

# Fever in the Adult Patient

*Summary from Rosen's By Ben Cooper*

## Epidemiology

- Generally self-limited in younger adults with mortality < 1%
- 70-90% of those > 65 are hospitalized, 7-9% mortality within one month

## Pathophysiology

- PGE2 mediated via cytokines (IL-1, IL-6, TNF, interferon) in the hypothalamus (antipyretics inhibit PGE synthesis)

## Differential Diagnosis

- The vast majority of serious causes are infectious in origin. Immediate threats to life such as septic shock, meningitis, or peritonitis should be treated empirically.
- Noninfectious Causes
  - Critical Diagnoses
    - Acute Myocardial Infarction
    - Pulmonary Embolism
    - Intracranial Hemorrhage
    - Cerebrovascular Event
    - Neuroleptic-Malignant Syndrome
    - Thyroid Storm
    - Acute Adrenal Insufficiency
    - Transfusion Reaction
    - Pulmonary Edema
  - Emergent Diagnoses
    - Congestive Heart Failure
    - Dehydration
    - Recent Seizure
    - Transplant Rejection
    - Pancreatitis
    - Deep vein thrombosis
  - Nonemergent Diagnoses
    - Drug Fever
    - Malignancy
    - Gout
    - Autoimmune Diseases

## Signs and Symptoms

- In the ED, rectal and bladder temperature are most practical and accurate. Rectal temperatures are typically 1C higher than oral temperatures
- Inconsistently, fever may be associated with tachycardia by an increase of 10 for each 0.55C (1F)
- Bradycardia may be associated with beta-blockers, drug-related fever, brucellosis, leptospirosis, rheumatic fever, Lyme disease, viral myocarditis, endocarditis

## Work-up

- Guided by physical exam
- Two most useful tests, especially in elder patients, are urinalysis and chest radiography
- Consider cultures ONLY if admitting
- Cerebrospinal fluid analysis when mental status changes, headache, or meningismus are present
- Thyroid function tests when thyroid storm is suspected
- Arterial or venous gas studies (arterial rarely provides information beyond what venous can provide)
- CT can be helpful for presumed intra-abdominal sources
- Ultrasound for potential cholecystitis

## Empiric Management

- >41C requires prompt treatment with antipyretics and possibly external cooling measures
- No evidence for improved outcome by use of antipyretics, may make patient feel better though
- Early recognition, fluids, and antibiotics for suspected sepsis

## Disposition

- Outpatient treatment for localized bacterial infections in young, otherwise healthy patients
- Admission often necessary for older patients with chronic illness
- Admit neutropenic patients, and those with unstable vital signs or life-threatening infections

# Weakness

Summary from Rosen's By William Fox

## Epidemiology

- Linked closely to epidemiology of many other disorders; majority of cases are secondary to underlying medical condition
  - Higher likelihood of weakness as a secondary symptom in patients with cardiovascular or pulmonary dysfunction, diabetes, and cancer
- CNS causes are much less common

## Pathophysiology

- Causes of global weakness include alterations in plasma volume and composition, changes in blood composition, variations in cardiac output resulting in decreased substrate delivery, and toxin exposure modifying metabolic demand, global CNS function, or mitochondrial activity
- Localized weakness more likely due to decreased substrate delivery due to hemorrhage or ischemia, aberrant inflammatory processes, in addition to neoplasms and toxin exposures

## Differential Diagnosis (with critical diagnoses in bold)

- Dehydration
- Hypo/hyperglycemia
- Electrolyte imbalance
- **Myocardial ischemia**
- **Vasodilatory shock/sepsis**
- **Organophosphate/Botulism exposure**
- Vitamin deficiency
- Cord compression (entrapment syndrome, compressive plexopathy)
- **Ischemic or hemorrhagic CVA**
- **Inflammatory demyelination (GBS)**
- **Disc herniation**
- **Cord compression (internal)**
- Tumor
- **Rhabdomyolysis**
- Alcohol
- Trauma

## Signs and Symptoms

- Specific signs and symptoms depend on underlying cause of weakness
  - General symptoms of weakness include tachycardia or tachypnea, temperature abnormalities, and hypotension
- Important to differentiate global weakness from neuromuscular weakness; global weakness can be accompanied by other signs of myocardial dysfunction such as orthopnea or other CHF symptoms
  - Weakness secondary to anemia may present with history of melena or hematochezia, blood per rectum, pallor, or vital sign abnormalities
- Weakness symptoms secondary to lesions in motor neurons depend on the location of the lesion
  - Upper motor neuron lesions can result in limb spasticity, hyperreflexia, pronator drift, and upgoing Babinski reflexes
  - Lower motor neuron lesions result in flaccidity, decreased reflexes, fasciculation, and muscle cramping
- Patterns of neuromuscular weakness often reveal location of lesion (See Rosen Figure 13-1 for full analysis of patterns)
  - Bilateral Lower extremities → Anterior cord compression/GBS/cauda equina
  - Bilateral Upper extremities → Central cord pattern → hyperextension injuries/syringomyelia
  - Bilateral All extremities, no facial involvement → Cervical cord injury
  - Bilateral Proximal extremities only → Rhabdomyolysis/polymyositis/dermatomyositis
  - Bilateral Distal extremities only → GBS/chronic peripheral neuropathy
  - Unilateral weakness in leg/hand/arm with ipsilateral face → Contralateral cerebral cortex
  - Unilateral weakness in leg/hand/arm with contralateral face → Brainstem lesion → look for CN involvement
  - Unilateral weakness in leg/hand/arm with no face → Brown-Sequard/Internal capsule or homuncular lesion
  - Unilateral weakness in one limb → spinal cord or peripheral nerve lesion
  - Unilateral facial droop → CNVII neuropathy/Bell's palsy/mastoiditis/parotitis
  - Non-CNVII neuropathy facial weakness → brainstem lesions/NMJ problems

## Work-up

- Most laboratory testing is best to exclude non-neuromuscular causes of weakness
  - EKG, troponins, lactate, hemoglobin, complete metabolic panel, CK
- Characterization of issue relies on practitioner following the weakness from the myofiber to the CNS and characterizing it as unilateral or bilateral
  - Physical exam should focus on muscle strength and reflexes of specific groups
- Imaging is crucial for new onset weakness attributed to spinal or cerebrovascular incident
  - CT, consider MRI

## Empiric Management

- For non-neurological causes of weakness, correction of the underlying issue is important
  - Suspected MIs/CVAs require appropriate, aggressive intervention
- New spinal cord weakness calls for immediate imaging
- Rhabdomyolysis, dehydration, and electrolyte imbalance are treated with fluids

## Disposition

- Depends on extent of weakness
  - Mild LMN weakness determined to be benign on work up may be discharged
  - More serious cases of weakness require more aggressive intervention, especially if airway or breathing is compromised
  - Progressive LMN or new-onset UMN weakness should be admitted for full work-up

# Cyanosis

Summary from Rosen's By William Fox

## Epidemiology

- Rare in ED, commonly seen in patients with known cardiopulmonary disease or in patients with tissue hypoperfusion

## Pathophysiology

- Seen when deoxygenated hemoglobin reaches ~5g/dL
- Can also be as a result of formation of methemoglobin due to oxidation of ferrous iron ( $\text{Fe}^{2+} \rightarrow \text{Fe}^{3+}$ )
  - Methemoglobin's  $\text{O}_2$  dissociation curve is shifted to the left, resulting in decreased oxygen delivery to the tissues
    - NADH reductase prevents the formation of methemoglobinemia
  - When methemoglobin makes up 10-25% of total hemoglobin, cyanosis results

## Differential Diagnosis

### Central cyanosis

- Decreased arterial saturation (altitude, decreased pulmonary function, V-Q mismatch, respiratory compromise)
- Anatomic shunts (Pulmonary, cerebral, hepatic, or congenital cardiac defects)
- Abnormal hemoglobin (met-/sulf-hemoglobinemia)

### Peripheral cyanosis

- Low cardiac output (Shock, LV failure, hypovolemia)
- Environmental exposure (cold)
- Arterial occlusion (thrombosis, embolism, vasospasm)
- Venous obstruction
- Redistribution of blood flow

See Box 14-2 and Figures 14-3/4 in Rosen for complete explanations

## Signs and Symptoms

- Important to note onset, duration, time, and any previous precipitating factors
  - Changes in temperature, activity level, or altitude
- Exposures to dyes or heavy metals can result in pseudocyanosis
- Any current or prior cardiac issues such as cardiovascular disease, congenital heart defects
- Symmetrical clubbing of the fingers and toes can indicate chronic hypoxemic states
- Nail bed hemorrhages and end-organ damage (specifically in the eye or kidney) can indicate thrombotic processes

## Work-up

- Complete blood count with smear, can consider D-dimer if PE is on DDX, CXR, EKG
- Pulse oximetry is not reliable, ABG testing needs specific CO-oximetry measurements
- Look for changes in condition after administration of high-flow  $\text{O}_2$ 
  - If improvement noted, diffusion impairment is likely cause
  - No improvement on  $\text{O}_2$  likely due to V-Q abnormalities and respiratory status should be reassessed (auscultation, CXR, CT)

## Empiric Management

- High-flow  $\text{O}_2$  is first line treatment, along with fluids if hypovolemia or polycythemia (goal Hct~45%) is suspected
- If methemoglobinemia is secondary to aniline dye exposure, decontamination followed by  $\text{O}_2$  and methylene blue IV 1-2mg/kg/5 min if symptomatic or methemoglobin/hemoglobin>30%
- If peripheral cyanosis, consider the following treatments
  - Raynaud's phenomenon is treated by warming affected digits and administration of vasodilatory agents
  - If no improvement with supplemental  $\text{O}_2$  and warming, consider vascular occlusion and administer heparin

## Disposition

- All primary episodes of cyanosis should be admitted for full evaluation
  - Consider cardiology consult for new onset cyanosis in children
  - Vascular surgery consult for acute arterial occlusion
- Patients with known primary pulmonary disease, vasospasm-related peripheral cyanosis, and methemoglobinemia less than 15% can be discharged after observation over several hours

# Syncope

*Summary from Rosen's By Ian Bodford*

## Epidemiology

- Prevalence is 19% in general population. 32% are admitted from ED. Persons older than 65 are 80% of those admissions.
- Risk factors are cerebrovascular disease, cardiac medications, and hypertension. Most causes are benign, especially in young adults and adolescents.

## Pathophysiology

- Transient dysfunction of bilateral cerebral hemispheres or the brainstem (RAS), usually from hypoperfusion.
- Other mechanisms include hypoglycemia, toxins, metabolic abnormalities, failure of autoregulation, and 1° neurologic derangements.

## Differential Diagnosis

- Huge array of causes. Must distinguish from other causes of apparent sudden LOC (seizures, cataplexy, etc.).
- Three categories of causes
  - Focal hypoperfusion of CNS structures → cerebrovascular disease, hyperventilation, subclavian steal, SAH, basilar artery migraine, and cerebral syncope
  - Systemic hypoperfusion resulting in CNS dysfunction → outflow obstruction, reduced cardiac output, vasomotor, carotid sinus sensitivity, miscellaneous reflexes (sneeze, postmicturition, etc.), orthostatic hypotension, anemia, & drug-induced
  - CNS dysfunction with normal cerebral perfusion → hypoglycemia, hypoxemia, seizure, narcolepsy, psychogenic, and toxic (drugs, CO, etc.)
- Must rule out life-threatening causes first, i.e. dysrhythmias, MI, stroke, etc.

## Signs and Symptoms

- Character of syncope → rate of onset, position at onset, duration, and rate of recovery
- Events preceding syncope → exertion, heat exposure, emotional state, micturition, neck stimulation, palpitations, or aura
- Events during episode → tonic-clonic movements or trauma during fall
- Associated symptoms → chest pain, dyspnea, diaphoresis, light-headedness, graying of vision, tongue biting, incontinence
- PMHx → CHF, CAD, CVA, diabetes, HTN, medications causing syncope
- Signs → orthostasis, carotid massage, rectal examination for blood

## Work-up

- EKG in all patients unless patient is adolescent with clear vasovagal origin.
- If benign dysrhythmia is suspected but not seen on EKG, can use a Holter monitor as outpatient. If severe dysrhythmia is suspected, consider echo, continuous EKG, or stress testing before discharge.
- CBC, BMP with glucose, UDS, and urine pregnancy test when warranted by history and exam. CXR and BNP if CHF suspected. CT of head only when SAH is suspected. EEG if seizure is suspected. Orthostatic vital signs should be performed but may be unreliable to assess volume status. Carotid sinus massage could potentially elicit presyncope/syncope.

## Empiric Management

- By definition, syncope is a transient event and most patients do not have symptoms in the ED. Patients with non-emergent diagnoses can be treated as outpatients.
- San Francisco Syncope Rule → outpatient disposition considered in patients without abnormal EKG findings, shortness of breath, hypotension, anemia, or history of heart failure. Safety and efficacy of this rule have not been established.
- Patients with an abnormal EKG, abnormal vital signs, recurrent syncope, or concerning associated symptoms (chest pain, hypotension, etc.) should be stabilized in the ED and admitted to the ICU or a bed with telemetry.

## Disposition

- Unfortunately, the ED evaluation of syncope is often inconclusive. Up to 50% will not have a diagnosis at discharge.
- Patients less than 45 years old without worrisome symptoms, signs, or EKG findings may be treated as outpatients.
- Discharged patients should be warned of the hazards of recurrent syncope occurring during activities such as driving or working at heights. Recurrence may be as high as 50%.

# Depressed Consciousness and Coma

Summary from Rosen's by Richard B. Moleno

## Epidemiology

- Manifests as a wide spectrum of disease, i.e. sleepiness to decreased alertness to frank coma.
- Can be categorized into metabolic/systemic, structural, and psychogenic causes.
- Most cases caused by metabolic/systemic, followed by structural and then psychogenic causes.

## Pathophysiology

- Consciousness controlled by the ascending reticular activating system (ARAS), the neuroanatomical structure responsible for arousal and cortical activation. ARAS is located in the dorsal part of the brainstem.
- Structures are vulnerable to metabolic derangements, toxins, and mechanical injury. Depressed consciousness due to cerebral cortex injury is usually due to both hemispheres being affected.
- This contrasts injury to the brainstem, which must be totally intact for arousal to be unaffected.
- ARAS becomes impaired → cerebral cortex cannot be aroused → depressed consciousness/coma occurs.

## Differential Diagnosis

- Critical Dx
  - Hemorrhage
  - Meningitis/Encephalitis/Septic Shock
  - Hypoglycemia
  - Toxic: CO, Cyanide, Heroin, other drugs
  - Cerebral Edema
  - Heat Stroke/Hypothermia
  - Anaphylaxis
  - PE
  - Cardiovascular causes
    - MI, Dissection, Tamponade, Shock
- Nonemergent Dx
  - Concussion or contusion
  - Marijuana, LSD, Mushrooms, NSAIDs
- Very unusual for ischemic stroke to cause depressed consciousness.
- Pay special attention to elderly patients, susceptible to alterations in medication dosages and drug-drug interactions and infections.
- Emergent Dx
  - Subdural/Epidural hematomas (really anything that occupies space)
  - Acute hydrocephalus
  - Venous Sinus Thrombosis
  - CNS vasculitis
  - Brain abscess
  - Hyperglycemia (DKA)
  - Nutrition def (thiamine)
  - Electrolyte abnormalities (Na, Ca, hyperammonemia)
  - Myxedema coma
  - Uremia, Thyrotoxicosis

## Signs and Symptoms

- Patients often found down, important to rely on family, friends, prehospital personnel for history and information.
- Pay special attention to the rate of symptom onset (rapid onset usually indicates structural causes whereas slow onset favors a metabolic cause), hx of trauma, exposure to drugs or toxins, new meds/dosage changes. Perform thorough neurological exam, pay particular attention to the eyes.

## Work-up

- Neuro exam
- Dolls eye reflex and cold water calorics can provide information about brainstem function.
- Bedside glucose must be done, as well as serum electrolytes, CBC, CMP, ABG, urinalysis including urine tox screen. Also can do lumbar function for CSF analysis, thyroid studies and ammonia level. Get coags before any invasive procedure.
- Imaging is important as well if you suspect trauma or structural lesions. Consider noncontrast head CT to visualize blood (initial imaging of choice), MRI to evaluate masses or edema, CT angio or venography for obstruction or shunt malfunction. Also get a CXR, Perform an EKG.

## Empiric Management

- ABCs initially. GCS < 8 u must intubate (unless cause is easily reversible, i.e. hypoglycemia, opioids).
- Coma cocktail includes dextrose, naloxone, and thiamine can quickly reverse hypoglycemia, opioid overdose and thiamine deficiency. Avoid giving glucose before thiamine, as thiamine is a cofactor for glucose metabolism and can worsen or precipitate Wernicke encephalopathy.
- If cause is suspected to be infectious, empirical administration of broad spectrum Abxs should be started as soon as possible.
- Clear signs of impending herniation → stat neurosurgery consult, initiate measures to decrease ICP → elevate head of bed, start mannitol, increase RR to achieve a pCO<sub>2</sub> ~35.

## Disposition

- Pts with structural lesions on imaging require immediate neurosurgical consultation and will frequently require surgical intervention.
- Vast majority of pts with depressed consciousness or coma require admission to the hospital for further tx and workup.
- Pts who have returned to their baseline mental status following correction of hypoglycemia or opioid overdose may be discharged from the ED following a period of observation.
- Pts with depressed consciousness secondary to alcohol or recreational drug intoxication may be discharged once clinically sober.

\*Please see figure 16-3 for algorithm for diagnostic approach to altered mental status and coma.

# Confusion

Summary from Rosen's By John R Corker

## Epidemiology

- Incidence is chronically underestimated – often incidental or secondary to the primary diagnosis.
- 2% of ED patients, 10% of hospitalized patients and 50% of elderly hospitalized patients.
- For elderly ED patients, Delirium is an independent predictor of increased mortality within 6 months of d/c.

## Pathophysiology

- Largely a disturbance in the *content of consciousness*, NOT in the level of arousal or activity, with varying severity.
  - Extreme confusion – which incorporates arousal/activity – Is termed *delirium* (hyperactive or hypoactive).
- Thought to be caused by widespread cortical dysfunction, often secondary to:
  - Substrate deficiency (hypoglycemia, hypoxemia)
  - Neurotransmitter or Circulatory dysfunction

## Differential Diagnosis

- Underlying cause often falls into 1 of 4 categories:
  - Systemic disease affecting the CNS
  - Primary intracranial disease
- Exogenous Toxins
- Drug w/d states
- Focal cortical dysfunction (stroke, tumor) rarely causes confusion, but can cause receptive/expressive aphasia.
- Subcortical/brainstem dysfunction causes a change in the level of alertness.

## Signs and Symptoms

- Most acutely confused patients do not require immediate intervention, but critical exceptions include:
  - Hypoglycemia
  - Hypoxia
- Fever
- Unstable vital signs

## Work-up

- Initial work-up should center on a complete H&P and rapid bedside assessment, including:
  - Updated Vital Signs (including Temp and O2 Sat)
  - Bedside blood glucose reading
  - From family:
    - When the patient last exhibited “normal” cognition and behavior?
    - Any recent illnesses, drug use or changes in medication?
- Patient must be protected from self-harm (medication or restraints may be required).
- Careful evaluation is necessary to differentiate between organic and psychiatric causes:
  - Oriented?
    - Quick Confusion Scale (QCS) used most commonly in the ED
  - Hallucinations?
    - Visual (delirium) vs. Auditory (psychiatric)
- Laboratory tests and diagnostic imaging are less often helpful.
  - Serum electrolytes (BMP) testing is indicated in all cases.
  - Urinalysis (UTI) and CXR (pneumonia) are common tests for suspected infection.

## Empiric Management

- Treat the underlying cause! Observe for clinical improvement, and d/c when confusion has resolved.

## Disposition

- If no identifiable or reversible cause, and confusion persists, consider admission for further work-up beyond the means of the ED.

\*\*Figures 17-3 and 17-4 on page 154 of Rosen Volume 1 (8<sup>th</sup> Ed.) represent helpful algorithms for diagnosis and management.



# Seizures

Summary from Rosen's By John R. Corker

## Epidemiology

- >10% of the United States' population will experience at least 1 seizure in their lifetime.
- 3% of EMS transports, 2% of Pediatric ED visits, and 1% of overall ED visits.
- 3% of the US population diagnosed with epilepsy, 7% of patients in ED with seizures are in status epilepticus.
- Febrile seizures occur in 2-5% of children age 6 months to 5 years.
  - 20-30% of these have at least one recurrence.

## Pathophysiology

- Abnormal increased activity of cortical initiating neurons that activate adjacent neurons, propagating the abnormal signal.
  - This abnormal neuronal activity can remain localized (focal) or propagate to subcortical structures, affecting consciousness (generalized).
- At the cellular level, pathophysiology is poorly understood.

## Differential Diagnosis

- Nonepileptic convulsions (pseudoseizures)
- Syncope
- Primary Seizure – unprovoked and not linked to an inciting event
  - *Epilepsy* – recurrent, unprovoked seizures caused by genetically determined or acquired brain disorder
- Secondary Seizure: trauma, infection, intoxication and poisoning, organ failure, metabolic disturbances (hypoglycemia most common), cerebral tumors, pregnancy, supratherapeutic levels of some AEDs

## Signs and Symptoms

- Post-ictal state (exception atonic seizures)
- Retrograde amnesia
- Incontinence (can also occur with syncope)
- Tongue laceration or buccal maceration
- Ictal events have six properties: Abrupt onset (no aura), Brief duration (90-120 seconds), LOC (except focal seizures), Purposeless activity, Unprovoked ((exceptions: fever in children and substance withdrawal in adults), Post-ictal state (*except* focal and absence seizures)

## Work-up

- Patient should be taken to a monitored area of the ED and prepped for immediate physician evaluation.
  - IV access, bedside glucose, medication review, protection from self-harm
- If patient actively seizing on arrival:
  - Secure and maintain airway (nasopharyngeal often adequate with patient on his/her side)
  - Confirm pulse, cerebral blood flow and adequate oxygenation
  - Administer benzo's (1<sup>st</sup> line therapy) if in status epilepticus
- Blood tests and EEG are generally unhelpful, although *serum sodium is always important to assess*.
  - Serum anticonvulsant levels, LFT's and tox screen only when those etiologies suspected.
- Serum anticonvulsant levels, LFT's and tox screen only when those etiologies suspected.
- CT scan indicated for (any of the following):
  - First seizure
  - Fever and/or headache
  - Acute intracranial process suspected
  - H/o acute head trauma
  - Immunocompromise
  - H/o anticoagulation
  - New focal neurological deficit
  - Age > 40 years w/o h/o epilepsy
  - Focal onset b/f generalization
  - Persistent AMS

## Empiric Management

- Reversible causative disorders should be considered first and treated.
- Acute antiepileptic therapy depends on odds of recurrence, underlying predisposing disease and medication risks.
- Chronic antiepileptic therapy should be discussed with a Neurologist at f/u after ED d/c

## Disposition

- Admit: New-onset seizures with abnormal CT or persistent focal deficits
- D/c home with early referral to Neurologist if: normal neuro exam, no significant medical comorbidities, no known structural brain disease, do not require AEDs or multiple benzos in ED, can reliably comply, given state-specific guidelines re driving and other potentially dangerous activities

# Dizziness and Vertigo

Summary from Rosen's By Lindsay Schroeder

## Epidemiology

- 7.5 million people in ambulatory settings annually, 2.5% of ED visits

## Pathophysiology

- collaboration of functions of visual, proprioceptive (muscles and joints), and vestibular (otic labyrinths)
- vestibular apparatus- 3 semicircular canals (movement and angular momentum) plus the utricle (position relative to gravity) and saccule in the inner ear/ petrous temporal bone
  - impulses go via the vestibular part of CN VIII → enter the brain stem just below the pons/anterior to the cerebellum → 4 vestibular nuclei in the brainstem then cerebellum → medially longitudinal fasciculus (MLF) and vestibulospinal tract → motor neurons in the extremities
- vertigo- inner ear disease causing asymmetric signals from the bilateral semicircular canals
- nystagmus- unbalanced vestibular signals result in asymmetrical stimulation of the medial and lateral rectus, if vestibular most often unidirectional and horizontal (cerebral or brain stem lesion if vertical)
  - slow- towards the side of the stimulus
  - fast- cortical correction to midline, this direction is used for naming
- vestibular nuclei connected to the autonomic system explaining the associated perspiration, nausea, vomiting

## Differential Diagnosis

- peripheral vs central vs systemic causes (see box and tables 19-1 and 19-3 for examples and characteristics) but generally central causes are less common but more serious than peripheral
  - most common peripheral causes of true vertigo: BPPV, vestibular neuritis, labyrinthitis, and Ménière's disease
  - acute suppurative labyrinthitis is the only peripheral cause that needs urgent intervention, rest are benign
  - don't forget that posterior circulation stroke/bleed can cause vertigo
  - vestibulotoxicity-aminoglycosides, anticonvulsants, alcohols, quinine, quinidine, and minocycline
- sometimes dizziness is a metaphor for malaise (ddx: anemia, infection, depression)

## Signs and Symptoms

- vertigo- patient spinning vs room spinning
- near syncope- feeling faint or lightheaded
- disequilibrium- unsteady gait
- nonspecific dizziness- related to anxiety
- hypotension, carotid bruit, asymmetric pulse/BP in upper extremities, serous otitis media, pupillary abnormalities, papilledema, CN VI palsy, intranuclear ophthalmoplegia, nystagmus
- table 19-2 details distinguishing features of nystagmus to differentiate between central and peripheral lesions

## Work-up

- complete neuro exam
- Dix-Hallpike testing- dx posterior canal BPPV
- roll test- dx horizontal canal BPPV
- head thrust/impulse test- dx vestibular neuritis and labyrinthitis
- fingerstick glucose if history of DM
- CBC, BMP, EKG if concern is near syncope rather than vertigo
- if stroke risk factors or nystagmus suggestive of central lesion- head CT(A) or MRI(A)

## Empiric Management

- if positive Dix-Hallpike, treat with Epley maneuver (figure 19-4)
- if positive roll test, treat with bbq roll maneuver (page 168)
- stop offending medications
- start new med if cause warrants (meclizine, antiemetics, migraine tx, steroids, antibiotic...)

## Disposition

- discharge if: symptoms resolve with peripheral cause
- admit if: cannot walk, intractable symptoms, concern for cerebellar hemorrhage or infarction, vertebrobasilar insufficiency, acute bacterial labyrinthitis
- primary care follow up, consider ENT or neurology follow up if suspected acoustic neuroma or perilymphatic fistula

# Headache

Summary from Rosen's By Benjamin Trevias

## Epidemiology

- ED Presentation: Tension headache (50%), Headache of unidentified origin (30%), Migraine-type pain (10%), and headaches from other potentially serious causes (8%)
- The most commonly encountered life-threatening cause of severe sudden head pain is subarachnoid hemorrhage (SAH)
  - It is estimated that 25 to 50% of SAH are missed on the first presentation to a physician.

## Pathophysiology

- Brain parenchyma is insensitive to pain. Much of the pain is mediated by the 5th CN.
- Pain-sensitive areas of the head include the meninges, the blood vessels, and tissues lining the cavities within the skull.

## Differential Diagnosis

- Critical Diagnoses
  - Subarachnoid Hemorrhage
  - Carbon Monoxide Poisoning
  - Temporal Arteritis
  - Bacterial Meningitis
  - Encephalitis
- Emergent Diagnoses
  - Shunt Failure
  - Tumor/Mass
  - Subdural Hematoma
  - Mountain Sickness
- Emergent Diagnoses continued
  - Glaucoma
  - Brain Abscess
  - Hypertensive Crisis
- Nonemergent Diagnoses
  - Migraine
  - Trigeminal Neuralgia
  - Post-lumbar puncture
  - Dental, TMJ
  - Tension
  - Febrile headaches

## Signs and Symptoms

- Pattern/Onset- suddenness of onset/"worst ever" warrants a consideration of SAH. Onset on exertion.
- Location- Unilateral pain is more suggestive of migraine/localized inflammatory process in the skull. Occipital h/a's are associated with HTN. Temporal arteries, TMJ, dental infxns, sinus infxns are highly localized. Meningitis, encephalitis, SAH and severe migraine are more diffuse.
- Exacerbating/Alleviation- H/A's that come and go with the environment (Carbon Monoxide). H/A's on awakening are typical with brain tumors.
- Associated sx's/Risk Factors- Nausea and vomiting are nonspecific. Immunocompromised pt's can be at risk for unusual infectious causes.
- Prior History- Important to know if pt had prior work up for severe disease. Migraine, tension, and cluster tend to recur.
- General Appearance – Altered mental status, severe nausea/vomiting
- Vital Signs- HTN with normal HR/bradycardia, tachycardia, unexplained fever
- HEENT- Tender temporal arteries, meningismus
- Fundi-loss of venous pulsations or presence of papilledema- Increased CSF, subhyaloid hemorrhage, acute red eye (severe ciliary flushing) and poorly reactive pupils, enlarged pupil with 3rd nerve palsy
- Neurologic- Lateralized motor/sensory deficit, acute cerebellar ataxia

## Work-up

- Brain CT can miss 6 to 8% of patients with SAH, especially patients with minor (grade I) SAH, who are most treatable. The sensitivity of CT for identifying SAH is reduced by nearly 10% for symptom onset greater than 12 hours and by almost 20% at 3 to 5 days.
- Abnormal mental status, signs of increased intracranial pressure, papilledema, focal findings on neurologic examination, or any other indication suggestive of a focal intracranial mass lesion requires CT before lumbar puncture.

## Empiric Management

- Mild-Mod: Oral NSAID. If you suspect intracranial infections; empiric antibiotics before CSF.
- Opioids generally are not first-line management for any type of headache pain, except when ICH (including SAH) is thought to be present, conditions for which opioid analgesia is effective and beneficial.

## Disposition

- Appropriate analgesia and follow-up, unless evaluation has determined a serious underlying condition

# Diplopia

*Summary from Rosen's By Andrew Fredericks*

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## Epidemiology

- Represents 1.4% of ophthalmologic emergencies
- Majority are binocular w/ Cranial Nerve palsies being most common cause

## Pathophysiology

- Monocular diplopia: double visions that persists when the other eye is closed is related to distortions in the light path
- Binocular diplopia: double vision that results when either eye is closed is the result of misalignment in the visual axes with many causes—CNs, intra/supranuclear lesions in brainstem or above

## Differential Diagnosis

- Critical: Basilar artery thrombosis, Botulism, Basilar meningitis, Aneurysm
- Emergent: Vertebral dissection, Myasthenia Gravis, Wernicke's encephalopathy, Orbital apex syndrome
- Urgent: Brainstem tumor, Miller-Fisher syndrome, Multiple sclerosis, Thyroid myopathy, Ophthalmoplegic migraine, Ischemic neuropathy, Orbital myositis, pseudotumor, Orbital apex mass

## Signs and Symptoms

- Double Vision
- Monocular: diplopia resolves when pinhole is used, persists in affected eye when normal eye closed
- Restrictive, mechanical orbitopathy: gradual onset, sensations of mass effect/pain, fever if infectious, Graves' suggested w/ worse diplopia in morning, proptosis swelling, edema, hyperemia, palpebral swelling, abruptly restrict eye movement away from the muscle
- Neuroaxial process: can be sudden, vertical diplopia w/out vertical skew deviation suggest brainstem lesion
- Neuromuscular disorder: atrophy or weakness, weakness on forced eyelid closure normal reflexes and no sensory deficits

## Work-up

- Neuro Exam w/ special attention to CNs,
- Pupillary and facial examination for signs of pupillary asymmetry, ptosis, lid lag, conjunctival injection or chemosis, periorbital swelling, or proptosis and assessment of overall head positioning
- Use questions below to narrow down dx:
  1. Is the diplopia monocular?
  2. Is the binocular diplopia a result of a restrictive, mechanical orbitopathy?
  3. Is the binocular diplopia a result of a palsy of the oculomotor CNs (III, IV, or VI) in a single eye?
  4. Is the binocular diplopia a result of a neuroaxial process involving the brainstem and related CNs?
  5. Is the binocular diplopia a result of a neuromuscular disorder?
- Monocular: Slit Lamp, Optho consult/referral
- Neuromuscular process: Ice Test, Endrophonium challenge
- Restrictive mechanical orbitopathy (myositis, tumor, orbital inflammatory,): Contrast-Enhanced MRI or CT of Orbits
- Isolated CN palsies: MRA/CTA/DSA brain, Contrast MRI/CT of orbits +/- Brain
- Consider LP for meningitis and Miller-Fisher Syndrome

## Empiric Management

- Little primary treatment beyond addressing the primary disorder
- Signs of stroke: IV Fluid Bolus, Stroke evaluation
- Signs of infection: empiric abx pending CT, LP and confirmation of infection
- Signs of Wernicke's: Administer Thiamine

## Disposition

- Typically requires admission for further w/u and tx of underlying disorder
- CN II and CN VI palsy from microvascular ischemia is generally self-limited w/ complete resolution in a few days in 95% of patients. The can be discharged home with close output f/u

# Red and Painful Eye

Summary from Rosen's By Gray Millsap

## Epidemiology

- Nontraumatic ophthalmologic conditions (glaucoma, PVD leading to retinal ischemia) become more common as age increases.
- Traumatic eye injuries are the leading cause of visual impairment and blindness in the United States.

## Pathophysiology

- Redness is caused by dilation of blood vessels in the conjunctiva, sclera, and retina.
- Ocular pain originates from irritation or inflammation in the conjunctiva, cornea, iris, or vasculature.

## Differential Diagnosis

- Critical Diagnoses
  - Caustic injury (alkaline worse than acidic)
  - Acute angle-closure glaucoma
  - Retrobulbar hematoma (often from trauma)
- Emergent Diagnoses
  - Keratitis (inflammation of the cornea)
  - Anterior uveitis
  - Scleritis
  - Endophthalmitis
- Urgent Diagnoses
  - Penetrating ocular trauma
  - Spontaneous or trauma hyphema

## Signs and Symptoms

- Important to differentiate between itching, burning, dull pain, sharp pain, and sensation of a foreign body as it guides the diagnostic algorithm (Figure 22-10).
- Important findings associated with a more severe diagnosis in patients with red and painful eye: 1) severe ocular pain 2) persistent blurred vision 3) reduced ocular light reflection 4) proptosis 5) corneal epithelial defect or opacity 6) limbal injection 7) unreactive pupils 8) wearer of soft contacts 9) neonate 10) worsening signs s/p 3 days of pharmacologic treatment 11) immunocompromised host.
- Headache and nausea are common signs in acute angle-closure glaucoma.
- Complete eye physical exam includes visual acuity, visual field testing, external exam, extraocular muscle function, pupillary evaluation, pressure determination, slit-lamp, and fundoscopic exam.

## Work-up

- Application of a fluorescein solution will often identify damage to corneal epithelium (corneal ulcers, corneal abrasions).
- A dendritic pattern on the cornea is often present with a herpetic infection.
- Relief of eye pain with topical anesthetic can help you determine origin of pathology. Complete abolition of pain suggests a corneal lesion, modest relief suggests conjunctival process, and no relief suggests an intraocular origin.
- Elevated CRP and ESR are common in temporal arteritis.
- When an infectious process is likely, CBC and cultures are unnecessary in the ED.
- U/S is more sensitive than CT for detecting intraocular foreign bodies.

## Empiric Management

- Quick and prolonged irrigation of the eye with normal saline is strongly recommended for caustic injuries of the eye. For alkalis, irrigation should be a minimum of 4 liters and 40 minutes; for acids, 2 liters and 20 minutes.
- Retrobulbar hematoma – if IOP > 30mmHg, may require lateral canthotomy and inferior cantholysis in ED.
- Acute angle-closure glaucoma – ↓ production of aqueous humor pharmacologically and consult Ophtho if IOP > 20mmHg
- Pain relief with topical anesthetic may help with patient cooperation with an anterior eye injury, but may interfere with a complete eye assessment.
- With proven bacterial conjunctivitis, a topical broad-spectrum abx has shown benefit (trimethoprim + polymyxin B).

## Disposition

- Vast majority of patients with eye complaints are discharged and asked to follow-up in 1-2 days w/ ophthalmologist.
- Reasons for admission: procedural intervention, parenteral antibiotics, intractable pain, and further diagnostic evaluation.  
\*\*\* Please see Table 22-2 for management and disposition considerations for various diagnoses causing a red and painful eye.

# Sore Throat

*Summary from Rosen's By Brandon Morshedi*

## Epidemiology

- Over 2 million visits per year to ED and ambulatory care centers for sore throat or “throat-related” complaints

## Pathophysiology

- Inflammation of the soft tissues of the nasopharynx, oropharynx (most common) or hypopharynx
- Regional infections, both viral and bacterial, trigger inflammatory changes in lymphatic tissues within Waldeyer’s Ring (i.e. pharyngeal, tubal, palatine, and lingual tonsils)

## Differential Diagnosis

- Infectious
  - Viral (i.e. rhinovirus, adenovirus, coronavirus, HSV 1/2, Influenza A/B, Parainfluenza, CMV, EBV, VZV, hepatitis)
  - Common Aerobes (i.e. Strep pyogenes, Peptostreptococcus species, non-GAS, Neisseria gonorrhoeae, Neisseria meningitidis, Mycoplasma pneumonia, Arcanobacterium haemolyticum, Chlamydia trachomatis, Staph aureus)
  - Uncommon Aerobes (i.e. Haemophilus influenzae, Haemophilus parainfluenzae, Corynebacterium diphtheria, Streptococcus pneumonia, Yersinia enterocolitica, Treponema pallidum, Francisella tularensis, Legionella pneumophila, Mycobacterium species)
  - Anaerobes (i.e. Bacteroides, Peptococcus, Clostridium, Fusobacterium, Prevotella)
  - Other (i.e. Candida species)
- Noninfectious
  - Systemic (i.e. Kawasaki disease, SJS, cyclic neutropenia, thyroiditis, connective tissue disease)
  - Trauma/Misc (i.e. penetrating injury, angioneurotic edema, retained foreign body, anomalous aortic arch, laryngeal fracture, calcific retropharyngeal tendinitis, retropharyngeal hematoma, caustic exposure)
  - Tumor (i.e. tongue, larynx, thyroid, leukemia)

## Signs and Symptoms

- Most commonly erythema and edema of pharynx, dysphagia, andodynophagia, with or without petechiae or exudates
- Severe cases: difficulty breathing, muffled voice, sensation of tightness in the throat, drooling, stridor, signs of dehydration, and the sniffing position

## Work-up

- Assessment of the airway and patient’s general appearance, direct visualization of the pharynx and pharyngeal structures.
- If unable to visualize source, and symptoms are severe, consider nasopharyngoscopy or laryngoscopy with setup for rescue airway (i.e. cricothyrotomy, due to risk of laryngospasm) to evaluate for infectious or structural causes of obstruction.
- If patient is stable and there is concern for epiglottitis, consider plain film for screening, looking for “thumb sign”.
- Consider CT in stable patients with secure airway. Plain film sensitive for disease.
- HIV Status
- Centor criteria for GAS (history of fever, tonsillar exudates, tender anterior cervical adenopathy, absence of cough).
- Consider rapid strep test or Monospot test if clinically relevant.
- Symmetrical distribution of tonsillar erythema or exudates without airway involvement suggests acute tonsillitis
- Unilateral swelling and contralateral uvular deviation suggests peritonsillar abscess
- Involvement of entire oropharyngeal area suggests pharyngitis
- Significant symptoms with no clear oropharyngeal pathology on exam suggests hypopharynx etiology, especially epiglottitis

## Empiric Management

- Manage airway compromise or impending airway compromise first.
- Titrate analgesic meds (i.e. topical anesthetic sprays, APAP, NSAIDS, opioids). Consider corticosteroids.
- In setting of clinical pharyngitis, a fluctuant unilateral peritonsillar mass should be drained when possible.
- Antibiotics in cases of unilateral swelling and redness that appears not to be fluctuant (i.e. “peritonsillar cellulitis)
- Patients with severe, systemic illness should receive antibiotic coverage for streptococcal and anaerobic bacteria
- Acute pharyngitis should not typically be treated with antibiotics as the majority of cases are viral in origin, and suppurative complications following strep infections are both easily treated and rare in industrialized nations (i.e. use antibiotics in endemic setting of rheumatic fever). Additionally, the use of antibiotics has not been shown to be superior to NSAIDS in reducing pain. However, guidelines in the U.S. still recommend a combination of clinical assessment and bacteriologic testing with the goal of treating with antibiotics when GAS is identified or strongly suspected.
- Educate patients on 1) the self-limited nature of infectious pharyngitis, 2) the lack of symptomatic benefit with antibiotics, and 3) the potential harm of antibiotics (individual and population resistance, fungal infections in women, rashes, GI effects, recurrence of pharyngitis, and occasionally dangerous allergic reactions).

## Disposition

- With mild symptoms of pharyngitis and no airway compromise, provide symptomatic treatment and education, d/c with f/u as needed. Consider antibiotics only if high likelihood of GABHS in endemic, epidemic setting of rheumatic fever.
- If moderate illness or unable to determine source, admit and consider advanced imaging and/or empiric steroids and antibiotics.
- If severe illness and signs of airway compromise, provide disease-focused therapy and admit. Consider nasopharyngoscopy and/or advanced airway management if needed. Provide empiric steroids and antibiotic coverage.

# Hemoptysis

*Summary from Rosen's By Brandon Morshedi*

## Epidemiology

- Most cases of hemoptysis in the ED are small-volume, usually from blood-tinged sputum or minute amounts of frank blood.
- Only 1-5% of hemoptysis patients have “massive or life-threatening” hemorrhage, defined as 100-600mL of blood loss in any 24-hour period, resulting in up to 80% mortality.

## Pathophysiology

- Trace hemoptysis occurs via disruption of tracheobronchial capillaries from vigorous coughing or minor bronchial infections.
- Massive hemoptysis occurs via disruption of pulmonary or bronchial arteries. The bronchial arteries are small-caliber and high-pressured and are the culprit in 90% of massive hemoptysis.

## Differential Diagnosis

- Airway Disease (i.e. bronchitis, bronchiectasis, neoplasm, trauma, foreign body)
- Parenchymal Disease (i.e. TB, pneumonia, lung abscess, fungal infection)
- Vascular Disease (i.e. PE, AVM, aortic aneurysm, pulmonary HTN, vasculitis)
- Hematologic Disease (i.e. coagulopathy, DIC, platelet dysfunction, thrombocytopenia)
- Cardiac Disease (i.e. congenital heart disease, valvular heart disease, endocarditis)
- Miscellaneous (i.e. cocaine, post-procedural injury, tracheal-arterial fistula, SLE)

## Signs and Symptoms

- Ranging from blood-tinged sputum to massive amounts of frank blood from the oropharynx
- Patient may be either coughing up blood (hemoptysis) or vomiting blood (consider GI sources of bleeding)

## Work-up

- Primary survey and stabilization
- Remainder of work-up guided by physical exam findings
  - Focal adventitious breath sounds = pneumonia or lung abscess
  - New heart murmur, especially in a febrile patient = infectious endocarditis with septic PE
  - Signs and symptoms of a DVT = pulmonary embolus
  - Ecchymoses and petechiae = coagulopathy and thrombocytopenia, respectively
- CBC, coagulation studies, type and screen or crossmatch, renal function tests if vasculitis suggested or CT planned
- Plain film CXR has poor sensitivity, but can identify sources of infection or malignancy
- CT chest in high-risk patients (smokers, oncology patients), or in pt with moderate to severe bleeding even if the initial CXR is negative. Do not delay CT scan to obtain a CXR.
- Angiography is first-line study when cause of hemoptysis is known (e.g. malignancy) and embolization is contemplated

## Empiric Management

- Primary survey, including airway management and maintenance of hemodynamic instability
- Further management determined by CXR or CT results
- Bronchoscopy may benefit stable pts with mild to moderate hemoptysis by facilitating airway mgmt and allowing for balloon and topical hemostatic tamponade, thermocoagulation, and injection of vasoactive agents
- Bronchial arterial embolization is a first-line therapy for pts with massive hemoptysis who are unable to tolerate surgery and for whom bronchoscopy has been unsuccessful
- Emergency thoracotomy for life-threatening bleeding uncontrolled by bronchoscopy or percutaneous embolization

## Disposition

- Healthy patients with minor bleeding and resolution of symptoms and stable vital signs → d/c with outpatient f/u
- High-risk patients with minor bleeding OR all patients with moderate to large bleeding → CT → admit for obs → consider bronchoscopy
- All patients with massive hemoptysis → admit ICU



# Dyspnea

Summary from Rosen's By Ashley Phipps

## Epidemiology

- Very common presenting sx, occurs in all ages, causes range from benign to life-threatening

## Pathophysiology

- Actual mechanism unknown
- Normal breathing controlled centrally by respiratory center in medulla oblongata and peripherally by chemoreceptors near carotid bodies and mechanoreceptors in diaphragm/skeletal muscles
- Sensation of dyspnea thought to be 2/2 any imbalance leading to  $\uparrow$  ventilatory demand  $>$  capacity,  $\uparrow$  WOB, and  $\uparrow$  respiratory drive

## Differential Diagnosis

- Extensive differential with many pulmonary and non-pulmonary causes:
- Critical
  - Airway obstruction
  - PE
  - Noncardiogenic edema
  - Anaphylaxis
  - Ventilatory failure
  - MI
  - Cardiac tamponade
  - Toxins (CO & organophosphates)
  - DKA
  - Epiglottitis
  - Tension pneumothorax
  - Flail chest
  - Acute chest syndrome
  - CVA, intracranial insult
- Emergent
  - Spontaneous pneumothorax
  - Asthma
  - Cor pulmonale
  - Aspiration
  - Severe pneumonia
  - Pericarditis
  - Anemia
  - Pneumothorax or hemothorax
  - Diaphragmatic rupture
  - Renal failure
  - Electrolyte abnls/met acidosis
  - Sepsis
  - Bowel obstruction
  - Hypotension
  - MS, Guillain-Barre, Tick paralysis
- Non-emergent
  - COPD
  - Pleural effusion
  - Neoplasm
  - Pneumonia
  - Congenital heart dz
  - Valvular heart dz
  - Cardiomyopathy
  - Pregnancy
  - Ascites, Obesity
  - Somatization disorder
  - Hyperventilation syndrome
  - Panic attack
  - Thyroid dz
  - Fever
  - Rib fractures
  - ALS, porphyria, polymyositis

## Signs and Symptoms

- Patient descriptions of dyspnea vary greatly, consider duration, onset, and positional changes including orthopnea and paroxysmal nocturnal dyspnea
  - Dyspnea may be associated with exertion, trauma, fever, cough, sputum production, hemoptysis, anxiety, chest pain, diaphoresis, and nausea/vomiting
- Key physical exam findings are tachypnea, cyanosis, tachycardia, hypotension, fever, AMS, obesity, barrel chest, "tripod" positioning, clubbing, pale skin/conjunctiva, stridor, JVD, retractions, wheezes/rales, decreased breath sounds, friction rub, subcutaneous emphysema, cardiac murmurs or gallops, muffled heart sounds, unilateral leg edema, focal neuro deficits, and diffuse weakness

## Work-up

- All patients should be placed on a pulse oximetry monitor to determine if hypoxia is present
- Most patients should be placed on noninvasive waveform capnography, cardiac monitors, and have an EKG performed
- Depending on the severity and most likely cause other studies may be warranted such as an ABG, serum electrolytes, CBC, bedside ultrasound, CXR, cardiac markers, D-dimer, BNP, CT angiography, and duplex venous ultrasound

## Empiric Management

- Supplemental oxygen and assisted ventilation as necessary
- Critical diagnoses should be stabilized immediately including decompression of the chest for a tension pneumothorax, intubation if patient's airway or ability to protect it is compromised, parenteral epinephrine if significant wheezing or anaphylaxis, continuous beta-agonist nebs and steroids if severe asthma exacerbation

## Disposition

- Critical or unstable patients will require admission to ICU
  - Emergent patients may require admission to an intermediate care unit versus an observation unit depending on their improvement with ED management and their risk of deterioration
  - For most non-emergent patients, the symptoms will abate and they can be discharged home with medical follow-up
- \*\* Figure 25-1 and 25-2 in Rosen's volume 1, 8<sup>th</sup> edition, pages 211-212 show diagnostic and management algorithms

# Chest Pain

Summary from Rosen's By Liang Liu

## Epidemiology

- Chest pain accounts for approximately 6 million patients visits to the ED annually, that is 9% of all patients seen in the ED
- A number of risk factors are associated with increased probability of certain life-threatening pathologies
  - Acute coronary syndrome – family or personal history of CAD, age diabetes mellitus, hypertension, smoking, dyslipidemia, sedentary lifestyle, obesity, cocaine abuse
  - Pulmonary embolism – prolonged immobilization, recent history of surgery, prior VTE, pregnancy, oral contraceptive use with smoking, congestive heart failure, COPD, obesity, and history of hypercoagulability
  - Aortic dissection – HTN, congenital disease of the aorta or aortic valve, inflammatory aortic disease, connective tissue disease, pregnancy, arteriosclerosis, smoking
  - Pericarditis or myocarditis – infection, autoimmune disease, acute rheumatic fever, recent MI or cardiac surgery, malignancy, radiation therapy to mediastinum, uremia, drugs, and prior pericarditis
  - Pneumothorax – prior pneumothorax, Valsalva's maneuver, chronic lung disease, smoking

## Pathophysiology

- Afferent visceral fibers from heart, lungs, great vessels, and esophagus enter the same thoracic dorsal ganglia → nonspecific location of pain
- Dorsal segments overlap three segments above and below a level → Disease from the thoracic region can result in pain anywhere from the jaw to the epigastrium
- Somatic afferent fibers synapse in the same dorsal root ganglia as the thoracic viscera → Radiation of pain

## Differential Diagnosis

- Wide variety of causes including cardiovascular, pulmonary, GI, musculoskeletal, and neurologic
- Critical diagnosis – Acute myocardial infarction (MI), acute coronary ischemia, aortic dissection, cardiac tamponade, pulmonary embolus, tension pneumothorax, esophageal rupture
- Emergent diagnosis – Unstable angina, coronary spasm, Prinzmetal's angina, cocaine-induced pericarditis or myocarditis, pneumothorax, mediastinitis, esophageal tear (Mallory-Weiss), cholecystitis, pancreatitis
- Nonemergent diagnosis – Valvular heart disease, aortic stenosis, mitral valve prolapse hypertrophic cardiomyopathy, pneumonia, pleuritis, tumor, pneumomediastinum, esophageal spasm, esophageal reflux, peptic ulcer, biliary colic, muscle strain, rib fracture, arthritis, costochondritis, spinal root compression, herpes zoster, postherpetic neuralgia, etc.

## Signs and Symptoms \*

- Findings on history
  - Severe, crushing, pressure, substernal, exertional, radiation to jaw, neck, shoulder, arm – acute MI, coronary ischemia, unstable angina, coronary spasm
  - Tearing, severe, radiating to or located in back, maximum at onset, migrates to neck – aortic dissection
  - Pleuritic – esophageal rupture, pneumothorax, cholecystitis, pericarditis, myocarditis
  - Indigestion or burning – ACS, esophageal rupture, esophageal tear, cholecystitis
  - Sudden onset, unilateral chest pain, associated with respiratory distress – pneumothorax
  - Associated symptoms
    - Hemoptysis – pulmonary embolism
    - Near-syncope and syncope – aortic dissection, PE, acute MI, pericarditis, myocarditis
    - Dyspnea – ACS, pulmonary embolism, pneumothorax, pericarditis
    - Nausea and vomiting – esophageal rupture, ACS, esophageal tear, cholecystitis
- Physical exam findings
  - Pulmonary embolism – Dyspnea, diaphoresis, tachycardia, fever, hypoxemia, JVD, pleural rub, unilateral leg swelling/warmth/pain/tenderness /erythema
  - Pneumothorax – Dyspnea, unilateral diminished/absent breath sounds, subcutaneous emphysema
    - Tension Pneumothorax – hypotension, tachycardia, hypoxemia, JVD
  - ACS – Dyspnea, diaphoresis, new murmur, S3/S4 gallop, JVD, rales
  - Pericarditis – Hypotension, tachycardia, fever, narrow pulse pressure (with effusion), pericardial rub, JVD
  - Myocarditis – Hypotension, tachycardia, fever
  - Mediastinitis – Tachycardia, Hamman's sign, subcutaneous emphysema
  - Aortic dissection – Diaphoresis, hypertension (early), hypotension (late) , tachycardia, significant difference in upper extremity blood pressures, new murmur, focal neurologic findings
  - Esophageal rupture – Diaphoresis, hypotension, tachycardia, fever, Hamman's sign, subcutaneous emphysema, epigastric tenderness
  - Esophageal tear (Mallory-Weiss) – Tachycardia, epigastric tenderness

## Work-up \*

- Obtain 12-lead electrocardiography (ECG) – should be performed w/i 10 minutes of arrival
  - Classic MI – ST segment elevation (>1 mm) in contiguous leads, new LBBB, Q waves > 0.04 sec duration
  - Subendocardial infarction – T wave inversion or ST segment depression in concordant leads
  - Pericarditis – Diffuse ST segment elevation, PR segment depression
  - Pulmonary embolism – Right ventricular strain pattern
  - Pericardial effusion/tamponade – Low voltage on ECG
- Chest radiograph (CXR) – indicated in any patient with chest pain
  - Wide mediastinum or ill-defined aortic knob – acute aortic dissection
  - Pleural effusion, subcutaneous air, mediastinal air-fluid level – esophageal rupture
  - Increased cardiac silhouette – pericarditis, cardiomyopathy
  - Pneumomediastinum – esophageal rupture, mediastinitis
- Other imaging: CT, pulmonary angiography, VQ scan, cardiac ultrasound, transesophageal echocardiogram, MRI
- Laboratory testing: Elevation in troponins (I and T) identify patients with ACS with highest risk for adverse outcome
  - CK-MB is more specific for cardiac ischemia than CK but Is still considered secondary to troponins

## Empiric Management

- ACS – Initial management with oxygen therapy, aspirin, nitroglycerin
  - If STEMI – heparin or LMWH, IV nitroglycerin, beta blocker, revascularization via fibrinolysis or GP IIb/IIIa inhibitor + percutaneous coronary intervention (PCI)
- Aortic dissection – Beta blockade, IV antihypertensive therapy, decrease contractility, immediate surgical consult
- Pulmonary embolism – IV heparin or SQ LMWH, thrombolysis if severe cardiovascular instability
- Tension pneumothorax – Needle decompression or tube thoracostomy
- Esophageal rupture – IV fluid resuscitation, analgesia, IV antibiotics, early surgical consultation
- Pericarditis – US for effusion or tamponade risk, NSAIDs, corticosteroids, cardiology consultation

## Disposition

- Will vary greatly based on cause of chest pain
  - Surgical consultation – aortic dissection, esophageal rupture
  - Admission with cardiology consult – myocardial ischemia, pericarditis, myocarditis, etc.
  - Discharge with follow up – musculoskeletal causes, stable angina, low risk chest pain

\*See Table 26-6 for further discussion on signs/symptoms and workup on potentially life-threatening causes of chest pain

# Abdominal Pain

Summary from Rosen's By Kevan Beth Meadors

## Epidemiology

- Accounts for 10% of all ED visits, and <40% receive the diagnosis of nonspecific abdominal pain
- Elderly patients (>65yo), immunocompromised patients and women of reproductive age deserve special consideration
- Immunocompromised patients are often worked up in the ED and require a broad differential diagnosis due to misleading labs and highly variable presentations

## Pathophysiology

- Most common sources of pain perceived are the GI and GU tracts
- Pain derived from one of three distinct pain pathways: visceral, somatic, referred
  - Visceral: results from autonomic nerve stimulation, usually earliest manifestation of disease, poorly characterized and hard to localize
  - Somatic: occurs with irritation of the parietal peritoneum; usually caused by infection, chemical irritation or other inflammatory processes; conducted by peripheral nerves; better localization than visceral pain; often intense and constant
  - Referred: pain felt at a distance from its source, makes localization difficult, both visceral and somatic can manifest as referred

## Differential Diagnosis

- Intraabdominopelvic causes (intraperitoneal, retroperitoneal, pelvic)—ex: appendicitis, cholecystitis, pancreatitis
- Extraabdominopelvic causes—ex: pneumonia, MI, ketoacidosis, toxicologic
- RUQ Pain
  - Biliary colic
  - Cholecystitis
  - Gastritis
  - GERD
  - Pancreatitis
  - Hepatic Abscess
  - Acute Hepatitis
  - Hepatomegaly (due to CHF)
  - Perforated Ulcer
  - Retrocecal Appendicitis
  - Myocardial Ischemia
  - Appendicitis in pregnancy
  - RLL Pneumonia
- RLQ Pain
  - Appendicitis
  - Meckel's Diverticulitis
  - Cecal Diverticulitis
  - Aortic Aneurysm
  - Ectopic Pregnancy
  - Ovarian Cyst
  - PID
  - Endometriosis
  - Ureteral Calculi
  - Psoas Abscess
  - Mesenteric Adenitis
  - Incarcerated/ Strangulated Hernia
  - Ovarian Torsion
  - Tubo-ovarian Abscess
  - UTI
- LUQ Pain
  - Gastritis
  - Pancreatitis
  - GERD
  - Splenic Pathology
  - Myocardial Ischemia
  - Pericarditis
  - Myocarditis
  - LLL Pneumonia
  - Pleural Effusion
- LLQ Pain
  - Aortic Aneurysm
  - Sigmoid Diverticulitis
  - Incarcerated/ Strangulated Hernia
  - Ectopic Pregnancy
  - Ovarian Torsion
  - Mittelschmerz
  - Ovarian Cyst
  - PID
  - Endometriosis
  - Tubo-ovarian Abscess
  - Ureteral Calculi
  - Psoas Abscess
  - UTI
- Diffuse Pain
  - Peritonitis
  - Pancreatitis
  - Sickle Cell Crisis
  - Early Appendicitis
  - Mesenteric Thrombosis
  - Gastroenteritis
  - AAA
  - Intestinal Obstruction
  - Diabetes Mellitus
  - IBD
  - Irritable Bowel

## Signs and Symptoms

- Perform a focused history, asking high yield questions
- Abrupt onset usually represents a more serious case
- Surgical causes often manifest with pain first, then N/V, and are rarely diffuse
- Classic pain descriptions:
  - Bowel Obstruction—diffuse, severe, colicky
  - Mesenteric Ischemia—"pain out of proportion to exam"
  - Pancreatitis—pain radiating from epigastrium straight through to midback
  - Splenic pathology, Diaphragmatic irritation, or Free intraperitoneal fluid—pain radiating to left shoulder

- Perforated gastric/duodenal ulcer, ruptured aortic aneurysm or ectopic pregnancy—pain associated with syncope
- Thorough review of PMH is key, including medications that can be causing current disease state (immunosuppressive, anticoagulation, anti-inflammatory, narcotics)

## Work-up

- Measure vital signs, although interpret in context of entire presentation because they can sometimes be misleading
- Perform a thorough abdominal exam with patient supine and abdomen exposed
- Consider rectal exam if concerned for gastrointestinal hemorrhage, prostatitis or perirectal disease
- Perform a pelvic exam in females with lower abdominal pain and a GU exam in males
- Choose a pelvic ultrasound to evaluate uterine and ovarian pathology and a CT for intra-abdominal pathology
- Exams may be repetitive, especially when presentations are atypical
- UA and UPT testing are time- and cost-effective; CBCs are frequently ordered but rarely helpful for a diagnosis
- Plain radiography is only useful if bowel obstruction or foreign body is suspected
- CT is used often in elderly patients and is the imaging modality of choice with nonobstetric, nonbiliary abdominal pain
- Bedside transabdominal/transvaginal ultrasounds are useful to identify intrauterine pregnancy or diagnose non-life-threatening conditions such as gallstones

## Empiric Management

- Main goals: physiologic stabilization, control of symptoms and expeditious diagnosis with or without consultation
- Analgesics, antacids, anticholinergics, antiemetics, NGT suctioning or broad-spectrum antibiotics are given according to symptoms and suspected disease process

## Disposition

- Surgical vs. Nonsurgical consultation and management
- Admission for observation (using information gained from H&P, test results, suspected disease, likelihood of follow-up after discharge, patient's ability to return if symptoms worsen)
- Discharge if clinically stable with appropriate follow-up care arranged and following criteria met:
  - No serious organ pathology or peritoneal irritation suspected
  - Normal or near-normal vital signs
  - Pain and nausea controlled
  - Patient can take fluids by mouth
  - Patient informed about what to do if circumstances change after discharge

# Jaundice

Summary from Rosen's By Chris Chase

## Epidemiology

- Jaundice is a manifestation of elevated serum bilirubin and requires an understanding of normal metabolism for evaluation and management.

## Pathophysiology

- Bilirubin is generated from heme products. Heme is oxidized to biliverdin, converted to bilirubin. It forms tight, reversible bonds with albumin and is taken up by hepatocytes where it undergoes glucuronidation, Conjugated fraction secreted into biliary system, emptied in gut and metabolized into urobilinogen and stercobilin.
- Urobilinogen reabsorbed and excreted in urine, while stercobilin is excreted in stool.
- Remaining conjugated bilirubin deconjugated and reenters portal circulation where it's taken back up by hepatocytes (enterohepatic circulation).
- Clinical jaundice not evident until total serum bilirubin concentration > 2.5 mg/dL and observed in albumin-rich tissues.
- Physiology altered in three areas: 1. Overproduction of heme products (hemolysis) 2. Hepatocellular dysfunction 3. Obstruction of biliary excretion into intestine

## Differential Diagnosis

- **Critical**
  - Fulminant hepatic failure
  - Hepatic toxin
  - Virus
  - Alcohol
  - Hepatic ischemic insult
  - Reye's syndrome
  - Cholangitis
  - Sepsis
  - Heatstroke
  - Obstructing AAA
  - Budd-Chiari syndrome
  - Severe CHF
  - Transfusion reaction
  - Preeclampsia/HELLP syndrome
  - Acute fatty liver of pregnancy
- **Emergent**
  - Hepatitis of any cause with confusion, bleeding, coagulopathy
  - Wilson's disease
  - Autoimmune hepatitis
  - Liver transplant rejection
  - Infiltrative liver disease
  - Drug induced
  - Toxin ingestion/exposure
  - Bile duct obstruction (stone, inflammation, stricture, neoplasm)
  - Sarcoidosis
  - Amyloidosis
  - Graft-versus-host disease
  - Right-sided CHF
  - Venous-occlusive disease
  - Hemolytic anemia
  - Massive malignant infiltration
  - Inborn error of metabolism
  - Pancreatic head tumor
  - Metastatic disease
  - Hyperemesis gravidarum
- **Nonemergent**
  - Hepatitis with normal mental status and VS, and no active bleeding
  - Post-traumatic hematoma resorption
  - TPN
  - Gilbert's syndrome
  - Physiologic neonatal jaundice
  - Cholestasis of pregnancy

## Signs and Symptoms

- Patients may be asymptomatic or have nonspecific symptoms: pruritus, malaise, or nausea.
- Jaundice with abdominal pain suggests biliary obstruction or significant hepatic inflammation. New-onset painless jaundice classic for neoplasm involving head of pancreas.
- Ill-fitting clothing complaints due to weight loss or increasing abdominal girth due to ascites.
- Personality changes or confusion may suggest hepatic encephalopathy.
- Jaundice first apparent sublingually, in conjunctiva and on hard palate (Cephalocaudal progression). Cutaneous findings of liver disease may be present (angiomas, caput medusa, excoriations from pruritus).
- Abdominal exam can reveal distension, indicating presence of ascites. Enlarged, tender liver-hepatic inflammation or engorgement due to biliary obstruction. Enlarged, non-tender liver-malignant infiltration. Nonpalpable liver-fibrosis due to cirrhosis. Splenomegaly-hemolysis, malignancy or portal hypertension.
- Neurologic evaluation may show signs of hepatic encephalopathy: depressed mental status, confusion, asterix (specific in HE)

## Work-up

- Begin with a thorough history with emphasis on: liver disease, viral prodrome, alcohol/IVDU, biliary tract surgery, fever, abdominal pain, pregnancy, toxic ingestion, malignancy, recent blood products, occupational exposure, recent trauma, and cardiovascular disease.
- Physical exam with emphasis on abdominal, skin and neurologic exams.
- Laboratory tests: CBC with platelets, PT/PTT, Hepatic panel: transaminases, alkaline phosphatase, bilirubin with fractionation, amylase, ABG, alcohol /acetaminophen level, Hep panel, pregnancy test. Serum ammonium and glucose checked with patients with AMS. If ascites present and suspect SBP, paracentesis is diagnostic.
- Abdominal imaging helpful if obstruction is suspected. Ultrasound best screen with high likelihood of biliary disease and benign obstruction. CT is preferred if entire abdomen needs to be evaluated or high likelihood of malignancy.
- Indirect bilirubinemia points to a hematologic cause, whereas direct bilirubinemia indicates hepatobiliary pathology.
- Elevated AP parallel to transaminases with direct bilirubemia->**Obstructive process**
- Highly elevated transaminases with normal to mild increase in AP->**Hepatocellular/cholestatic**

## Empiric Management

- Specific therapies depend on the presumptive cause of the jaundice.
- Supportive therapy with IV fluids, analgesics, antiemetics.
- Remove hepatotoxic drugs and treat for acetaminophen toxicity with NAC therapy if indicated. Evaluate and treat hepatic encephalopathy.
- If hemolytic cause, consider transfusion based on patient's ability to oxygenate. Urgent hematology consult.
- SBP treated empirically with a 3<sup>rd</sup> generation cephalosporin.
- Pregnant women with jaundice should be treated in conjunction with obstetric and GI specialist. IV hydration for hyperemesis gravidarum. Ursodeoxycholic acid for pruritus in intrahepatic cholestasis of pregnancy. Acute fatty liver will require prompt delivery.

## Disposition

- In absence of liver failure, patients with encephalopathy or unstable vital signs should be admitted. Hospitalize patients with new-onset jaundice if transaminases >1000 IU/L, bilirubin >10mg/dL or evidence of coagulopathy.
- Patients with hepatitis or cholestatic jaundice managed as outpatient if normal mental status, stable vital signs, ability to take oral fluids and no evidence of active bleeding/coagulopathy or complicating infectious process.
- Patients with extrahepatic obstructive jaundice admitted for drainage with ERCP.
- Consultation with surgery, GI, hematology, OB as indicated by cause of jaundice.
- Fulminate hepatic failure patients should be admitted to ICU.

# Nausea and Vomiting

Summary from Rosen's By Christina Smith

## Epidemiology

- Most common causes in adults: Acute gastroenteritis, Febrile systemic illness, Drug effects
- Most common causes in pediatrics: Infections (mainly of the GI tract)

## Pathophysiology

- Emetic Center Brainstem: lateral reticular formation of the medulla
  - Chemoreceptor Trigger Zone Area Postrema rich in dopamine and serotonin receptors: Response to exogenous and endogenous molecules
  - Lateral Vestibular Nuclei: Histamine Receptors, Muscarinic Receptors, Labyrinth input
  - Efferent inputs: Vagus (Esophagus, Stomach, Duodenum), Phrenic (Diaphragm), Spinal nerve (Abdominal rectus, Intercostals)
  - Afferent inputs: Vagus and Sympathetic nerves from GI Tract, Heart, Testicles

## Differential Diagnosis

- **Critical Diagnosis**
  - Boerhaave's
  - Ischemic Bowel
  - GI Bleeding
  - Intracerebral Bleed
  - Meningitis
  - DKA
  - MI
  - Sepsis
  - Aspirin
  - Antibiotics
  - Erythromycin
  - Ibuprofen
  - Chemotherapy
  - Narcotics
  - Narcotic withdrawal
  - Alcohol
  - UTI
- **Emergent Diagnosis**
  - Gastric outlet obstruction
  - Pancreatitis
  - Cholecystitis or Cholangitis
  - Bowel obstruction or ileus
  - Ruptured viscus
  - Appendicitis
  - Peritonitis
  - SBP
  - Migraine
  - CNS Tumor
  - Raised ICP
  - Adrenal insufficiency
  - Uremia
  - Acetaminophen
  - Digoxin
  - Aspirin
  - Theophylline
  - Gonadal torsion
  - Carbon monoxide
  - Electrolyte disorders
  - Organophosphate poisoning
- **Nonemergent diagnosis**
  - Gastritis
  - Gastroparesis
  - PUD
  - IBD
  - Biliary colic
  - Hepatitis
  - Gastroenteritis
  - Thyroid
  - Pregnancy



## Signs and Symptoms

- Acute <1 week vs Chronic >1 month
- Stable vs Unstable: Hemodynamic stability, Level of consciousness, Neurologic assessment, Vital signs
- Sequela of vomiting: Hypovolemia, Metabolic alkalosis, Hypokalemia, Mallory-Weiss, Boerhaave's syndrome, Aspiration

## Work-up

- **Labs:** CBC, Electrolytes, BUN, Cr, Serum lipase, Urine pregnancy, UA, Blood & Urine cultures, Liver Function, Ammonia, Serum drug level
- **Imaging:** Flat & upright radiograph, CT abd (Obstruction), Abd US (Cholelithiasis, Cholecystitis, Pyloric stenosis, Intussusception), CT Head or MRI (CNS trauma, tumor, infectious)

## Empiric Management

- If Unstable: Airway, Monitor, ECG, Labs, Oxygen, Fluid resuscitation
- Rehydrate: Oral fluids vs IV fluids
- Nasogastric tube: Refractory vomiting, Gastroparesis, Pancreatitis, Bowel obstruction
- Drugs
  - Antidopaminergic: **Prochlorperazine** (Compazine) → Side effect Akathisia and Dystonia that is mitigated with Diphenhydramine and Benztropine, **Metoclopramide** (Reglan) → Useful in children but black box warning of tardive dyskinesia
  - Antiserotonergic: **Ondansetron** (Zofran) → Effective in ED and pediatrics
  - Antihistamines: **Promethazine** (Phenergan)

## Disposition

- Admission: Significant underlying disease, Unclear diagnosis, Responds poorly to fluid and antiemetic, Refractory emesis
- Discharge: No serious underlying illness, Response to fluid and antiemetic therapy, Able to take clear liquids, Follow-up is favorable with PCP in 24-48 hours, Instruction to return to the ED with recurrence, change, deterioration

# GI Bleeding

Summary from Rosen's By Meaghan Dehning

## Epidemiology

- >1 million hospitalizations annually in the US
- Upper GI bleeds (proximal to ligament of Treitz) incidence is 165 per 100,000; 13-14%
- Lower GI bleeds (distal to the ligament of Treitz) incidence is 20.5 per 100,000; mortality 4%

## Pathophysiology

- Hematemesis – bloody or coffee-ground emesis. Occurs secondary to an upper GI bleed
- Melena – dark, tarry stools. Occurs secondary to upper GI bleed.
- Hematochezia – bright red or maroon blood per rectum. Most often occurs secondary to a lower GI bleed, but may be seen in rapid upper GI bleeds

## Differential Diagnosis\*\*\*

- Upper GI Bleed – Adult
  - Peptic ulcer disease (>50% of acute upper GI bleeds in ED)
  - Erosive gastritis (more common in inner city populations)
  - Esophageal varices (more common in inner city populations)
- Lower GI Bleed – Adult
  - Hemorrhoids
  - Colonic diverticula
  - Angiodysplasia
  - Colitis secondary to ischemia, infection, IBD
  - Aortoenteric fistula\*
- Mimickers
  - Hematemesis – Epistaxis, dental bleeding, red food coloring
  - Melena - Bismuth and Fe containing medications
  - Hematochezia – vaginal bleeding, gross hematuria, partially digested foods (beets)
- Upper GI Bleed – Pediatric
  - Gastric/duodenal ulcers
  - Esophagitis
  - Gastritis
  - Esophageal varices\*
  - Mallory-Weiss tears

\*may rapidly lead to exsanguination and death.
- Lower GI Bleed – Pediatric
  - Anorectal fissures
  - Infectious colitis
  - Intussusception
  - Meckel's diverticulum

## Signs and Symptoms

- Upper GI Bleed : Hematemesis, melena
- Lower GI Bleed: Hematochezia (may be seen in rapid upper GI bleed)
- Physical Exam/Vitals
  - Hypotension and tachycardia suggest impending shock
  - Assess for pallor, cool/clammy extremities, ecchymoses/petichiae, jaundice, palmar erythema, spider angiomas
  - Abdominal exam – assess for peritonitis, tenderness to palpation
  - Rectal exam, evaluation of external anus, anoscopy

## Work-up

- Thorough History: Context i.e. recent history of severe retching; Quantity – passage of clots, streaks of blood on toilet paper (often better assessed via vitals); Appearance – hematemesis, melena, hematochezia.
- Past Medical History – assess for bleeding risk factors (NSAIDs, Coumadin), previous bleeding episodes, EtOH use\*\*
- Basic Laboratory Studies: Hemoglobin (<10 + correlate with increased rates of rebleeding and mortality), BUN (BUN:creatinine ratio >36 without renal failure is suggestive of upper GI bleed), coagulation studies, platelets
- Additional Studies
  - Occult blood/guaiac testing (may have false positive with ingestion of red meat, turnips, horseradish, vitamin C)
  - EKG – especially in elderly or high risk for ischemic event
  - Abdominal CT – identify viscus perforations (no use for plain films)

## Empiric Management

- Type and screen or cross-match (If unstable O neg to women of childbearing age; O pos to others)
- Sengstaken-Blakemore Tube- bedside balloon tamponade if exsanguinating likely variceal bleed without immediate endoscopy available
- Medications
  - High dose PPI (80mg bolus omeprazole iv, 8 mg/hr x 3days)
  - Somatostatin and Octreotide – splanchnic vasoconstrictors that reduce portal hypertension. Begin empirically in GI bleeds with a history of EtOH or abnormal LFTs (50 microgram bolus, 50 micrograms/hour iv)

## Disposition

- Hemodynamically unstable with severe upper GI bleeding → urgent GI consult
- Lower GI bleed or exsanguinating → also consult surgery
- Ongoing bleeding, significant comorbidity → admit to ICU
- Identifying high risk patients – requiring a blood transfusion, endoscopy, or surgical intervention – using clinical and laboratory data to risk stratify
  - Blatchford score -99.6% sensitive
  - Rockall score – 90.2%

\*\*See table 30-2 in Rosen's for additional characteristics of high risk bleeders

\*\*\*See Figure 30-1 for helpful diagnostic algorithm

# Diarrhea

Summary from Rosen's By Nnenna Ejeseime

## Epidemiology

- 4% of all deaths each year
- 3.5 million cases/ year of childhood diarrhea are caused by rotavirus
- 90% of diarrheal cases in the U.S. are caused by norovirus

## Pathophysiology

- Infectious (viral mc, bacterial, protozoal, ect) vs Noninfectious (autoimmune, toxins, anatomic)
- Secretory – cytotoxins increase membrane permeability and over secretion of water and electrolyte
- Inflammatory – aka invasive, severe, or dysentery diarrhea s/s attributed to mucosal cell damage, leading to secondary hyper secretion of water, electrolytes, blood and mucus.
- Osmotic – solutes induces osmotic movement of water into the lumen
- Abnormal Motility – decrease contact time between absorbing mucosa and luminal contents.

## Differential Diagnosis\*

- Infections
  - Viral (60% of cases) → Rotavirus, Norovirus, Coronavirus, CMV, HSV
  - Bacterial (20%) → E. Coli, Salmonella, Shigella, Campy, C. Diff, Campylobacter, V. Cholera, Yersenia
  - Parasitic (5%) → Cryptosporidium, Cyclospora, Giardia, Schistosoma
- Noninfectious – ACE Inhibitors, Diuretics, Antibiotics, Antidepressants, Sorbitol, Shellfish neurotoxin, Ethanol, Carcinoid Tumor, Hyperthyroidism, VIPoma, Diabetes, Cystic Fibrosis, IBS, Crohn's. UC, Diabetes

## Signs and Symptoms

- Duration:
  - Acute < 14 days, typically infectious
  - Persistent 14-30 days, suggests bacterial or protozoal
  - Chronic > 30Days, typically noninfectious
- Associated w/ Dehydration → tachycardic, hypotensive, poor skin turgor, diaphoretic, dry mucous membranes
- Associated with electrolyte disturbance → Kussmaul breathing. Muscle cramping, seizures, AMS
- Stool: melena, mucus in stool → Invasive, inflammatory. Watery, loose, drug changes → Osmotic. watery, rice like → secretory. Pale → hepatobiliary
- Pediatrics : sunken eyes, depressed fontanel, decreased urine output, decreased activity

## Work-up<sup>1</sup>

- Labs typically unnecessary in the Acute setting ; illness is usually self limiting
- Invasive or 'non-noroviral' suspicion → CBC, CMP, TSH, Serum lipase, Urine HcG, Hemocult and fecal cell count, C diff Toxin, E. Coli 0157:H7 toxin assay, Stool Culture, , and stool O&P,
  - Others: Giardia Antigen Assay, U/A
  - r/o Surgical Abdomen → Abd Series, or Abd CT, or laparotomy

## Empiric Management

- Specific treatments are directed towards cause
- General → Oral Rehydration for Mild to moderate fluid loss.
  - Antimotility drugs still undecided → Loperamide safest for sx relief
  - Flora Restoration → Probiotics with Lactobacillus
- Bowel Rest is no longer suggested and may worsen diarrhea
- Avoid Dairy products b/c some of the infectious pathogens cause a transient lactase deficiency worsening symptoms
- Avoid foods high in simple sugars
- Pediatrics → BRAT (Bread, Rice, Apple, Toast) diet is suggested
  - *Abx treatment in severe cases may lead to HUS or TTP → AVOID ABX IN PEDI PT'S!!*
- Severe or invasive diarrhea aka the pt looks toxic → Cipro 500mg bid or levo 500mg daily for 3-5 days
- Suspicion of C. Diff → Metronidazole (Flagyl) or Vancomycin
- Suspicion of amebic inf → Metronidazole (Flagyl)

## Disposition

- Uncomplicated acute diarrhea → d/c home after symptomatic relief, improvement of general exam, or stable. Follow up with PCP if symptoms worsen or do not improve
- Severe dehydration, hemodynamically unstable, toxic appearing, high risk patient → Admit to Floor for further management

# Constipation

*Summary from Rosen's By Nnenna Ejeseime*

## Epidemiology

- Prevalence is 16% in the U.S. → Women > Men and increased in the elderly population

## Pathophysiology

- Any structural, metabolic, mechanical, neurologic, or behavioral abnormality may cause constipation.

## Differential Diagnosis\*

- Hirschsprung
- Imperforated Hymen
- Anorectal atresia
- IBS
- Spinal Cord Injury
- Diabetes, Hypercalcemia, Hypothyroidism
- Intussusception, rectal prolapse
- Calcium supplements, Opiates, Anticholinergic
- Abuse, Eating d/d
- Dehydration, drugs, pregnancy, post-Op pain

## Signs and Symptoms

- Decreased number of Bowel movements beyond pt's norm
- Alarm Sx: fever, anorexia, nausea, vomiting, melena, anemia, weight loss > 10lbs, FamHx of colon cancer, constipation after 50

## Work-up\*\*

- Thorough H&P (abdominal and rectal exam)
- Typically no testing are necessary in the acute setting
- May order abd series/ abd plain films, CBC, CMP if suspicion of underlying secondary cause of constipation

## Empiric Management<sup>1</sup>

- It is not recommend to use stool softeners as the initial treatment in the acute setting
- Increase fluid intake and fiber Or → stool softener, Osmotic laxative, Lubricants, Suppositories to aid
- Failed Laxatives → Enema or dis-impaction may be helpful in treatment
- Refractory Opioid induced Constipation → Methylnaltrexone (Relistor) → blocks GI mu receptors without compromising central mu receptors
- Recalcitrant Constipation → benefit from biofeedback intervention and bowel training

## Disposition

- If stable or mild constipation → d/c home
- If abnl presentation, alarm symptoms are present, moderate Constipation or significant pain → admit the patient for further evaluations

<sup>1</sup>Treatments for constipation: Table 32-1 & Box 32-2

\*See Box 32-1

\*\* SeeFigure 32-1

# Pelvic Pain

*Summary from Rosen's By Justin Yuan*

## Epidemiology

- Pelvic pain common and presentation may be diffuse or lower abd pain, pelvic pain, or low back pain
- >1/3 of reproductive age women will have non-menstrual pelvic pain
- Ectopic account for 2% 1<sup>st</sup> trimester pregnancies, but among ED visits the incidence is as high as 18%
- Incidence of heterotopic pregnancy is 1 in 8000 and of special concern for women undergoing fertility treatment (1/100)

## Pathophysiology

- Visceral pain afferents that supply pelvic organs also supply appendix, ureters, colon
- Pain 2/2 inflammation, distention, ischemia, or blood, pus, or other material in the pelvis
- Parietal pain due to afferent nerves in the parietal peritoneum adjacent to an affected organ are stimulated

## Differential Diagnosis

- MCC fit into 3 categories:
- 1) Reproductive: Ovarian torsion/cyst, PID, TOA, Endometriosis, Endometritis, Uterine perforation, Fibroids, Dysmenorrhea,
  - Pregnancy 1<sup>st</sup> Tri: Ectopic, threatened abortion, nonviable pregnancy, ovarian hyperstimulation syndrome
  - Pregnancy 2/3<sup>rd</sup> Tri: Placenta previa/abruption, round ligament pain, labor/Braxton-Hicks, Uterine Rupture
- 2) Urinary: Pyelonephritis, Cystitis, Ureteral Stone
- 3) Intestinal: Appendicitis, diverticulitis, ischemic bowel, perf viscus, bowel obstruction, hernia, IBD, GE, IBS
- Other possible causes: septic pelvic thrombophlebitis, ovarian vein thrombosis, pelvic congestion syndrome, depression, herpes zoster
- ED prevalence of potentially catastrophic causes of pelvic pain: ectopic (common), ruptured ovarian cyst (uncommon), Torsion (uncommon), appy (common), PID (common), TOA (uncommon)

## Signs and Symptoms

- Location of pain and radiation pattern helpful in focusing diff dx (i.e. lateral pelvic pain often in tube or ovary)
- Abnormal vaginal d/c in vaginitis, cervicitis, endometritis, PID, and retained FB
- CMT most commonly indicates reproductive tract inflammation but also of adjacent structures
- Fundal tenderness difficult to distinguish from cystitis but can suggest PID, endometritis, necrotic fibroids
- Unilateral adnexal mass/tenderness suggest ovarian cyst, ectopic, TOA, torsion
- Constellation of adnexal/uterine tenderness and CMT is classically PID

## Work-up

- Complete H&P including LMP, menstrual patterns, sexual activity, obstetric history,
- Pelvic exam performed in almost all patients including pregnant patients <20weeks; >20weeks w/vag bleeding should undergo TA pelvic US for placental localization b/f pelvic, have FHR measured, and may need timely OB consult
- Pregnancy test in all childbearing age patients w abdominal pain
  - If positive- bedside or formal US to r/o ectopic (should visualize a yolk sac or embryo for confirmation of IUP)
- UA (absence of hematuria does not r/o ureteral stone but does lower likelihood and pyuria can be seen in extravesicular conditions such as appy)
  - Should be performed in all pregnant patients w/ pelvic pain
- Possible hemorrhage- H&H, type and crossmatch, pregnant patients with vaginal bleeding require blood typing

## Empiric Management

- Critical most likely hemorrhaging and needs rapid resuscitation with fluid, blood products, FAST, and most likely surgical intervention
  - <20 weeks pregnant- presumed ectopic: rapid resuscitation, STAT gyn consult, FAST, and RhoGAM prn
- If in septic shock- volume resuscitation, abx, surg/gyn consult, imaging when stabilized

## Disposition

- Non sick patients with a sound diagnosis may be d/c with close f/u and precautions
- Pregnant patients >20 weeks should be referred to OB for observation as well as ones with abdominal trauma

# Vaginal Bleeding

Summary from Rosen's By Alex Cohen

## Epidemiology

- About 5% of women aged 30-45 will see a physician for vaginal bleeding annually
- Menorrhagia secondary to anovulation is seen in 10-15% of all gynecologic patients
- About 10% of postmenopausal bleeding is due to endometrial cancer
- Approximately 20% of pregnant patients have vaginal bleeding before the 20<sup>th</sup> week
- There is vaginal bleeding in 50-80% of ectopic pregnancies
- Vaginal bleeding after 20 weeks occurs in about 4% of pregnancies
  - 30% due to abruption placentae and 20% due to placenta previa

## Pathophysiology

- Nonpregnant patients → Ovulatory, anovulatory, or nonuterine
  - Ovulatory: Single episode of spotting between regular menses
  - Anovulatory: Overgrowth of the endometrium due to estrogens stimulation without progesterone which results in persistent proliferative endometrium
  - Nonuterine: Lesions of the vulva, vagina or cervix as well as uterine tumors, adnexal masses, and urethral, rectal and anal disorders

## Differential Diagnosis

- |                               |                                   |  |
|-------------------------------|-----------------------------------|--|
| • Vaginitis                   | • Coagulopathy                    | • Atrophic vaginitis                               |
| • Anovulation                 | • Uterine leiomyoma               | • Vulvar, vaginal, cervical tumors                 |
| • Genital trauma/Foreign body | • Cervical and endometrial polyps | • PCOS/Hemorrhagic cysts                           |
| • Pregnancy                   | • Thyroid dysfunction             | • Urethral furuncles/Infected urethral diverticula |
| • Ectopic pregnancy           | • Endometrial cancer              | • Rectovaginal fistula                             |
| • Exogenous hormone use       |                                   |  |

## Signs and Symptoms

- The volume, duration, and timing of bleeding must be ascertained
  - Associated symptoms of n/v, abd pain, breast tenderness, urinary frequency, and history of trauma are also key
- Must perform vital signs, abdominal and pelvic exams, and in pregnant patients fetal heart tones and fundal height
  - Fetal heart tones below 100 indicate fetal distress
  - After 20 weeks, must perform ultrasound BEFORE pelvic exam
- Beta-HCG is reported as positive when concentration is greater than 20mIU/ml in urine or 10mIU/ml in serum

## Work-up

- Most important factor is to determine pregnancy status
  - If pregnant, then obtain bedside transabdominal or transvaginal ultrasound followed by pelvic exam and quantitative B-HCG
  - If not pregnant, perform pelvic exam followed by coagulation studies
- Be sure to also inspect urethra, perineum, and rectoanal areas
- Obtain cultures on speculum exam of the cervix

## Empiric Management

- All patients should be resuscitated with oxygen, IV crystalloids, and blood as needed
- Pregnant patients:
  - If viable, consider emergent cesarean section in the OR
  - If non-viable, consider D&C
  - Postpartum hemorrhage can be controlled with Pitocin
  - If bleeding is persistent and life threatening, hysterectomy can be performed
- Non-pregnant:
  - NSAIDs are the mainstay of treatment
  - Unstable patients can be treated with conjugated estrogen 25mg, repeated every 4-6 hours
    - Can use pediatric Foley inserted into cervical os to tamponade if the above is unsuccessful

## Disposition

- Patients with persistent symptomatic bleeding, especially with persistent hypotension and low hematocrit should be admitted
- Stable vaginal bleeding can be discharged with close follow-up
- In preadolescents, always rule out abuse before discharge

# Back Pain

*Summary from Rosen's By Stace Breland*

## Epidemiology

- 97% of pts complaining of low back px ultimately are Dx w/ mechanical back px (strain and/or degenerative in origin) but a very small % end up having more serious concerns like herniated disc, spinal stenosis, fracture, and congenital disorders. About 1% of all back px pts will have "true" sciatica.
- 72% will have completely recovered in 1 yr.
- For those with chronic back px (lasting longer than 3 mths) it is common to have persistent px or recurrence within 12 mths

## Pathophysiology

- Numerous causes including vascular, visceral, infectious, mechanical, or rheumatologic with pain possibly originating from spinal column, cord or root, musculature, or visceral organs.
- 95% of herniations occur at either L4-L5 and/or L5/S1 disc spaces with the majority being posterior herniations due to posterior thinning of the annulus fibrosus in those areas.
- Anatomic disc bulging and focal annulus tears with focal disc protrusion are usually asymptomatic whereas disc extrusion (a small % of overall instances) is usually symptomatic.

## Differential Diagnosis

- Vast majority of causes are non-emergent but consideration should always be given first to the possible emergent life threatening causes and should be addressed immediately.
- Emergent Diagnoses
  - Aortic Dissection
  - Cauda Equina Synd.
  - Epidural Abscess or hematoma
  - Meningitis
  - Ruptured or expanding Aortic Aneurysm
  - Spinal fracture or subluxation w/ cord or root impingement
- Numerous other Referred or Visceral causes such as Cholecystitis or Biliary Colic, Esophageal Disease, Nephrolithiasis, Ovarian Torsion/mass/tumor, pancreatitis, Peptic Ulcer Disease, Pelvic Inflammatory Disease, Endometriosis, Pleural Effusion, Pneumonia, Prostatitis, Pulmonary Embolism, Pyelonephritis, Retroperitoneal hemorrhage or tumor.
- Urgent Diagnoses
  - Back px w/ Neuro deficits
  - Disc Herniation causing neuro compromise
  - Malignancy
  - Sciatica with nerve root compression
  - Spinal fracture without cord compression
  - Spinal Stenosis
  - Transverse myelitis
  - Vertebral Osteomyelitis
- Common/Stable Diagnoses
  - Acute muscle strain
  - Acute ligamentous injury
  - Ankylosing Spondylitis
  - Degenerative Joint Disease
  - Herpes Zoster
  - Intervertebral disc disease without impingement
  - Pathologic fracture without impingement
  - Seropositive arthritis
  - Spondylolisthesis

## Signs and Symptoms

- Abnormal vital signs may suggest a life-threatening process (e.g., hypotension and tachycardia with ruptured abdominal aortic aneurysm, hypertension with aortic dissection, fever with abscess, osteomyelitis, or diskitis).
- Assess gait, ROM, and standing posture and palpate the area of complaint for indications on px severity
- Neurologic assessment may be abnormal in more serious etiologies of back pain.

## Work-up

- Guided primarily by HPI and physical exam.
- One of the most useful exams is the neuro exam and should include checks for asymmetry of reflexes, dermatomal sensory loss, and focal muscle weakness with any abnormal findings a cause for serious concern and greater investigation.
- Palpation of peripheral pulses for symmetry/quality, pulmonary and cardiac auscultation, and abdominal exam for tenderness/masses should be completed.
- Straight Leg Test should be performed if possible as it is the classic test for sciatic nerve root irritation. It is 92% sensitive but only 28% specific for disk disease.
  - This test is often negative in pts with Spinal Stenosis!



- A rectal examination can assess sphincter tone and anal wink. Testing for perianal sensation is necessary if there is any history of bowel or bladder dysfunction.
- Laboratory tests- Often of little use for mechanical causes but consideration should be given to ESR, WBC, and Urinalysis if etiology is possibly infections or possibly renal in nature but even these have poor prognostic values.
- Imaging- The vast majority of pts do not require radiographic evaluation in the ED. Exceptions to this are when “red flag” signs/symptoms are present or there is a Hx of trauma or bony tenderness in which case radiography may be warranted and based on the specific red flag noted.
  - Plain film- used for straightforward evaluation for fracture, or as a screening tool for other issues when the prior probability of serious injury or pathology is low.
  - CT- used if a spine fracture has been identified or a more complex fracture is suspected.
  - MRI- used if a disk condition, hematoma, infection, or a mass is suspected to be the cause of the problem.
  - \*\*\*Emergency MRI, CT, or, rarely, myelogram (in order of preference) is indicated **ONLY** if an acute, significant neurologic deficit such as motor loss, loss of reflexes, cauda equina syndrome, or one of the serious nonmechanical back pain sources is suspected.

## Empiric Management

- The initial empirical management of acute back pain depends on the presenting vital signs and the patient’s overall appearance.
  - Stable Pts- Early effective pain management can be of significant value.
  - Unstable Pts- Treat the underlying cause.
- Px management should be the goal in stable pt’s with non-life threatening causes with the choice of analgesic dictated by the patient’s and physician’s perception of the degree of pain. NSAIDs should be considered for non-severe px while opioids such as morphine or hydromorphone may be considered for severe px.
  - There is no credible evidence supporting the use or effectiveness of “muscle relaxants” or “antispasmodic agents,” such as methocarbamol and cyclobenzaprine, and they have significant adverse effects. The best approach is to provide adequate analgesia for the patient, supplemented by a benzodiazepine when the pain syndrome is particularly severe.

## Disposition

- Depends on the diagnosis. Obviously those with life threatening causes require admission and further treatment and some (cord compression, epidural abscess/hematoma) will require neurosurgical consultation.
- Patients who are unable to walk or require continued IV analgesics for adequate pain control should be considered for admission to the hospital or observation unit.
- If pain can be adequately controlled with oral analgesics, patients can be discharged with appropriate follow-up so long as adequate discharge instructions detailing do’s and don’ts are provided.
- Repeat visits to the ED for chronic back pain should prompt counseling of the patient regarding the necessity of management by the primary care physician or a pain management center.

\*\*\* Opioid medication should **NOT** be administered or prescribed in the ED for such patients, absent a new, acute condition.