exposure
- Elderly more at risk d/t decreased renal functioning, and more use of ASA for common ailments
- Affects GI mucosa, coagulation, neurological system, and acid-base status.
- Peak levels approx 6 hours post-ingestion
- Toxic: 150-200 mg/kg
- Lethal: >500 mg/kg

### ASSESSMENT
- Long term vs short term exposure
- Reason for exposure (intentional?)
- Type and amount
- N/V, tinnitus
- **PMHX**
  - Psych Hx
  - Chronic pain syndromes
  - Liver disease

### EXAM
- Tachypnea/cardia, hyperthermia, diaphoresis
- Skin: ecchymosis
- Lungs: crackles

### DIAGNOSTICS
- CBC, Chemistries
- Salicylate level:
  - On arrival and 6 hours post ingestion
  - Compare w/nomogram
- ABGs: acidosis, alkalosis
- Coagulation studies
- U/A, UPT, Tox panel
- Occult blood testing
- EKG, CXR as indicated

### INTERVENTIONS
- ABCs, IV, Monitor, Pulse Ox, O2 PRN
- Hyperventilation for acidosis
- NGT prn
- Gastric lavage of questionable benefit
- Possible whole bowel irrigation
- Prepare for admission, possible hemodialysis
MEDICATIONS
Activated charcoal (if NO GI bleeding)
Sodium Bicarb: alkalinize urine
Potassium replacement
D50 for hypoglycemia

TRICYCLIC ANTIDEPRESSANT (TCA) POISONING
Extremely lethal
Aggressive management is key
Anticholinergic and alpha-adrenergic blocking properties
Produce cardiotoxic effects, and CNS depression
Once absorbed, highly bound
Death is d/t cardiac effects
Uses: antidepressants, enuresis in peds

ASSESSMENT
Amount and time of ingestion
Types
Co-ingestions?

PMHX
Psych Hx
Neurological disease
Cardiac disease

EXAM
LOC; alert to comatose, possible seizures
Hypotension
Anticholinergic Crisis Memory Cue:
“DRY as a BONE, MAD as a HATTER,
RED as a BEET”

Dysrhythmias:
Sinus tach (early)
PVCs, SVT, VT, QRS widening

DIAGNOSTICS
CBC, Chemistries
Cardiac markers
ABG’s
U/A: UPT, tox panel, myoglobin
EKG

INTERVENTIONS
ABCs, IV, Monitor, Pulse Ox, O2 prn
RSI and/or airway management
NGT, lavage controversial
Seizure precautions
Prepare for admission; if not then must observe for at least 6 hours

MEDICATIONS
Activated charcoal
Benzodiazepines: seizure control
Vasopressors: Hypotension management
# Substance Abuse Emergencies

## Amphetamines

- Synthetic sympathomimetic drugs
- CNS stimulation
- Oral, intranasal, parenteral forms

**Normally used for:**
- Appetite suppressants
- Mood elevation
- Alertness
- ADHD

"Ice" is smoked, “meth”, “crank”

- Crystal meth
- Dextroamphetamine
- Methylphenidate (Ritalin)

### Assessment

- Restless, anxious, paranoid
- Seizures
- Tachycardia, hyperthermia
- Pupils: mydriasis
- Tremors
- Thin, emaciated
- Skin lesions: formication?
- Heart: murmurs

### Interventions

- Normothermia
- Protect staff/pt.
- Benzodiazepines for sedation
- Antipsychotics for agitation/psychosis

## Cocaine

- Snorting is most common
- Also can be smoked or injected
- “Crack”, “rocks”; purified - rush similar to IV use

**CNS and autonomic stimulation**

- Increased catecholamines release

**Blocks reuptake of dopamine and norepinephrine**

**Tx is supportive corrective measures**

### Assessment

- Time ingested; short lived
- Poly-substance ingestion? (PSA)
- Moods: euphoria, energy, agitation, aggression
- Cardiac: palpitations, chest pain, angina
- Pupils: dilated are hallmark
- Seizures
- Restless, anxious, jittery
- Perforated nasal septum
- Skin infections/abscesses
- Lungs: crackles, rhonchi
- Heart: rubs, murmurs
INTERVENTIONS
Benzodiazepines for symptoms control
If in a hypertensive urgency, DO NOT give
Nitroglycerin or B-Blockers
This will result in a profound,
unopposed alpha cascade and a
HYPERTENSIVE CRISIS
Antipsychotics for delirium or psychosis

HALUCINOGENS (GHB, 'X')
Originally an anesthetic agent
"Designer drug"
Dance clubs, “rave” scene
Date rape drug
Effects: sedation, amnesia, resp. depression, altered LOC

ASSESSMENT
Route of exposure
Circumstances: recreational? Date rape?
Concurrent ETOH or other drugs
Time ingested: rapid onset w/in 15 min
LOC; lethargy to deep coma
Seizures?
Resp depression

INTERVENTIONS
Do you RSI? Do you Intubate?
-Consider risk/benefits of this
Benzodiazepines for seizures

ALCOHOL
Most commonly used drug in US
Assoc w/70% of overdose cases in ER
Alcoholism: physiologic, psychological, social dysfunction
Metabolized by liver; affects ALL body parts

ASSESSMENT
Trauma, falls, head injuries
LOC changes
How much ingested, when?

PMHX
Seizures
Liver dz
GI bleeding, varices
Hx of suicide, depression, anxiety
Family Hx of alcoholism?

Social: ETOH Hx
Pattern, types, amounts
When last drink?
Blackout Hx
Change in tolerance
Prior attempts to discontinue
Periods of abstinence?
"Heavy drinking" = > 5 drinks one sitting

**EXAM**
- LOC, alert to stuporous
- Odors: ETOH, mints, mouthwash
- Hygiene: unkempt to well-groomed
- Nystagmus?
- Jaundice, icteric sclera
- Abdomen: ttp, ascites

**INTERVENTIONS**
- Quiet environment dec. stimuli if DT's
- Fall precautions
- Seizure precautions

**MEDICATIONS**
- D50 for hypoglycemia
- Thiamine for Wernicke-Korsakoff Syndrome
- Electrolyte replacement: K+, Mag
- Benzodiazepines: agitation, tremors, seizures
- Antipsychotics: withdrawal psychosis

Delirium Tremens (DT's)/ The "Skakes"
- Account for only 5% of ETOH w/d
- Can be fatal; mortality up to 35% if untreated
- Confusion, disorientation, agitation, hallucinations
- Autonomic instability
- Medical admission

**DRUG TOXICITY EMERGENCIES**

**LITHIUM**
- Tx depression, mania-bipolar
- Narrow therapeutic range
- Common symptoms: nausea, headache, tremor
- Toxic symptoms:
  - Ataxia, nystagmus, renal impairment, seizures
  - Dehydration and/or renal insufficiency are common causes
- Treatment: rehydration, supplemental sodium, supportive care

**DIGOXIN**
- Rx to inc. cardiac contractility.
- Treat A-fib and PAT
- Toxic levels d/t: overdose, hypokalemia, dec. renal functioning
- Narrow therapeutic margin

- Symptoms:
  - Visual disturbances, YELLOS HALOS, weakness, N/V, palpitations
- Treatment:
  - Digoxin immune Fab (Digibind):
  - Tx only for life threatening arrhythmias.
<table>
<thead>
<tr>
<th>Other Anti-dotes: (Fill in the blank w/antidote):</th>
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<tbody>
<tr>
<td>Coumadin:</td>
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<td>Calcium channel blockers:</td>
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<tr>
<td>Ethylene glycol/methanol:</td>
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<tr>
<td>Beta-Blocker, CCB:</td>
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<tr>
<td>Oral sulfonylurea:</td>
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<tr>
<td>Heparin:</td>
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<tr>
<td>Hydrofluoric acid:</td>
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</tbody>
</table>
ENVIRONMENTAL EMERGENCIES
COLD-RELATED/HYPOTHERMIA

MILD: 93-95 F
MODERATE: 86-93 F
SEVERE: <86 F

Life threatening complications:
- Apnea
- V-fib
- Acidosis

Risk factors:
- Age
- Medications
- Alcohol
- Traumatic injury
- Shock
- Diabetes

ASSESSMENT
- Ambient temp, length of exposure
- Water exposure?
- Paradoxical undressing
- Shivering diminished/absent at 86 F
- Dysrhythmias

INTERVENTIONS
- Not dead until warm and dead
- Meds/ALS may not work at cold temps
- Airway management, RSI
- Humidified, warmed O2

Mild hypothermia:
- Passive rewarming: blankets, environment
- Body to body contact, radiant lights

Moderate hypothermia:
- Active core rewarming
- Warmed IVF
- Warmed peritoneal lavage

Severe hypothermia:
- Active core rewarming as above, PLUS
- Warmed thoracotomy/mediastinal lavage
- Possible bypass if possible

Warm all patients to a minimum of 86 F

FROSTBITE
- Tissue freezing; ice crystals
- 90% involves hands, feet
- Others: cheeks, nose, ears, penis too
Factors:
Vasoconstriction
Inc. viscosity of blood
Thrombus formation
Vascular stasis

4 degrees based on appearance after re-warming
1st: central pale area, surrounding erythema
2nd: blisters, erythema and edema
3rd: hemorrhagic blisters and eschar
4th: necrosis and tissue loss

Treatment:
Rewarming
Pain mgmt and wound care

SUBMERSION INJURY
True incidence unknown; many not reported
Defined as hypoxic d/t submersion
Can be dry chemicals or even grain (grain silos)

Risk factors:
Inability to swim
Intoxication
Seizures
Hypothermia
Stroke
MI
Child abuse

Progression:
Panic, followed by breath-holding, followed by
hyperventilation, then aspiration and then hypoxia

EXAM
LOC; awake to comatose
Pale, cyanotic
Gastric distention
Consider other injuries (ie diving)
Lungs: crackles (d/t aspiration)

INTERVENTIONS
BLS/ACLS per protocols
RSI and airway management PRN
Rewarming as per hypothermia guidelines
Warm to at least 86 F

HEAT-RELATED EMERGENCIES
When body can't regulate normal temp. through
normal processes
Heat normally dissipates through:
- Convection
- Radiation
- Evaporation

Evaporation (sweating) primary mechanism
Sweat can cause Na+ and K+ losses
Subsequent dehydration possible

**A) Heat Cramps**
Depletion of fluid/electrolytes in exerted muscles
Not enough PO fluid replacement
C/O pain in exerted muscles & thirst
Tx: stop activity, cool off, PO fluid replacement

**B) Heat Exhaustion**
Major exertion in hot weather
Large amounts fluid/electrolytes lost through sweating
Core temp= high normal up to 104 F
Pale, ashen, sweating profusely
Weakness, LOC changes possible
Tx: cool off, IVF replacement, monitor for arrhythmias, check for rhabdomyolysis

**C) Heat Stroke**
Emergency
Can no longer dissipate heat
Failure of thermoregulation mechanisms
Primarily young athletes
Seen with core temps > 102.5, poss to 106
LOC; stupor, coma
Skin: hot and dry
Tx: rapid cooling
Avoid shivering
Check electrolytes, check for rhabdomyolysis
DIC is a possible complication

**HAZ-MAT EMERGENCIES**
**RADIATION**
Gamma, neutron rays:
Penetrate bodies/structures

Alpha/beta particles:
Weaker, stopped by clothing, skin, barriers
Alpha/beta particles considered a contaminant

The risk of injury r/t: time, distance, shielding
If symptoms occur immediately after exposure = critical

Acute Radiation Syndrome (ARS)
F/C/N/V/D
Malaise, anorexia
Bleeding, bruising
Decontamination is important, but don't delay life saving treatments; apply the principles of time, distance, shielding.

**PETROLEUM DISTILLATES**

Hydrocarbons, gasoline, kerosene, pain thinner etc.

**ASSESSMENT**

Route: inhalation, topical  
Circumstances, type of product  
Cough, choking  
LOC  
Petroleum odor  
Lungs: crackles, rhonchi, wheezes

**INTERVENTIONS**

High concentration FiO2  
Remove contaminated clothing  
Copiously irrigate  
Needs 6 hours of observation

**ORGANOPHOSPHATES**

Inhibits acetylcholinesterase, which causes a build up of ACTH at the neur-muscular junction  
Found in insecticides and WMD

**ASSESSMENT**

"MUDDLES"  
M-iosis  
U-ri nation (increased)  
D-efecation  
D-iaphoresis  
L-acrimation  
E-xcitation  
S-alivation

"SLUDGEM"  
S-alivation  
L-acrimation  
U-ri nary (inc.)  
D-efecation  
G-astrointestinal (emesis)  
E-xcitation  
M-iosis

**INTERVENTIONS**

ABCs, IV, Monitor, Pulse Ox, O2  
Protective clothing for tx team  
Decon. Pt with soap and water

**MEDICATIONS**

Atropine:  
Anti-Cholinergic; large doses  
Pralidoxime (2-pam):  
Frees up bound/deactivated Ach  
Benzodiazepines: seizures
**REVIEW QUESTIONS:**

1. For what common toxidrome is Activated Charcoal contraindicated?

____________________________________________________________________

2. What are three “classic” findings of tricyclic overdose?

____________________________________________________________________

3. When is it most crucial to draw a serum Tylenol level?

____________________________________________________________________

4. What are some possible routes of exposure for cyanide toxicity?

____________________________________________________________________

5. For the hypothermic patient, to what degree should the patient be re-warmed?

____________________________________________________________________

6. What medication is used for cocaine overdose when it is causing cardiovascular symptoms?

____________________________________________________________________

7. What are the “classic” findings of nerve agent or organophosphate toxic exposure?

____________________________________________________________________
SHOCK AND MULTISYSTEM EMERGENCIES

SHOCK
Defined as:
"Clinical manifestation of the body’s inability to perfuse its tissue adequately"

Inadequate tissue perfusion - Types of Shock:
Hypovolemia
Cardiogenic
Distributive
Obstructive

When interventions fail, the end result is the same, multiple organ failure and eventually death.

PATHOPHYSIOLOGY
Compensated:
Sympathetic nervous system is stimulated
Aerobic to anaerobic metabolism

Uncompensated:
Failing compensatory mechanisms
Cellular derangement and death irreversible
No treatment to reverse this process
Severe cellular destruction

Common final pathway:
Impairment of cellular metabolism

Impaired Oxygen Use:
Cells not receiving or unable to use oxygen

Impaired Glucose Use:
Impaired glucose delivery or impaired glucose uptake

Some compensatory mechanisms activated by shock contribute to decreased glucose uptake by the cells

HYPOVOLEMIA
Fluid loss (Blood, plasma, other)
MOST COMMON

Causes:
Trauma
GI hemorrhage
Dissecting aneurysm
Vaginal hemorrhage
Burn victims
Vomiting/diarrhea
Diabetes mellitus/Insipidus
Ascites
Excessive diuretic use
ASSESSMENT
Consider those conditions relevant to blood or volume loss:
Chest: hemothorax, pneumothorax, flail chest
Abdomen: bruising, tenderness, rigidity
Musculoskeletal: displacements, open fx’s, femur, humerus
Signs of burn injury
Obvious/visible blood loss

PHYSICAL EXAM
LOC: restless, depressed
Behavior: dizzy, lethargic
Vitals: low blood pressure, tachycardic, tachypnea
Obvious signs of bleeding or deformities
Burns
Skin color: pale, cool, moist, cyanotic
Lung sounds: decreased, diminished, absent, wet
Heart sounds: muffled, distant
Palpation: swelling, tenderness, rigidity
Pulses: central versus peripheral, rate, quality

INTRAOSSEOUS INFUSION
Indications:
Need emergent vascular access
3 failed peripheral attempts
Spent more than 90-120 seconds
Contraindications:
A fractured bone or previously penetrated bone
Areas of cellulitis, burns, or infection
Osteogenesis imperfecta and osteoporosis
Sites:
Distal femur
Proximal tibia (most common)
Medial malleolus
Iliac crest
Sternal

Technique
Proximal tibia site:
Approx 1 to 3 cm below tibial tuberosity and medially on the tibial plateau.
This flat, antero-medial surface of the tibia is the preferred site for infants and children.

Intraosseous infusions - medications:
If you can put it in an IV, you can put it in an IO

DIAGNOSTICS
Looking for the causes of the hypovolemia
CBC, Type and cross
FAST abdominal U/S
CT appropriate body part
Port CXR looking for:
Pneumothorax, tension Ptx, hemothorax,
multiple rib fxs, sternal Fx, widened mediastinum

Pelvis xray: general visualization of bony pelvic structures

INTERVENTIONS
Aimed at treating the underlying cause
Still follow ABC's
In primary survey, control “life threatening” bleeding

Fluid Resuscitation 3:1 rule:
3ml of fluid for every 1 ml lost

Use warmed LR or NS initially
NS preferred as it will mix w/blood
Avg adult 1-2 liters bolus initially
Peds: 20 ml/kg bolus x2

If no response after first two boluses, consider going to blood products

Control bleeding definitively; surgery

PHARMACOLOGICAL
RSI meds: sedative, paralytics
Vasopressors: but not while acutely bleeding
Antibiotics (for septic shock)
Analgesics
Tetanus immunization

GIVING BLOOD PRODUCTS
Universal donor O-negative
Give blood with NS
Need blood filter
Level one rapid infuser
Autotransfusion: for hemothorax
Intraosseous site can be used for blood

REASSESSMENT/EVALUATION
Repeat primary survey: ABC's
Improvement in mental status
Recheck central peripheral pulses
Labs: lactate, base excess/deficit

Urinary output:
GOOD = 30ml/hr
BETTER = 1-2 ml/kg/hr

CARDIOGENIC SHOCK
Alteration in cardiac pump function
“Pump failure”
Causes:
Severe myopathy (CHF)
Valvular failure
Toxins

Results in decreased cardiac output
Body compensates:
   Catecholamines,
   Anti-diuretic hormone release,
   Renin-angiotensin
Cardiac oxygen demands increase
Further decreases in cardiac output
Decreased tissue perfusion
Anaerobic metabolism
Myocardial tissue decreased perfusion
Myocardial dysfunction

ASSESSMENT
PMH
Recent MI (LV failure)?
Recent chest trauma (blunt cardiac injury)?

PHYSICAL EXAM
Ill, weak, shocky, decreased mental state
Restlessness
Tachycardia, hypotensive
Lungs: crackles, rales
Heart sounds:
   S3/S4/Murmurs: think CHF, MI, Endocarditis
   Muffled/Distant: think Tamponade
Bowel sounds: hypoactive
Skin: clammy, pale
Decreased capillary refill, peripheral pulses

DIAGNOSTICS
ECG
Cardiac biomarkers
Serum lactate
Chemistries
Clotting studies
ABG's
Chest x-ray

INTERVENTIONS
ABC'S
RSI and ventilatory support PRN
Fluid resuscitation; judiciously d/t risk of overload

PHARMACOLOGICAL
Drugs that decrease PRELOAD:
Diuretics (Lasix)
Nitroglycerin
Morphine
Sodium Nitroprusside (Nipride)
Drugs that decrease AFTERLOAD:
Vasodilators
Sodium Nitroprusside (Nipride)
Ace-Inhibitors
Nitroglycerine (at higher doses)
Hydralazine

Drugs that Increase AFTERLOAD:
Vasoconstrictors
Norepinephrine
Epinephrine

Drugs that increase CONTRACTILITY:
Inotrope
Dopamine
Dobutamine
Inocor

DISTRIBUTIVE SHOCK
Disruption of the sympathetic control of vessel tone
Normal “resting” state of vessels; dilated
Also called “high space” shock, or “all over shock”
Blood is maldistributed to the periphery
Increased parasympathetic control of heart

TYPES OF DISTRIBUTIVE SHOCK
A) NEUROGENIC
Loss of sympathetic outflow

   Spinal shock
   A specific type of neurogenic shock
   Arreflexia, flaccid
   Skin WARM below level of injury
   Bradycardia or normal heart rate

B) SEPTIC
Bacteremia
Diffuse dilation of vessels r/t circulating toxins
Most commonly; untreated infection

C) ANAPHYLAXIS
Antigen-antibody reaction
Hypersensitivity reaction
Massive histamine release (vasodilator)
Constriction of pulmonary vasculature

DIAGNOSTICS
CBC (sepsis)
Lactate (anaerobic cell destruction)
ABG’S (pH)
UA (infection/sepsis)
Cultures (sepsis)
LP (rule in out infectious causes)
EKG; normal heart rate down to bradycardia
<table>
<thead>
<tr>
<th>Chest xray: infiltrates (infection), thoracic spine damage</th>
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<tbody>
<tr>
<td>C-spine: spinal cord injury</td>
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</table>

**INTERVENTIONS**
ABC's, IV access, 100% O2

**PHARMACOLOGICAL**
- Vasopressors
- Antibiotics
- Antipyretics
- Analgesics

**ANAPHYLAXIS Treatment**
- Epinephrine:
  - SQ & ET 1:1000
  - IV 1:10,000
- Antihistamines: diphenhydramine
- Histamine blockers: rantidine (Zantac)
- Bronchodilators (albuterol)
- Corticosteroids (solumedrol)

**OBSTRUCTIVE SHOCK**
Inadequate circulating blood volume
D/T compression of great vessels or heart

**POSSIBLE CAUSES:**
- Cardiac Tamponade
- Aortic Aneurysm
- Tension pneumothorax
- Pulmonary embolus

**ASSESSMENT**
- Pt possibly in extremis
- Altered, decreased mental status
- Tachycardia
- Hypotension
- Diaphoretic
- Cyanotic
- Tracheal deviation?
- Jugular venous distention?
- Lung sounds: present? Absent?
- Heart sounds: muffled, distant

**DIAGNOSTICS**
- ABG's for PE, TPtx
- D-dimer for PE
- Echocardiogram for Tamponade
- Chest CT: for PE, TensionPTX, Anueryism
- VQ Scan: PE
- EKG: Tamponade, PE
- CXR: TensionPTX, Anuerysm
INTERVENTION
ABC's, IV access, O2
Needle decompression (tension pneumothorax)
Pericardiocentesis (cardiac tamponade)
Open thoracotomy
Surgery
Balloon pump
QUICK REVIEW
(1) What are the four main types of shock?

(2) Name one way to decrease after load

(3) Name one way to decrease preload

(4) Describe some differences between spinal shock and neurogenic shock

(5) Describe the procedure for inserting an intraosseous needle

(6) Describe the process for performing needle chest decompression

(7) What type of shock would you expect a tension pneumothorax to lead to?

(8) What’s concentration of epinephrine do you use for subcutaneous and administration?
(9) What are the classic findings of Beck’s triad? What condition is it associated with?

________________________________

(10) You are treating your patient who is in hypovolemic shock. You are monitoring his urine output. How much urine output would indicate adequate fluid resuscitation and organ perfusion?

________________________________
# Asthma

## Chronic, Reversible Obstructive Pulmonary Disease

### Hallmarks of Condition:
- Airway inflammation
- Bronchoconstriction/spasm

### #1 Chronic Childhood Illness

## Assessment

- Dyspnea, cough, wheezing, restlessness
- Tightness in chest
- Possible concurrent resp infection sx

### PMHX

- Prior asthma problems
- Hx of intubation or admission
- Current medications

## Exam

- Tachypnea, Tachycardia
- Children in distress: bradycardia, apneic periods
- Orthopneic posturing/tripod
- Prolonged expiratory phase
- Accessory muscles
- Wheezing on exhalation
- Worsens to inhalation wheezing

## Diagnostics

- CBC, Chemistries, ABG’s PRN
- CXR
- Peak flow: The best objective measurement

## Interventions

- O2, Monitors, IV access
- Positioning

## Medications

### Bronchodilators:
- Epinephrine: Status Asthmaticus
- Mag Sulfate: airway smooth muscle relaxant

### Rescue Medications:
- Short Acting beta-agonists
- Albuterol, Proventil, Xopenex

### Anticholinergics:
- Atrovent

### Corticosteroids:
- Solu-Medrol, Prednisone

### Antibiotics

- For concurrent infections if indicated

### Anxiolytics:
- Ativan
May be useful as an adjunct

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<tr>
<th>CEN REVIEW NOTES</th>
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</table>
**DISPOSITION/EDUCATION**
- Allergen/triggers reduction
- Medications; finish steroids, don't over use MDI
- Home nebulizers and peak flow meters
- Use of spacer
- Smoke-free environment
- Follow up

**ACUTE BRONCHITIS**
- Inflammation of bronchi/trachea
- D/T irritants (pollen, smoking etc)
- Usually concurrent with a URI (viral)
- Excess production of bronchial mucus

**ASSESSMENT**
- Dyspnea, wheezing
- Cough: dry or productive
- Fever possible
- Pain in chest/back

**PMHX**
- Recent URI
- Pulmonary disease
- Smoking Hx
- Medications
- Immunizations (Pneumovax)

**EXAM**
- Tachypnea
- Accessory muscle use
- Prolonged expiratory phase
- Sputum
- JVD: Cor Pulmonale
- Barrel chested
- Orthopneic positioning
- Rhonchi; may improve w/cough

**DIAGNOSTICS**
- CBC, Chemistries
- CXR
- ABG's

**INTERVENTIONS**
- O2, Monitor, IV
- EKG if indicated
- IPositioning

**MEDICATIONS**
- Inhaled bronchodilators
  - Albuterol, Proventil, Xopenex
- Inhaled anti-cholinergics
  - Atrovent
- Corticosteroids
  - Solu-medrol, Prednisone
### Anxiolytics
- Ativan

### Expectorants/antitussives
- Guifenessin, Dextromethorphan, Tessalon
- Avoid codeine/hydrocodone

### Antibiotics if indicated
- All pneumonia tx aimed at: *Streptococcus*
- Think PCN’s
- Smokers can get “atypical” bacteria
  - *Chlamydia Pneumoniae, Mycoplasma, Legionella*
  - Think Macrolide (Zpack, Biaxin, Emycin), Sulfa, Doxy

### DISPOSITION/EDUCATION
- Clinical improvement
- No more serious underlying cause
- Access to medications
- Complete steroids/antibiotics
- Smoking cessation

### BRONCHIOLITIS
- Lower resp tract
- Inflammatory obstruction
- Peds <2y.o.
- 90% d/t Respiratory syncytial virus (RSV)
- Winter months

Obstruction leads to:
- Air trapping, resistance, atelectasis
- Hypoxemia, fatigue

Associated with later asthma development
- First attack, gen. the worst

### ASSESSMENT
- URI prodrome with progressive dyspnea
- Increasing cough
- Poor feeding, irritability

### PMHX
- Chronic pulmonary diseases
- Congenital heart disease
- Immunodeficiency
- Smoking exposure

### EXAM
- Tachypnea (apnea periods poss in infants)
- Tachycardia
- Fever
- Grunting, nasal flaring
- Accessory muscles (retractions)
- Peripheral/central cyanosis
- Wheezing
- Signs of dehydration
<table>
<thead>
<tr>
<th>DIAGNOSTICS</th>
<th>MEDICATIONS</th>
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</thead>
<tbody>
<tr>
<td>CBC, Chemistries</td>
<td>Nebulized Meds:</td>
</tr>
<tr>
<td>Consider ABG’s</td>
<td>May give trial, but only continue</td>
</tr>
<tr>
<td>Consider blood cx</td>
<td>if documented improvement</td>
</tr>
<tr>
<td>CXR: hyperinflation, infiltrates</td>
<td>Adrenergic/Beta agonist: Albuterol, Xopenex</td>
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<tr>
<td>Possible nasal swab for RSV</td>
<td>Anticholinergics: Atrovent</td>
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<tr>
<th>INTERVENTIONS</th>
<th>Others</th>
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<tr>
<td>Supplemental O2/Blow-by (humidified)</td>
<td>Antibiotics: if indicated</td>
</tr>
<tr>
<td>Monitor, IV access</td>
<td>Claforan, Rocephin (3 gen ceph)</td>
</tr>
<tr>
<td>IV fluids</td>
<td>Antivirals: not routinely</td>
</tr>
<tr>
<td></td>
<td>Corticosteroids: no proven benefit</td>
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<thead>
<tr>
<th>DISPOSITION/EDUCATION</th>
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<tr>
<td>Hospital admission:</td>
<td></td>
</tr>
<tr>
<td>Based on risk factors (prematurity) and response to tx</td>
<td></td>
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<tr>
<td>If concurrent bacterial infection (pneumonia)</td>
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<tr>
<td>Dehydration</td>
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<tr>
<td>Poor response to therapy</td>
<td></td>
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<tr>
<td>Persistent SpO2 &lt;92-94%</td>
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| D/C Home                    |                          |
| Home nebulizers             |                          |
| Fever management            |                          |
| Feedings/fluid balance      |                          |
| Should have f/u within 24 hours |                          |

<table>
<thead>
<tr>
<th>CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)</th>
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<tbody>
<tr>
<td>Chronic/recurrent airflow obstruction</td>
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<tr>
<td>Smoking #1 causative factor; others</td>
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<tr>
<td>Inc. bronchial mucus production</td>
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<tr>
<td>Airway muscle contraction and decreased recoil</td>
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<tr>
<td>Results in difficulty exhaling</td>
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<tr>
<td>Impaired gas exchange</td>
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Includes:
Asthma: reactive airway
Chronic Bronchitis: airway inflammation
Emphysema: airway collapse
All three present - one dominates
| CHRONIC BRONCHITIS |  |
|---------------------|  |
| Inflammation of bronchi |  |
| Inc. mucus prod. |  |
| Chronic cough |  |
| Air trapping and chronic hypoxia |  |
| Hypercapnia |  |
| COPD and the respiratory drive: |  |
| Regulated by increases of PaCO2 |  |
| (Hypercarbia is prime stimulus to breathe) |  |
| Over time body switches the drive to decreases in PaO2 levels |  |
| Hypoxia now becomes stimulus to breathe |  |
| The theory is that increasing FiO2 may obliterate that drive |  |
| Pulmonary HTN may develop (Cor Pulmonale) |  |
| “Blue bloater” |  |
| Periph. edema, anasarca and JVD |  |
| EMPHYSEMA |  |
| Impaired exhalation |  |
| Over distention of air spaces |  |
| Airway collapse |  |
| Loss of elastic recoil |  |
| Progress from DOE to DAR |  |
| (Dyspnea on exertion - Dyspnea at rest) |  |
| Cough not typical |  |
| Resp. infections common |  |
| ASSESSMENT |  |
| Dyspnea, Tachypnea, Tachycardia |  |
| Current URI |  |
| “Pink Puffer” |  |
| PMHX |  |
| Pulmonary disease |  |
| Recent URI |  |
| Cardio-vascular Dz |  |
| Smoking Hx |  |
| Medications |  |
| Home O2 |  |
| Immuniz: Pneumococcal Vacc. (Pneumovax) |  |
| EXAM |  |
| Speaks incomplete sentences |  |
| Position/tripod |  |
| Tachycardic, dysrhythmias on monitor |  |
| Pursed lip breathing |  |
| Lungs: crackles, rhonchi, wheezes |  |
| DIAGNOSTICS |  |
| ABG’s |  |
| CBC, Chemistries |  |
BNP (brain natriuretic peptide)  
---WHY?

CXR: flat diaphragm, long, narrow heart,  
over inflation, barrel chested

INTERVENTIONS  
O2, Monitor, IV access  
Positioning  
CPAP/BiPAP  
Peak flows

MEDICATIONS  
Inhaled Beta Agonists:  
Albuterol, Proventil, Xopenex

Inhaled Anti-cholinergic:  
Atrovent

Mucolytics:  
Dextromethorphan, Guifenessin

Corticosteroids: Solumedrol

Antibiotics: if indicated

Anxiolytics: Ativan

Nicotine replacement

DISPOSITION/EDUCATION  
Admit:  
Lack of clinical response  
Concurrent condition requiring admit  
Inability to wean from O2

Home Instruction:  
Small frequent meals  
Exercise  
Deep breathing, coughing exercise  
Medication compliance  
Home O2 safety  
Follow up: PCP or consult Pulmonologist  
Immunizations  
Smoking Cessation

PNEUMONIA  
Major cause morbidity and mortality

Types:  
Community Acquired Pneumonia  
VAP  
HAP/Nosocomial
60-90% viral
Bacterial higher mortality (*Streptococcus*)
Pneumonia 6th leading cause of death in US

**ASSESSMENT**
- Cough, +/- fever, chills
- Chest pain, pleuritic
- Tachypnea, tachycardia
- Cyanosis
- Splinting chest wall
- Sputum: purulent to blood-tinged
- Lungs: coarse crackles
- Pleural rub?

**DIAGNOSTICS**
- Labs, Blood Cx, ABG's PRN
- CXR: possible negative early on
- EKG PRN, Cardiac Biomarkers

**INTERVENTIONS**
- Cardiac/Pulse Ox Monitor PRN
- Supplemental O2
- IV access
- CPAP-BiPAP possible
- Antibiotics within 4 hours ER arrival
  ***NOT from time Dr sees pt!***
- Inhaled bronchodilators
- Antipyretics

**HYPERVENTILATION**
- Rapid breathing
- Decreased PaCO2 = 
  Cerebral vasoconstriction (refer to head injuries and hyperventilation!)
- Respiratory alkalosis
- Anxiety common cause

Other causes:
- MI, PE, intracerebral bleed
- ketoacidosis, salicylate OD

**ASSESSMENT**
- SOB, air hunger
- Tingling in extremities
- Tingling in face/lips (“circumoral”)
- Lightheaded/dizzy
- HA
- Chest discomfort/pain
- Fever

**PMHX**
- Cardiac disease
- Diabetes
- Panic/anxiety disorder
- Consider exposures/toxins
- Medications/OTC products
- Allergies
EXAM
Anxious or panicked appearance
Tachypnea
Carpopedal spasms
Lungs: clear

DIAGNOSTICS
Pulse Ox unreliable
Toxicology panel PRN
D-dimer PRN
EKG, Cardiac enzymes PRN

INTERVENTIONS
Positioning
Use abdominal breathing
Cardiac monitor/Pulse Ox PRN
IV access/Labs PRN

MEDICATIONS
Anxiolytics:
Benzodiazepines; Ativan, Valium

DISPOSITION/EDUCATION
Recognize/avoid triggers
Avoid benzodiazepine Rx's (suggest Vistaril)
Referral to PCP or Mental Health PRN

PNEUMOTHORAX (PTX)
Air in intrapleural space
Causes:
Trauma
Primary spontaneous, no disease (tall, thin)
Secondary spontaneous, w/disease (COPD, bleb)
Iatrogenic (subclavian needle insertion)

ASSESSMENT
Weakness, dyspnea
Pain: chest/thorax
Cyanosis
DOE

PMHX
Concurrent diseases/illness
Smoking Hx
Substance abuse Hx
Medications
Prior pneumothorax

EXAM
Tachypnea, tachycardia
Poss hypoxia, cyanosis
Lungs: decreased, diminished, absent
Subcutaneous emphysema
DIAGNOSTICS
CXR, ABG’s PRN
CBC, Chemistries if indicated
EKG if indicated

INTERVENTIONS
ABC’s
ABSENT BS = EMERGENCY
O2, Monitor, IV access
Assist with chest tube
CXR immediately following and 4 hours later

MEDICATIONS
Procedural Sedation (chest tube)
Antibiotics: if indicated

DISPOSITION/EDUCATION: Admission

HEMOTHORAX
Blood accumulation in intrapleural space
Usually d/t chest wall trauma
Usually a degree of PTX exists also
Potential for hypovolemia

“Consider” an open thoracotomy:
If 1000 ml initially out via chest tube -OR-
if >200ml/hr for 3-4 hours

Tracheal shift: LATE SIGN
JVD (Jugular venous distention)
May be difficult to assess in large necks
Head of bed at 40-45 degrees to assess

Treatments:
Fluid, blood replacement, autotransfusion
Chest tube
Surgical intervention

TENSION PNEUMOTHORAX
Life threatening
Air enters pleural space and cannot exit
Increasing air on one side causes mediastinal shift
Heart/great vessel compression, decreased venous return
Leads to hypotension, shock, vascular collapse

What kind of shock:? _______________________

MARKEDLY DECREASED or ABSENT BS
Needle thoracentesis:
14 ga needle
2nd ICS, MCL affected side
Reassess
Followed by chest tube insertion
Surgical repair needed
PULMONARY EMBOLUS
Thrombus from leg, pelvis or R side heart
Lodges in branch of pulmonary artery (PA)
Total or partial occlusion
Potential infarct
3rd most common cardiovascular cause of death
10% Acute Pulmonary Embolus (APE) die in 60 min

ASSESSMENT
Dyspnea, cough, chest pain
Hemoptysis +/-

PMHX
Hx of thrombosis
Immobility
Obesity
Pregnancy
Recent Trauma/Surgery
Meds; OCPs
Social: Smoking

EXAM
Tachypnea, tachycardia
Possible hypotension, cyanosis, petechiae
Lungs: crackles, pleural rub
Decreased SpO2
Dyspnea at rest

DIAGNOSTICS
CBC, SED Rate, D-Dimer
ABG’s PRN
CXR: initially normal

Hi-Res Chest CT:
Comparable to pulmonary angiography
Consider if renal disease present

V/Q when CT unavailable or renal insuff.

INTERVENTIONS
ABC’s
O2, Monitor IV

MEDICATIONS
Anticoagulants: Heparin, LMW Heparin (Lovenox)
Fibrinolytics: TNKase, r-TPA
Analgesics
**ADULT RESPIRATORY DISTRESS SYNDROME (ARDS)**
Sudden, progressive, severe
Diffuse bilateral infiltrates
Freq assoc. w/pulmonary contusion
40-70% fatal
D/t direct injury or systemic illness
Diffuse inflammatory response in lung tissue
Followed by increasing capillary/alveolar permeability with fluid leakage
Pulmonary edema results
Atelectasis and decreased compliance leads to respiratory failure

**ASSESSMENT**
Sudden marked resp. distress
Recent trauma
Drowning
Inhalation injury
Aspiration
Pregnancy related HTN
Sepsis

**PMHX**
Cardio-pulmonary disease
Surgeries
Smoking hx

**EXAM**
Tachypnea, hypoxia
Hypotension, tachycardia
Marked distress, cyanosis, retractions
Lungs: fine to coarse crackles

**DIAGNOSTICS**
ABG’s, CBC, Chemistries
Consider Blood Cx (sepsis possible cause)

**CXR:**
No infiltrates poss. early
Bilat. diffuse, white infiltrates, NO CMG
Complete “white out” later

**INTERVENTIONS**
ABC’s
O2, Monitor, IV Access
Positioning
RSI prn
CV catheter prn
Chest tube prn
Aggressive pulmonary management

**RESPIRATORY TASKS**

**ENDOTRACHEAL INTUBATION**
Oral/nasal routes
Indications/Contraindications?
Possible complications:
- Aspiration, laryngospasm
- Cardiac arrhythmias
- Hypoxemia
- Oro-pharyngeal trauma
- Tracheal stenosis, necrosis, erosion
- Failure to wean
- Ventilator pneumonia

Contraindications:
- Orofacial injuries
- Cervical spine injury ---- relative

**RAPID SEQUENCE INTUBATION (RSI)**

**The SEVEN P’s**
1) Prepare
   - Start IV/O2/Monitors; prepare meds

2) Pre-oxygenate
   - 100% O2 for 3 min w/NRB

3) Pre-treatment
   - Give sedative (Etomidate, Propofol, Ketamine)
   - Give adjuncts (Lidocaine, atropine)
   - Defasculating agent

4) Paralysis
   - Short acting NMB (Succinylcholine, Vecuronium)
   - Start mechanically ventilating pt

5) Placement
   - Sellick maneuver, Intubate, Inflate

6) Proof
   - Observation, auscultation, ETCO2, CXR

7) Post-intubation
   - Secure tube, continuous ETCO2 monitoring
   - Continue sedation/paralysis PRN
   - Monitor pt’s responses

Confirming placement 10 ways....
1) ____________________________
2) ____________________________
3) ____________________________
4) ____________________________
5) ____________________________
6) ____________________________
7) ____________________________
MECHANICAL VENTILATION
Positive or negative pressure
Volume cycled: based on volume
Pressure-cycled: based on resistance
Time-cycled: based on time

Alarms:
Frequently check that they are “ON”
If cause for alarm cannot be easily identified;
take the pt off and manually ventilate

Frequent assessments
Re-assess cardiopulmonary every 2-4 hours
VS, auscultate
Pulse Ox, ETCO2, hemodynamic parameters
Re-assess pt’s need for sedatives/neuro-muscular
blockers

Problem with ETT/VENT? Think “D.O.P.E.”

1) Dislodgement
   How to check for: ______________________
   How to fix: ______________________

2) Obstruction
   How to check for: ______________________
   How to fix: ______________________

3) Pneumothorax
   How to check for: ______________________
   How to fix: ______________________

4) Equipment

END-TIDAL CO2 MONITORING
Measures CO2 at end expiration
Provides early detection of hypercapnia
Can monitor apnea
Normals: 35-45 mm Hg
Report a 10% inc/dec. in trend readings

CHEST TUBES
Inserted into pleural space
Allows blood, pus, fluid or air to drain
Lung re-expands
4th ICS anterior axillary line (drains air & blood)
The negative pressure suction, pulls air out
The water seal prevents air going back in

CARE OF CHEST TUBES
Routinely assess functioning
Notify MD if drainage is >200 ml in 1 hour

NORMALLY:
The water seal level RISES on inspiration
DECREASES on expiration (“ROI, DOE”)
IF ON PPV It’s opposite

WHEN TO CLAMP
While changing the drainage system
If system cracks
Never clamp > 1 minute
Risk of tension PTX developing

BUBBLE = TROUBLE?
Clamp intermittently from proximal to distal
Bubbles stop when the air leak is between
the clamp and the pt.
If bubbles never stop when clamping; there
may be a crack in the drainage system
OBSTETRICAL EMERGENCIES

SPONTANEOUS ABORTION
Loss of pregnancy before viability
Before 20 weeks or < 500 grams
Always consider with vaginal bleeding
In US 10-15% of all pregnancies
Cause unknown in majority
Main “rule out” = Ectopic pregnancy

ASSESSMENT
Pain: abdominal, back, pelvic
Crampy, pressure
Vaginal bleeding: color, tissue, amount
Had + preg test yet?
Fatigue, vertigo, syncope

PMHX
LMP, Estimated date of confinement (EDC)

Nagel’s Rule:
LMP + 9 months + 7 days

Reproductive Hx
Contraceptive Hx
Prenatal Care
Medications
Known blood type?

EXAM
Orthostatics (r/t hemorrhage)
Pelvic examination assess cervix/bleeding
Abdominal examination

DIAGNOSTICS
Urine/Serum HCG
CBC
Type and RH
U/A
Consider STD screening
OB Ultrasound

INTERVENTIONS
ABCs, IV, O2 PRN
IVF bolus (hypovolemia)

MEDICATIONS
Rh Immune globulin (RhoGAM):
For all Rh negative pt’s
Oxytocin (Pitocin) or Methergine:
For hemorrhage control

EDUCATION
Bed rest 24-48 hours
Pelvic rest, pads only
Arrange follow up
DISPOSITION
Home if stable and IUP verified
Admit if ectopic or if needs D&C
Admit for IVF, dehydration

PLACENTA PREVIA
--------> “PAINLESS PRIVATES”
A serious cause of vaginal bleeding
2nd-3rd trimester
Placenta covers the internal cervical os
Painless, bright-red bleeding

PLACENTAL ABRUPTION
--------> “ALWAYS PAINFUL”
Emergency OB condition
Placenta separates before fetal delivery
Accounts for 15% of all perinatal deaths
Can be partial or complete abruption
Bleeding visible or concealed
Massive hemorrhage possible
Compromises fetal circulation
Can lead to DIC
Uterine pain/tenderness, with bleeding

ASSESSMENT
OB, reproductive, prenatal Hx
LMP
Ultrasound yet?
S/Sx of shock?

DIAGNOSTICS
CBC, Coags, Type and Cross
Kleihauer-Betke
Pelvic U/S
Fetal Monitoring

INTERVENTIONS
ABCs, O2, IVF bolus
Position: left lateral decubitus
Administer RhoGAM if indicated
IF hemodynamically stable, xfer to L/D
Prepare for emergency C-Section
Prepare for neonate resuscitation

ECTOPIC PREGNANCY
Implantation outside the uterus
95% will implant in the tubes, R>L
Risk of tube rupture
Subsequent internal hemorrhage and shock
The #1 cause of maternal death in pregnancy

ASSESSMENT
Abdominal pain assessment
Possibly diffuse, unilateral/bilateral
Vague or sharp
Vaginal bleeding?
Dizziness, syncope
**EXAM**
- Abdominal exam: tenderness, rebound, rigid?
- Pelvic exam: masses, tenderness

**DIAGNOSTICS**
- Serum and urine preg test
- Quant B-hcg
- CBC, Type and Rh
- Transvaginal U/S to r/in or out

**INTERVENTIONS**
- ABC's, IV access
- Possible fluid resuscitation
- Assist with pelvic
- Prepare for U/S
- Prepare for admit/OR possible

**MEDICATIONS**
- Methotrexate: terminates the EP
- RhoGAM: if indicated
- Opioid Narcotics: if indicated
- Antiemetics

**PIH: PREECLAMPSIA; ECLAMPSIA; & HELLP SYNDROME**

PIH is HTN unique to pregnancy
PIH synonymous with Pre-eclampsia
Exact cause is unclear
Placental dysfunction with systemic vasospasm
Then increased in peripheral vascular resistance
Pt becomes volume contracted
Vasospasm causes decreased placental/fetal circulation
Primarily only with the first pregnancy

**Preeclampsia:**
- HTN, Proteinuria, and nondependent edema
- Occurs after 20th week of pregnancy

**Eclampsia:**
- Extension of preeclampsia
- Convulsions, coma or both
- Can occur in early postpartum too

**HELLP**
- Hemolysis, Elevated Liver Enzymes, and Low Platelets
- Severe form of preeclampsia
- Must be after 20 weeks gestation
- 20% may go onto DIC

**Hypertension:**
- 140/90 or more -OR-
- A SBP inc of 30 or more, -OR-
- A DBP inc of 15 or >
DIAGNOSTICS
U/A: looking for proteinuria
CBC: shows anemia and thrombocytopenia
Liver panel: elevated
Check coags (risk for DIC)
Abd/pelvic U/S (fetal stability, hemorrhage)
CT Head: edema, bleed

INTERVENTIONS
ABC's, O2, Monitor
2 IV access sites
Position left lateral decubitus
Prep for possible emergent delivery, esp if > 30 wks.
Delivery is THE definitive treatment

MEDICATIONS
Magnesium Sulfate:
Blood pressure reduction
Seizure prevention

Benzodiazepines:
For seizures resistant to Mag

Anti hypertensives IV:
Hydralazine (Apresoline)
Labetalol (Trandate)
Nitroprusside (Nipride)
Goal: maintain DBP 90-100

HYPEREMESIS GRAVIDARUM
Severe vomiting before 20th week
Incidence peaks at 8-12 wks gestation
Occurrence overall 2%
True condition, usually needs admission
Frequent, sustained vomiting for 4-8 weeks
Leads to: wt loss, dehydration, metabolic acidosis
Complications: GI bleeding, Mallory-Weiss tears
At risk: primiparous, young, multiple gestations

DIAGNOSTICS
CBC, Chemistries
Serum ketones
U/A: ketonuria

INTERVENTIONS
ABC's, supplemental O2 PRN
IV access: give 1-2 L NS
Then switch to dextrose containing solution
Possible NGT if severe
Prepare for admission
Attempt gradual oral rehydration

~ 62 ~ CEN Review Course
MEDICATIONS
Anti-emetics:
Reglan, Zofran (safe)
H2 Blocker: IV Zantac (safe)

POSTPARTUM HEMORRHAGE
Bleeding delivery of placenta and membranes
A degree of hemodynamic instability
Occurs with vaginal or c-section deliveries
90% of all cases d/t uterine atony
Late PPH (6-10 days later): retained products

INTERVENTIONS
ABC’s, O2 as indicated
Check orthostatics
Cardiac monitor if indicated
IV access
Atony: firm manual uterine massage
Possible D & C for retained fragments

MEDICATIONS
Pitocin (oxytocin); increases uterine tone
Methergine; for persistent bleeding

EMERGENCY DELIVERY
Get a rapid Hx
Imminent delivery:
Contractions greater than 30 seconds -OR-
Intervals q5 min or <

Stage 1: contraction onset to cervical dilation
Stage 2: cervical dilation to infant delivery
Stage 3: infant delivery to placental delivery

INTERVENTIONS
Secure ABC’s
IV access
When head emerges, tell mom “pant like a puppy
Support head as it’s delivered
When head delivered, feel around neck for cord
If cord present; gently slip around infants neck
If cord too tight; clamp x2 and cut NOW
Wipe NB face; suction...
#1 MOUTH, then #2 NARES
Support head; deliver shoulders
Anterior then posterior
Hold head-dependent at level of introitus
Suction again as needed
## NEONATAL RESUSCITATION

Required if s/sx of CV or respiratory compromise

**Hi risk:**
- Preterm
- Breech birth
- Multiple gestations
- Meconium staining
- APGAR at 1 and 5 min
- Lack of infant's ability to start extrauterine circulation

**FILL IN THE BLANKS:**

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MOST neonates are born w/o asphyxia
MOST only require: warming, suctioning and mild stimulation
ONLY 6% of all NB require further resuscitation

### ASSESSMENT
- Maternal OB Hx
- Current other conditions

### EXAM
- APGAR at 1 and 5 minutes
- Meconium staining?

### INTERVENTIONS
- Treat mom too!
- ABCs
  - Quickly dry off, put under heat source
  - Keep on back or side; neck neutral

  Absent, slow or difficult respirations:
  - Suction trachea
  - Stimulate: flicking feet, rubbing back
  - Resp slow or shallow:
    - 100% blow by O2
  - Resp gasping or ineffective:
    - POSITIVE PRESSURE ambu @ 40-60/min
  - HR < 60 and not increasing with PPV
    - Chest compressions
  - Consider intubation
MEDICATIONS
Epinephrine: Inc CO; vasoconstriction

Narcan: “Consider” if: skin color and HR normal but respiratory depression persists AND mother has received an an opiate within 4 hours of delivery. NOT for long-term opiate use!

Glucose: As indicated

Sodium bicarb: For prolonged resuscitation

DYSFUNCTIONAL UTERINE BLEEDING (DUB)
Many causes:
Uterine fibroids
Menstrual irregularities
Trauma
Infection
Hormonal imbalances

Steady painless bleeding w/o clots or tissue
Basic labs and pelvic to rule in/out: pregnancy, infection

Tx: Low dose OCP (progesterone)
Ensure f/u to re-assess response to therapy
Must do this to ensure NOT cancerous

PELVIC INFLAMMATORY DISEASE (PID)
Non specific term
Infection of higher reproductive structures
Acute, subacute, or chronic
Common pathogens: Gonorrhea, Chlamydia
Damage to fallopian tubes over time results
Hi-risk for future ectopics

No single piece of history, exam or lab test is highly sensitive or specific

Long term consequences:
Chronic pelvic pain
Dyspareunia
Infertility

Risk factors:
Prior PID
Multiple sexual partners
Younger age
Frequent sex
IUD use
Douching
Smoking
ASSESSMENT
Abdominal pain; worse w/movement
Dull aching to sharp, persistent
Vaginal d/c: mucopurulent, malodorous
Vaginal bleeding possible
N/V and/or F/C

EXAM
Fever?
Lower abdominal TTP; rebound, heel jar
Pelvic:
Abnormal d/c
Cervical tenderness
Uterine/adnexal tenderness
May be difficult to “clear the appendix”

INTERVENTIONS
IV access for fluids, meds if indicated
Assist with pelvic specimen collection
MUST rule out pregnancy!

Main differentials:
Ectopic
Tubo-ovarian abscess (TOA)

MEDICATIONS
Antibiotics:
Rocephin treats Gonorrhea
Zithromax treats Chlamydia
Flagyl treats trichomoniasis

Analgesics:
Opiates, non-opiates

DISPOSITION/EDUCATION
Complete all home antibiotics
PID goes home with 10-14 d of Doxycline
Should make arrangements for “test of cure”
Consider HIV testing
Safe sex recommendations
Return if fever, intractable vomiting or worse

URINARY TRACT INFECTION (UTI)
Symptomatic bacturia anywhere along the
GU tract
Upper UTI’s: Pyelonephritis
GU tract normally sterile
Uncomplicated lower UTI = cystitis
80% UTI’s = E. Coli
Dysuria or frequency in men < 50 usually = a STD
ASSESSMENT
Hx of dysuria, frequency, urgency, hematuria
Suprapubic/lower abdominal pain
Possible f/c, n/v (pyelonephritis)

PMHX
Prior UTI's
Sexual activity/STD Hx
LMP, vaginal symptoms
Concurrent conditions (Diabetes)

EXAM
Suprapubic/lower abd TTP
CVA tenderness

DIAGNOSTICS
U/A
In/out cath if on menses or vag. d/c
UTI: nitrites, leukocytes and/or blood
C/S to guide therapy (esp if recurrent)
Preg test if indicated

MEDICATIONS
Antibiotics:
Depends on local pathogen prevalence
Bactrim (SMX-TMP) still good for most
Quinolones are over kill
Macrobid/Macrodantin okay in pregnancy
(only goes to urine)

Urinary agents:
Pyridium: local analgesic effect on bladder and
urethral tissues

EDUCATION/DISPOSITION
Finish all antibiotics
Front to back perineal cleansing
Don't avoid voiding
Avoid bubble bath/perfumed soaps
Void after sex

PYELONEPHRITIS
Upper UTI involving renal tissues
D/T ascending lower UTI unchecked
80-90% d/t E. Coli
If occurs in pregnancy, then assoc with
maternal/fetal problems

Complications of pyelo: renal insuff, failure,
perinephric abscess, bacteremia

ASSESSMENT
UTI like symptoms
Pain: back, flank, abdominal
Possible F/C, N/V, malaise
PMHX
Diabetes
Immunosuppression
Recurrent UTI's

EXAM
Febrile, tachycardic
Uncomfortable
CVA tenderness or suprapubic tenderness

DIAGNOSTICS
U/A, urine preg, and consider Urine C/S
CBC, Chemistries

INTERVENTIONS
IV access for fluids/medications
Consider IVF boluses
Bladder catheterization if unable to void
Possible pelvic exam assistance

MEDICATIONS
Antibiotics:
Quinolones usually (Cipro, Levaquin)

GU analgesics:
Pyridium

Opiate analgesics:
if indicated

Anti-emetics: many

DISPOSITION/EDUCATION
D/c if can take PO okay
Good idea send Rx anti-emetic
Recheck in 24-48 hours
Complete all antibiotics
Rest and plenty of fluids.

URINARY CALCULI
One of the most common GU problems seen in ED
Stones can occur anywhere in the GU tract
Renal pelvis most common location
Pain exists when stone descends via the ureter
Pain not dependent on size alone
80-85% pass spont w/o problems or intervention

Risk factors:
Sedentary
Gout
Prior stones
Frequent UTIs
High protein intake
Pregnancy
Dehydration
An obstruction must be ruled in/out
Obstruction WITH pyelo is an emergency
Complications:
Perinephric abscess
Urosepsis
Death

ASSESSMENT
Severe, colicky flank pain
Constant, dull underlying pain
Possible radiation: back to front, into groin
No relieving factors; can't get comfortable
Possibly associated with dysuria, N/V
Febrile?

EXAM
Tachycardia
Moderate to severe distress, diaphoretic
CVA tenderness
Normal GU exam

DIAGNOSTICS
U/A for hematuria, infection
Degree of hematuria not relevant
CBC, Chemistries
Renal CT w/o contrast now test of choice

INTERVENTIONS
ABC's, IV access
IVF boluses do not "push the stone out"

MEDICATIONS
Non-narcotic analgesics:
Ketorolac (Toradol):
Caution in kidney disease

Opioid analgesics:
Dilaudid, Morphine

Anti-emetics:
Zofran, Compazine

DISPOSITION/EDUCATION
Admit if:
Intractable pain or n/v
Infection w/stone
Single kidney
Underlying renal insufficiency
Stone larger than 6mm
Can't arrange f/u in 24 hours

Discharge:
Straining urine instruction
Rest, fluids
Should get adequate Rx for pain/nausea meds
Instructions to return if fever or can't take PO
TESTICULAR TORSION
Strangulation of testicle d/t twisting
Obstruction of arterial flow
2/3 of all cases between 12 to 18 y.o.
Detorsion within 6 hours = 80-100% salvage
True urological emergency

ASSESSMENT
Pain; rapid onset, severe
Poss. radiate to lower abdomen
Unrelieved with elevation
Can even occur during sleep
N/V and low grade fever (LGF) possible

PMHX
Prior episodes?
Spontaneous resolution by 40% with priors

EXAM
Moderate to severe distress/discomfort
Colicky (think kidney stone)
Diaphoretic, tachycardic, N/V poss.

Scrotum
Possibly warm, swollen, and tender
Maybe higher riding
Compare side to side
Check for hernias
Palpate epididimis
Observe for signs STD's

DIAGNOSTICS
U/A, CBC
Doppler U/S, looks at size/shape and flow

INTERVENTIONS
IV access
If urology tries manual detorsion,
may need conscious sedation
Prepare for admission possibly

MEDICATIONS
Narcotic Analgesics
Antiemetics
Procedural sedation meds

EPIDIDYMITIS
Inflammation/infection
Common 19-40 y.o.
< 35 y.o. most likely STD (C. Trachomatis)
>35 y.o. usually E. Coli
Complication: abscess
Difficult to distinguish: torsion, carcinoma
ASSESSMENT
Pain: gradual, dull, aching, scrotal
Worse with sex
Improve with elevation
Irritative voiding sx
N/V, F/C, abdominal pain all possible
Urethral d/c possible

PMHX
Prior urethritis or STDs
Hi-risk sexual behaviors

EXAM
Tachycardia, hypertension poss
Possibly febrile
“Duck waddle”
Edematous epididimis/scrotum
Scrotal erythema
Urethral d/c

DIAGNOSTICS
U/A: +WBC's, +bacteria
CBC possible
Consider RPR
Urethral swab
Doppler U/S (r/o torsion)

INTERVENTIONS
IV Access possible
Positioning

MEDICATIONS
Antibiotics
Analgesics
Anti-emetics

DISPOSITION/EDUCATION
Complete all antibiotics
Urology or PCP f/u: test of cure
Consider HIV testing
Bed rest until pain decreases
Scrotal support
Safe sex education

PROSTATITIS
Infection/inflammation prostate gland
Can lead to sepsis if not tx'd appropriately

3 classes:
Acute bacterial (#1 seen in ER)
Chronic bacterial/Nonbacterial

Prostatodynia
Men 30-50 y.o.
Diabetes, immunosuppressed or HD pt's at risk
Usually d/t ascending UTI
ASSESSMENT
F/c, n/v very possible
Pain: low back, low abdomen, scrotal, peri-anal
Dull, aching
Myalgias
Urethral d/c

PMHX
Prior prostate problems
Urological interventions
Diabetes

EXAM
Febrile, tachycardic
Mild/moderate discomfort
Rectal exam: prostate = boggy, tender, hot

DIAGNOSTICS
CBC, Chemistries, U/A
Consider urethral swabs
Pelvic CT for suspected abscess

INTERVENTIONS
IV Access, fluids, meds
Catheterize if needed for obstructive sx

MEDICATIONS
Antibiotics: quinolones, STD coverage
Analgesics

DISPOSITION/EDUCATION
Possible admission if “sick”
Sitz baths
Complete antibiotics
Stool softeners
Follow up/recheck

RENAL TRAUMA
Seldom occurs by itself
Consider if: chest, abdominal or back trauma
Most common cause: blunt force
MVCs & GSWs = 80% of renal injuries
Consider with lower rib or spinal process fxs
85-90% are minor and don't need surgery
If surgery indicated: must be within
12 hours d/t risk of ischemia
ASSESSMENT
Mechanism of injury
Pain: CVA tenderness, or abdominal
Quality of pain varies
Frank hematuria
**A heme NEGATIVE U/A, does NOT r/o injury**

PMHX
Renal disease, HTN
Solitary kidney
Prior urological/renal procedures/surgery

EXAM
Possible hypotension, tachycardia
Flank TTP, abdominal/back TTP
Flank swelling, hematoma or ecchymosis:

DIAGNOSTICS
CBC, Chemistries
Possible serial Hcts
Coags
U/A (1/3 will have normal U/A)
Hcg
FAST
Abd/Pelvic CT

INTERVENTIONS
ABCs, supplemental O2 if indicated
IV access; 2 large-bore
Fluid resuscitation if indicated
Blood replacement if indicated
NGT, F/C, Cardiac/pulse ox monitors

DISPOSITION
Surgery
Admit/observe
Transfer out

BLADDER RUPTURE
Most bladder trauma d/t blunt forces
80% bladder ruptures concurrent w/pelvic fxs
Bladder contusion much more common
Distended bladder inc. the chances of rupture
Seldom occurs alone

If penetrating trauma:
Bowel probably involved
Much higher mortality (up to 60%)

Peritonitis/sepsis follow d/t urine/blood
invading abdomen
ASSESSMENT
Pain: abd, pelvic, dull, constant
Difficulty/inability to void
Possible frank hematuria

PMHX
Renal disease
Prior urological problems

EXAM
Possible shock symptoms
Mod/severe distress
Lower abd/perineal hematoma
Abd distention, tenderness, rigidity, rebound
Poss pelvic instability
Blood at meatus possible

DIAGNOSTICS
CBC, Chemistries
Coags, serial Hcts
U/A, UPT

Abd/Pelvic CT:
Better than cystography
Urethral intactness established
Then contrast goes in via urethra

Pelvic U/S if indicated

INTERVENTIONS
ABCs, supplemental O2 prn
IV access: 2 large-bore
Fluid resuscitation PRN
Blood replacement PRN
Cardiac/pulse ox monitors
NGT
F/C: if no blood at meatus and if prostate intact

DISPOSITION
Surgery, Admit/obs or Transfer

BARTHOLIN'S CYST
Labia minora 4 & 8 o’clock
Normally nonpalpable
Secretes mucus keeping vestibular surface moist
Cysts affect women of reproductive age
Ductal obstructed d/t inflammation or trauma
Acute, rapidly progressive pain

ASSESSMENT
Acute painful, unilateral labial swelling
Pain with walking/sitting
EXAM
Exquisitely tender, warm, fluctuant mass
Possible cellulitis also

INTERVENTIONS
Cultures rarely helpful in these
Usually required I/D, irrigation, packing
"Word" catheter is an option

DISPOSITION
Analgesics
Sitz baths
PCP or GYN f/u
Typically antibiotics not needed
-but are usually given)

SEXUAL ASSAULT
Physical force does not have to be present
Injuries do not have to be present
May present with a myriad of c/o's
Ideally a trained SANE available
Law enforcement:
Do not have to be in room during exam
Discouraged

Victim advocate: Ok

Even if pt does not wish to speak
with law enforcement, reporting the
event is still mandatory

ASSESSMENT
Date, time, location of assault
Use of penetrating objects or weapons
Any consensual intercourse in preceding
5 days (any route)
Any post assault hygiene
Any voluntary substance or ETOH use
Any injuries left on perpetrator
Contraceptive/lubricant use by perpetrator

PMHX
Prior OB/GYN/GU illness/injury
LMP
Meds

EXAM
LOC/Mental State
Affect (alert to comatose)
Possible crying, calm, angry or anxious

Head to toe:
Lacerations abrasions, "hickeys"
Bite marks, fingernail marks
Genital/perineal trauma
DIAGNOSTICS
CBC, U/A, Urine pregnancy, RPR
Toxicology panel
Possible DNA swabs (oral, vaginal, rectal)
STD swabs

U/V light exam:
Seminal fluid sometimes shows; swab if seen

INTERVENTIONS
Provide privacy immediately
DO NOT put pt back in Lobby
Evidence collection: pt's clothes in paper bags
Chain of custody procedures
Labeling of evidence
Pt removes clothing standing on paper sheet
Each article in separate paper bag
Photographs per policy
Fingernail scrapings
  (scraper goes w/specimens)
Pubic hair combings
  (if shaved, use evidence tape)
Pelvic exam and evidence collection
Anal/rectal exam and evidence collection

Male: swab glans, shaft and base or penis
Offer pt shower after exam

MEDICATIONS
STD prophylaxis per CDC guidelines

Pregnancy prevention medication:
“Plan B”
Must have NEG Hcg
Must be given w/in 72 hours post-assault

DISPOSITION/EDUCATION
Support pt recovery
Follow up with PCP 1-2 weeks
Victim advocate support ongoing

TRAUMA IN PREGNANCY
22% of maternal deaths d/t major trauma
Pregnancy = higher risk aortic dissection
Most common injury
  = blunt force trauma r/t MVC
  -60% of all major injuries

17% suffer trauma from another individual
60% have ongoing domestic violence issues

After 24 weeks gestation, fetus much more susceptible to injury
Head injury & shock are most frequent causes of maternal death
THE MOST COMMON CAUSE of fetal death is maternal death. Second most common = maternal shock. Fetal demise occurs approx 80% of the time.

Stabilization of mother is first priority. Best chance for fetal survival is maternal survival.

Changes in Pregnancy
A) Cardiovascular
Increased blood volume and cardiac output
Uterine shunting of blood
“Anemia” of pregnancy

B) Respiratory
Increased minute volume (=dec. PCO2)
Increased tidal volume (=dec. HCO3)
This causes partially compensated resp. alkalosis

Elevation of diaphragm:
Increases risk for hypoxia
Decreased pulmonary reserves

C) Gastrointestinal
Decreased motility (risk for aspiration)
Distended abdominal wall can mask intra-abdominal injury

DIAGNOSTICS
Indicated imaging tests should not be deferred d/t pregnancy.
If possible avoid abdominal CT in early pregnancy. FAST, Abd, Pelvic and Trans-vaginal U/S

INTERVENTIONS
ABCs, supplemental O2 PRN
IV access; 2 large-bore as indicated

Position:
Left lateral decubitus
Displaces gravid uterus off vena cava
Up to 30% of venous return can be compromised
**GENERAL MEDICAL TOPICS**

**ANEMIA**
Low Hemoglobin and Hematocrit
Caused by:
- RBC loss/destruction -OR-
- RBC decreased formation/production

Decreased production:
- Iron deficiency (most common),
- Folic acid deficiency

Loss/Destruction:
- Acute blood loss (most common),
- Hemolytic anemia

Hgb resp for oxygenation,
Hgb is on the RBC
Anemia means less O2 carrying capacity

**IDIOPATHIC THROMBOCYTOPENIC PURPURA (ITP)**
Decreased production of platelets -OR-
Increased destruction or sequestration

ITP most common acutely in children following a viral illness

Chronic ITP: women 20-40 y.o.
At risk for bleeding

**ASSESSMENT**
- Ecchymosis
- Petechiae
- Bleeding gums
- Hematuria
- Signs of GI bleeding

Labs: CBC, Coags
Avoid punctures, handle pt gently
Ice to injured areas
Possible admission if severe for possible:
- Plasmapheresis
- Platelet transfusion
- Spleenectomy

Ig treatment: increases platelets rapidly
Steroids: alternative

**DISSEMINATED INTRAVASCULAR COAGULATION (DIC)**
Inappropriate or accelerated activation of the coagulation cascade
Results in thrombosis and subsequent hemorrhage
DIC a complication of serious conditions
Primarily trauma and/or sepsis

S/Sx: bleeding, rash, bruising, joint pain

Risk factors:
Liver disease
Massive blood transfusions
Pregnancy
Snake bite
Anticoagulation meds

DIAGNOSTICS
Thrombocytopenia
Prolonged PT/PTT, inc INR
H/H decreased
BUN/Cr elevated
D-dimer inc

TREATMENTS
Warmed PRBCs
Platelet concentrates
FFP/Cryoprecipitate

MEDICATION
Heparin:
To inhibit thrombin and Xa factor
Slows the excessive clotting cascade

Activated Protein C:
Inhibits factors Va and VIIIa
Inhibits plasminogen

HEMOHILIA
Inherited, sex linked disorder
Most freq in boys/men
Women are carriers

Four types: each affects a different factor
Hemophilia A: (Classic) Factor VIII
Hemophilia B: (Christmas dz) Factor IX
Hemophilia C: (Rosenthal’s) Factor XI
Von Willebrand’s: Factor VIII

Usually present with a bleeding complication:
Bleeding after trauma/dental
Spontaneous hemorrhage
Hemarthrosis
Melena

DIAGNOSTICS
CBC, PT/PTT
X-rays if joint/trauma affected, CT if head injury
TREATMENT
Factor replacement is the key
Possible platelet infusions

Hemarthrosis:
Ice, immobilize, elevate
Mild compressive dressing

Analgesics as indicated (no ASA, NSAIDs)
Minimize punctures

May be Rx’d F VIII for home use to terminate an early bleeding episode

SICKLE CELL CRISIS
Inherited genetic disorder
Abnormal Hgb makes up 35% of total Hgb
Crisis begins with microcirculation obstruction (vaso-occlusive crisis)
Stressors can trigger the sickling

COMMON TRIGGERS/CAUSES:
Dehydration
Stress
Infection
Heat/cold extremes

Vascularity obstruction:
Leads to ischemia
Then infarction

Oxygenation resolves the sickling process in about 80% of cells

Impaired growth/development
Char. by periods of exacerbation/remission
May have signs of heart failure or other organ damage

**RED FLAGS***
Acute Chest Syndrome/Acute Abdomen

DIAGNOSTICS
Dec. H/H (chronic - often very low),
Increased reticulocytes (immature RBCs)

TREATMENT
O2 only useful if hypoxic
Possible admit for plasmapheresis or transfusions
IV hydration

MEDICATIONS
Oral hydroxyurea:
Impedes sickling
Stimulates production of Fetal Hgb F
- high affinity for O2
Parenteral analgesics, anti-emetics
### Childhood Illnesses

#### Measles
- Rubeola virus: late winter, early spring
- Direct contact with droplets or airborne

**Contagious:**
- 3-5 days before rash
- Until 4 days after rash appears

**Rash:**
- Maculopapular
- Starts face, then spreads trunk
- Extremities last

C/O fever, rash, cough
**Complications:**
- Otitis media
- Diarrhea
- Pneumonia
- Bronchiolitis
- Encephalitis

HS or College age should be re-vaccinated;
Unless had disease or two doses in childhood

#### Diagnostics
- CBC, LP, CXR, Septic w/u as indicated

#### Intervention
- Resp isolation
- APAP
- No outside family contacts until no longer contagious
- Rest, fluids

#### Mumps
- Benign, viral infection
- Paramyxovirus
- Swelling and tenderness of salivary and/or parotid glands

**Transmission by direct contact into nose or mouth**
- Peaks January-May

**Complications:**
- Viral meningitis
- Arthritis
- Pancreatitis

#### Assessment
- Fever, malaise, HA, earache
- Parotid gland tenderness
- Possible orchitis in males
- Abdominal pain: possible pancreatitis
- Trismus and/or painful chewing
INTERVENTIONS
Airborne/resp isolation
APAP/IBU
No work/school until swelling gone

PERTUSSIS
Whooping cough
Highly contagious, bacterial
*Bordetella pertussis*

Airborne droplet transmission
Vaccine immunity less than 12 years
New guidelines for DTaP!!!
Contagious for 3 weeks
Presents similar to common cold,
but then progressively worsens

CHICKEN POX
Highly contagious
Varicella Zoster Virus
Direct contact and airborne transmission

Contagious:
1-2 d before rash
Ends when lesions crusted over
Usually 4-5 days

Age of onset in peds <10 y.o.
Complications:
Bacterial infection
Pneumonia
Renal failure
Reye’s Syndrome

Prodrome 48 hours before rash:
Fever, cough, malaise, headache

Rash:
Starts on trunk
Teardrop vesicles, open, dry then crust
Palms and soles spared

TREATMENTS
APAP, antihistamines, consider antivirals
Socks/mittens at night to prevent scratching
Trim nails short

FEBRILE SEIZURES
Seizure with fever in infancy or childhood and
no evidence of other underlying cause

Overall age of onset: 6mo-6y.o.

Simple:
Single episode in 24 hr, less than 15 min
85% of febrile seizures are these
Complex:
Multiple episodes in 24 hours
Associated focal findings and lasting >15 min

**ASSESSMENT**
Temp usually 102.2 (39) or greater
Rate of change maybe more important
Tonic-clonic activity
Usually occurs within hours of fever onset

Causes:
Viral/bacterial illnesses
Immunizations (MMR, dPT)

Having a sibling with Hx of febrile sz’s:
  increases risk 2-3 times
Workup: must find cause
May include full septic w/u

**INTERVENTIONS**
Tepid sponge bath to cool
Supplemental O2 prn
IV access/fluids

Temp control:
APAP 10-15 mg/kg q6h
IBU 10 mg/kg q6h

**EDUCATION**
Febrile Seizures (FS) don’t cause developmental
delays/retardation

FS don’t cause death

Risk of epilepsy for FS only slightly more than
general population

Risks for subsequent FS:
Onset if lower temp
Onset less than 12 months
Family Hx of FS
Multiple initial FS

33% develop recurrent FS
95% of recurrences happen within 1 year

**FLUIDS & ELECTROLYTES**

**HYponatremia**
D/T actual Na deficits or dilutional causes

Dilutional:
Excess H2O intake
Freshwater drowning
Inappropriate antidiuretic hormone secretion
Psychogenic polydipsia
Deficits:
- Diaphoresis
- Diuretics
- Wound drainage
- Dec. aldosterone
- Hyperlipidemia
- Renal disease.

Usually asymptomatic until Na = 120-125 mEq/L
S/Sx:
- Confusion
- Seizures
- Dizziness
- Tachycardia
- Flat neck veins

**HYPERNATREMIA**
Less common than Hypo Na
Indirect causes: NPO status or increased fluid loss
Direct: excess Na intake
- Too much Na/HCO3
- Too much Na
- Renal failure, steroids, Cushing’s syndrome

Na > 145 mEq/L
Mortality 40-60%

Osmolarity rises
Causes cellular dehydration and shrinkage.
Brain cells shrink causing CNS Symptoms
ICH may occur due to shrinking cells and mechanical stresses/tears

**TREATMENT**
- Hypo-osmolar solution
- Decrease total body Na
- Goal is to decrease 2 mEq/L/hr

**HYPERKALEMIA**
Excess serum K+ > 5.5 mEq/L
Cardiac effects are the highest concern

HyperK+ causes hyper-polarization of cardiac cells and inability to re-polarize

Worse form = ventricular asystole

**INTERVENTION**
- D50: fuel for K+ Pump
- Insulin: allows D50 to be used
- Calcium: only PRN myocardial irritability

**Boswell Emergency Medical Education Technology ~ 85 ~**
HYPOKALEMIA
Serum K+ < 3.5 mEq/L
Causes:
Decreased K+ intake
Excessive K+ losses (V/D)

ASSESSMENT
weakness, cramps
SOB, hypotension
Decreased/shallow respirations
Hyporeflexia to flaccid paralysis

Cardiac:
Sagging ST
T wave depression
PVCs, PACs, and 2nd/3rd blocks possible

INTERVENTIONS
K+ replacement
Cardiac monitoring
Max physiological dose = 40 mEq/Hr
Central vs peripheral IV sites

SIADH
(SYNDROME OF INAPPROPRIATE ANTI-DIURETIC HORMONE SECRETION)

DEFINED AS:
Hyponatremia -PLUS-
eliminated urine osmolarity
-excessive urine Na
-decreased serum osmolality

PEARL:
The hyponatremia is a result of excess water and not a Na deficiency!!!
The pituitary releases ADH
ADH affects the kidney at vasopressin receptors to retain/re-absorb free water
Too much vasopressin causes too much fluid to be retained, and makes concentrated urine
Na+ continues to excrete
Thus the circulation has an excess of H2O

Idiopathic causes are the most common
Presenting sx are r/t the Hyponatremia
Treatment: correct the Na deficit first
May add Lasix
Search for underlying cause

ENDOCRINE DISORDERS
ADDISON’S DISEASE
Adreno-cortical insufficiency
Destruction or dysfunction of the adrenal cortex
Untreated leads to Addisonian Crisis
May be fatal
Present with sx of corticosteroid deficiencies:
Skin changes
Vitiligo
Hyperpigmentation
Weakness
Fatigue
Poor appetite
Possible n/v
Dizziness, syncope

Crisis state may see:
Hypotension
Dehydration
Orthostasis
Hypoglycemia
Circulatory collapse

About 80% cases are idiopathic or autoimmune
Increasing numbers d/t TB/HIV

DIAGNOSTICS
“Rapid Adrenocorticotrophic Hormone test”
ACTH is infused
Timed labs at 30/60 minutes
Looking at cortisol and aldosterone response

In crisis situation, don’t wait to test
Treat ASAP
Hydrocortisone 100mg/100cc over 8 hours

HHNK (HYPEROSMOLAR HYPERGLYCEMIC NONKETOTIC COMA)
More common in older people with NIDDM
Pt unable to maintain adequate hydration

Ketoacidosis doesn’t usually develop because
there is still enough endogenous insulin to
allay ketogenesis

Often precipitated by infection, stroke or sepsis
May be first presentation for new onset Type-2 DM
Presents in dehydration state

DIAGNOSTICS
Ph: “usually” normal
Glucose: “usually” >600

Hyper/hyponatremia
Ketones: slightly elevated
U/A: elev glucose, small ketones,
elevated specific gravity

IVF rehydration paramount
1-2 liters possibly - using NS
Start glucose containing fluids at glucose = 300
K+ replacement as indicated
Regular insulin as indicated
**DKA**
Result of insulin deficiency  
Mostly occurs in IDDM pt's  
Lack of insulin = dec. cellular glucose uptake  
Then release of free fatty acids and increased hepatic gluconeogenesis  
Hyperglycemia causes osmotic diuresis and dehydration and electrolyte depletion  
Free fatty acids converted to ketone bodies  
Onset varies; 24 hours to 2 weeks  
Look for preceding bacterial/viral illness  
Nausea/vomiting, pain, fever possible  
Lethargy, weakness, fatigue  
Signs of profound dehydration possible  

**DIAGNOSTICS**
K+ elevated  
Ph: acidotic  
Na: decreased  
Acetone: elevated  

Bolus IVF NS 1-2 liters  
IV regular insulin: keep giving until ketosis resolves  
First dose 0.15 u/kg IV  
Maintenance 0.1 u/kg/hr drip  
Flush tubing and discard  
May give bicarb if ph <7  
Start K+ replacement when drops to 5.5 mEq/L  

**HYPOGLYCEMIA**
The most common endocrine emergency  
Defined as:  
Glucose less than 50 in males -AND-  
Less than 45 in females  
Too rapid a glucose reduction doesn’t allow for ketogenesis to provide an alternate source  
Below 35, the brain cannot extract O2 and hypoxia ensues  
Can be due to multiple reasons  
All pt’s with altered LOC should be considered for hypoglycemia  
Glucose replacement is only temporary  
Must search for underlying cause  
Must be monitored in ER to see if the hypoglycemia recurs  
If so, admission is warranted
### MEDICATIONS
- Thiamine IM/IV if malnourished
- D50
- Glucagon:
  - Only effective if glucagon stores are present
  - Not present in alcoholics

### RENAL FAILURE
Acute renal failure (ARF) develops over hours to days

Accumulation of nitrogenous wastes

Disrupts:
- ECF volume,
- Electrolyte balance, and
- Acid base status

Not a disease by itself, but rather a potential complication of other processes

### CATEGORIES
- **Pre-renal:**
  - Decreased blood flow to kidneys
  - Leads to ischemia
- **Intrarenal:**
  - Actual tissue damage to the kidney
- **Postrenal:**
  - Obstruction of the urinary collecting system

### CAUSES
- **Prerenal:** shock, heart failure, PE, sepsis
- **Intrarenal:** Nephritis, acute GMN, ATN, RAS
- **Postrenal:** Ureteral/bladder cancer, renal calculi, prostatic CA, cervical CA, urethral stricture

Goal of tx to address the underlying cause

### F/E disturbances seen in ARF:
- HyperK+
- HypoNa
- Hypocalcemia
- Hyperphosphatemia
- Volume overload
- HTN
- Pulmonary edema
### ASSESSMENT
Look for signs of fluid overload or F/E imbalances
- U/A: presence of myoglobin
- RBC casts = glomerular disease
- Uric Acid Crystals suggest Acute Tubular Necrosis (ATN)
- Calcium Oxalate crystals possible
- BUN/Cr: greater than 20:1 ratio
- CPK: rhabdomyolysis

Judicious IVF d/t risk of overload
Possible central pressure monitoring

### MEDICATIONS
- Anti-hypertensives
- Vasopressors
- Inotropes
- Potassium binding agents
- Diuretics
- Nitrates
- Calcium channel Blockers

### GRAVES DISEASE
Hyperthyroidism
A reaction to excess thyroid hormone
- Thyroid stimulating Igs bind to TSH receptors
- Results in excess secretion of T3 and T4
- Goiter is common
- Women 3:1 over men

### ASSESSMENT
- Nervousness, palpitations, sweating
- Heat intolerance, tremor
- Dyspnea, fatigue,
- Exophthalmous

### DIAGNOSTICS
- TSH below normal
- T3/T4 elevated

Thyrotoxic Crisis Treatment:
- Propylthiouracil (PTU)
- Solution of Potassium iodide
- Beta Blocker
- Anti-thyroids
- Thyroidectomy/Radiation

### ALLERGIC REACTION/ANAPHYLAXIS
Usually present with urticaria and pruritis
Often can’t identify
Can occur from hours to 3-4 days later
The faster the reaction develops,
the more likely to be severe

- Anaphylaxis - Life threatening
- Angioedema
Hypotension
Bronchospasm
Rapid onset

Release of mediators:
Histamine, leukotriene, prostaglandin

Causing:
Inc mucous secretion
Incr. bronchial muscle tone
Airway edema

Most common anaphylaxis causes:
IV antibiotics (penicillins)
IV contrast
Hymenoptera stings
Foods

ASSESSMENT
Description of precipitating event if known
SOB, vomiting, abd pain, CP, pruritis
What pre-hospital care given?

LOC: anxiety, restlessness
Hypotension, cyanosis
Tachypnea, tachycardic, stridor
Angioedema: tongue, lips, facial
BS: wheezing, hoarseness

PMHx
Prior reactions, Tx’s
Shellfish/iodine allergy
Iodine allergy - doesn’t exist
Use of LMW contrast - reactions approx 0.5%

INTERVENTION
O2, Monitor, IV access as indicated

MEDICATIONS
Epinephrine: SQ or IV
Inhaled B-2: Albuterol
Histamine-2 (H2) blocker (Zantac)
Antihistamine (Benadryl)
Steroids (Solumedrol)

DISCHARGE
Sx improvement
No respiratory component
Education: use of benadryl
Use of Epi-Pen
### Infectious Diseases

**Hepatitis A**
- Fecal-oral route
- Infectious period:
  - 2 weeks before and 1-2 weeks after jaundice
- Total duration: 4-8 weeks
- People at risk: Travelers/immigrants
- IG effective if given within 1-2 weeks of exposure
- Vaccination pre-exposure; 2 doses

**Hepatitis B**
- Parenteral transmission, sexual contact
- Duration approx 8 weeks
- HBsAg (surface antigen) appears early
- HB core antibodies, if persistent, indicate chronic infection
- Acute infection treatment: HepB IG & vaccination

**Hepatitis C**
- Parenteral transmission mostly (IVDU)
- Detected by anti-hepatitis C virus AB
- About 50% become chronic, and no immunity
- Duration (acute illness): 8 weeks

**Hepatitis All Forms**
- All present in similar conditions
- Jaundice common indicator of liver dysfunction
- Low grade fever, myalgias prominent also
- HBV: sometimes a rash

**Laboratory Tests (LABS)**
- LFT (AST, ALT) elevated
- Bilirubin: elevated
- Alk Phos: elevated
- Coags: prolonged

**HIV/AIDS**
- HIV: a retrovirus
- AIDS: end stage of HIV disease state
- Opportunistic infections:
  - Pneumocystis Pneumonia (PCP),
  - Cytomegalovirus (CMV),
  - Kaposi’s sarcoma
- Acute HIV infection characterized by mononucleosis type presentation
- Acute phase resolves and pt returns to baseline
- Current average time between HIV and AIDS approx 10 years
- Earlier treatment = better long term survival

**Meningitis**
- Bacterial, viral or fungal infection of meninges
- Viral: usually mild and short lived
- Bacterial: severe and life threatening
**Bacterial Pathogens:**
* S. Pneumoniae,
* H. flu,
* Neisseria Meningitides,
* Listeria

Fungal
Usually just in immunocompromised pt’s

Pathogens enter after colonizing the nasopharyngeal airways

Infants and elderly mask typical symptoms
Some can be fatal within hours

**ASSESSMENT**
- Headache
- Photophobia
- Fever
- Malaise, lethargy
- Decreased LOC possible late
- Petechial rash possible

**Kernig’s Sign**
What do you flex?__________________________

_________________________________________

What do you look for?______________________

_________________________________________

**Brudzinski’s Sign**
What do you flex?__________________________

_________________________________________

What do you look for?______________________

_________________________________________

**Spinal tap results…**
A) Bacterial infection:
   Glucose - decreased

B) Viral infection:
   Glucose - normal
   Protein slightly inc,
**MEDICATIONS**
- IV Rocephin
- Anti-epileptics if needed
- Tylenol (APAP)
- Anti-Emetics

**MONONUCLEOSIS**
Acute Viral Illness
Duration 3-4 weeks
Pathogen: Epstein-Barr Virus (EBV)
Member of the herpes virus family
Transmitted by oropharyngeal route
Age: 15-24 y.o
Uncommon in adults d/t probable prior immunity

Complications (mostly in younger pedds):
- Glomerular nephritis (GMN),
- Pericarditis, hepatitis, Group-B Strep (GBS),
- Meningitis, pneumonia

Rare:
- Splenic rupture,
- Thrombocytopenia,
- Hemolytic anemia

Prodrome 3-5 days of malaise
Then followed by possible N/V, F/C
Sore throat nearly always present
Fever range varies

**ASSESSMENT**
Red throat w/exudate,
Palatal petechiae
Tender posterior cervical/occipital nodes
Abd tenderness d/t poss. splenomegaly

**DIAGNOSTICS**
Monospot, CBC, Strep screen

**MEDICATIONS**
NSAIDs, APAP, Steroids

**EDUCATION**
No isolation necessary
Contact sports precautions.

**TINEA**
Fungal dermatophyte infection
Transmitted by direct contact

Characteristic rash:
- Sharply defined annular pattern
- Central clearing
- Dry, scaly active at margin
- Occasionally vesicles at margin
### MEDICATIONS
- Topical antifungals for most
  - Continue 2 weeks, or until rash gone +1 week
- Antihistamines for pruritis

### SCABIES
- Contagious infestation by the mite *sarcoptes scabiei*
- Transmitted by personal skin-to-skin contact

### ASSESSMENT
- Generalized itching, intense at night
- Burrows in finger webs
- Excoriated and non-excoriated papules,
  - Predominately on hands and flexor surfaces
- Vesicles and papules

### TREATMENT
- **Elimite:**
  - A neurotoxin to the mite
  - Apply neck to soles
  - Leave on 8-14 hours then wash off
  - Also Lindane, Kwell
  - Repeat either Tx 1 week later

- **Antihistamines:**
  - For pruritis

- Treat all intimate contacts, close household,
  - and family members

- Wash all clothing, linens and towels in a normal
  - wash cycle

### GONORRHEA
- Yellow, purulent discharge
- No lesions
- Males c/o: dysuria
- Females c/o: asymptomatic possible
- Treatment: Rocephin
  - (Cipro falling out of favor)
- Home tx: none

### CHLAMYDIA
- IF IT’S NOT A UTI - IT’S CHLAMYDIA UNTIL PROVEN OTHERWISE!
- Mucopurulent D/C
- No lesions
- Males/Females c/o: dysuria
- Treatment:
  - Zithromax 1gm x1 -OR-
  - Rx Doxycycline 10 d
**SYPHILIS**  
No discharge  
Chancre sore on genitals  
Poss. fever, lymph node enlargements  

Treatment:  
Primary: Bicillin x1  
Secondary: Longterm Biicillin  
Home Tx: Doxycycline

**TUBERCULOSIS**  
*Mycobacterium tuberculosis*  
An acid-fast bacillus (AFB)  
Commonly lungs - but can infect anywhere  
Droplet transmission  

Latent TB: asymptomatic, but PPD will be ‘+’  
Active TB: pt is symptomatic and infectious  

**ASSESSMENT**  
Fever, chills, night sweats, fatigue, hemoptysis  

**RISK FACTORS**  
Nursing home, jail, homeless, alcoholic or drug abuser  

**DIAGNOSTICS**  
CXR - infiltrates, esp. posterior upper lobe  
Sputum for AFB smear  
Routine labs as indicated  

**INTERVENTION**  
O2, Monitor, IV as appropriate  
Isolation for first 2 weeks of Tx  

**MEDICATIONS**  
INH (isoniazid)  
Rifampin  
**DISCOLORS BODY FLUIDS-STAINS CONTACTS**  
Ethambutol  
Pyrazinamide (PZA)  
All pt’s with active Dx should have DOT (Drug-observed-therapy), even HCW
<table>
<thead>
<tr>
<th>REVIEW/TEST YOURSELF</th>
<th>CEN REVIEW NOTES</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are the mainstays of Tx for a pt in SSC?</td>
<td></td>
</tr>
<tr>
<td>What is the most common type of anemia?</td>
<td></td>
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<tr>
<td>How long is a child with chicken pox contagious?</td>
<td></td>
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<tr>
<td>What is a risk factor for future febrile seizures?</td>
<td></td>
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<tr>
<td>Reye’s syndrome is associated with what?</td>
<td></td>
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<tr>
<td>What ECG finding suggests hyperK+</td>
<td></td>
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<tr>
<td>What are the three types of dehydration?</td>
<td></td>
</tr>
<tr>
<td>What is the max mEq/hour to replace K+ by?</td>
<td></td>
</tr>
<tr>
<td>A pruritic rash with red, scaly borders, expanding and centrally clearing suggests?</td>
<td></td>
</tr>
<tr>
<td>What are the “classic findings” of SIADH?</td>
<td></td>
</tr>
<tr>
<td>Spinal fluid for meningitis - which has a normal glucose level (bacterial or viral)?</td>
<td></td>
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</tbody>
</table>
The rash of measles progresses from what to what to what?
CARDIOVASCULAR

ATHEROSCLEROSIS
Injury to the endothelium
Symptoms present when 75% or more of blood supply affected/occluded

Risk factors:
Age
Family
Gender
Tobacco use
HTN
Physical inactivity
Diabetes mellitus
Obesity

CORONARY ARTERY DISEASE

Ischemia
Reversible cellular damage d/t imbalance between oxygen supply and demand

Injury
EKG changes indicate more extensive damage
Still a degree reversible

Infarct
Prolonged ischemia
Irreversible damage

ACUTE CORONARY SYNDROME/UNSTABLE ANGINA

ASSESSMENT
Chest pain at rest -OR-
CP >20 minutes -OR-
Previous diagnosis of angina that has become more severe, prolonged or frequent

EKG Changes
Transient ST segment or T-wave changes may occur
Troponin: Normal early

NSTEMI
ASSESSMENT
CP, N/V, dyspnea, unexplained weakness
fatigue, dizziness, syncope

EKG changes
ST depression
T-wave abnormalities

Troponin: Elevated

STEMI
ASSESSMENT
CP, N/V, dyspnea, unexplained weakness,
fatigue, dizzy, syncope
EKG changes
ST elevation
New L BBB

Troponin: Elevated

**EKG GUIDELINES**
Perform within 10 min of presentation
AND MD interpretation

Infarction Patterns:
Inferior: II, III, AVF
Septal: V1, V2
Anterior: V1, V2, V3, or V4
Lateral: I, aVL, V5, V6

Posterior MI:
Reciprocal changes: V1, V2
Tall R wave
ST depression
Upright T wave

***Right Side EKG

**DIAGNOSTICS**
CBC, Chemistries
Mag, BUN, Creatinine
Coagulation profile
Lipid panel

**Cardiac Biomarkers**
Troponin proteins
Elevates 2–12 hrs after MI onset
Peaks at 24 hr
Returns to normal 5–12 days
May be elevated in pts with renal failure/CHF

Other Cardiac Biomarkers:
Creatine Kinase Isoforms (CK-MB)
Creatine Kinase (CK)
Myoglobin
Lactate dehydrogenase (LDH)

**INTERVENTIONS**
Cath lab preferred
Door to Balloon = 90 Minutes (D2B)
If cannot achieve D2B time (IE: need to transfer pt.) - then consider fibrinolytics

Other testing considerations for Non-acute MI
Cardiac catheterization
Stress test
Echocardiogram
PT EDUCATION TOPICS
- Nitroglycerine use
- Activity
- Smoking cessation

**MEDICATIONS USED**
- Oxygen
- Aspirin preferred, Plavix okay if ASA allergic
- Metoprolol
- Nitrates
- Morphine
- Glycoprotein IIb/IIIa inhibitors
  - Integrillin, Reopro
- Heparin or LMWH
- Antidyshyrrhythmics
- Vasopressors
- Vasodilators
- Inotropes

**FIBRINOLYTICS**
- If cannot achieve D2B of 90 min
- Goal: prompt restoration of coronary patency

**Exclusion criteria/Contraindications:**
- >12 hrs onset
- Cardiogenic shock
- Hx of CVA in past year
- Hx of Intra-cerebral hemorrhage (ICH)
- SBP>180; DBP>110

Which Tpa product is ONE TIME IV PUSH (no drip)?

---

Watch for signs of bleeding, limit veinpunctures and invasive procedures

Watch for dysrhythmias d/t ventricular remodeling/repurffusion
**RIGHT VENTRICULAR INFARCTION**

Occlusion of what vessel? ____________
Associated with Inferior wall MI
Right sided EKG needed

**INFERIOR WALL MI**

TREATMENT
Fluids
Inotropes to inc. ventricular contractility;
Vasodilators to decrease afterload

**HEART FAILURE**

Common causes:
Acute MI
Coronary artery Disease
Chronic HTN (overall most common cause)
Diabetes
Valvular disorders

Left sided HF usually d/t hypertension
In acute exacerbation, presents as
pulmonary edema, and dependent edema
(lower extremities)

Right sided heart failure usually starts secondary
to a pulmonary problem (COPD and pulmonary
hypertension)
R ventricular failure causes profound, systemic
venous congestion.
Pt presents with HF symptoms and/or generalized
edema/anasarca

HF may progress from either Right side to
Left Side failure or vice-versa.

**ASSESSMENT**

Heart Sounds
S1 – Loudest at apex
S2 – Heard over aortic area
S3 – Heard over pulmonic area
S4 – Heard over tricuspid area

Murmur:
Turbulent blood flow
Stenotic (restricted) or
Incompetent (regurgitant) valves
Valve dysfunction causes the associated heart
chamber to work harder, thereby hypertrophy
ensues and progressive dysfunction

**DIAGNOSTICS**

CBC, Chemistries
Hyponatremia - often seen severe heart failure
Hypokalemia - frequently associated w/diuretics
BUN/Creatinine
BNP (“B-type” or “Brain” Naturietic Peptide)
ABG’s

~ 104 ~ CEN Review Course
Chest radiography:
- Cardiomegaly
- Pulmonary edema (Left HF)
- Pleural effusion(s) (Left HF)
- Pulmonary vascular congestion (Right HF)

ELECTROCARDIOGRAM (ECG/EKG)
Acute HF causes may show as:
- MI, ischemia, conduction defects
Chronic failure causes may show as:
- Ventricular hypertrophy, atrial enlargement

ECHOCARDIOGRAPHY
Used to distinguish systolic from diastolic dysfunction and left versus right-sided
Also assesses for valvular dysfunctions

INTERVENTIONS
- O2, Monitor, IV Access
- Bipap/Cpap
- Positioning

PHARMACOLOGY
Diuretics: decrease preload by reducing circulating fluid volume
Morphine: reduces myocardial workload by decreasing preload
Reduces sympathetic (adrenergic) stimulation
Vasodilators: Decrease afterload
Venodilators: Decrease preload
Arteriolar dilators: Decrease afterload

Other Medications
- Combined arteriolar/veno-dilators
- ACE Inhibitors
- Inotropes
- Bronchodilators

Goal of Tx: Decrease workload of heart and maximize pumping efficiency
**HYPERTENSIVE EMERGENCIES**

**Defining characteristics:**
- Rapidity of the rise in BP
- Signs of end-organ dysfunction
  - Eyes, brain, kidney, heart
- Systolic BP > 140 mm Hg
- Diastolic BP > 90 mm Hg

**Acute disorders that can precipitate a HTN crisis:**
- Pheochromocytoma
- Intra-cranial hemorrhage (ICH/Stroke)
- Acute aortic dissection
- Pregnancy-induced HTN
- Autonomic hyper-reflexia
- Certain drug use (sympathomimetics)

**ASSESSMENT**
- Headache, CP, dizziness, visual complaints
- Symptoms of heart failure possible
- May be asymptomatic
- LOC, VS, hemiparesis
- Retinopathy, visual changes
- Auscultation for murmurs
- Lungs/BS – crackles, rales
- Abdomen - bruits, pulsations

**DIAGNOSTICS**
- CBC, Chemistries, UA,
- CXR
- EKG
- CT Head/CT Thorax/CT Abdomen

**INTERVENTIONS/PHARMACOLOGY**

**Nitroprusside (Nipride):**
- Arterial & venous vasodilation
- Decreases preload & afterload

**Nitroglycerin:**
- Preferred in patients with CAD or cardiac ischemia

**Labetalol (Trandate/Normodyne):**
- Slower onset
- Avoid for HF patients, due to HR reduction

**Hydralazine (Apresoline):**
- Arterial vasodilator
- Mainly used in pregnancy induced HTN or pre-eclampsia/eclampsia

**Regitine:**
- Specifically indicated for pt in catecholamine crisis such as Pheochromocytoma (a tumor on the adrenals)
**Inderal/Brevibloc:**
Short-acting Beta blockers

**Diuretics**
Lasix, Bumex

**Goal:**
Decrease BP 20-30% over 2-3 hrs in patients with chronic HTN.
Acute HTN can be more aggressively lowered.

**Prevention of end organ damage**

**ENDOCARDITIS**
Affects endocardium and heart valves

**Causes:**
Native valve (vegetative growths)
Prosthetic valve (thrombosis, emboli)
Infective endocarditis
Affects endocardium and heart valves

**Risk factors:**
Mitral valve prolapse
Congenital heart defects
Rheumatic heart disease
Prosthetic valve
Body piercing, Tattoos, IV drug use
Dental procedures without antibiotic prophylaxis
Long term venous access devices

**Prevention of end organ damage**

**ASSESSMENT**
(Symptoms often vague and non-specific)

**Complications:**
May progress to heart failure
Conduction disturbances

**Fever**

**Anorexia**

**Night sweats**

**Fatigue/malaise pain**

**Headaches**

**Confusion**

**Hemoptysis**

**Hemorrhoids**

**Diabetes**

**Fever**

**Anorexia**

**Night sweats**

**Fatigue/malaise pain**

**Headaches**

**Confusion**

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**Diabetes**

**Fever**

**Anorexia**

**Night sweats**

**Fatigue/malaise pain**

**Headaches**

**Confusion**

**Hemoptysis**

**Hemorrhoids**

**Diabetes**
ASSESSMENT
Auscultation: Heart murmur, crackles, rales
Splenomegaly possible due to emboli to spleen

DIAGNOSTICS
CBC, Chemistries
Blood cultures (may require 3-6 cultures)
Sed Rate (ESR) elevates in inflammatory endocarditis
Echocardiogram
EKG

INTERVENTIONS/MEDICATIONS
Antibiotics
Anti-pyretics for fever control
NSAIDS/Narcotic Analgesics

PERICARDITIS
Idiopathic inflammation of the pericardium
Inflammation causes a build up of fluid in pericardial sac
Worst complication: cardiac tamponade

ASSESSMENT
Pleuritic CP radiates to left shoulder
Relieved by leaning forward
Friction rub
EKG changes elevation of ST segments in all leads
CXR: Pericardial effusion

INTERVENTIONS
NSAIDS
Usually will resolve spontaneously
Pericardiocentesis if indicated
Assess for tamponade

CARDIAC TAMPOANE
Pericardial sac engagement causing compression on the myocardium.
Increasing compression leads to decreased filling followed by decreased cardiac output.

ASSESSMENT
Impending doom
Localized chest pain
Dyspnea
Consider mechanism of injury if trauma pt.
(steering wheel, dashboard etc)
Falling arterial pressure

Beck’s Triad:
JVD, muffled heart tones, decreased BP
Pulsus paradoxus:
Systolic BP decreases > 10 mm/Hg
during inspiration
Due to changes in intra-thoracic pressure during
respiratory cycle

DIAGNOSTICS
EKG: progressive decrease in voltage of
ECG complexes
F.A.S.T. U/S: looks at pericardial sac for fluid
Portable CXR
Possible Chest CT Scan if stable/time permitting

INTERVENTIONS
Pericardiocentesis, pericardial window or
emergency thoracotomy procedure
CVP and hemodynamic status monitoring
Vital signs trending

BLUNT CARDIAC INJURY
AKA - Cardiac Contusion
Most common cause - direct impact to the chest
into the steering wheel in an MVC
High risk for dysrhythmias

ASSESSMENT
Inspection of chest for contusion/ecchymosis

DIAGNOSTICS
CBC, cardiac serum markers
CXR
ECG - Sinus tachycardia; ST segment and
T-wave changes in anterior leads

MEDICATIONS
Analgesics
Antidysrhythmic medications

INTERVENTIONS
Observe for developing cardiac tamponade
Monitor for dysrhythmias or decreased cardiac
output
CVP and hemodynamic status monitoring possible
Vital signs trending
**PERIPHERAL ARTERIAL DISEASE**
Atherosclerosis of arteries
Risk factors same as for coronary artery disease

**ACUTE ARTERIAL OCCLUSION**
Sudden cessation of blood flow d/t emboli.
Common locations include:
- Femoral artery
- Iliac artery
- Aorta
- Popliteal
- Tibioperoneal arteries
- Thrombus in situ

**VENOUS THROMBUS**
A thrombus present in a superficial or deep vein
Accompanied by an inflammatory process

Risk Factors:
- Stasis
- Vascular damage
- Hypercoaguability
- Trauma
- Immobilization
- Pregnancy (3rd trimester and 1st mo. postpartum)

**ASSESSMENT**
- Calf pain
- Unilateral swelling, warmth, erythema, tenderness
- Homan's sign; not reliable
- Palpable cording possible
- Measure and compare mid calf and ankle circumferences

**DIAGNOSTICS**
- D-Dimer - sensitive though not specific
  - More useful to rule out than rule in
- Duplex venous ultrasonography, doppler
- Venography

**INTERVENTIONS**
- Directed at preventing pulmonary embolism
- Bed rest, elevate extremity
- Anticoagulants: Heparin, Lovenox
- Warfarin: goal INR of 2-3 for 3-6 months
- High risk patients - Filter

**AORTIC ANEURYSM**
Dilation of vascular wall
Aortic, thoracic or abdominal
Risk factors
- Atherosclerosis
- Man
- Age>50
- HTN
### ASSESSMENT
- Usually asymptomatic
- Pain usually an indicator of rupture
- Decreased quality of pulses
- Discrepancies in BP in upper extremities for aortic aneurysm

### DIAGNOSTICS
- Routine labs including T&S; T&C
- Chest xray: aortic aneurysm may show a widened mediastinum
- Aortogram

### INTERVENTIONS
- Operative repair
- Prosthetic graft

### AORTIC DISSECTION
- Tear of the intima layer of the vessel
- Risk factors:
  - systemic HTN
  - age 60-70s
  - male

### ASSESSMENT
- Sudden onset of “tearing” or “ripping” pain
- Signs of hypoperfusion:
  - diaphoresis
  - syncope
  - dyspnea
  - hyper or hypotension
  - loss of pulses

### DIAGNOSTICS
- Aortogram
- Echocardiography, CT

### INTERVENTIONS
- Must urgently decrease stress/pressure on vessel wall
- Beta blockers, Nitroprusside
- Ca Channel blockers (if above contraindicated)
- Surgical Correction

### ARTERIAL LINES
- Indication: any patient receiving a vasoactive substance or for blood pressure monitoring
- Sites: radial or femoral
- Allen’s test
- Transducer:
  - Levelled
  - Zeroed
  - Calibrated
Phlebostatic Axis:
Horizontal to the 4th ICS, Mid-axillary line
Coincides with the atria of the heart
Must be relocated each time pt. is moved

Goal MAP - 70-105

Complications of an A-line (List 3)
1) __________________________________
2) __________________________________
3) __________________________________

Characteristics of a good waveform
   Rapid upstroke
   Clear dicrotic notch
   Definite end diastole
NEUROLOGICAL EMERGENCIES

HEADACHES
4% of ER visits
Main goal: identify emergent conditions
Causes: When pain receptors fire due to:
Traction
Pressure
Displacement
Inflammation
Dilation

Primary HA:
No organic cause
Migraines, tension, cluster
50% of these are migraines

Secondary HA:
Associated w/an organic cause
Tumor
Sinusitis
Aneurysm
Meningitis
Temporal arteritis

HA “red flags”:
Sudden onset (peaking < 1 minute)
“Thunderclap”
Change from prior HA's
Concurrent evidence of infection
Altered mental status
HA with exertion
Age > 50 (regardless of prior HA's)
Immunosuppression
"Focal neurological deficits”
Stiff neck
Toxic appearance

MIGRAINE
With or without aura
Usually: unilateral, pulsating
Moderate to severe
Associated with nausea/vomiting
Photo/phonophobia
Women more than men
Strong family Hx

CLUSTER
No aura
Peri-orbital/temporal locations
Each attack less than 1 hr each
Unilateral tearing and nasal congestion
Eyelid edema, red eye, sweating (poss)
Pain:
 Burning, severe
 Sharp, excruciating, exquisite
Location: Strictly unilateral
Frequency: "clusters"
1-8 per day, 15-180 min duration
Usually ongoing for weeks-months
Men more than women

TENSION-TYPE
Dull/non-pulsating
Bilateral, back to front
Tight/band-like
Possible neck involvement
Nausea vomiting rare
No photo/phonophobia

TEMPORAL ARTERITIS
AKA "Giant Cell Arteritis"
Inflammatory disease of cephalic arteries
Frequently associated with:
  Lupus, Rheumatoid Arthritis, or infections
Unilateral, severe pain, constant
Tender over temporal artery (classic finding)
Female: Male - 3:1
Average onset 65-70 y.o.
EXTREMELY RARE < 50 y.o.
EMERGENT condition
Arteritis may lead to blindness
Sed Rate (ESR) VERY HIGH;
  but may be NORMAL in 20%

Treatment:
Biopsy (for confirmation)
Hi-dose steroids
Analgesics
Needs neuro or vascular consult
Possibly admission

ASSESSMENT (Headaches in general)
What were you doing
Emotional?
ETOH/Caffeine withdrawal/abuse
Sleep deprivation
Menstrual cycle
Food related
Position changes
Recent head trauma
Muscle spasm
Aura prodrome
Nausea/vomiting
Photo/phonophobia
Visual changes

PMHX
Current/pre-existing dz/illness
Prior HA Hx
Family Hx of HAs
Seizure disorder
Prior Hx of sinus problems
**EXAM**
- General appearance
- How uncomfortable?
- LOC, vitals
- Toxic appearing?

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<tr>
<th>Skin:</th>
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<tbody>
<tr>
<td>Color, rashes, sores</td>
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<tr>
<th>Neuro:</th>
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<tbody>
<tr>
<td>Gross assessment (GCS), pupils, cranial nerves</td>
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<th>ENT:</th>
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<tbody>
<tr>
<td>Sinus tenderness</td>
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**DIAGNOSTICS**
- Sed Rate: elevates with TA
- CBC: for suspect infection or TA
- Lumbar puncture: SAH, meningitis
- CT head w/o contrast

<table>
<thead>
<tr>
<th>CT indications:</th>
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<tbody>
<tr>
<td>SUDDEN onset (usu. &lt;1 min)</td>
</tr>
<tr>
<td>Onset with exertion/cough</td>
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<tr>
<td>Focal neurological deficits</td>
</tr>
<tr>
<td>Stiff neck</td>
</tr>
<tr>
<td>New onset (esp Hx Cancer)</td>
</tr>
<tr>
<td>Immunocompromised</td>
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<tr>
<td>New onset &gt; 55 y.o.</td>
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<tr>
<td>Change in “usual” HA pattern</td>
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<table>
<thead>
<tr>
<th>LP notes:</th>
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<tbody>
<tr>
<td>If suspected bleed despite normal CT</td>
</tr>
<tr>
<td>CT usually done first</td>
</tr>
<tr>
<td>Contraindicated if signs of ICP</td>
</tr>
</tbody>
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<table>
<thead>
<tr>
<th>Meningitis:</th>
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<tbody>
<tr>
<td>Cell Count elev, decreased glucose</td>
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<tr>
<th>Intra-cranial HTN:</th>
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<tbody>
<tr>
<td>Cell count normal, protein normal</td>
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<tr>
<th>SAH:</th>
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<tr>
<td>Blood positive</td>
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**INTERVENTIONS**
- Environmental (dec. lights/sounds)
- IV access
- Labs
- CT Head
- Medication administration

**PHARMACOLOGY**
- *Try to treat cause*
- Frequent narcotic use may precipitate Has or withdrawal syndromes
Consider IVF boluses as well
May need vol. replacement d/t N/V
Helps offset possible hypotension of other meds
Dehydration a common cause of migraine

Ergot based meds:
Imitrex, DHE 45 etc.
Useful within hours of onset
Caution with known CAD

Compazine:
Indicated for migraines
Treats N/V well
Some sedation/relaxation as side effect
Give with anti-histamine (Benadryl) to reduce dystonias
Can give with or without narcotics if indicated

Toradol
PO/IM/IV routes
Anti-inflammatory, prostaglandin inhibition
Caution in renal impairment
No euphoria/sedation
No studies comparing against other NSAIDS
Consider giving with anti-emetic as well

Zofran
Good adjunct for N/V
PO/SL/IM/IV
Safe in pregnancy
Wide dosing range

Reglan
Good adjunct for N/V
IM/IV/PO
Safe in pregnancy
Some dystonic side effects possible

Phenergan
Moving away from
Was NEVER approved for IV use
Possibly addicting (euphoria possible)
IM route still quite acceptable
PR route for d/c home if poss N/V continue

Opiate Narcotics:
Dilaudid, Morphine etc
Avoid Demerol whenever possible
Dilaudid, least sedating
Side effects: hypotension, dec. LOC
Concerns about frequent narcotic use
Concerns about “masking” more serious conditions

Rocephin
First line IV antibiotic for suspected meningitis
If blood cultures and LP delayed - Give the ABX!
When in doubt, give the drug!
Consider PCN allergy
Only cross-reactive if prior anaphylaxis
And then only 15%

DISPOSITION/EDUCATION
Ensure adequate follow up
Education about “triggers”
Medication education
Admission if suspected infection or intractable

STROKE
Interruption in vascular supply to cerebral tissue
Ischemic (80%) and hemorrhagic
50% of all strokes fatal
50% of survivors have permanent deficits
700,000 new/repeat strokes/year
Third leading cause of death

Types:
Ischemic (92%):
Atherosclerosis
Plaques,
Emboli
Vessel spasm

Hemorrhagic (8%):
“Bleed”
SAH or ICH

Either way….it results in…
Interruption in blood supply (oxygenation)
Ischemic and inflammatory processes
Subsequent neuronal ischemia and infarction

Treatment goals:
Prevent further damage
Possible reversal of ischemia
Re-establishing vascular supply

Hypothermia as treatment:
Some successes
Some failures and complications though
Ongoing research and protocols necessary

Risk factors:
Hyperlipidemia
Cardiac valvular disorders
A-fib and other dysrhythmias
Diabetes Mellitus
Hypertension
Obesity/Smoking
Substance abuse (cocaine)
Patent Foramen Ovale (PFO)
## ASSESSMENT

**Onset:** important r/t possible TPA admin.
- Witnessed/unwitnessed?

**Symptom progression/change over time**
- Associated Sx prior to onset
- Paresis/weakness
- Sensory changes: usually face-arm-leg

**Visual changes:** diplopia
- Incontinence
- Vertigo, gait
- Speech changes

---

**FINDINGS MORE OFTEN ASSOC. W/BLEED***

- Nausea and/or vomiting
- Photo/phonophobia

---

## PMHX

**CV problems:**
- A Fib, HTN, CHF, Rheumatic HD, Valve problems, MI, CABG

**Neuro:** Migraines
- Diabetes
- Prior CVA/TIA

---

## EXAM

**General appearance**
- LOC, behaviors
- Orientation
- Speech

**Vital signs (HTN common)**
- Motor
  - Facial asymmetry
  - Pronator drift
- Cranial nerve dysfunction
- Gross motor/sensory deficit/change

**Cardiovascular**
- Cardiac Monitor: possible Afib
- Carotid bruits
- Possible murmur if valve disease

---

## DIAGNOSTICS

**CBC:** leukocytosis, anemia, thrombocytopenia
**Chemistries:** BUN/Creatinine, glucose
**Coagulation studies:** baseline
**Cardiac enzymes:** if poss onset r/t cardiac event
**Tox panel:** PRN

**Emergent Non-contrast CT head**
- Goal: In CT within 25 min of arrival
- Goal: Read by radiologist within 45 min of arrival
- Used to rule-in/out hemorrhage or other gross causes
- Ischemic CVA CT usually "normal" for up to 6 hours
- 5% of SAH may have normal CT scan
- Needs LP for "definitive" rule-in/out
INTERVENTIONS
ABCDs, GCS < 8 = intubate
O2 PRN

Rapid IV access; 2 sites
Cautious fluid administration

Position:
HOB 30-40 deg if not contra-indicated
Consider risk of aspiration
Consider dec. cerebral perfusion

Monitoring
Cardiac, pulse oximetry & vs
Frequent neuro checks
NIHSS if trained

Consider NGT (r/t risk of aspiration)
If not, maintain NPO until swallowing is assessed

MEDICATIONS
Anti-hypertensives
Maintain SBP < 185
Maintain DBP < 110

Labetalol:
Trandate
Non selective Beta Blocker

Nicardipine:
Cardene
Ca Channel Blocker

FIBRINOLYTICS
Tpa, Alteplase
Risk of ICH (bleed) approx 6% overall
IDEALLY given within 3 hours of Sx onset
Can be “considered” within 4-6 hours
-no hard evidence; studies ongoing

Dosed by Weight
An initial bolus/push
The rest over the following 60 min

Must meet qualifying criteria per protocols
Still have to consider risk/benefit ratio
Family consultation

If given, pt will be at risk for bleeding.
An ischemic CVA can complicate with a subsequent bleed.
DISPOSITION/EDUCATION
Future risk reduction:
Start antiplatelet: ASA, Plavix, Aggrenox
Start statin

Consider TIA's to be admitted or held in observation status for further evaluation

TIA’s - HIGH recurrence rate
TIA’s - HIGH incidence of subsequent stroke

DEMENTIA
Organic disorder
Decreased cognitive function
LOC remains unchanged
Memory most commonly affected initially

Causes:
Alzheimer’s: 55%
Vascular: 20%

#1 Risk factor = increasing age
Other risk factors: same as those for CVA
Some familial component

ASSESSMENT
Memory loss; get details
Ability to do ADL’s safely
Agitation/Mood changes
Note baseline
Confusion
Sleep problems
NO CHANGE IN LOC

PMHX
Alzheimer’s
CVA, CAD
Parkinson’s
Psychiatric conditions
Head injury
Medications: polypharmacy

EXAM
General: LOC, behaviors, affect, grooming
Neuro: Mini-Mental State Exam

DIAGNOSTICS
CBC: infection, anemia
Chemistries: electrolyte abnormalities
Consider: Thyroid function, U/A, B12 levels

CXR - if indicated
Non-contrast CT head
Especially if a change from baseline

INTERVENTIONS
O2, Monitor PRN
IV fluids if indicated
Treat current infections
Treat electrolyte disturbances
Medications: anxiolytics, psychotropics PRN

DISPOSITION/EDUCATION
Admit for acute change or new onset
If d/c to home -consider pt safety in at home
Primary care follow up important
Consider family ability to care for patient

VENTRICULO-PERITONEAL SHUNTS
Placed to relieve inc. ICP due to hydrocephalus
Excess CSF “shunted” from ventricle to peritoneal cavity
Common complications: infection and malfunction

ASSESSMENT
Type of shunt, when placed
Prior problems
Reason it was placed
Neuro changes
Fever
Nausea/vomiting

PMHX
Concurrent conditions

EXAM
General: LOC, behavior, lethargy, inconsolable
Fever?
Abdomen: distended?
Fontanels (infants): tense/bulging?
Abdomen: tender? (Infection?)

DIAGNOSTICS
CBC (infection)
Chemistries/Glucose (esp Peds)
Xrays: “Shunt series” - Skull, CXR, KUB
Possible CT of head, abdomen
LP if suspect infection

INTERVENTIONS
IVF cautiously to prevent fluid overload
Antibiotics: infection
Anti-emetics: N/V

DISPOSITION/EDUCATION
For shunt revision (malfunction)
For parenteral antibiotics and shunt removal or replacement (infection)
To manage secondary problems (inc. ICP)
Family education: what to watch for and report

SEIZURES
Sudden discharge of neuronal impulses
Temporarily impairs movement, sensation or cognition
Can occur with or without a LOC
Causes:
Electrolyte imbalances (pH changes)
Metabolic changes (fever, hyperglycemia)
Stress, fatigue

Nerve cell structure changes
d/t hypoxia, tumors, trauma
Medication non-compliance

ETOH/Benzodiazepine withdrawals

The International Classification of Seizures:
Partial
Generalized
Unclassifiable (including idiopathic)

Age groups most affected <2 y.o. and >65 y.o.

40% of new onset are <18 y.o.
Most are febrile related
Febrile seizures - strong family Hx
Genetic vs. environment?

At risk Populations:
Hx of head injury
Stroke
CNS infection
Degenerative CNS conditions

STATUS EPILEPTICUS
Series of seizures without recovery period
Near constant focal or generalized contractions
Diaphragm/intercostals involvement
EMERGENCY d/t risk for hypoxia

Treatment:
High doses benzos and/or neuroleptics
Ativan/valium
Phenytoin/fosphenytoin (Cerebyx)

NO PARALYTICS!
The neuronal discharge will still continue
but will just be "masked"
Neuronal cellular changes/destruction
will continue

PMHX
Hx of seizures
Recent infection/illness
Diabetes
Neurological disease
Congenital problems
Medications
Substance abuse
EXAM
General: LOC, behavior, affect, post-ictal state
Vitals: fever?
GU: incontinence
Lungs: wet, aspiration
ENT: intra oral bleeding, trauma
Musculoskeletal: apparent injury or trauma
Skin: rash

DIAGNOSTICS
CBC: infection
Chemistries: electrolyte disturbances
Tox panel
Drug levels
Head CT if new onset or markedly different
LP PRN if suspect infection

INTERVENTIONS
Supplemental O2 if recurrent seizure/status
Suction PRN
Protect from trauma during seizure
Risk for aspiration
RSI if respiratory compromise possible
IV access
Cardiac, pulse ox monitoring
NGT/Foley PRN

PHARMACOLOGY
Benzodiazepines:
Ativan, Valium
Consider IM/rectal if no IV
May have to give higher than usual dosages

Neuroleptics:
Dilantin, Depakote, Tegretol,
Phenobarbital
Check pre-existing levels

Antibiotics:
If infection is suspected cause

DISPOSITION/EDUCATION
New onset, not necessary to admit
...BUT will need follow up
Might need Rx for neuroleptics (patient specific)
No driving

If not new onset -AND-
No identifiable cause found, may consider D/C
Might need loading dose of maintenance
anti-seizure medication if level is low
Ensure follow up
Education:
Follow ups
Drug level monitoring
Home safety/treatment, driving

Pediatrics:
Educate on sound fever control

**INTRA-CRANIAL PRESSURE - TRAUMA**
Managing secondary problems r/t increased ICP is paramount

The goal is to maintain a physiological CPP (Cerebral Perfusion Pressure)
70-100 mmHg

Many head injuries can lead to increases in ICP

Intracranial pressure (ICP)
Sum of the pressures in the cranium:
Cerebral tissue
Blood
CSF

As the pressure (volume) of 1 rises the others decrease to compensate

Cerebral perfusion pressure:
Amount of pressure to perfuse cerebral tissues
Normal 70-100 mm Hg
CPP = MAP-ICP

IE: The “average” systolic blood pressure minus the “resistance” (ICP)

Compensatory mechanisms strive to keep CPP at physiological levels

CPP compensation
Initially, a fair amount of change is compensated and tolerated

As compensatory resources are depleted; a rapid decline ensues

In the LATE stages of increasing ICP; small changes in pressure, can cause big changes in volume
HOW TO MANAGE INCREASING ICP
Increase the MAP:
Fluids, blood, pressors

Decrease the intra-cranial volume:
Diuretics, promote outflow
Elevate HOB

Decrease vasodilation
(which increases volume)

Cerebral ischemia causes pH changes
Increased concentrations of CO2 (an acid) causes vasodilation (bad)

Hyperventilation….?….Or Not?
NOT hyper-oxygenation
We LIKE hyper-oxygenation
Gives more O2 to ischemic tissues
Hyper-ventilation is increasing the minute ventilation

IE: More ventilation means more CO2 is diffused out of circulation

Just like it sounds
(think anxious female hyperventilating)

There are problems however...
Increasing intrathoracic and subsequent ICP increases with increased ventilations

Also may constrict cerebral arteries thus decreasing blood flow

Current guidelines:
“Consider” hyperventilation “if” no clinical improvement with….
-Sedation
-Diuretics
-CSF drainage (when possible)
-Positioning
-Maintain normothermia
-Prevent/treat seizures

In Other Words:
Do the things which are proven to be more effective AND have less risks BEFORE doing something which is more controversial.

HERNIATION
When volume can’t be compensated for any more, risk of herniation ensues
Herniation directly affects brainstem
***CHANGES IN LOC ARE THE MOST IMPORTANT ASSESSMENT FINDING R/T RISING ICP***

**CUSHING’S RESPONSE/TRIAD**
Dr Harvey Cushing (neurosurgeon)  
(NOT: Syndrome -- an adreno-cortical problem)

Indicates impending herniation  
Hypertension (really high)...WHY?  
Bradycardia...WHY?  
Some say: Irregular respirations ....WHY?  
Some say: Widening pulse press....WHY?  
Some say: Pupillary changes...WHY?

Immediate intervention is mandatory!

Findings of Increasing ICP

**EARLY:**  
LOC, behavior, anxiety, restlessness, agitation  
Decreasing responsiveness  
Pupils: sluggish response to light  
Motor: pronator drift, decreasing strength  
Vitals: occasionally tachycardic, some HTN seen

**LATE:**  
LOC, decreasing arousability  
Pupils: fixed and/or dilated  
Motor: hemi-paresis, posturing or no response

Cushing's Response  
Posturing  
Decerebrate/Decorticate  
-which is worse?

Respiration changes r/t ICP increases

**CONCUSSION**  
Temporary impairment of mental state

**SYMPTOMS**  
HA, dizziness  
Amnesia, altered verbal or ocular responses  
Possible N/V  
May or may not have a LOC  
Other trauma sustained

The trauma initially affects the RAS  
(Reticular Activating System)  
Responsible for sleep-wakefulness

“IF” LOC occurs…  
it will resolve in minutes to hours  
Evidence does not support LOC as a useful predictor
GRADES:
Mild = no LOC
Moderate = +LOC

Post-concussion syndrome:
Symptoms persist for a prolonged period of time
Not r/t severity of the initial concussion

Even with apparently minor injuries…
always consider more serious problems:
SDH, SAH, Secondary impact syndrome

ASSESSMENT
Mechanism of injury
Other injuries
LOC changes; current and immediately following
Duration of LOC changes (witness preferred)
Associated sx: vertigo, N/V, amnesia

PMHX
Current other disease/illness
History of other falls, trauma, or head injuries
Medications:
Anticoagulants, antiplatelets, steroids, NSAIDS
Social: ETOH

EXAM
General: LOC, behavior, affect, “blank stare”
Cognitive: memory, recall, attention
Neuro: gait, speech, gross motor
Musculoskeletal: other signs of trauma/injury

DIAGNOSTICS
Non contrast CT head for:
Focal neurological findings
GCS < 15
Seizure activity
Current meds which affect bleeding time
“Consider” based on age or reliability of pt.
Consider ETOH, tox screens PRN

INTERVENTIONS/MEDICATIONS
Seizure precautions if indicated
Cardiac, pulse oximetry if indicated
IV access/labs if indicated
Positioning (HOB elevated)
Regular neuro checks
Document progression of any symptoms

DISPOSITION/EDUCATION
Return to play guidelines
Restrict medications which may impair
neuro functioning/mask sx
Complete recovery expected in 24-48 hours
Supervision round-the-clock x24 hours
Home neuro checks (“wake-ups”)
**INTRA-CRANIAL HEMORRHAGE**
Can be d/t trauma or other (HTN)
Based on lesion location

SDH: sub-dural
SAH: sub-arachnoid
Epidural: above dura
ICH: intra-cerebral; “inside” the brain tissue

Think of “layers” of tissue

<table>
<thead>
<tr>
<th>Lesion: ICH</th>
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</thead>
<tbody>
<tr>
<td>Tissue: Cerebrum</td>
</tr>
<tr>
<td>Tissue: Pia Mater</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Lesion: SAH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tissue: Arachnoid Layer</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Lesion: SDH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tissue: Dural layer</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Lesion: EDH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tissue: Skull/Cranium</td>
</tr>
</tbody>
</table>

**Intra-cerebral**

A) Cerebrum (gray stuff)

B) Pia Mater (#1)

**Sub-Arachnoid Hemorrhage/hematoma**
More common w/contusions
Bleeding will contaminate CSF

Complications:
Seizures
Increased ICP
Cerebral vasospasm
Hydrocephalus

Could be result or cause (of trauma)
Occurs in up to 40% of major head injuries

C) Arachnoid layer (#2)

**Sub-Dural Hemorrhage/hematoma**
More likely venous; bridging veins
More gradual/insidious onset
Often overlooked
Higher overall mortality
Onset over hours-days-weeks
Symptoms progressive

High risk:
Alcoholics
Alderly
Those on anticoagulants/anti-platelets
**Epidural Hemorrhage/hematoma**
Most often d/t middle meningeal artery
Commonly r/t temporal skull Fx
Arterial in nature; rapidly expanding
Approx 50% mortality
“Talk and die” phenomenon (lucid interval)
Rapid presentation/Progression of Sx

**SKULL FRACTURES**
Linear:
Approx 70% of all Fxs
Usually benign

Depressed:
Damage to underlying cerebral tissue

Basilar skull Fxs:
Occur in one of the fossae
Clinical sx depends on area affected

Possible complications of all skull Fx’s:
Infection
Hematoma
CSF leaks
Anosmia (what is this??)

Hearing problems
Seizures
Pneumocephalus
Consider maltreatment/abuse in peds with these

**HEAD INJURIES - OTHER POINTS...**
Do good facial assessment also if skull Fx possible
Good neuro checks, including pupils and EOMs
Consider orbital Fx’s also

Terms:
Battle’s sign
Raccoon eyes

Don’t pack ears/nose if CSF leaking
Elevate HOB
Ice packs PRN
Ensure c-spine protection if indicated
Manage ICP increases
Avoid nasal intubation or NGT if facial Fx’s
Aggressive airway management

Terms to know:
Otorrhea:
Rhinorrhea:
Hemotympanum:
Skip the skull xray - get a CT -AND-
Consider doing the facial also
Consider doing the C-spine also

**SPINAL CORD INJURIES**
Bruising or tearing of spinal cord tissue
Nearly 100% r/t trauma

<table>
<thead>
<tr>
<th>Forces:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Axial loading</td>
</tr>
<tr>
<td>Hyperflexion</td>
</tr>
<tr>
<td>Hyperextension</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Intrinsic damage:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemorrhage</td>
</tr>
<tr>
<td>Edema</td>
</tr>
<tr>
<td>Hypoxia</td>
</tr>
<tr>
<td>Biochemical</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Extrinsic damage:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bony and soft tissue injury</td>
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</tbody>
</table>

Can lead to/cause neurogenic shock

Classified as complete or incomplete

<table>
<thead>
<tr>
<th>Complete:</th>
</tr>
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<tbody>
<tr>
<td>Transection</td>
</tr>
<tr>
<td>NO sensory/motor function</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Incomplete:</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Central Cord syndrome</td>
</tr>
<tr>
<td>Sensory/motor sx &gt; in upper extremities</td>
</tr>
<tr>
<td>2) Anterior Cord syndrome</td>
</tr>
<tr>
<td>Loss of motor, pain and temp. sensation</td>
</tr>
<tr>
<td>Preservation of vibration, touch and position perception</td>
</tr>
</tbody>
</table>

**Brown-Sequard Syndrome**
**Same side:**
Paralysis/paresis
Loss of pressure, touch, vibration

**Other side:**
Loss of pain and temperature perception

SCI and respiratory...
Phrenic nerve controls diaphragm
Exits at C3-4-5; thus C4 = “breathe no more”
Intercostal nerves for accessory muscle breathing
They exit T1-T12
Controls deep breathing, coughing, sighing
C-SPINE INJURIES

What "clears" a c-spine?
A) Xray/CT ONLY proves NO Fx’s OR DISPLACEMENTS

B) The patient must demonstrate no
motor/sensory deficits

C) The pt exam must elicits no painful or
tender places to suggest injury

So therefore....
You need negative xrays and a normal neuro
exam in an AWAKE, ALERT, COOPERATIVE patient...

And...
The patient must have a clear sensorium and not
have any other markedly PAINFUL or DISTRACTING
conditions present...

Clinical judgment prevails...

Backboards do not protect C-spines
Probably don’t protect thoracic & lumbar areas

…but, they sure make a good transfer device!
…no conclusive science has proven them

Still, early removal, if clinically indicated,
and if appropriate, is beneficial

SCI and Steroids
Given to:
PRESERVE
PROTECT-AND-
"Possibly" RESTORE spinal cord function

Use is controversial
There are risks to these recommended
MEGA-DOSES of steroids

National Spinal Cord Institute still recommends
Solu-Medrol: 30mg/kg over 15 min
Example: 100 kg male (220 lbs) = 3 GRAMS!
Data supports use within EIGHT hours of injury

SPINAL CORD INJURY – OTHER POINTS...
Good pulmonary/respiratory support
Good skin/breakdown care/prevention
Protect spine/c-spine until clinically AND
radiographically cleared
Frequent VS/Neuro checks
Strong family/patient support
Best outcomes: prevention!
### AMYOTROPHIC LATERAL SCLEROSIS

**Lou Gehrig’s disease**

- Progressive, usually fatal
- Neuro-degenerative disease
- Affects MOTOR neurons (voluntary movement)
- Subsequent weakness/atrophy
- Cognitive function is generally spared
- Affects all races/sexes equally
- No consistent cause

Possible causes:
- Viral
- Neurotoxin
- DNA
- Immune system causes

#### ASSESSMENT

- Symptoms gradual, subtle
- Initially stiffness, cramping, twitching of muscles
- NO bowel/bladder involvement:
  - autonomic control remains

Later muscles have weakness and atrophy
- 75% have “limb onset”
- More often legs: frequent tripping, stumbling, falls

- 25% “bulbar onset”
- Difficulty with speech
- Followed by swallowing and tongue problems
- Loss of protective airway reflexes

- Both will progress to all body parts
- Some cases have an arrested progression
  - young males often

#### TREATMENT

- No cure yet
- Riluzole: FDA approved
- Lithium currently being investigated

#### NURSING IMPLICATIONS

- Similar to any pt with weakness or paralysis
- Skin protection/breakdown
- Airway protection
- Ventilatory support
- Pulmonary toilet/hygiene
- Prevent/treat secondary complications
- Team approach
- Social work
- Physical/occupational therapy
**MULTIPLE SCLEROSIS**  
Autoimmune condition  
CNS is attacked  
Demyelination results  
Leads to physical AND cognitive disabilities  
Starts in young adulthood  
More common women  

Loss of myelin causes ineffective/loss of nerve impulse conduction  

Suggested causes:  
Epstein-Barr virus  
Vit-D deficiency in youth  
NOT considered genetic or hereditary  

No cure  

Can have either PROGRESSIVE disease, or episodic ATTACKS  

Therapy aimed at maximizing function and treating secondary complications  

Life expectancy approaches that of the unaffected population  

**ASSESSMENT**  
Changes in sensation, muscle weakness or spasms  
Coordination, balance, gait problems  
TREMOR: At rest  

Occasionally:  
Dysarthria, dysphagia, fatigue, visual problems  

May also be associated with other chronic pain syndromes  

**THE MOST COMMON initial symptom reported is:**  
Change in sensation or arms, legs or face (33%)  
Diplopia may occasionally be a first symptom  

**INTERVENTIONS/TREATMENT**  
During acute episodes (exacerbations):  
Steroids (solu-medrol)  
Analgesics (muscle pain, soreness)  
Muscle relaxants (muscle twitching, spasticity)  

Disease modifying agents (DMAs):  
ininterferon and interferon-like
### MYASTHENIA GRAVIS

- Means "muscle weak"
- Autoimmune disorder
- Antibodies block ACETYLCHOLINE receptors
- Leads to fluctuating muscle weakness and fatiguability

### ASSESSMENT

- Hallmark: muscle weakness during periods of increased activity, improving with rest
- Eyes, eyelids, facial muscles commonly affected
- Chewing, talking, swallowing muscles affected
- Can involve diaphragm
- COMMONLY- first symptoms affect eye muscles

### MYASTHENIC CRISIS:

- Paralysis of respiratory muscles

### DIAGNOSTICS

- Must rule out other conditions first
- Neuro testing may be ordered
- Possibly serology can identify acetylcholine receptor antibodies (sometimes)

- **"Tensilon" test:**
  - IV edrophonium causes a temporary increase in ACTH at the NMJ
  - A rapid/brief return of function is noted
  - Not usually used anymore
  - EMG’s helpful
  - A good history and documentation of symptomatology are useful

### INTERVENTIONS/TREATMENT

- Cholinesterase inhibitors (neostigmine)
- Immunosuppressants (prednisone, cyclosporine)
- Plasmapheresis
- Thymectomy d/t inc incidence of Thyomas in MG

### PARKINSON’S DISEASE

- Degenerative disease of CNS
- Affects basal ganglia
- Decreased numbers of dopamine secreting cells
- Decreased efficiency of motor movements

- No diagnostic testing
- Pt history and exam are the most useful tools!
ASSESSMENT

Tremor: AT REST

Rigidity: stiffness, inc. muscle tone, cogwheel rigidity with passive ROM

Akinesia: slowness of movement or absent

Postural instability: postural reflexes fail leading to balance problems; falls

INTERVENTIONS/TREATMENTS

No cure

Dopamine replacement or augmentation
L-dopa is base form of dopamine

Combined with Carbidopa:
Maximizes the amount of drug converted to it's useful form (Sinemet)

Dopamine Agonists:
Stimulate dopamine response
Many side effects
Bromocriptine, Pergolide

OTHER NEUROLOGICAL CONCEPTS

GCS
Measures the BEST:
EYE function: 1-4
VERBAL function: 1-5
MOTOR function: 1-6

----> A rock gets a 3
----> A corpse gets a 3
ABDOMINAL/GI EMERGENCIES

GASTRITIS

Inflammation of stomach

Common causes:
- NSAIDS
- ETOH
- Steroids
- Caustic ingestions
- Infected food

Other causes:
- Physical/emotional stress
- Tobacco
- Radiation
- Bacterial/viral infections
- Chronic infection (H.Pylori)

Acute episodes:
- Epigastric pain
- N/V/D
- Anorexia

Chronic Gastritis leads to:
- Ulcers
- Hemorrhage
- Anemia
- Perforation

HISTORY

Pain usually relieved by food
- Squeezing, burning, dull, gnawing
- Sudden or gradual

EXAM

Possible orthostatic
- Hematemesis
- Bowel sounds: normal to hyperactive
- Epigastric TTP, guarding

DIAGNOSTICS

CBC, CMP
- Lipase, Amylase
- Urine Hcg
- Hemocult
- Abd series xrays

TREATMENT

“Rule out” causes
- NPO, NGT possibly
- IV Fluids
- PO challenge

MEDICATIONS

Antiemetics
- Antacids
- H2 blocker
- PPI
EDUCATION
Clear liquids - advance as tolerated
Avoid causative agents

ULCERS
Linings of the upper GI break down
Erosions and open wounds develop and bleed
GI bleeding can be occult and go unnoticed

The pathogen H.Pylori has been implicated in chronic ulcer disease conditions

Ulcers more common:
Men
Smokers
NSAID users

ASSESSMENT
Pain: associated with eating or fasting
Squeezing, burning, gnawing
Epigastric pain, poss radiation to back

Duodenal ulcer:
Pain before meals - AND -
Relieved by eating or antacids

Gastric ulcer:
Usually pain after eating

PMHX
ETOH, caffeine, tobacco use
NSAID use, or ASA

EXAM
Vomiting
Hematemesis
Epigastric tenderness

DIAGNOSTICS
CBC, Chemistries
Type/cross
LFTs, Amylase, Lipase
Hemocult stool

TREATMENTS
IV access, possible fluid replacement
NPO, consider NGT

Medications:
Narcotics
Antiemetics
Antacids
H2 Blockers/PPI

EDUCATION
Bland, low fiber diet
Avoiding causative factors

~ 140 ~ CEN Review Course
BOWEL OBSTRUCTION
Normal movement of intestinal contents is impaired/blocked

May be partial or complete

Causes include:
- Strictures
- Hernias
- Adhesions
- Paralytic ileus
- Cancer

Obstruction causes intestinal materials, fluids, and gas to accumulate

Normal GI absorption is decreased
Gastric acid production is increased
Fluids and electrolytes are imbalanced

Increasing pressure causes decreased perfusion to the living intestinal lumen

Leads to possible:
- Edema, congestion
- Bowel necrosis
- Rupture or perforation

ASSESSMENT
Abdominal pain
Colicky, crampy, intermittent
Vague, poor localization

PMHX
Hx of abdominal surgery
Concurrent conditions
Change in bowel habits

EXAM
Sepsis like findings
Can be very ill appearing
Abdominal distention
Vomiting: bilious, fecal material possible
Bowel sounds: high pitched to absent
TTP diffusely
Tympanic to percussion

DIAGNOSTICS
CBC, Chemistries
Amylase, lipase
Type and cross
Abdominal series x-rays
Abdominal CT possible
TREATMENTS
IV access
Fluid resuscitation
NPO, NGT possible
Admission/surgical consult

MEDICATIONS
Antiemetics
Antibiotics
Narcotics

GASTROENTERITIS
Inflammation of the linings of the stomach and/or intestine

Most causes are infectious:
Viral
Bacterial
Parasitic

Also possible d/t imbalance in normal GI flora secondary to recent antibiotic use

Usually self limiting

Routes of infection:
Person to person
Fecal contact
Ingestion of contaminated foods
Raw or under prepared foods

Infectious diarrhea produces enterotoxins that override the colon’s ability to reabsorb fluids (C. diff, Giardia, E. coli)

Inflammatory diarrhea agents invade the intestinal mucosa and release toxins that destroy the mucosal lining (Rotavirus, Norwalkvirus, Salmonella, E. coli, Campylobacter)
The result is bloody diarrhea, abdominal cramping and fever.

“Food Poisoning”
Usually caused by bacterial toxins:
S. Aureus, Clostridium and Bacillus
Short incubation (2-6 hours).
Prominent vomiting and epigastric distress

ASSESSMENT
Pain: cramping, colicky, diffuse
PMHX
Concurrent disease states
Stool pattern
Travel
Recent antibiotic use
Consumption of raw/undercooked foods

EXAM
Fever possible
Vomiting, diarrhea
Hyperactive bowel sounds

DIAGNOSTICS
CBC, Chemistries
Stool for WBC, culture, ova/parasites

TREATMENTS
IV access and fluid replacement
PO challenge eventually
NPO until vomiting under control

MEDICATIONS
Antiemetics
Narcotics
Anticholinergics
Antibiotics

EDUCATION
Clear liquids, advance as tolerated
Hand washing
GI referral for prolonged, recurrent, protracted episode

GASTRO-ESOPHAGEAL REFLUX DISEASE (GERD)
Back flow of gastric contents into esophagus
Seen commonly in overweight/obese and those over 40 y.o.

MECHANISMS
Dec. lower esophageal sphincter (LES), tone
Slowed motility or peristalsis
Delayed gastric emptying

Many go on to develop esophagitis.
May occur concurrently with a hiatal hernia.

ASSESSMENT
Pain usually aggravated by meals or activities
Burning sensation classic
May radiate to chest, back, neck, jaw
Often occurs within 30-60 min of eating
### PMHX
- Peptic ulcers
- Cardiovascular disease
- Obesity
- Tobacco use
- Caffeine
- NSAID use

### EXAM
- Belching, hoarseness
- Epigastric TTP

### DIAGNOSTICS
- CBC, Chemistries
- Lipase, amylase
- Liver functions

#### **CARDIAC RULE OUT**
- EKG, Cardiac biomarkers

### TREATMENTS
- IV access, fluid replacement

### MEDICATIONS
- Antacids (GI Cocktail)
- H2 receptor antagonists
- PPI's
- Anticholinergics

### EDUCATION
- Diet: small meals, low-fat foods
- Raise HOB 6-8 inches
- Wt loss as appropriate
- Tobacco cessation

#### Limit:
- Caffeine
- Alcohol
- Chocolate
- Peppermint
- Spicy foods

### BOWEL INTUSSEPTION
Segment of bowel “telescopes” within itself
Causing a mechanical bowel obstruction
Most commonly in infants and small children

- Usually starts at a defect area:
  - Ileo-cecal valve
  - Location of a tumor
  - Meckel's diverticulum

- Without treatment, can be fatal
- Can lead to bowel ischemia, necrosis
- Ultimately gangrene and sepsis
The classic presentation triad of vomiting, colicky abdominal pain and currant jelly stool only presents in 25% of all patients.

**ASSESSMENT**
Pain: colicky, spasmodic, diffuse
Out of proportion to exam
Sudden onset, explosive, comes-n-goes

**PMHX**
Diarrhea
Constipation
Appetite changes

**EXAM**
Fever possible late
May be critical appearing if sepsis pending
Paroxysms of acute abd pain
Knees pulled to chest during acute periods
Rectal blood or currant jelly like w/mucous
Bowel sounds: hyperactive

Palpation:
“Sausage shaped” mass possible RLQ and MLQ

**DIAGNOSTICS**
CBC, Chemistries
Hemocult
Abdominal series x-rays

**TREATMENTS**
IV Access/fluids
NPO/NGT
Barium enema with fluoro or sonography
Guided air enema

**MEDICATIONS**
Analgesics
Antispasmodics/anticholinergics

**PYLORIC STENOSIS**
Most common cause of intestinal obstruction during infancy

95% of the time it is found in the first 1-3 months of age

Hypertrophy, hyperplasia and narrowing of the lower gastric compartment results in the blockage

Causes are hereditary and environmental
4:1 male to female incidence
30% are first-born male children
Lack of diagnosis and treatment can lead to dehydration, shock and death

**ASSESSMENT**
- Projectile vomiting with feedings
- Can be intermittent however
- Poor wt gain or even wt loss
- Hunger

**EXAM**
- Hypotension/tachycardia (r/t shock)
- Jaundice
- Bowel sounds; hypoactive
- Palpation: “Olive shaped” mass

**DIAGNOSTICS**
- CBC, Chemistries
- LFTs, bilirubin
- Abdominal series x-rays, U/S

**INTERVENTIONS**
- IV access/fluids
- NPO/NGT
- Prepare for admission for surgery

**APPENDICITIS**
- Inflammation or obstruction of the appendix
- Leads to ischemia, necrosis or perforation
- Subsequent peritonitis

Must have a high index of suspicion
No specific assessment or diagnostic test can accurately identify conclusively

Occurrence peaks in late teen years

**ASSESSMENT**
- Pain: with movement
- May progress from dull to colicky

Early:
- Poorly localized/peri-umbilical

Later:
- May localize to RLQ (McBurney's point)
- Nausea/vomiting possibly

**EXAM**
- Fever possible
- Vomiting
- Bowel sounds; hypoactive
- TTP RLQ/McBurney's point
- Rebound tenderness, peritonitis findings

**Rosving’s sign:** LLQ pressure increases RLQ pain
**DIAGNOSTICS**
- CBC, chemistries
- Abdominal CT scan: most precise
- Abdominal U/S: false negative possible

**INTERVENTIONS**
- IV access
- NPO
- Prepare for admission
- Surgery consultation

**MEDICATIONS**
- Analgesics
- Anti-emetics
- Antipyretics

**PANCREATITIS**
Inflammation of the pancreas
Release of digestive enzymes causes autolysis

May present with acute episodes or chronic exacerbations

Common causes for acute:
- Mechanical obstruction (gallstones)
- Injury
- Infection
- Alcoholism
- Medications (steroids, diuretics, sulfonamides)

Respiratory symptoms may accompany acute episodes
- Atelectasis, pleural effusions

Chronic pancreatitis is an inflammatory state that results in irreversible damage

The organ function is preserved until late in the disease state

Hypocalcemia may be seen as a complication due to free fatty acids binding with free calcium

**ASSESSMENT**
- Pain: aggravated by eating, ETOH intake
- Unrelieved by antacids
- Sharp, boring pain
- Epigastric, abdominal, chest, back pain
- Usually abrupt onset

**PMHX**
- Concurrent conditions
- Previous episodes
- ETOH intake, esp recent binge drinking
- Recent/current gallbladder disease
EXAM
Hypotension/tachycardia - shock state poss.
Possible fever
Moderate to severe distress
Abdominal distention
Fatty stools
Jaundice
Vomiting
Bowel sounds: hypoactive or absent
Breath sounds: crackles, rhonchi poss
TTP over epigastrium

DIAGNOSTICS
CBC, Chemistries
Amylase, lipase
LFTs
CXR, Abdominal series x-rays
Abdominal CT: most reliable
**CARDIAC RULE OUT!**

INTERVENTIONS
IV access, fluids
NPO/NGT
Airway control
Prepare for admission

MEDICATIONS
Narcotics
Antibiotics
Antiemetics

CHOLECYSTITIS
Inflammation of the gallbladder
Most often d/t blockage of the duct by stones
Other causes: infections, tumor

ASSESSMENT
Pain: aggravated by breathing
Sharp, constant, colicky
RUQ or epigastric, possibly referred to right
scapula or shoulder
_________________’s Sign

Frequently onset after a large/fatty meal
Indigestion, nausea, vomiting

EXAM
Fever possible
Jaundice
Bowel sounds; hypoactive or absent
RUQ TTP, guarding, rigidity
Murphy’s sign

DIAGNOSTICS
CBC, Chemistries
Amylase, Lipase
LFT's
Abdominal series x-rays
Abdominal U/S
Abdominal CT

TREATMENTS
IV access, fluids
NPO/NGT
Prepare for admission
Surgical consultation

MEDICATIONS
Antiemetics
Analgesics (Avoid Morphine if poss)
Antibiotics

EDUCATION
Low fat diet, avoid causative factors

DIVERTICULITIS
Inflammation of colon diverticula
Small mucosal pouches along the colon wall
Entrapment of undigested food/fecal material
Bacteria present may lead to inflammation and infection
Can possibly perforate the diverticula wall
Affects the sigmoid colon
Almost exclusively only in pt's over 50 y.o.

ASSESSMENT
Pain: aching, crampy, vague
May localize to the LLQ
Can be abrupt or onset over a few days
Assess bowel patterns
Bloody stools

EXAM
Possible low-grade fever
Hemocult positive usually
Bowel sounds hyper/hypoactive
LLQ TTP; guarding

DIAGNOSTICS
CBC, Chemistries
Hemocult
Abdominal CT scan
Sigmoidoscopy

TREATMENTS
IV access, fluids
NPO, consider NGT
Prepare for admission
Possible surgical consultation
### MEDICATIONS
- Analgesics
- Antibiotics
- Anticholinergics

### EDUCATION
- Recommend high fiber diet
- GI follow up

### ESOPHAGEAL VARICES
Dilated veins of the lower esophagus
May rupture and bleed
Cause: obstructed portal circulation
Associated with liver cirrhosis d/t chronic alcoholism
Hemorrhaging may be occult and unnoticed until severe

### ASSESSMENT
- Pain; dull, possibly radiate to chest
- Onset: gradual, sudden, constant or intermittent
- Nausea, vomiting
- Hematemesis

### PMHX
- Alcoholism, liver disease
- Gastritis, Ulcers, Bleeding disorders

### EXAM
- Shock like S/Sx
- Pallor
- Hematemesis
- Melena
- Hepatomegaly
- Splenomegaly
- Ascites

### DIAGNOSTICS
- CBC, Chemistries
- LFT’s, PT/PTT/INR
- Serum Ammonia
- CXR, Upper GI series
- Type/cross

### TREATMENTS
- IV access, fluids
- NPO/NGT
- Endoscopy or mechanical tamponade
- If mechanical tamponade: intubate
- Surgical/GI emergent consultation

### MEDICATIONS
- Vasopressors
- Vitamin K
- Analgesics
- RSI meds
ENT & OCULAR EMERGENCIES

CONJUNCTIVITIS
Inflammatory condition of the membrane lining the eyelids

Inflammation causes marked hyperemia (injection) and discharge

Causes:
Bacterial, viral
GC/Chlamydia
Allergens
Chemical burn, flash burns
Foreign bodies

Common organisms:
Streptococci, H. Flu,
Staphylococcus,
Pneumococci and Gonococci

Spread commonly by direct contact.
Good hand washing paramount

Contact wearers: susceptible to Pseudomonas

Neonates at risk for Chlamydia and N. Gonorrhea from birth.

ASSESSMENT
Redness of affected eyes
Sudden, abrupt onset
Pain: “irritated”
Foreign body “gritty”
Discharge: clear or purulent, “matting”

PMHX
Steroid use: may worsen infections
Close contact w/others affected

EXAM
Visual acuity should be normal
Cornea/pupil: clear/normal
Conjunctiva: red or pink
Discharge: purulent or mucopurulent
Eyelid edema possible

DIAGNOSTICS
None usually indicated
Consider cultures of discharge
Corneal staining to rule in/out foreign body

INTERVENTIONS
Visual acuity
Cultures if indicated
Ophthalmology referral for recurrent cases -OR-
If suspected Herpes infection
MEDICATIONS
- Bacterial: Broad spectrum
- Contact Lens wearers: Fluoroquinolone
- Viral: NSAID
- Allergic: Antihistamines
- Suspect Gonococcal: Systemic Abx

EDUCATION
- Eye med administration
- Frequent hand washing
- Avoid eye makeup, discard
- Contacts out until treatment completed

UVEUTUS/IRITIS
Uveal tract inflammation:
- iris, ciliary body and choroid

- Pain, reddened, edema, lacrimation
- Photophobia
- Usually idiopathic

- Pain in affected eye with light into opposite eye d/t consensual constriction of irritated iris

- Hallmark finding:
  - Inflammatory cells in anterior chamber on slit-lamp exam

- W/O treatment, uveitis can lead to glaucoma, pupillary abnormalities, cataracts and macular dysfunction
- Prompt ophthalmologic referral indicated

ASSESSMENT
- Decreased visual acuity
- Blurred vision
- Edema of eyelid
- Red eye
- Intense photophobia
- Lacrimation/tearing

PMHX
- Rheumatoid arthritis
- Ankylosing spondylitis
- Syphilis
- Family Hx of rheumatic diseases

EXAM
- Decreased visual acuity
- IOP: low to minimally elevated
- Pupils: miosis, small, sluggish
- Cornea: clear-hazy

DIAGNOSTICS
- Visual acuity
- Slit-lamp exam
Fluorescein stain
Tonometry

INTERVENTIONS
Dark environment
Warm compresses to eye(s)
Topical steroid agents per ophthalmologist
Cycloplegic agents to reduce ciliary spasm

EDUCATION
Shielding methods or dark glasses
Eye medication administration
Eye rest
Ophthalmological follow up

GLAUCOMA
Acute angle-closure glaucoma
Angle of junction between iris and cornea becomes narrowed/ blocked
Aqueous fluid cannot exit through canal of Schlemm
Precipitating factors:
Gradual inc. in pressure and sudden
dilation of pupil
Usually in a darkened environment
(movie theater then walks outside)
Unless treated within hours, blindness may result d/t optic nerve damage and decreased retinal circulation
Peak age: 55-70

ASSESSMENT
Red eye
Pain: severe, sudden onset, deep, unilateral
Headache
Blurred vision
Light halos
Photophobia
Nausea/vomiting

EXAM
Decreased visual acuity
Cornea: hazy
Affected pupil: poorly reactive, fixed
IOP: 40-80 mm Hg (more than the normal 20)

DIAGNOSTICS
Visual acuity
Tonometry
Slit-Lamp exam
## INTERVENTIONS
- IV access (possible IVF, IV pain meds)
- Immediate Ophthalmological consultation
- Definitive Tx: Iridotomy

## MEDICATIONS
- Narcotics
- Antiemetics
- Beta blocker eye meds: Diamox
- Pilocarpine eye drops
- IV diuretic: mannitol

## RETINAL ARTERY OCCLUSION
**Sudden, painless, unilateral loss of vision**

- Blockage of retinal artery:
  - Thrombus, embolus

- Can arise from carotid plaques or heart valves

- Can be preceded by "Amaurosis fugax"
  - sudden temporary vision loss
  - seconds to minutes

- To preserve vision, must be recognized and treated within 1-2 hours

- Average age: 50-70 y.o.

## ASSESSMENT
- Sudden, profound, unilateral loss of vision
- Painless

### PMHX
- Cardiovascular disease
- HTN
- Hx of CVA or TIA
- Cardiac valve disease
- Atrial fibrillation
- Atherosclerotic disease
- Diabetes
- Trauma

### EXAM
- Anxious
- Visual acuity limited to only light perception

### DIAGNOSTICS
- Fundoscopic exam
- Tonometry
- CBC, Chemistries
- Coagulation profile
- Glucose
- CT head
INTERVENTIONS
IV access
Immediate ophthalmological consultation
Admission/surgery

MEDICATIONS
Ocular hypotenstive drops (Beta blockers)
Systemic acetazolamide (Diamox)
Controversial: tPA, anticoagulants

RETINAL DETACHMENT
Vision-threatening emergency
Separation of retinal layers from choroid

Accumulation of vitreous fluid/blood can decrease blood and O2 supply to retina

Common cause is degenerative changes in either retina or vitreous body in the elderly

Direct head trauma and sports injuries may also be a cause

ASSESSMENT
Mechanism of injury
Cloudy, smoky vision
Flashing lights; floaters
Curtain, veil “coming down”
Gradual or sudden

PMHX
Diabetes
Sickle cell disease
HTN
Head or facial trauma
Prior eye surgeries

EXAM
Pupil reaction usually normal
Possible visual field deficits

DIAGNOSTICS
Visual acuity: diminished
Fundoscopy
Slit-lamp exam
Tonometry

INTERVENTIONS
Shield both eyes to decrease eye movement
Bed rest, avoid movement
Emergent ophthalmological consultation
Admission/surgery
CORNEAL ABRASION
From an injury to the corneal epithelium
Common causes: foreign bodies, contact lens wearing and UV burns
Range from superficial to deep
The Cornea itself is avascular so healing and re-epithelialization may be complicated

ASSESSMENT
Hx of injury or contact lens wearing
Pain, redness, tearing possible
Light sensitivity
Inquire about use of protective equipment

EXAM
Conjunctiva injected
Tearing, blinking
Evert eyelid to check for FB
Erythema or swelling of eyelids

DIAGNOSTICS
Visual acuity: usually normal
Fluorescein staining
Slit lamp exam

INTERVENTIONS
Eye patching controversial
If organic/vegetative material involved must cover for risk of Pseudomonas

Meds
Ophthalmic anesthetics
Cycloplegics (reduces ciliary spasm)
Ophthalmic antibiotic
Tetanus ......why?

EDUCATION
Eye rest 12-24 hours; sunglasses, dark room
Ophthalmology recheck in 24 hours

HYPHEMA
Blood in anterior chamber of eye
Usually due to trauma
Damaging to small vessels of iris
Usually resolves
Requires ophthalmologic follow up

ASSESSMENT
Mechanism of injury/associated injuries
Visual changes/acuity
Check PERRLA, EOM's
Possible tonometry
Slit lamp examination
**TREATMENT**
- Eye shield
- HOB 30-40 degrees
- Analgesics
- Ophthalmological consultation and follow up

**GLOBE RUPTURE**
- Ocular emergency
- May require immediate ophthalmologic surgery
- Due to blunt or penetrating forces

**ASSESSMENT**
- Visual acuity/changes
- Other associated injuries
- Pain: minimal to severe
- Asymmetry of globes
- Extrusion of aqueous humor
- Possible enopthalmos
- “Teardrop” pupil possible

**DIAGNOSTICS**
- Visual acuity
- CT head/face for associated injuries

**TREATMENTS**
- Patch both eyes to disallow movement
- HOB elevated
- Analgesics
- Requires emergent ophthalmological consultation
- Prepare for admission
## ENT EMERGENCIES

### PHARYNGITIS

**Sore throat**

Usually a viral URI causes inflammation of the pharynx

Most resolve with symptom treatment only

Transmission by droplets

Common fall, winter, spring

Possible fine scarlatiniform rash

### ASSESSMENT

Mild-mod sore throat

Possible referred pain to ears, neck, jaw

Painful swallowing

Possible fever/chills

Possible cough

Fatigue, myalgias

### EXAM

Congested

Fever possible

Enlarged tonsils possible

Injected posterior pharynx

Cervical nodes enlarged/tender

### INTERVENTIONS

Throat culture

CBC, BMP possible

### MEDS

Symptomatic

OTC NSAIDS, Lozenges

Consider glucocorticoid (inflammation)

Consider narcotics

Consider antitussives/decongestants

To cover *Streptococci* PCN is drug of choice

Benzathine PCN 1.2 million units IM x1

PO Abx 10 days!

Azithromycin for PCN allergic

### EDUCATION

Warm saline gargles

Good hand washing

Cover mouth, nose

### EPISTAXIS

Most due to bleeding from Kiesselbach's Plexus

Superficial blood vessels

Less often due to bleeding arterioles from ethmoid arteries
Factors causing:
Localized trauma
Forceful nose blowing
Foreign bodies
Rhinitis
Drying of nasal mucosa
Antiplatelet medications

Anterior epistaxis:
Pinching nostrils for 10-30 minutes
Topical anesthetics and/or vasoconstrictors
Follow with by Siliver Nitrate cautery
Packing may follow

Posterior epistaxis:
Most always requires packing

DIAGNOSTICS:
To rule in/out causes:
CBC, Coags, Type and cross if severe
CT scan if tumor suspected

ODONTALGIA
Frequently the result of a cavity involving the pulp and nerve tissue
Sudden or gradual
Sharp or dull
May radiate to ear, jaw, temple, neck

Untreated, the decaying tissue leads to necrosis and possible abscess formation

Treatments aimed at relieving pain and preventing further tooth decay.
Antibiotics and analgesics usually prescribed.
Dental follow up and assessment is mandatory

DENTAL ABSCESS
Localized build up of pus in a tooth cavity
May extend to deep tissues: tooth, roots, bone
Pain, localized or radiating

Swelling, warmth, tenderness of maxillary/mandibular tissue
Fever/chills possible
Fetid breath
Malaise

DIAGNOSTICS
CBC
Facial/Neck CT
<table>
<thead>
<tr>
<th>TREATMENT</th>
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<tbody>
<tr>
<td>Incision and drainage if fluctuant</td>
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<tr>
<td>Wound culture/sensitivity</td>
</tr>
<tr>
<td>IM/IV antibiotics</td>
</tr>
<tr>
<td>Analgesics</td>
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</tbody>
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<tr>
<th>DISPOSITION</th>
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<tbody>
<tr>
<td>Maxillary abscess:</td>
</tr>
<tr>
<td>Concerning for sinus or cranial extension</td>
</tr>
<tr>
<td>ENT consult, possible admit</td>
</tr>
<tr>
<td>Mandibular abscess:</td>
</tr>
<tr>
<td>Appropriate antibiotics</td>
</tr>
<tr>
<td>Ensure adequate follow up with ENT or Oral Surgeon</td>
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<table>
<thead>
<tr>
<th>LUDWIG'S ANGINA</th>
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<tbody>
<tr>
<td>Usually an extension of a pre-existing dental infection.</td>
</tr>
<tr>
<td>Infection and cellulitis extends into the submandibular, sub-mental and sublingual tissues</td>
</tr>
<tr>
<td>Potential for airway compromise d/t swelling</td>
</tr>
<tr>
<td>Risk for sepsis</td>
</tr>
<tr>
<td>Infection can progress and swell into the tissue planes of the neck and inferiorly into the mediastinum</td>
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<table>
<thead>
<tr>
<th>ASSESSMENT</th>
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<tbody>
<tr>
<td>Sick/toxic appearing</td>
</tr>
<tr>
<td>Tongue swollen, elevated</td>
</tr>
<tr>
<td>Possible trismus, dysphonia, dysphagia</td>
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<tr>
<td>Fever/chills</td>
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<thead>
<tr>
<th>DIAGNOSTICS</th>
</tr>
</thead>
<tbody>
<tr>
<td>CBC, BMP Blood cultures, ESR</td>
</tr>
<tr>
<td>CT Maxillo-facial, neck</td>
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<table>
<thead>
<tr>
<th>TREATMENTS</th>
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<tbody>
<tr>
<td>Urgent/prompt ENT/Oral Surgeon consultation</td>
</tr>
<tr>
<td>Incision/drainage (usually in O.R.)</td>
</tr>
<tr>
<td>Parenteral antibiotics</td>
</tr>
<tr>
<td>Prepare for airway management (RSI)</td>
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</tbody>
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<thead>
<tr>
<th>TOOTH FRACTURE/AVULSION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Usually associated with some trauma</td>
</tr>
<tr>
<td>Possible injury to tooth and supporting tissues</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Classification:</th>
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<tbody>
<tr>
<td>Ellis I: minor injury, enamel only affected</td>
</tr>
<tr>
<td>Not very painful</td>
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</table>

| Ellis II: involves enamel and part of dentin |
| Increased pain |
Needs dental referral within 24 hours
Risk of infection into pulp

Ellis III: involve enamel, dentin and pulp
Dental emergencies
Immediate consultation required
High risk for infection and ischemia

ASSESSMENT
Mechanism of injury
Other signs/symptoms of associated injury
Airway/oral: swelling, bleeding, debris

TREATMENT
Oral suctioning
Calcium chloride or Zinc Oxide to cover exposed tissues
Analgesics
Antibiotics

ACUTE OTITIS EXTERNA (AOE)
“Swimmer’s Ear”
Inflammation of external auditory canal (EAC)
Swelling, drainage and maceration
Followed by pruritis, pain and swollen glands
Exudate sometimes present

Causes:
Bacterial
Fungal
Viral
Perforation
Foreign Bodies
Pseudomonas

ASSESSMENT
Ear pain, possibly radiating
Swollen lymph nodes
Tender with movement of pinna/tragus
Impaired hearing
Pruritis in ear
Low grade fever possible
Drainage may/may not be visible
Edema, tenderness and erythema of EAC
TM's normal

TREATMENT
Topical antibiotics
Topical/oral analgesics
Ear wick
Warm moist compresses

ACUTE OTITIS MEDIA
Disease of middle ear
Commonly bacterial infection
Most also have concurrent URI
Most common in infants/children
### ASSESSMENT
- Tugging/pulling at ears
- Febrile
- Irritability or malaise
- Fever/chills
- Localizing ear/facial pain

### PMHX
- Previous ear infections
- Pressurized environment
- Upper respiratory infections
- Immune status

### EXAM
- Possibly febrile
- Dehydration?
- Red/dull/bulging TM
- Possibly ruptured TM with drainage

### TREATMENT
- Analgesics
- Hydration
- Antibiotics
- Consider ENT f/u for recurrent cases
- Tympanocentesis
- Eustachian tubes

### COMPLICATIONS
- Mastoiditis
- Meningitis
- Osteomyelitis
- Facial nerve paralysis
- Intracranial abscesses

### RUPTURED TYPANIC MEMBRANE
Most caused as complication of infection
Some traumatic causes too
If secondary to increased pressure; pain usually resolves with rupture

### ASSESSMENT
- Bloody or purulent drainage or discharge
- Hearing loss
- Possible infection signs/symptoms also

### TREATMENT
- Removal of blood or debris from canal
- Analgesics (PO)
- Antibiotics (PO)
- May place cotton in EAC opening
- Referral to ENT for follow up
- Most cases heal without complications

### MENIERE’S DISEASE
- Inner ear-vestibular system disorder
- Age of onset approx 40-60’s
- Damage to hearing and balance structures
May be episodic or recurrent

Causes:
- Trauma
- Post-infection
- Allergies
- Degenerative changes

ASSESSMENT
- Vertigo
- Tinnitus
- Unilateral sensorineural loss
- Possible nystagmus
- Nausea/vomiting d/t vertigo

DIAGNOSTICS
- Labs as appropriate
- CT imaging as appropriate

TREATMENT
- Symptom management
- Antihistamines (Antivert)
- Diuretics (to reduce pressure)
- Steroids
- Anticholinergics

EDUCATION/DISPOSITION
- ENT follow up
- Driving/position change precautions
- Sedation precautions

LABRYNTHITIS
- Inflammatory response of middle ear
- Bacterial and/or viral infections
- Symptoms usually sudden and limited
- Any age affected

ASSESSMENT
- Vertigo, esp with head/body movement
- Possible nausea/vomiting
- Tinnitus or other hearing abnormalities
- Ear pressure
- Headache
- Nystagmus

PMHX
- Prior ear problems/infections
- HTN
- Neurological disorders
- Migraines
- Meniere's syndrome
- Head trauma

DIAGNOSTICS
- Labs as indicated
- CT imaging as indicated
- Dix-Hallpike maneuvers
TREATMENT
Antiemetics
Antibiotics
Sedative/anxiolytics
Primary care, ENT or Neurology referrals PRN

PERI-TONSILLAR ABSCESS
Infection extending beyond tonsillar tissue
Invades surrounding pharyngeal tissues
Usually streptococcus germs
Considered an ENT emergency
Risk for airway compromise

ASSESSMENT
Unilateral phyaryngeal/tonsillar swelling
“Hot potato” voice
Drooling possible
Severe throat pain
Possibly septic looking
Uvula displacement
Possibly dehydrated
Concurrent URI usually
Trismus

DIAGNOSTICS
Good ENT examination
Plain xray; soft tissue neck possible
Contrasted CT neck possible
Labs as indicated

TREATMENT
Airway precautions
Antibiotics (parenteral)
Analgesics
Steroids
ENT consult for possible drainage

DISPOSITION/EDUCATION
Possible admit if septic
Consider if ED can handle ENT I/D or aspiration
At home, warm saline gurgles
Sleep with HOB elevated
Ensure ENT follow up

EPIGLOTTITIS
AKA: Supraglottitis

Infection and inflammation of epiglottis, vallecula and surrounding tissues

Frequent cause: H Influenza B (HiB)
Predominately peds: age 2-7 y.o.
Abrupt onset fever, severe sore throat
Males 3:1 over females
Mortality: Peds <1%, Adults approx 7% (?)
#1 Concern: Airway obstruction

ASSESSMENT
Fever, chills  
Pain  
Dysphagia, odynophagia  
Muffled voice, cough, SOB  
Sudden onset 12-24 hours  

LOC, Anxiety, restless, irrritable  
Tripod positioning possible  
Drooling and stridor  
**DO NOT ASSESS PHARYNX - NOTHINg IN MOUTH**  

DIAGNOSTICS  
Lateral soft tissue neck  
“Thumb print sign” - epiglottic swelling  
CBC, BMP, Blood Cultures possible  
Hi-index for suspicion  

TREATMENTS  
Least invasive as possible  
Blow-by O2  
Defer IV access/lab draw until airway secure  
Airway equipment, skilled operator at ready  
Inhaled Nebulizer meds possible  
Prompt ENT consultation  
Asmission required  

CROUP/Laryngotracheobronchitis (LTB)  
Acute viral syndrome  
Swelling, inflammation of the vocal cords  
Starts with URI symptoms and progresses  
Age: 6 months to 3 years old  
Boys > Girls  
Peaks in late fall/early winter  
Typically occurs or recurs at night  
Airway obstruction is rare  

ASSESSMENT  
“Barking” cough  
Prodromal URI 1-2 days  
Low grade fever  
“Well appearing”  
No drooling  
Stridor  

DIAGNOSTICS  
Labs as indicated  
Soft tissue A/P xray neck: “Steeple sign”  

TREATMENTS  
Humidified O2/blow-by  
Pulse ox monitoring  
Steroids  
Nebulized albuterol, racemic epi  
Observation period in ED 2 hours +/-  
Antibiotics not usually indicated  
Consider admission if recurrent