2020 LLSA Articles

BRADFORD L. WALTERS, MD, FACEP
WILLIAM BEAUMONT HOSPITAL, DEPT. OF EMERGENCY MEDICINE, ROYAL OAK, MI
BLWALTERS@BEAUMONT.EDU

Article set

- 13 selections for 2020.
- The newest iteration is with the list from ABEM there a two questions with answers included with each article.
- Are those questions going to be on the test? Time will tell.
Diarrhea in Adults

- **Acute Diarrheal Infections in Adults.**
- Acree M, Davis AM.
- JAMA 2017;318:957.
- One of the JAMA clinical guidelines synopsis articles in the Clinical Review and Education series.
- This was a short, to the point, 2 page summary that focused on Cochrane reviews from 2000 and 2010.

Summary of the Clinical Problem

- An acute diarrheal illness is defined as **< 14 days of symptoms.**
- Includes 179 million cases of gastroenteritis and 47.8 million cases of food poisoning yearly in the U.S.
- *Clostridium difficile* is the leading cause of mortality is not covered in this summary.
- While **most cases** in patients who have not travelled abroad have **no identifiable cause**, *norovirus* is the most common cause of gastroenteritis (26% of cases) with mortality typically confined to patients over age 65 (90%).
Summary of the Clinical Problem

- Only 9% of cases are found to have a bacterial etiology that would be amenable to antibiotics include:
  - Shigella.
  - Salmonella.
  - Campylobacter.
  - Shiga-toxin producing Escherichia coli strains.
  - Vibrio parahemolyticus.
  - Enterotoxigenic E. coli.
- In traveler's diarrhea some 50-94% have an identified etiologic source, mostly bacterial.

Travelers’ Diarrhea

- A Cochrane review from 2000 showed clear benefit with antibacterials in shortening the duration of illness in moderate-severe TD.
- Agents found to be effective over a placebo control included:
  - Fluoroquinolones.
  - Azithromycin either as a single dose or 500mg/day x 3 days.
  - Rifaximin (ineffective for invasive Salmonella, Shigella, Campylobacter).
Probiotics, Pepto-Bismol, Loperamide, Prophylaxis

- In a 2010 Cochrane review showed **probiotics can result in a reduction in the mean duration of diarrhea** and fewer cases lasting > 4 days but the evidence was not strong enough to recommend a specific probiotic.
- **Bismuth subsalicylate provided symptom relief** in travelers to Mexico and loperamide was effective in travelers to Latin America and was favored over bismuth subsalicylate the further south one went.
- **Prophylactic antibiotics are only recommended for patients going outside of the U.S. or Europe who are at risk of TD.**
- Agents recommended are rifaximin (lower complication rate, safer, lower risk of developing C. diff or ESBL-PE); fluoroquinolones can also be used.

Testing

- **Testing** for an etiology is recommended for patients with severe disease, co-morbidities such as immunocompromised, dysentery, and disease lasting > 7 days.
- Polymerase chain reaction (**PCR**) is more sensitive than traditional stool testing but, it cannot distinguish between live disease causing organisms from those that are dead – its role in diarrheal illness work up is not quite established at this time.
- Cultures takes much more time but does **allow antibiotic susceptibility testing** that allows better antibiotic treatment choices.
Benefits

- Recommendations on an empirical approach based on:
  - Presence of grossly bloody stools (dysentery).
  - **Severity of illness** –
    - Moderate: forced change in activities.
    - Severe: total disability due to diarrhea.
  - Fever > 38°C (101°F).
  - Travel outside the U.S. or Europe.
- Bismuth subsalicylate in mild to moderate diarrhea to reduce stool frequency and loperamide should be used.
- Empiric antibiotics *only* in cases of **moderate to severe illness** in patients with a high likelihood of a bacterial source including the above symptoms and travel outside of the U.S. and *not* in cases of community acquired diarrheal illness.

Harms

- Risks of antibiotic treatment for diarrheal illnesses include inducing an infection with C. diff or extended-spectrum beta-lactamase-producing *Enterobacteriaceae* (ESBL-PE) particularly if using a fluoroquinolone.
- A study in Quebec found a **hazard ratio of 3.44 for patients treated with a fluoroquinolone for C. diff illness.**
- This emphasizes that treatment for mild diarrheal illness (community or traveler acquired) should be symptomatic.
Attached Questions

- ________________ is not recommended for routine acute diarrheal infection or mild traveler-associated diarrhea.
- Disabling traveler-associated diarrhea with fever should be treated with ________________.
- In patients receiving antibiotics for traveler-associated diarrhea, use ________________ to decrease the duration of diarrhea and increase chance of cure.

Answers:
- Empirical antimicrobial therapy
- Azithromycin
- Adjunctive loperamide therapy

Summary

- Diarrheal illness is common and pervasive with millions of cases per year in the U.S.
- Treatment is symptomatic in most cases of community acquired disease with norovirus being the most common etiology, mortality for norovirus infections is primarily in patients > 60 years old.
- Empiric antibiotics are strongly discouraged in such cases.
- Bacterial etiologies for diarrheal illness is seen in only 9% of cases and is prevalent in travelers' diarrhea where antibiotic treatment would be indicated in more severe cases.
- For TD recommended antibiotics include rifaximin, azithromycin, and the fluoroquinolones (that class of agents can increase the risk of C. diff or ESBL-PE).
Summary

- More serious illness would manifest with dysentery, fever, incapacitating disease, and travel.
- Symptomatic treatments include:
  - Probiotics.
  - Bismuth subsalicylate.
  - Loperamide.
- Prophylactic antibiotics are only indicated for patients traveling to high risk areas outside of the U.S. or Europe and would include rifaximin and the fluoroquinolones.
- Testing for patients with more serious illness, co-morbidities, dysentery, or > 7 days of diarrhea is primarily culture with the use of PCR not established.

AMI

- Acute Myocardial Infarction.
- Anderson JL, Morrow DA.
- This is a review of the presentation and hospital management of type 1 acute myocardial infarction (AMI).
- Much of the article focused on therapeutic considerations well beyond the time and scope of the ED.
Definitions and Types

- AMI is diagnosed and assessed on the basis of clinical evaluation, EKG, labs, invasive/non-invasive imaging, and pathological evaluation.
- Classification is based on the presence or absence of ST-segment elevation and further classified into six types:
  - Type 1 – due to coronary atherothrombosis.
  - Type 2 – due to supply-demand mismatch.
  - Type 3 – sudden death, no opportunity for lab/EKG confirmation.
  - Type 4a – infarct related to PCI.
  - Type 4b – infarct related to stent.
  - Type 5 – infarct related to CABG.

Epidemiology

- Since 1987 there has been a 4-5% decrease in hospitalization for or death due to AMI and/or CAD.
- ~550,000 first AMI’s and 200,000 recurrent AMI’s occur per year.
- Interestingly, looking globally 80% of CAD and AMI now occurs in low and middle-income countries and has decreased in the high-income ones.
Pathology

- Usual etiology of AMI is either rupture or erosion of an atherosclerotic plaque whose thrombogenic core is now exposed resulting in acute thrombosis and occlusion of the coronary artery.

- Current data shows erosion being more common than acute plaque rupture.

- Total occlusion leads to a STEMI.

- Partial occlusion or occlusion with collateral circulation results in an NSTEMI or unstable angina.

- However, in 10% of AMI cases occur without critical CAD with various mechanism proposed but, none found to be definitive.
Initial Evaluation

- Symptoms are not always typical ischemia-type chest pain – nausea, dyspnea, weakness are often seen along with combinations of symptoms that may or may not indicate ACS.
- ACC-AHA class I recommendations include an EKG within 10 minutes of presentation with cardiac troponin and serial troponins ordered and sent to the lab.
- Newer high-sensitivity assays make it possible to rule out AMI in 1-2 hours, even with just a single sample – the risk of the high-sensitivity assays is a lower specificity (high false positive rate).
- Keep in mind the disorders with elevated troponin without CAD - myocarditis, respiratory or renal failure, stroke, ICH, septic shock, or chronic structural heart disease.

Risk Assessment

- Risk assessment involves addressing whether the presenting symptoms are in fact due to CAD along with the risk of an early adverse outcome.
- Models including TIMI and GRACE are useful and are an ACC-AHA class IIa recommendation.
- TIMI model uses: age > 65, >3 CAD risk factors (HTN, high cholesterol, DM, FH, smoker), known CAD, ASA use within 7 days, severe angina, EKG ST changes > 0.5mm, + cardiac marker.
- The GRACE model is more complex and uses: age, HR, SBP, creatinine, arrest at admission, ST segment deviation, abnormal cardiac enzymes, Killip class.
Prehospital Recommendations

- **Early notification** is probably the most effective “therapy” EMS can do shaving 15 minutes off getting the patient to PCI however, there is a high rate of false activation (36%).
- The administration of **ticagrelor or cold fluids** in the pre-hospital setting with cardiac arrest has not been shown to provide benefit.
- **ASA and NTG** started in the field are obvious treatments that can provide benefit and in the case of ASA is time sensitive.

ED Care

- The ED’s primary job is **diagnosis, notification**, and moving the patient rapidly to **definitive care** – usually PCI.
- **Oxygen** is given to most patients but, current evidence does not support benefit unless the patient is **hypoxic** (Pox < 90%).
- SL or IV **NTG** for symptom relief is endorsed.
- **Beta-blocker** therapy should be gauged carefully and avoided in patients at risk for cardiogenic shock.
- Later upon admission, within 24 hours, lipid and ACEI therapy can be initiated.
STEMI Treatment

- Restoration of perfusion is the goal with two general therapies:
  - **PCI** - goal < 90 minutes, advantages of lower mortality, lower reinfarction, lower ICB – radial artery approach is now recommended.
  - **IV fibrinolytic therapy** - should be initiated if PCI would be delayed > 120 minutes, particularly important if PCI is not available at the initial hospital.
  - Proof of the utility of this approach is a reduction in 30-day mortality from 20% to now 5%.
  - Various stents have added to improvements with now cobalt chromium drug-eluting stents the recommended stent with best outcomes.
  - Stenting of non-culprit lesions is somewhat controversial and beyond ED consideration or care.
  - Manuel clot aspiration has not been shown to be beneficial.

Treatment of ACS Without ST-Segment Elevation - NSTEMI

- Invasive strategies leads to improved outcome with the timing dependent on high-risk features.
- If the initial medical therapy stabilizes the patient’s symptoms and hemodynamics catheterization can be delayed 12-24 hours or even 25-72 hours in low risk patients.
- In the unstable patient the approach would be immediate PCI.
- Often the culprit vessel is difficult to determine and multi-vessel stenting is recommended if the patient is stable.
- **Thrombolytic therapy is contra-indicated in NSTEMI.**
Antiplatelet Agents

- **Non-enteric ASA 162-325mg** is recommended at the time of initial contact for all patients and continued indefinitely at 81-325mg/day.

- **Oral P2Y-12 inhibitors** (clopidogrel, prasugrel*, ticagrelor) is recommended in high-risk patients and STEMI patients going for PCI given as a loading dose as early as possible and continued for a year. *(recent data supports prasugrel as the superior platelet inhibitor)*

- For **non-ST-segment elevation ACS** clopidogrel or ticagrelor is recommended at the time of presentation.

- Glycoprotein IIb/IIIa inhibitors have a limited role in ACS.

Anticoagulation Therapy

- An ACC-AHA **class I level A** recommendation is that patients who present with an ACS be **anticoagulated with unfractionated heparin, enoxaparin, bivalirudin, or fondaparinux**.

- **Enoxaparin is felt to more effective** than unfractionated heparin particularly if a non-invasive approach is chosen.

- There is controversy as to whether bivalirudin or heparin is better and fondaparinux alone does not provide adequate anticoagulation to support PCI but is a better choice if the risk of bleeding is high.

- The duration of therapy is at least 2 days, preferably the duration of hospitalization, until PCI is performed, or maximum 8 days.
Combined Therapy and Discharge Care

- The article goes on to discuss guidelines regarding antiplatelet therapy plus vitamin K antagonist anticoagulation – none of that is relevant to emergency medicine.
- Finally late inpatient and pre-discharge care is discussed – again not relevant to emergency medicine.

Attached Question

- For a patient with a STEMI, when PCI is delayed by more than ___________ fibrinolytic therapy should be administered if it is not contraindicated.
- 120 minutes.
Summary

Six initial assessments and management decisions are recommended in this article:

1. **Triage** using an ACS pathway on the basis of Hx, exam, EKG, and cardiac troponin results.
2. **Assess risk** of death or recurrent ischemia on the basis of clinical features, EKG, and troponin testing with an integrated risk score – TIMI or GRACE.
3. Initiate general care including ASA, NTG, and statin with consideration of O₂, beta-blocker, or morphine.
4. Choose an **invasive or non-invasive strategy**.
5. Select a **second antiplatelet agent** to add to ASA.
6. Choose an **anticoagulant agent** based on initial management and risk of bleeding.

Child Abuse

- **Physical Abuse of Children.**
- Berkowitz CD.
- Dr. Berkowitz is from the UCLA Medical Center.
- This a Clinical Practice review of common clinical problems in NEJM starting with a clinical scenario –

   - 4 month old male brought to the ED by EMS because mother found the child limp in the crib, her boyfriend was caring for the child while she was at work. Vitals: T – 37°C, HR – 114, BP – 90/68, RR – 28. Exam was normal except for decrease muscle tone, 1cm bruise to the left cheek.
Child Abuse

- The codification of child abuse as a medical entity was in 1962 when the term *battered-child syndrome* was first elucidated – clearly child abuse has existed since ancient times and even institutionalized with the Industrial Revolution.*
- The medical definition includes *physical, sexual, emotional, and neglect of children.*
- Data mandated by the Federal Child Abuse Prevention and Treatment Act in 1988 suggests there are ~ 700,000 cases of child abuse per annum.
- Assessments of the child are best in a multi-disciplinary fashion.
- The *legal requirement to report* suspected child abuse can be difficult as the result of uncertainty of the diagnosis or if an injury is in fact accidental as opposed to inflicted.

* At the turn of the 20th Century the first cases of child abuse were prosecuted under the animal cruelty laws.

Abusive Head Trauma

- Some 30 years ago the term *shaken-baby syndrome* was described, since 2009 it is called *abusive head trauma (AHT).*
- Incidence is estimated at 20-30 cases/100,000 infants < 1 year old.
- Symptoms can be non-specific including:
  - A brief unexplained event that has resolved.
  - Apnea.
  - Altered level of consciousness, altered tone.
  - Vomiting.
  - Seizure.
  - Poor feeding.
  - Scalp swelling.
Abusive Head Trauma

- **One third** of AHT patients will have **seen a physician within 3 weeks** of the ultimate diagnosis for non-specific symptoms.
- 25% of those presented with **poor weight gain, ecchymoses, fractures, or other conditions not felt to be inflicted**.
- Assessment should include careful inspection of the head, oral cavity, the entire body for trauma, and fundoscopic exam that the author suggests be done by a pediatric ophthalmologist (clearly the author is from a major medical center).
- **Retinal hemorrhages** are reported in 85% of AHT.
- Imaging included an **HCT and full skeletal survey** looking for an ICB, rib fractures, metaphyseal fractures of legs from being jerked back and forth.

Abusive Head Trauma

- The author recommends **immediate HCT if there are CNS findings but other suggests MRI to avoid the radiation and greater sensitivity for parenchymal injury**.
- Parents/caregivers most often have no explanation for the clinical status typically saying the child was fine when put to bed and suggest actions beyond the ability of child that age.
- **Short falls, i.e. from the changing table or bed, are unlikely to result is such trauma to the brain**.
Abdominal Trauma

- Abdominal trauma is less common than head trauma and can occur in any age though more often seen in older toddlers with a mean age of 2.6 years.
- There is a high mortality mostly due to delayed diagnosis.
- Any organ in the abdomen can be injured but the solid organs are most commonly seen (liver and spleen).
- A bruise to the abdomen though uncommon raises suspicions with the work-up including LFT’s, lipase, UA, and CT with contrast.

Cutaneous and Intraoral Injury

- Bruises are common in younger children particularly toddlers.
- Significant medical problems can result in easy bruisability and should be considered in selected cases.
- Falls typically causes bruising over bony prominences like the forehead or shins but, less commonly the trunk, hands, or buttock.
- Non-ambulatory child should not have bruises to the face or trunk with intraoral injuries being sentinel finding of child abuse.
- Patterned bruises that outline a hand, belt, or brush are also sentinel findings.
- Burns to the buttocks consistent with immersion should be considered ipso facto child abuse.
Normal toddler bruises to the anterior aspect of shins

Bruise patterns that cannot be explained by typical childhood activities.

Fractures

- **Skeletal surveys** are recommended in all children < 2 years old as part of the abuse work-up or in older children where the injury and the history are incongruent.
- Rib fractures particularly to the lateral and/or posterior aspects of the ribs and *bucket-handle* metaphyseal lesions are considered specific for inflicted injury.
- **Spiral fractures are more difficult to call** as they can occur from accidents i.e. the spiral fracture of the tibial called the *toddler fracture*.
- One entity to keep in mind is osteogenesis imperfecta as such patients have commonly been considered to be child abuse before the diagnosis was made.
Healing rib fractures from being squeezed forcefully is consistent with child abuse.
Issues Not Mentioned

- As the author was not an emergency physician several issues were not mentioned but are familiar to emergency physicians:
  - ER shopping – it is not uncommon that the abused child will be taken to multiple local ED’s so no one facility sees the patient with any frequency.
  - The “new boyfriend” is not uncommonly the perpetrator.
  - Drugs or alcohol prominently in the household.
  - Not considering the diagnosis in patients from higher income households – child abuse crosses all economic lines.

Suggested Assessment

- Obtain a careful history including witnesses to the event(s), can the event account for the injuries, is the child’s development consistent with the injuries, a delay in seeking medical attention.
- Exam – fully unclothed, note clinical status, look in the mouth, check for retinal hemorrhages, get photographs.
- Work-up – CT/MRI brain, CT abdomen if needed, CBC/BMP/LFT/coag’s, full skeletal survey, ask ophthalmology for a fundoscopic exam with photographs.
- Notify Child Protective Services, admit, refer to child abuse team if available, repeat studies i.e. bone scan, repeat skeletal survey, blood for other medical issues.
Attached Questions

- Retinal hemorrhages are reported in approximately _____% of children with abusive head trauma.
  - 85%
- Physically abused children, particularly _____, may present with nonspecific symptoms and signs, such as _____; the possibility of abusive head trauma requires consideration in such cases.
  - Infants
  - Vomiting or apnea

Summary

- Child abuse may present with non-specific symptoms and signs including vomiting, apnea, alerted MS, head trauma should be considered.
- Physical findings include intraoral injuries particularly a torn frenulum, bruises to the head/face/torso particularly in non-ambulatory infants.
- Work-up – complete exam, particular attention to mouth, skin, abdomen; HCT, labs, abdominal CT, skeletal survey.
- Mandated reporting to child protective services in cases of reasonable suspicion.
LGI Bleed

- **Acute Lower Gastrointestinal Bleeding**
  - Grainek IM, Neerman A, Strate LL.
  - This was a review article as part of the NEJM continuing series on clinical practice – again not written by emergency physicians or from the emergency medicine perspective.

Clinical Problem

- GI bleeding is the most common gastrointestinal issue for which patients are admitted to a hospital – **30-40% are LGI bleeding** defined as bleeding from the colon or rectum.
- An UGI is defined as proximal to the ligament of Treitz with middle GI bleeding from that ligament to the ileocecal valve.
- Typically the bleeding ceases without intervention but advanced age and co-morbidities increase mortality/morbidity as one would expect.
- Typically the **blood passed is maroon or grossly red blood as melena is classically from an UGI bleed however, a LGI can uncommonly present with melena and a brisk UGI bleed as gross blood per rectum.**
Initial Patient Assessment

- History should include stool color, amount, frequency, and duration of bleeding.
- Associated abdominal pain might suggest an inflammatory or infectious etiology while weight loss might suggest a malignancy.
- Past history of GI disease along with cardiac, renal, or hepatic conditions that would increase the risk of a poor outcome.
- Medications including aspirin, NSAID’s, steroids, or anticoagulants goes without saying.
- CBC, BMP, coagulation studies, LFT’s, TxS with a BUN/creatinine > 30/1 suggesting an UGI source.
- Risk factors include unstable vital signs, creatinine > 1.7, age > 60, though risk factor models have performed poorly in terms of predicting outcome.

Table 1. Etiology of Lower Gastrointestinal Bleeding in Adults

<table>
<thead>
<tr>
<th>SOURCE</th>
<th>FREQUENCY (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diverticula</td>
<td>26-56</td>
</tr>
<tr>
<td>Vascular ectasias/angiodysplasia/AVM</td>
<td>2.7-30</td>
</tr>
<tr>
<td>Cancer</td>
<td>7-14</td>
</tr>
<tr>
<td>Inflammatory bowel disease</td>
<td>2-10</td>
</tr>
<tr>
<td>Hemorrhoids</td>
<td>1.4-3.6</td>
</tr>
<tr>
<td>Ischemic colitis</td>
<td>2-9.7</td>
</tr>
<tr>
<td>Polyps</td>
<td>1.4-22</td>
</tr>
<tr>
<td>Unknown</td>
<td>1-14</td>
</tr>
</tbody>
</table>

Abbreviation: AVM = arteriovenous malformation
Initial Management

- Addressing unstable vital signs with fluid resuscitation is obvious for the emergency physician.
- **Early transfusion for a Hgb < 7 gm/dl** has been shown to be beneficial in UGI bleeds with no studies looking at a similar threshold for LGI, however in the face of ongoing bleeding such would be prudent, a threshold of < 9 gm/dl is suggested for patients with more co-morbidities.
- **Colonoscopy** is the initial procedure of choice within 24 hours of presentation with the addition of EGD in patients in whom the bleeding source is undetermined.
- Colonoscopy preparation with 4L of PEG over 4 hours is advised in the stable patient.

Initial Management

- The **briskly bleeding patient** or one who is unstable represents a challenge in diagnosis.
- Radiographic imaging with **CT, CTA, a bleeding scan** is recommended in such patients.
- A retrospective study suggested a **bleeding scan followed by CTA resulted in higher diagnostic accuracy**, other studies did not confirm that result.
- Both CTA and a bleeding scan have bleeding detection rate of 0.3 ml/min with the CTA highly accurate in detecting the bleeding vessel if later angiography to occlude the vessel.
Therapies

- **Endoscopy hemostasis** with clips, bands, heat coagulation, or local injection of epinephrine are all used with diverticular bleeding, angioectasias, or post-polypectomy bleeding will most often benefit from such mechanical means to stop the bleeding.
- **Success** has been shown in 92%, early re-bleeding in 8%, and late re-bleeding in 12%.
- **Perforation, worsening bleeding, and CHF** as adverse events are seen in 0.3-1.3%.
- LGI bleeding secondary to ischemic colitis, inflammatory disease, or neoplasms are less amenable to endoscopic hemostasis and may require surgical or endovascular control.

Angiography, Surgery

- Allows both bleeding site localization and intervention.
- Angiography can be negative in slow bleeds < 0.5 mg/min or with intermittent bleeding.
- Case series show a success rate of 73-100% with re-bleeding in 11-50% and a risk of ischemic bowel with embolization of 1-4%.
- Surgery is the last resort with failure of other means or the exsanguinating patient.
- Localization of the bleeding is extremely helpful in deciding the surgical approach and preserving as much bowel as possible.
Antiplatelet Agents and LGI Bleeding

- All the antiplatelet agents increase the risk of LGI bleeding ~ 3x over the risk of UGI bleeding.
- Risk of cardiovascular death is the concern in patients on daily aspirin with no significant increase in bleeding risk in PUD in which the bleeding was controlled and the patient resumed low-dose aspirin.
- This was not the case with LGI where continued ASA use was associated with a 19% 5-year increased risk of re-bleeding compared to non-ASA using patients however, the overall mortality still showed a benefit in those patients who continued ASA use.
- Data is lacking on what to advise patients on dual-anti-platelet therapy.
- No comment was made by the authors on the use of platelet transfusion in such patients nor reversing anticoagulation in patients on coumadin or a DOAC.
Attached Questions

- Colonoscopy should be the initial procedure for most patients presenting with acute lower gastrointestinal bleeding. It should generally be performed within _____ after presentation, after hemodynamic resuscitation and colon cleansing.
  - 24 hours
- Hematochezia in the context of hemodynamic instability may represent an upper gastrointestinal bleeding event; therefore, _______ should be considered.
  - Upper endoscopy

Summary

- As in any bleeding issue stabilization and resuscitation of the patient is paramount.
- Consideration of transfusion with a Hgb < 9 gm/dl and certainly < 7 gm/dl is recommended.
- A careful history of the bleeding, quality, quantity, frequency, and risk factors starts the process of localizing the bleeding from upper vs. lower.
- Maroon or gross blood per rectum is typically lower though melanotic stool can occur with LGI bleeding and gross blood with a brisk UGI bleed can occur.
Summary

Once the patient has been stabilized diagnostic studies begin with labs – CBC, BMP, LFT, coag’s, Txs.

The diagnostic procedure of choice in the first 24 hours is colonoscopy after cleansing with EGD in selected patients where the source of the bleeding is unknown.

For the unstable patient or one bleeding severely radiographic imaging can localize the bleeding source with a bleeding scan, CTA, or both – the CTA can be particularly helpful if endovascular embolism is a consideration.

To a large extent after stabilization and basic diagnostic studies therapy for LGI bleeding is in the province of GI and/or surgery.

Necrotizing STI’s

Necrotizing Soft-Tissue Infections.

Stevens DL, Byrant AE.


This was also a review article from the NEJM series and again from the non-emergency medicine perspective.
Definitions

- Necrotizing fasciitis (NF) is a surgical diagnosis characterized by friable fascia, dish-water grey exudate, notable absence of pus.
- Can be due to major trauma, minor skin/mucosa injury, routine Ob/Gyn procedures, and in immunocompromised patients.
- NF types are distinguished by cause:
  - **Type I** – polymicrobial, less common, incidence is growing, both aerobic and anaerobic bacteria, more often in elderly, DM, decubiti, hemorrhoids, Ob/Gyn or urologic procedures.
  - **Type II** – monomicrobial, 55-87% of cases, often with gas in tissues, hard to distinguish from gas gangrene, often in DM especially with foot infections, but can occur in younger patients with no underlying disease.

Definitions

- Type II most commonly due to Group A strep (GAS) followed by MRSA.
- *Aeromondas hydrophila* and *Vibrio vulnificus* are less common etiologies of Type II NF.
- Related syndromes include:
  - **Ludwig’s angina** – infection in the fascial compartments of head/neck.
  - **Lemierre’s syndrome** – thrombosis of jugular vein most often associated with Ludwig’s angina infections.
  - **Fournier’s gangrene** – perineal/abdominal wall NF that rapidly spreads.
- Both **Group A strep and clostridial** infections are mediated by potent **exotoxins** and the host response to those.
Exotoxins

Exotoxins are secreted into the bloodstream by gram-positive or gram negative bacteria, they are heat labile, highly cell toxic even in small amounts, are neutralized by antitoxins produced by the immune system, are amenable to having vaccines against the specific exotoxin.

Endotoxins are part of a cell wall of only gram-negative bacteria that are released as that cell is lysed.

Invasive Group A strep Infections

- In 2005 there were an estimated 18 million cases of invasive Streptococcus pyogenes infections worldwide.
- In developed countries incidence = 3-5 cases/100,000 population.
- Mortality overall = 29%, increasing to 38% and 45% in strep toxic shock and septic shock respectively.
- Group A strep has a very fulminant course, rapid progression of soft-tissue necrosis, and a high mortality.
- Two clinical presentations:
  - NF with defined portal of entry: varicella lesions, insect bites, cuts, IVDA, surgical incisions, childbirth, penetrating trauma - clinically the initial lesion is mildly erythematous, over 24-72 hours skin becomes dusky, purplish, bullae appear, progressing to frank gangrene.
  - NF that arises spontaneously, 50% have non-penetrating injury i.e. muscle strain with only fever and pain as symptoms, delayed diagnosis is the rule with mortality 70%.
Group A strep and NSAID’s

- In the 1980’s reports of an association of NSAID’s and GAS NF though experimental evidence is limited.
- Some theories involved NSAID suppression of neutrophil function and augmentation of TNF-alpha, a key mediator.
- Other experts opined that NSAID’s merely masked symptoms with ketorolac and ibuprofen felt to accelerate the course and worsen outcomes.

Clostridial Infections

- **Gas gangrene (clostridial myonecrosis)** can occur spontaneously but is typically (70%) due to deep penetrating trauma that compromises blood supply to the soft-tissue creating the anaerobic environment ideal for bacterial proliferation and spore germination.
- Other predisposing conditions include bowel/biliary surgery, IM epinephrine injections, retained placenta, and prolonged rupture of membranes.
- **Clostridium perfringens** is seen in 80% of infections with other clostridial species (septicum, novyi, histolyticum) in the rest.
- **C. septicum** is the common etiology of spontaneous gas gangrene often in the GI tract, clinically fever is absent, profound shock, diffuse capillary leak, hemoconcentration and leukocytosis (WBC 50-175K), mortality is 70-100%
Diagnosis

Early diagnosis is **confounded** by:

- **Lack of fever**, particularly with the use of NSAID’s.
- **Absence of early skin manifestations** particularly in spontaneous disease.
- **Attributing pain to injury or procedure**, severe pain out of proportion to the injury/surgery may suggest NF but is difficult to ascertain clinically.
- **Non-specific imaging** tests without gas in the soft-tissues and only edema.
- **Attributing systemic symptoms to other causes** such as diarrhea and vomiting can be early signs of toxemia.

Clinical manifestations of NF:

- **Soft-tissue edema** – 75%.
- **Erythema** – 72%.
- **Severe pain** – 72%.
- **Tenderness** – 68%.
- **Fever** – 60%.
- **Skin bullae or skin necrosis** – 38%.

GAS infection without a portal of entry begins in the deep tissues so crescendo pain is the most important clinical finding but, can be attenuated by the use of NSAID’s.

Authors state: "**all patients presenting with a sudden onset of severe pain in an extremity with or without a portal in entry or fever should be evaluated for severe soft-tissue infection on an emergency basis.**"
Imaging, Biopsy, Histology, Gram Stain Tests

- X-rays, CT, MRI can show soft-tissue swelling and gas once necrosis has occurred – just edema may not be particularly useful.
- Gram stain of the tissue and/or exudate is crucial for diagnosis but beyond the EM evaluation in most cases.
- CRP + modest leukocytosis with left shift + elevated total CK suggests severe GAS infection.
- A marked leukemoid reaction (WBC 50-150K) and profound hemoconcentration suggests C. sodellii infection.
- WBC > 15,400 + Na < 135 can distinguish NF from non-NF infection with a NPV = 99% but a PPV only 26%.
- There is a scoring system, Laboratory Risk Indicator for Necrotizing Fasciitis, but has not been used in the ED setting.

Laboratory Risk Indicator for Necrotizing Fasciitis

<table>
<thead>
<tr>
<th>Value</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>C-reactive protein, mg/dL</td>
<td></td>
</tr>
<tr>
<td>&lt;150</td>
<td>0</td>
</tr>
<tr>
<td>&gt;150</td>
<td>4</td>
</tr>
<tr>
<td>WBC count, cells/mm³</td>
<td></td>
</tr>
<tr>
<td>&lt;15 k</td>
<td>0</td>
</tr>
<tr>
<td>15–25 k</td>
<td>1</td>
</tr>
<tr>
<td>&gt;25 k</td>
<td>2</td>
</tr>
<tr>
<td>Hemoglobin level, g/dL</td>
<td></td>
</tr>
<tr>
<td>&gt;13.5</td>
<td>0</td>
</tr>
<tr>
<td>11–13.5</td>
<td>1</td>
</tr>
<tr>
<td>&lt;11</td>
<td>2</td>
</tr>
<tr>
<td>Sodium level, mmol/L</td>
<td></td>
</tr>
<tr>
<td>&gt;135</td>
<td>0</td>
</tr>
<tr>
<td>&lt;135</td>
<td>2</td>
</tr>
<tr>
<td>Creatinine level, mg/dL</td>
<td></td>
</tr>
<tr>
<td>&lt;1.6</td>
<td>0</td>
</tr>
<tr>
<td>&gt;1.6</td>
<td>2</td>
</tr>
<tr>
<td>Glucose level, mg/dL</td>
<td></td>
</tr>
<tr>
<td>&lt;80</td>
<td>0</td>
</tr>
<tr>
<td>&gt;80</td>
<td>1</td>
</tr>
</tbody>
</table>

Risk category: low <5 (<50% chance of NF), intermediate 6-7 (50-75% chance of NF), and high 8 (>75% chance of NF).
Treatment

- **Mainstay of treatment** is *early surgical intervention* (< 24 hours) so the primary role of EM is diagnosis and/or suspicion.

- **Antibiotic regimens** are determined by the gram stain, most often not available in the ED.

- **Antibiotics** would include:
  - Polymicrobial infection *vancomycin* or *linezolid* plus one of – *piperacillin-tazobactam*, *carbapenem*, or *ceftriaxone-metronidazole*.
  - GAS or spontaneous gas gangrene – *clindamycin + PCN*.
  - There are a few other antibiotic regimens but they require specific culture diagnosis again out of the range of information in the ED.

**Treatment**

- **Fluid requirements** can be massive due to toxins and host mediator factors that cause severe capillary leak.

- 10-12 liters of NS/day is common, the use of less acidotic fluids have not been evaluated (LR or Plasmalyte).

- Severe *hypoalbuminemia* is common so replacement is often necessary but, again not in the ED.

- Bacterial hemolysins cause rapid and striking reductions in hematocrit, it is suggested to follow the HCT instead of Hgb to gauge transfusion.

- **Global hypokinesia** results in a low cardiac output seen in strep toxic shock, it is difficult to treat as pressors increase afterload in an already weakened heart, LVAD’s or ECMO might be treatments as yet not well studied, a MAP of 65mmHg is a reasonable BP goal.

- Non-useful therapies in terms of mortality include hyperbaric oxygen, IVIG infusions, and inhibitor of bacterial superantigen.
Attached Questions

- Survival, among patients with necrotizing soft-tissue infections, is significantly increased among patients taken to surgery within 24 hours after admission as compared with those in whom surgery is delayed for more than 24 hours. Survival is further increased with _________.
  - Earlier surgical intervention (e.g. within 6 hours).

Summary

- Evaluation for necrotizing STI's starts with suspicion of skin injury with swelling, edema, and pain.
- Systemic signs are typically absent with cellulitis, erysipelas, or abscess.
- Tachycardia > 120, hypotension, elevated CK, CRP > 15, or LRINEC score > 6 suggest a necrotizing STI with further evaluation necessary.
- X-rays looking for gas in the soft tissues, CT/MRI are more accurate for subtle evidence of gas or edema.
- Immediate surgery for tissue inspection, debridement, and cultures/specimen collection.
- Empiric antibiotics – vancomycin or linezolid + pip/tazo or a carbapenem or ceftriaxone/metronidazole.
Summary

- Gram stain/culture determines therapy that is obtained at surgery or later with culture results:
  - Gram positive rods = clostridial myonecrosis, spontaneous gangrene, traumatic or gynecologic myonecrosis.
  - Mixed aerobes/anaerobes = NF type I – nonclostridial cellulitis.
  - Gram positive cocci = NF type II – Strep pyogenes, Staph aureus, Group A strep myonecrosis.
  - Gram negative rods = NF type III – Aeromonas (freshwater), Vibrio (saltwater)

Eye Injuries in Kids

- Nonpenetrating Eye Injuries in Children.
- Root JM, Gupta S, Jamal N.
- A review of eye injuries in children save for penetrating etiology that in all cases would be immediately referred to ophthalmology.
- Non-penetrating injuries can often be treated in the ED or referred to ophthalmology at a later date.
- Eye trauma in common in children with ~ 840,000 injuries/year with most being preventable with eye safety precautions.
Four Guiding Principals

- The authors state 4 guiding principals to the assessment:
  - Manage other life-threatening injuries – goes without saying.
  - Ensure the structural integrity of the globe – globe rupture.
  - Assess vision in both eyes.
  - Seek consultation when further assistance is necessary.
Initial Assessment

- Starts with **visual acuity** both eyes.
- If eye is so swollen that it cannot be opened **shining a light against the lid** will at least discern perception of light or in small children they will squint with a bright light.
- Refractive errors can be differentiated from traumatic vision impairment by having the patient look through a pinhole.
- Inspection of the **periorbital soft tissue** an lids for injury, ptosis, ecchymoses, and lacerations.

---

Initial Assessment

- **Globe rupture** can be **subtle**, history of an injury that could result in penetration or rupture of the globe would start that assessment.
- Blue, brown, or black material on the surface of the eye indicates a ruptured globe with the iris or choroid plugging the rupture.
- An **asymmetric pupil**, particularly teardrop shape along with circumferential hemorrhage or bullous hemorrhage strongly suggests a globe rupture.
- In more subtle cases the **Seidel test** with fluorescein can be helpful.
- Once integrity of the globe has been assured **dilation of the pupil** with phenylephrine 2.5% or tropicamide 1% along with proparacaine for topical anesthesia can be considered to visualize the retina.
Corneal Abrasions

- Approximately half of all ED visits for eye injuries are for eye contusions or abrasions of the cornea.
- Clinically patients present with sharp pain, photophobia, FB sensation, and discomfort with blinking - healing can be expected in 24-48 hours.
- Diagnosis is by direct visualization aided by fluorescein.
- Traumatic abrasions are usually linear or geographic in shape with multiple linear abrasions often due to a FB under the upper lid.
- Treatment includes:
  - **NSAID** drops for pain, diclofenac 0.1% or ketorolac 0.4%, reports of corneal melting have been reported so use is restricted to 2 days.
  - **Cycloplegics/mydriatics** reduce ciliary spasm, cyclopentolate 1%.
  - **Topical antibiotics** have not shown benefit but can lubricate the eye unless for contact lens abrasion where *Pseudomonas* is a risk with moxifloxacin or gatifloxacin preferred over ciprofloxacin.
  - **Patching** for large abrasions, not necessary from small ones or abrasions due to contact lenses.
Corneal Burns

- Generally due to chemicals splashed in the eye, are ~10% of all eye injuries.
- **Alkali substances** (lye, ammonia) cause far greater damage than acidic ones due to *liquefaction necrosis*.
- Immediate *copious irrigation* with water, saline, or LR – a Morgan lens is very helpful and one can instill proparacaine down it to reduce pain.
- Irrigate to neutral pH, then thorough eye exam, *cycloplegia* can reduce pain.
- Early *ophthalmology consultation* for acids, immediate for alkali burns.

**Alkali and acid burns to the eye.** Note the less severe injury of the acid exposure.

**Morgan lens facilitates irrigation of the eye.** One can instill proparacaine down the lens periodically to control pain.
Traumatic Hyphema

- Typically sports related secondary to **blunt trauma** to the globe with a significant risk of re-bleeding after the initial injury and glaucoma.
- Most can be seen by gross inspection, often layering out.
- Symptoms include **pain, blurred vision, increased pain with pupillary constriction**.
- Management in the ED includes an exam that includes measuring **IOP**, assessment for globe rupture, **eye shield**, **topical anesthetics to facilitate the exam**, **topical cycloplegics** (1% cyclopentolate, 1% atropine).
- Topical NSAID’s should be avoided and while topical steroids can help that should be the providence of the ophthalmology.

---

Traumatic Hyphema

- Secondary glaucoma due to a hyphema is common with IOP’s > 22-24mmHg should be addressed with topical beta-blockers, PO/IV carbonic anhydrase inhibitors (except in sickle cell patients), and IV mannitol.
- Surgery is usually for bleeding > 50% of the anterior chamber particularly in sickle cell trait/anemia or if the IOP remains elevated.
- In **older children** who can cooperate a hyphema can be **managed as an out-patient** with close follow-up.
- Patients with a hyphema who have **sickle cell disease or trait** deserve special mention as they are very difficult to manage and **should be hospitalized**.
Orbital Fractures

- **Facial injuries** can account for **11% of pediatric ED visits** with pediatric orbital fractures accounting for **15% of all orbital trauma**.
- Common etiologies are **MVA's, sports, falls, and violence**.
- Examination includes assessment of the pupils, globe, EOM's, visual acuity, and surrounding soft/bony tissues.
- **Orbital dystonia (orbits in different planes) or enophthalmos (posterior globe displacement) suggest orbital fractures.**
- Blowout fractures of the orbital floor can **entrap the inferior rectus** limiting upward gaze or in more severe cases enophthalmos.
- **Hypesthesia of the inferior orbital nerve** distribution can be seen with zygoma and blowout fractures.
Orbital Fractures

- As the frontal sinuses do not pneumatize until 6 y/o frontal bone fractures are actually cranial fractures with associated intracranial injury not uncommon.
- **ALARA (as low as reasonably achievable) CT scans** are more definitive than plain films that are unreliable in children particularly to identify blowout fractures of naso-orbital-ethmoid fractures that can result in saddle-nose deformities.
- ED treatment includes eye shielding, antibiotics if the fractures involve the sinuses or nose, pain medications, and referral to ophthalmology for consideration of operative repair.

Subacute Presentations

- Often post-traumatic conditions that present to the ED require a dilated fundoscopic exam by an pediatric ophthalmologist, particularly in younger patients that can require anesthesia.
- **Traumatic iritis or uveitis** typically presents 24-72 hours post-blunt injury.
- Clinically it is typically in male patients with dull, aching eye pain, photophobia, and eye redness.
- Slit lamp exam reveals WBC's and protein in the anterior chamber.
- Management includes steroid drops (requiring ophthalmology exam) and mydriatic medications.
- **Post-traumatic vision loss** suggests retrobulbar/posterior segment injury such as retrobulbar neuritis where the optic nerve becomes inflamed, this requires urgent intervention.
- Clinically patients complain of central vision loss, blurry vision, dull colors, pain with eye movement, and eye tenderness.
Subacute Presentations

- **Choroidal rupture** can occur with any trauma to the eye resulting in decreased vision, **white or yellow streaks seen in the optic disc**.
- **Traumatic retinal detachment** is suggested by **flashes of light, floaters, or a curtain over the field of vision** – prompt evaluation is necessary.
- **Commotio retinae** occurs when the shock waves from blunt globe trauma disrupts the photoreceptors seen as areas of retinal whitening, **no treatment** is necessary and vision clears over time.
- **Iridodialysis** is the **disinsertion of the iris** from the sclera seen as an **irregularly shaped pupil**, it is usually asymptomatic unless it causes glaucoma and surgery is necessary for large defects.

Attached Questions

- In the pediatric population, admission to the hospital is recommended if there is penetrating ocular trauma, secondary hemorrhage, suspected child abuse, hyphema greater than ____%, risk of noncompliant family, or patients with ______.
  - 50%
  - Patients with sickle cell disease or trait
- In non-verbal patients, careful attention should be paid to _____ that confirms light perception.
  - The reflex contraction of the eyelid
Summary

- Stepwise approach to ocular trauma:
  1. Assess **light perception** in both injured and uninjured eye.
  2. Assess **visual acuity** in both eyes.
  3. Inspect **periorbital and lid tissues** for bruising/laceration/ptosis.
  4. **EOM** assessment.
  5. Evaluate the **anterior surface** of the eye.
  7. Consider **dilating** the pupil.
  8. Examine **red reflex**.
  9. **Direct ophthalmoscopy** to assess for papilledema or retinal hemorrhage.

---

### Acute Ophthalmic Conditions

<table>
<thead>
<tr>
<th>Emergency</th>
<th>Very Urgent</th>
<th>Urgent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alkali burns</td>
<td>Globe penetration</td>
<td>Corneal abrasions</td>
</tr>
<tr>
<td></td>
<td>Globe rupture</td>
<td>Corneal ulcer</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Traumatic hyphema</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Intraocular FB</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Orbital fractures</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Retinal detachment</td>
</tr>
</tbody>
</table>
Opioid Policy

- Changes in Provider Prescribing Patterns After Implementation of an Emergency Department Prescription Opioid Policy.
- This study looked at opioid prescribing patterns in an urban ED with 23,000 annual visits after implementation of an opioid prescribing guideline.

Opioid Crisis

- We are all familiar with the grim statistics of what is now called the opioid crisis:
  - > 13,000 deaths nationally since 2007, exceeding MVA's.
  - 74% of overdose deaths involved prescription opioids.
  - 13% involved prescriptions from an ED.
- Washington state with the Washington ACEP chapter have been at the forefront of this issue and have developed the most comprehensive program of opioid prescription policy.
- This study looked at opioid prescriptions before and after implementing that policy, both in terms of number of prescriptions written but secondarily the amounts prescribed.
Washington ED Opioid Policy

- Highlights of the Washington policy include:
  - Not providing opioids to treat chronic pain nor provide parenteral opioids in the ED to patients with chronic pain.
  - Not to provide replacement prescriptions for opioids that are lost, stolen.
  - Not to prescribe long acting opioid formulations.
  - Sharing of ED visit history with other ED physicians and ED’s.
  - Coordination of care for frequent flyers.
  - Maintaining lists of clinics/facilities for opioid abuse problems.
  - For exacerbations of chronic pain contacting the PMD and only providing a few pills to allow the patient to follow-up with their PMD.
  - Screen for substance abuse prior to prescribing an opiate.
  - That the law requires screening exams for all patients it does not obligate the physician to use opioids to treat pain.

Results of Policy Implementation

- Between 1/2007 to 6/2014 with 116,676 ED visits 25,219 opioid prescriptions were written.
- After the opioid policy was in place and the physicians educated in in-serviced there was a 39.6% decrease in patients discharged with a prescription for a narcotic.
- The decrease was greatest for oxycodone with substantial decreases seen for hydrocodone, hydromorphone, and codeine.
- Patients aged 18-49 saw the largest reduction in prescriptions.
- The mean number of pills prescribed decreased 14.8% from an average of 19.5 to 16.6 pills.
- As importantly the decrease in prescription rate was sustained over the 2 years following the opioid guidelines.
CMS EMTALA Policy

- One ruling that Washington ACEP was able to obtain from the CMS district overseeing Washington state was the ability to post signs in the ED waiting room delineating their opioid policy without violating EMTALA.
- That ruling has not been universal in other CMS districts including here in Michigan.
- One part of this study was to post such signage but after consideration by the hospital and their legal department such signs were not put up.

Attached Questions

- Implementation of an ED prescription opioid policy was associated with a ________________ in total opioid prescriptions and in the number of pills per prescription.
  - Significant reduction
- The improvements were sustained ____ years after the intervention. Decreases were seen in all major opioids (hydrocodone, oxycodone, hydromorphone, and codeine). The number of pills prescribed also decreased ____%.
  - 2.5
  - 15
Summary

► Emergency departments can implement an opioid policy and expect that there will be significant reductions in the number of prescriptions written and a reduction in the amount of pills prescribed.

► Such guidelines can also be expected to maintain their efficacy over time.

Peds Airway

► The Pediatric Airway and Rapid Sequence Intubation in Trauma.
► Sulton CD, Taylor TR.
► Trauma Reports, Nov 2017.
► This was a comprehensive review of RSI by two PEM physicians from Emory.
► Of the articles selected for 2020 this is the one "must read" article.
Anatomy

- Pediatric airway has **3 basic structures**:
  - **Supraglottic segment** – most collapsible component.
  - **Glottic segment** – contains the vocal cords and upper trachea.
  - **Intrathoracic airway** – thoracic trachea and bronchi.
- The pediatric airway is **short** and **funnels** down, is more **anterior**.
- The **large occiput** in infants makes positioning more difficult.
- Cervical spine injuries are rare in young children but must be assumed particularly if there is neurologic deficit, subcutaneous emphysema, hoarseness, and neck hematomas.
- Oral and nasopharyngeal airways are helpful but, the usual backwards insertion of the oral airway then **rotating it can cause soft-tissue trauma**.

RSI Goals, Preoxygenation

- The goals of pediatric RSI are identical to that in adults in establishing a secure, definitive airway is the safest manner possible.
- The indications are also the same as in adults: **inadequate oxygenation, inadequate ventilation, airway compromise** or **loss of airway reflexes**.
- **Preoxygenation** to replace the 79% nitrogen in room air with oxygen is critical as ~ 33% of children desaturate to < 90% during RSI.
- The authors suggest using a NC, NRB mask, or BVM to preoxygenate for **~ 3 minutes** – they do not mention using both the NC + NRB mask.

*Presenter’s note: the authors do not mention that the apnea time in most children is ~ 2.5-3 minutes, that time evaporates rapidly during a difficult intubation or if the preoxygenation time is shortened.*
Preoxygenation in RSI should be “denitrogenation” as it is really replacing the usual 79% nitrogen of room air with as much oxygen as possible so the lungs become a reservoir of O2 the body can pull on during the apnea time of RSI.

Optimal denitrogenation gives one ~ 3.5 minutes of apnea time in the average child.

Equipment

- The necessary equipment is essential including:
  - Supplemental **oxygen**, **BVM** with appropriate sized masks.
  - **Endotracheal tubes** of appropriate sizes, **cuffed** in almost all cases, with the size = \( \text{age}/4 + 3.5 \) – 1 size larger and 1 smaller.
  - **Laryngoscope** with appropriate sized Miller and Macintosh blades (*video laryngoscopy not mentioned by authors*):
    - Premie – Miller 0, Term neonate – Miller 0, 1
    - 6-12 months – Miller 1, 1-2 years – Miller 1, Mac 2
    - 2-8 years – Miller 2, Mac 2, > 8 years – Miller 2, Mac 3
  - **Monitors** – pulse oximetry, end-tidal CO2.
Difficult Airway

- As in adults identifying the patient with a difficult airway is crucial to successful and safe RSI.
- **Markers of a difficulty airway** include:
  - < 1 years old are at high risk for difficulty in intubating.
  - Craniofacial abnormalities – Downs, Pierre Robbins, Treacher Collins, etc.
  - Trauma – facial or otherwise.
- Use of an **LMA** is suggested if a patient cannot be bagged - < 5kg – size 1, 5-10kg – size 1.5, 10-20kg – size 2, 20-30kg – size 2.5, 30-50kg – size 3.

  *Presenters note: Use of a pediatric bougie was not mentioned by the authors.*

Sedatives

- The authors suggest a wide-range of induction agents:
  - Etomidate – 0.3 mg/kg
  - Ketamine – 1.2 mg/kg
  - Propofol – 1 – 1.5 mg/kg
  - Midazolam – 0.2 – 0.3 mg/kg
  - Fentanyl – 2.4 mcg/kg
  - Morphine – 0.1 – m/kg
- **Etomidate and ketamine are the most stable induction agents** from a cardiovascular standpoint and are the ones used exclusively by the presenter to simplify the process.

  *Presenters note: Propofol + fentanyl or ketamine infusions post-intubation*
Sedatives

- **Etomidate** does have a temporary adrenal suppression effect, that effect is not completely known in terms of clinical problems, however it seems prudent not to use it in septic shock.

- **Ketamine** supports or mildly increases blood pressure and pulse, it also has a bronchodilatory effect, its use is cautioned with hypertension that is rarely seen in children*, the caution for use with increased intracranial pressure has been largely debunked.

- **Propofol** while an effective and rapid acting sedative tends to cause hypotension when given as a bolus and is best used post-intubation for continued sedation.**

  * presenter’s note: ketamine causes the release of endogenous pressors, such stores are typically completely released in most patients, even if hypertensive.

  ** the authors fail to mention continued sedation/analgesia post-RSI, a critical step.

Paralytics

- Recommended agents:
  - **Succinylcholine** – direct depolarizing agent – < 2 y/o - 2 mg/kg, > 2 y/o – 1.5-2.0 mg/kg.
  - **Rocuronium** – non-depolarizing agent – 1 mg/kg.
  - Vecuronium – non-depolarizing agent – 0.15-0.2 mg/kg.

- **Sux** can induce bradycardia and even asystole with repetitive doses, should additional SUX be necessary in children during RSI (rare) < 5 y/o giving atropine 0.01 mg/kg (minimum 0.1mg, maximum 0.5mg) – however recently this practice has been challenged, avoid in any case where hyperkalemia could be extent.

  Presenter’s note: given the complexity of SUX I suggest just using rocuronium as the main paralytic, it makes sense and is easier to calculate dose.
Complications

- **Missed intubations** – 1st pass success rates of 77% for EM residents or PEM fellows, 50% for pediatric residents – use of video laryngoscopy may alter that percentage – while not commented on by the authors it is suggested to – *Have a plan, a back-up plan, and a back-up plan to the back-up in every RSI case.*

- **Mainstem or endobronchial intubation** – more common in female and/or younger patients, with **cuffed ETT’s** it is suggested to advance it so the **balloon is just past the cords**, careful assessment post-intubation including CXR is important.

- **Pneumothorax** – are rare, < 1% of RSI cases, careful attention to bagging the patient and ventilator settings are helpful.

- **Aspiration** – also rare, having suction available is helpful, the authors do not comment on post-RSI NG tube placement or delayed-sequence intubation.

Trauma Considerations

- With blood loss children remain normotensive longer than adults and cardiac output is completely dependent on heart rate.

- Also blood and debris in the oropharynx can impair visualization along with facial trauma.

- **Induction medications**, fentanyl and morphine, can induce **hypotension** – the authors do not mention positive pressure on ventilator doing the same.

No suggestions were forthcoming from the authors aside from careful consideration should be made before intubating a pediatric trauma patient – delayed sequence intubation allowing oxygenation, fluid/blood resuscitation, and decompression of the stomach are advancements not covered in this article.
The pediatric airway differs greatly from the adult airway: it is ________ and __________. This can lead to a challenging direct visualization. ________ is a precursor to cardiac arrest in children. ________ is necessary prior to securing an airway.

- Shorter, more anterior, Hypoxia, Preoxygenation.

Nondepolarizing paralytic agents, such as ________, are increasing in popularity in pediatric emergency medicine because of their shorter half-life and more favorable safety profile.

- Rocuronium
Summary

- **SOAPME** – an organizational mnemonic:
  - **Suction** – available, appropriate Yankauer’s for age
  - **Oxygen** – preoxygenate with NRB + NC, BVM as second choice.
  - **Airway** – appropriate sizes of nasopharyngeal and oral airways, cuffed ETT sizes = age/4 + 3.5 (one larger, one smaller), appropriate sized laryngoscope and blades.
  - **Pharmacy** – sedatives (etomidate, ketamine), paralytics (succinylcholine, rocuronium)
  - **Monitors** – BP, cardiac, ETCO2, Pox.
  - **Extras** – LMA, video laryngoscopy, bougies as back-ups – **have lots of help**.

Pediatric Shock

- **Emergency Department Management of Pediatric Shock.**
  - Mendelson J.
  - This is a review of pediatric shock, diagnosis, and treatment from the perspective of a pediatric critical care physician from Banner-University Medical Center in Tucson – note this is not the emergency perspective and it differs in a number of significant ways – i.e. many of the patients that are admitted to the PICU have already undergone significant diagnostic and resuscitative efforts.
Definition of Shock

- **Shock** is a state of *acute energy failure* with a decrease in ATP production and a failure to meet cellular metabolic demands - essentially not enough oxygen to meet cellular demands.
- This *hypoxia is directly related to perfusion* and finally cell membrane pumps fail, intracellular edema ensues, pH regulation is inadequate, and the cell dies.
- **O2 delivery is directly related to CO + arterial oxygen content** and CO = SV x HR, SV is related to preload and afterload that is determined by systemic vascular resistance.
- SV in children is dependent on HR and arterial oxygen content on PaO2, Hgb, Pox.
- In compensated shock BP is normal and perfusion is preserved unlike in uncompensated shock where BP falls along with organ perfusion leading to death if not addressed rapidly.
- Sepsis and hypovolemia are the common etiologies of pediatric shock with a mortality rate of 11.4% compared to 2.6% in children not in shock.

**Haemodynamic Response to Hypovolaemia**

- Compensated Shock
- **Compensated Shock**
- **BP**
- **HR**
- **Child**
- **Adult**
Shock Classifications

Shock has been classified into four types:

- **Hypovolemic** - GI or renal losses, hemorrhage, 3rd spacing/burns – decreased preload and CO, elevated SVR.
- **Distributive** – sepsis, anaphylaxis, neurogenic – decreased preload and SVR, increased/decreased CO.
- **Cardiogenic** – congenital heart defect, arrhythmia, cardiomyopathy, severe anemia – increased preload and SVR, decreased CO.
- **Obstructive** – PE, pericardial tamponade, tension PTX, congenital heart lesions – increased/decreased preload, increased SVR, decreased CO.

In order to judge hypotension and tachycardia a chart with normal vital signs for age is particularly helpful.

Hypovolemic Shock

- The **most common** cause of shock in children.
- Either from intravascular fluid loss due to GI or renal loss, 3rd spacing, burns or hemorrhage that also results in low oxygen carrying capacity due to loss of Hgb.
- Hypovolemia **can occur very rapidly** and GI or renal losses can be unappreciated by parents.
- Hemorrhage shock can be broken down into the ATLS four classifications based on the % of blood loss and vital signs.

*presenter’s note – In my opinion that classification is often not a particularly helpful classification clinically in the initial few moments of evaluation in the ED.*
Distributive Shock

- In this type of shock a precipitous drop in SVR and vasodilatation creates a relative hypovolemic state and fall in CO.
- Causes include sepsis, anaphylaxis, or neurologic injury.
- Note that sepsis is some 10x more common in children < 1 y/o compared to older children.
- Clinically altered mental status, delayed capillary refill, decreased urine output is seen - hypotension is variably seen.
- The author notes that septic shock can be cold shock with a high SVR resulting in cold extremities with warm shock seen with a low SVR and warm extremities.

Cardiogenic Shock

- In children this is commonly due to congenital heart defects, cardiomyopathies, or myocarditis.
- This type of shock has two categories bases on the presence of venous congestion due to increased filling pressures or hypoperfusion due to decreased CO or myocardial contractility.
- With venous congestion findings include JVD, edema, hepatomegaly, ascites, S3 gallop, lung congestion.
- In hypoperfusion the patient looks more like cold shock cool extremities, weak pulses, narrow pulse pressure, altered MS, delayed capillary refill, and hypotension.
Obstructive Shock

- **Obstructive shock** is exactly what it says – *mechanical obstruction to blood flow* resulting in perfusion impairment and if severe enough to shock.

- **PE** is the quintessential example of such along with congenital heart lesions, *tamponade, tension pneumothorax, hypertrophic cardiomyopathy, and severe pulmonary hypertension.*

- Prompt recognition and addressing the source of the obstruction is emphasized by the author including those procedures to address a PTX, tamponade, or a ductal dependent cardiac lesion.

Recognition

- **History** can give clues as to the etiology of pediatric shock such as immunosuppressed state, steroid dependent, presumed sepsis in any child < 3 months.

- The calm, afebrile but persistently tachycardic child should be concerning for impending or occult shock.

- **Delayed capillary refill time** (CRT) is an important finding as studies have shown it be specific but not sensitive for mortality with a CRT > 2 seconds + hypotension (< 5th percentile for age) associated with a 27% mortality.

- **Altered mental status, low urine output** (often not available in the ED), metabolic acidosis, tachycardia, weak pulses, and worsening peripheral perfusion are all harbinger of shock.
Ultrasound

- The e-FAST examination for assessment of intra-abdominal fluid, cardiac tamponade, and PTX has been found to be a sensitive test in determining the etiology of pediatric shock.
- The combination of normal transaminases and a negative e-FAST have in a small study shown to define children unlikely to have an intra-abdominal injury.
- Vena caval collapsibility is a reliable measure of fluid resuscitation though measurements of the vena cava in children have not been established.

Treatment

- A shock is a state of inadequate oxygen delivery supplemental oxygen makes sense.
- Monitors and adequate IV's go without saying along with necessary labs including ionized calcium that if low could effect cardiac function.
- Fluid resuscitation starts with 20 ml/kg bolus of isotonic fluids (no comment by the author on NS vs less acidotic fluids) with as much as 40-60 ml/kg necessary in septic shock – the “push-pull” method of getting fluids rapidly into a child is highly recommended.
- In a neonate or child with suspected cardiogenic shock less fluid is given at 10 ml/kg boluses with frequent assessment for fluid overload (hepatomegaly, rales, S3 gallop).
- The author comments that central lines are unnecessary in the initial stages of resuscitation and using an IO if peripheral access is not quickly established.
Treatment

- **Vasopressors** will be necessary for shock *not responsive to fluids* run via any line that is available as establishing a central line in younger children can be difficult though a PICU study found extravasation occurred in only 2% of cases with none requiring treatment.

- The **choice of agent** is, of course, **controversial** but clearly the long standing recommendation of dopamine being the first choice is now challenged with a study showing epinephrine superior to dopamine in fluid-refractory shock and another finding no difference in mortality but quicker resolution of shock with epinephrine.

- While uncommon warm shock should be treated initially with norepinephrine.

- The addition of vasopressin has not shown benefit in trials.

---

Treatment

- **RSI** is often needed in pediatric shock and it is essential to get some fluids into the patient prior to intubation.

- **Ketamine** is a recommended induction agent, particularly in septic shock avoiding the adrenal issue with etomidate.

- A significant proportion of **metabolic demand** in pediatric shock patients is due to the work of breathing that can be **reduced with intubation and ventilator support**.

- **Antibiotics** in suspected septic patients makes sense.

- Steroids are recommended in pressor-resistant shock or in steroid dependent patient or one having been on steroid in the past 6 months.
Resuscitation Endpoints

The Surviving Sepsis Campaign (SSC) recommends the following for pediatric shock therapeutic goals:

- CRT < 2 seconds.
- Normal BP and pulse for age.
- Warm extremities.
- Normal urine output.
- Normal mental status.

The SSC goes to define targets for SvO2, perfusion pressure, lactate clearance, and cardiac index that require invasive lines and are less useful in the ED and primarily involve therapies once the patient is in the PICU, i.e. maintenance fluids with D/10

Resuscitation Endpoints

The SSC recommends the following resuscitation endpoints:

- Mixed venous saturation (SvO2) 70% or greater.
- Perfusion pressure (MAP– CVP) of 55 + (1.5 x age).
- Cardiac index of 3.3–6.0 L/min/m².

All of these require a central line, Swan-Ganz catheter, or a non-invasive perfusion monitor like the Vigileo device (not readily available in most ED’s and extremely difficult to use particularly in younger children).

This seems to reflect the author’s PICU background as opposed to practical ED constraints.
Attached Questions

- ________ is usually the preferred vasopressor in pediatric shock, especially in ________ fluid refractor shock.
  - Epinephrine
  - Cold shock
- In shock, for rapid fluid administration it is suggested that the “_______” method be strongly considered.
  - Push-pull
- Regarding foal directed therapy for shock, the American College of Critical Care Medicine recommends a SvO2 of ________% or greater.
  - 70%

Summary

- Addressing the child who presents to the ED with tachycardia, hypotension, or signs of shock would include:
  - Immediate stabilization of the airway, breathing, circulation.
  - Rapid assessment for historical clues to shock etiology.
  - Physical exam looking at perfusion, CRT, cool extremities, heart exam.
  - Laboratory studies, CXR, US (absolutely in all trauma patients).
  - Most shock states will require fluids with vasopressors added to fluid unresponsive shock patients.
  - Frequent re-assessments for progress (or lack thereof).
Reversing Dabigatran

- Idarucizumab for the Reversal of Dabigatran.
- Gottlieb M, Khishfe B.
- This was a review of literature of dabigatran and the use of Praxbind (way, way easier to say than idarucizumab) to reverse the anticoagulant effect of Pradaxa*.
- At least in my practice I rarely see a patient on dabigatran (Pradaxa) with rivaroxaban (Xarelto) and apixaban (Eliquis) dominating the marketplace to the point where I have never needed to reverse dabigatran to date.

*And this allows us to once again review the entire clotting cascade – yeah!

DOAC’s

- Dabigatran (Pradaxa) is only direct thrombin (F-IIa) inhibitor – all the other current products act as F-Xa inhibitors.
- It was introduced in 2010 for the treatment of prevent stroke and systemic embolization in studies for AF, dosing was 150mg BID, overall bleeding incidence was 16% with 1.5% life-threatening over a 1-year study period.
- Reversal of dabigatran could only be accomplish via stopping the drug or dialysis until the introduction idarucizumab (Praxbind) that is a monoclonal antibody with 350 times greater affinity for the thrombin binding site than thrombin itself resulting in near-reversible binding until the complex is renally excreted.
- Praxbind itself has a short half-life allowing relatively early resumption of anticoagulation and can cause hypercoagulability.
The authors review five articles (most by Glund) on Praxbind.

**Article 1** – Glund, et al – this was a dosing study in healthy volunteers comparing a placebo with doses from 20mg to 8g.

- adverse events were reported in 44.4% placebo and 36.1% Praxbind most commonly nausea, headache, back pain, skin irritation.
- No anticoagulation effect was seen with Praxbind itself.
- Peak concentrations were at 39-54 minutes.
Article Summary

**Article 2** – Glund, et al. – double-blind placebo controlled study of 47 healthy volunteers pretreated with Pradaxa and then reversed with Praxbind:

- Doses were 1, 2, 4 grams IV as a single dose or 5g IV then 2.5g 1 hour later.
- *Adverse events reported in 66%, only 7 felt to be drug related, all mild.*
- *Immediate restoration of clotting activity* was reported in all 5 groups and was sustained.

Article Summary

**Article 3** – Glund, et al – double-blind crossover study of 12 elderly patients with some degree of renal impairment, pre-treated with Pradaxa and then reversed:

- Doses were 1, 2, 5 grams or two doses of 2.5g 1-hour apart or placebo.
- A washout period of 6 days and subjects allocated to the opposite group – placebo or active drug.
- Age did not effect pharmacokinetics but *clearance was prolonged in patients with mild-moderate renal impairment*.
- All adverse events were mild.
- **Complete reversal was seen with all doses.**
Article Summary

**Article 4** – Glund, et al – this study looked at restarting Pradaxa anticoagulation 24 hours following a Praxbind reversal in 12 volunteers:

- Pretreated with Pradaxa, reversed with 2.5g, 5g, or placebo.
- Immediate reversal was achieved, it was sustained for 24 hours.
- **Re-starting Pradaxa at 24 hours resulted in restoration of anticoagulation.**
- This suggested that one could reverse Pradaxa for a procedure and re-start the drug 24 hours later and successfully anticoagulate the patient.

Article Summary

**Article 5** – Pollack, et al. – an interim analysis of a larger study assessing efficacy and safety of Praxbind in 90 patients with serious bleeding or the need for an urgent procedure:

- Primary end-point was maximal reversal of anticoagulation within 4 hours of giving the Praxbind with secondary endpoints of clinical hemostasis and adverse events.
- 51 patients with serious bleeding, 39 requiring a procedure.
- Patients were given 2.5g IV as 2 separate doses.
- **Median time to bleeding cessation was 11.4 hours with 100% reversal.**
- Of the 18 deaths none were felt due to a medication, 1 thrombotic event 72 hours post-reversal.
- Dabigatran levels were detectable in 6 patients at 12 hours and in 16 at 24 hours suggesting a second dose might be necessary in 24 hours.
Attached Questions

- Idarucizumab is a monoclonal antibody fragment specifically directed at dabigatran that binds to the thrombin site with ____ times greater affinity than thrombin, resulting in near-irreversible binding until _______.
  - 350
  - Renal excretion
- Given the _______ half-life of dabigatran, it is also important to consider the _______ of the last dose.
  - Short
  - timing

Summary

- Idarucizumab (Praxbind) is a direct antibody to dabigatran (Pradaxa) with a far higher affinity to bind the blocking site on dabigatran.
- It is highly effective, rapid onset, and has a low rate of adverse reactions including hypercoagulability, can be used in renal failure patients.
- One can restart anticoagulation with Pradaxa in ~24 hours.
- In a dose of 5g IV in divided doses the median time to bleeding cessation is 11 hours with detectable levels of dabigatran by 24 hours in many patients possibly necessitating a second dose of the reversal agent.
- Praxbind is expensive ($3,500).
- For life-threatening bleeding or the need for an emergency procedure the use of Praxbind seems well established.
MRI Safety with Cardiac Devices

- Safety of Magnetic Resonance Imaging in Patients with Cardiac Devices.
- This study from Pennsylvania School of Medicine was a prospective, nonrandomized study looking at whether an MRI is safe in patients with various cardiac devices (pacemakers, AICD’s).
- We automatically assume all implantable devices are MRI incompatible, this study proves that assumption is incorrect.

Introduction

- There have been some small studies that suggest pacers and AICD’s might not be as vulnerable to the magnetic field strength of an MRI as was assumed – the issue being erasing the programing of the computer chips in the device.
- In fact the majority of such devices are labelled as MRI incompatible.
- The study ran from 2003 – 2015 in patients with an implanted cardiac device who required an MRI in a 1.5 Tesla strength machine.
- By protocol all patients who were pacemaker dependent had the pacer reprogramed to asynchronous pacing mode to avoid inappropriate inhibition of pacing.
Assessments

- The pacemaker or AICD was assessed immediately after the MRI for:
  - Generator failure and power-on reset.
  - Changes in sensing and thresholds, inhibition of pacing.
  - Programming revisions.
  - Battery life and/or failure.
  - Any arrhythmia, inappropriate delivery of pacing or shock.
  - Patient discomfort, heating during exam, pain.

Results

- 2103 MRI’s were performed in 1509 patients, 58% with pacemakers, 42% with AICD’s – all were immediately interrogated and 63% were available for long-term follow-up.
- Device function:
  - Power-on reset occurred in 9 cases, all transient and generator function was fully restored.
  - 5 patients had the MRI terminated prematurely – 3 for image artifact due to the device, 2 for arrhythmias with 1 not felt due to the MRI.
  - In pacers with reed switch activation the pacer went into asynchronous pacing, all asymptomatic and no sequelae.
  - No device changed parameters either immediately or long-term.
  - 137 pacer dependent patients who were re-programed to an asynchronous mode and underwent the MRI without problem.
  - In 1% of patients P-wave amplitude was decreased.
Long-term Results

- At long-term follow-up:
  - 4% were found with decreased P-wave amplitude
  - 4% increased in atrial capture threshold
  - 4% with increase right ventricular capture threshold
  - 3% with increased left ventricular capture threshold
  - **96% had no changes in pacemaker or AICD function or programing.**

Attached Questions

- In 1189 scan of 1509 patients, ____% of all MRI's were performed without the occurrence of an event (power-on reset or early termination of the examination.
  - 96%
- The most common notable change in device parameters from baseline were decreases in __________ and increases in __________ and __________.
  - P-wave amplitude
  - Atrial capture threshold
  - Right ventricular capture threshold
  - Left ventricular capture threshold
Summary

- MRI does not interfere with pacemaker or AICD function to the extent that was previously thought with 96% of patients undergoing an MRI without difficulty or problem with the pacemaker.
- Patients who are pacemaker dependent would need their device reprogrammed temporarily to asynchronous mode.
- Interrogation of the pacemaker should occur right after the MRI and a few months later as 4% might have some change, 96% would be expected to have had no alteration in settings.

Spinal Cord Compression

- Acute Spinal Cord Compression
- Ropper AE, Ropper AH.
- This was a review of the subject out of the Department of Neurosurgery, Baylor University and as such has distinct shortcomings in terms of an emergency medicine prospective.
- It looked at acute compression due to trauma, tumor, abscess, and hematoma that can both narrow the spinal canal or damage the supporting discs/vertebrae causing instability that can also narrow the canal and compress the cord.
Clinical Features

- Clinical Features include:
  - **Symmetrical paralysis** of the limbs, areflexia.
  - **Urinary retention or incontinence**.
  - **Loss of sensation**, circumferential, c/w a sensory level.
  - **Hyperreflexia and Babinski sign** seen in intrinsic disease of the spinal cord is less evident in acute cord compression.
  - Localized neck/back pain
  - **Cauda equina** shows flaccid paralysis and early incontinence.
  - The authors caution a frequent physical exam deficiency was failing to assess sensation above the clavicle indicating cervical cord compression and omitting spinal percussion that can reveal metastatic disease.

Selected Cord Compression Syndromes

- **Spinal shock** – injury in the cervical/upper thoracic region, paralysis limbs, hypotonia, areflexia, loss of sphincter function, hypotension.
- **Central Cord** – grey matter injury of the cord, weak/areflexia arms, less severe weakness legs, loss pain/temperature sensation arms sparing vibration/properception arms and legs.
- **Hemicord (Brown-Sequard)** – one sided paralysis/hyperreflexia/reduced vibration, + Babinski injured side, loss pain/temperature sensation opposite side.
- **Conus medullaris** (compression L1-2) – weak legs/feet, variable reflexes, early loss sphincter function, loss sensation sacral and perineal dermatomes.
- **Cauda equina** (compression L2-S1) – sciatic or radicular pain, areflexia feet/legs, sphincter dysfunction, reduced sensation groin to legs
Traumatic Cord Compression

- **Acute trauma** results in cord compression due to retropulsed bone fragments, disc herniation, and subluxation of the vertebral body.
- **20% involve > 1 one level**, this is particularly true in the cervical spine.
- Classification is by the level and severity of the neurologic deficit.
- The **American Spinal Injury Association Impairment Scale** is a grading methodology with 5 levels – A = complete, no sensory or motor function to E = normal motor and sensory function.
- **With a neck injury if there is preserved motor/sensory function and no palpable neck pain or pain with full ROM** the incidence of an unstable fracture with risk of cord compression is highly unlikely - the authors suggest imaging is unnecessary.

American Spinal Injury Association Impairment Scale

<table>
<thead>
<tr>
<th>Level</th>
<th>Functional Impairment</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Complete</td>
<td>No motor or sensory function is preserved in S4–S5.</td>
</tr>
<tr>
<td>B</td>
<td>Incomplete</td>
<td>Sensory, but not motor function, is preserved below neurological level and includes S4–S5.</td>
</tr>
<tr>
<td>C</td>
<td>Incomplete</td>
<td>Motor function is preserved below neurological level, and more than half of key muscles below neurological level have muscle grade &lt;3.</td>
</tr>
<tr>
<td>D</td>
<td>Incomplete</td>
<td>Motor function is preserved below neurological level, and at least half of key muscles below neurological level have muscle grade of ≥3.</td>
</tr>
<tr>
<td>E</td>
<td>Normal</td>
<td>Motor and sensory functions are normal.</td>
</tr>
</tbody>
</table>
Traumatic Cord Compression

- In the patient with neurologic findings a **CT is the first imaging study** advised with **MRI as a complimentary study** to assess ligamentous, disc herniation, hemorrhage, or edema.

- **Treatment of traumatic injury:**
  - High dose steroids are "controversial" with "most institutions have abandoned the use".
  - **Hypotension in the first hours of injury worsens outcome** typically from loss of vascular autoregulation – use of fluids and vasopressors to rapidly reverse the loss of perfusion is recommended.
  - **Surgery** to relieve the pressure on the cord and stabilize the bony spine is best **within 24 hours**.

*The controversy over high dose steroids in traumatic cord compression is one fraught with poor science, recommendations from organizations that had no business entering that issue (AHA), and resulted in many physicians being sued for malpractice even when the data did not support its use.*

Neoplastic Epidural Compression

- External compression from **spinal metastases** is common with **pain** and tenderness in the area preceding the neurologic deficit sometimes by weeks.

- The **neurologic deficit evolves over time** (hours to days) highlighted by hyperreflexia and Babinski sign, rarely sphincter dysfunction.

- In **adults breast, prostate, and lung** cancers are the most frequent causes of metastatic cord compression followed by Hodgkin’s lymphoma, renal cell carcinoma, and myeloma.

- In **children sarcoma, neuroblastoma, and lymphoma** are the common cancers associated with cord compression.

- Sites of compression are **thoracic (60%), lumbar (25%), cervical (15%)** with multiple levels in 1/3 of cases and survival in cases with multiple level involvement is typically < 6 months.
Neoplastic Epidural Compression

- MRI imaging is the preferred test with/without gadolinium for a tumor/met of the entire spine with CT myelography in patients who cannot have an MRI.
- Treatment:
  - Surgery to decompress the spine and stabilize the vertebrae.
  - Steroids – to reduce edema, dexamethasone 100mg IV or alternatively 10mg IV and 4mg PO q-6-h tapering over a few weeks likely works as well.
  - Radiotherapy depending on tumor responsiveness.
  - Whether surgery is better than radiotherapy (studies favor surgery) are issues beyond the treatment in the ED.

Spinal Epidural Abscess

- SEA is “a treacherous condition” with a high rate of delayed diagnosis.
- Symptoms can appear abruptly or remain indolent for weeks.
- Fever and back pain are the hallmark symptoms with a bacterial site of infection in only 50% of cases with 25% not having an identifiable source of infection even at autopsy.
- Underlying conditions include DM, immunosuppression, cancer, IVDA, and ETOH abuse.
- Post-surgery SEA have a different bacterial, clinical, bacteriologic, and imaging characteristics than spontaneous SEA with an unknown proportion due to primary vertebral osteomyelitis.
Spinal Epidural Abscess

- **MRI** is again the imaging study of choice though small infections can be difficult to detect.
- The **common etiology is equally split between MSSA and MRSA** though a range of anaerobic/aerobic bacteria are found.
- Assessment of CNS rarely provides a diagnosis.
- **Lab findings include a leukocytosis, elevated CRP/ESR.**
- **Treatment:**
  - *Surgical drainage* and laminectomy is more effective than just antibiotics if done early before the onset of paralysis.
  - **Antibiotics guided by culture results of material obtained at surgery.**

Spinal Epidural Hematoma

- SEH is most often seen in patients on **anticoagulants** and/or the **trauma** patient resulting is **severe back pain, radicular pain, and then paralysis.**
- **MRI** is the most sensitive imaging test.
- **Surgical evacuation** and decompression is the treatment of choice along with **reversal** of anticoagulation in patients on VKA’s of DOAC’s.
Attached Questions

- As many as ____% of spinal injuries affect more than one level.
  - 20%
- For spinal epidural abscess, the _____ spine is most often affected.
  - thoracic
- The most common pathogen cultured from the blood or spine epidural abscess is __________, evenly split between __________ and __________ organisms.
  - S. aureus
  - Methicillin resistant
  - Methicillin sensitive

Summary

- Spinal compression can be due to a number of etiologies including trauma, metastatic lesions, epidural abscess or hematoma.
- *Time is spinal cord* so diagnosis and imaging as soon as a lesion is suspected is critical – 20% involve > 1 level.
- Symptoms include neurologic deficit that is often symmetrical and at a specific dermatome level along with pain and tenderness over the involved area.
- With SEA, SEH, and neoplastic lesions symptoms can have an acute or indolent onset while with trauma the neurologic findings are immediate.
Summary

- Assessment starts with suspecting the lesion, imaging with CT and MRI being the mainstays.
- **CT imaging** is the first choice with traumatic injury with MRI complimentary.
- **MRI** is recommended for neoplastic, infectious, or hemorrhagic causes of cord compression.
- Treatment is surgical in most cases with additional therapy in accordance with the etiology such as antibiotics with SEA, reversal of anticoagulation in trauma or SHE, and radiotherapy in neoplastic lesions sensitive for such.
- To a large extent the role of the ED is in diagnosis and imaging.

Thrombectomy for Stroke

- **Thrombectomy for Stroke at 6 to 16 Hours with Selection by Perfusion Imaging.**
  - Albers GW, Marks MP, Kemp S, et al.
  - This was the **DEFUSE 3 Trial** a study in 38 US centers of a randomized, open-label trial with blinded outcome assessment of thrombectomy in patients where last known to be well in 6-16 hours to presentation from 5/2017 – 5/2018.
Thrombectomy for Stroke

- **Endovascular thrombectomy** is indicated for acute ischemic stroke within 6 hours of symptoms for occlusion of the 1st segment of the middle cerebral and internal carotid arteries.

- The **DEFUSE 3 Trial** looked to test whether patients who had salvable brain as identified by perfusion imaging who were treated at 6-16 hours after onset of symptoms.

- The trial was at 38 US centers and patients were enrolled if their last known status was within 6-16 hours including patients with wake-up neurologic deficit if their last known well was within that timeframe.

- They had to have an infarct core volume of < 70ml with a ratio of volume of ischemic to infarcted tissue > 1.8 and an absolute volume of potentially reversible ischemic tissue of > 15ml with an occlusion of the intracranial internal carotid or proximal middle cerebral arteries as demonstrated by CTA or MRA.

Design

- The trial was a randomized, open-label trial but with blinded assessment of outcomes comparing endovascular + standard therapy compared to standard therapy alone.

- Randomization was 1:1 to either arm.

- Thrombectomy was done with a standard FDA approved device.

- Carotid angioplasty was allowed including with stenting, femoral puncture had to occur within 90 minutes of qualifying imaging.

- Intra-arterial t-PA was not allowed but IV t-PA was allowed if it was begun within 4.5 hours of symptom onset.
Primary outcome was the modified Rankin Scale at 90 days of function with secondary outcome of functional independence at 90 days along with safety outcomes (death at 90 days, ICB within 36 hours).

Imaging outcomes included infarct volume at 24 hours, lesion grown between initial and 24 hour imaging, reperfusion, and complete recanalization of the infarct artery at 24 hours.

Median time of symptoms to imaging was 10.5 hours endovascular group with median time to femoral puncture of 28 minutes.

<table>
<thead>
<tr>
<th>Modified Rankin Scale (MRS)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0  No symptoms</td>
</tr>
<tr>
<td>1  No significant disability, despite symptoms; able to perform all usual duties and activities</td>
</tr>
<tr>
<td>2  Slight disability; unable to perform all previous activities but able to look after own affairs without assistance</td>
</tr>
<tr>
<td>3  Moderate disability; requires some help, but able to walk without assistance</td>
</tr>
<tr>
<td>4  Moderately severe disability; unable to walk without assistance and unable to attend to own bodily needs without assistance</td>
</tr>
<tr>
<td>5  Severe disability; bedridden, incontinent, and requires constant nursing care and attention</td>
</tr>
<tr>
<td>6  Death</td>
</tr>
</tbody>
</table>
Results

- Endovascular + standard therapy was associated with a more favorable compared to standard therapy alone at 90 days – OR = 2.77.
- There was a higher percentage of patients functionally independent in the intervention group – 45% vs. 17%.
- Mortality was 14% in the endovascular group vs. 26% in the standard care group.
- No statistical difference in ICB – 7% vs. 4%.
- 5 patients died of ICB in the endovascular group, 2 in the standard care group.

Imaging outcomes showed the median growth in infarct volume at 24 hours was 23ml in the endovascular group vs. 33ml in the standard care.

- Reperfusion > 90% in the infarct artery was greater in the endovascular group – 79% vs. 18%.
- Complete recanalization of the occluded artery at 24 hours was also more common in the endovascular group – 78% vs. 18%.
- Benefit was seen in patients with a known time of symptom onset and those with an unknown time and favorable imaging.
- Benefit was also seen in patient treated in 9-12 hours and those treated > 12 hours.
Attached Questions

- Endovascular therapy plus standard medical therapy was associated with a ____________ favorable distribution of disability scores on the modified Rankin scale at 90 days compared to standard medical therapy alone.
  - more
- Regarding safety outcomes, mortality at 90 days was ____% in the endovascular group versus ____% in the medical therapy group.
  - 14%
  - 26%

Summary

- Endovascular therapy at 6-16 hours from symptom onset in patients with acute ischemic stroke due to large vessel occlusion (internal carotid and proximal middle cerebral) with findings of salvageable ischemic brain tissue can be expected to be associated with:
  - Lower mortality.
  - Higher functional and/or independent functioning at 90 days.
  - Some increase in ICB numerically but not statistically.
  - Greater recanalization of the infarct artery.
  - Safety measures are favorable with no higher incidence in injury due to the thrombectomy procedure.
Of course Winston and I wish you a wonderful day up here in the Michigan winter snows