2019 LLSA ARTICLES
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2019 LLSA ARTICLES
• 12 Articles
• From physician wellness to PE’s, to hyperemesis this year’s selections cover a wide range of topics with no central themes.

BRUE
• Tieder JS, Sonkowsky JL, Ettel RA, et al.
• Brief resolved unexplained events (formerly apparent life-threatening events) and evaluation of lower risk infants: executive summary.
• Pediatrics 2016;137:e20161488.
• This was a review of the topic with current recommendations from the AAP subcommittee on this topic.

Sudden Infant Death Syndrome – SIDS
• The original term for a sudden, unexplained “crib death”

Apparent Life-Threatening Event – ALTE
• The next term used in the literature but, there are definition problems that made the term difficult to use and research.

Brief Resolved Unexplained Events – BRUE
• The newest term and is the suggested description, the American Academy of Pediatrics has proposed rigorous definitions and has a suggested guideline to address this syndrome that is applicable to the emergency department.

BRUE
• Brue is defined as an event occurring in an infant < 1 year old that has resolved and has ≥ 1 of the following:
  • Cyanosis or pallor
  • Abnormal, decreased, or irregular breathing
  • Marked change in tone (hypertonia, hypotonia)
  • Altered level of responsiveness.
• This guideline looks to do two major things:
  • Identify lower-risk infants on the basis of Hx and PE for whom evidenced-based guidelines for evaluation and management are recommended.
  • Define risk infants where Hx or PE suggest the need for further work-up, monitoring, or treatment for which this guideline is not recommended.
BRUE GUIDELINE APPLICATION

• Guideline applies to infants defined as the following:
  • Age > 60 days.
  • Gestational age > 32 weeks and postconceptional age > 45 weeks.
  • Only 1 BRUE, no previous events or occurring in clusters.
  • Duration of event < 1 minute.
  • No CPR required by a trained medical provider.
  • No concerning physical exam findings.

BRUE DIAGNOSIS AND GUIDELINE

Initial assessment after a brief, resolved event that was observed by caregiver in a child ≤ 1 year old

Resolved episode has one or more of:
- Cyanosis or pallor
- Absent, decreased, irregular breathing
- Marked change in tone (hypo/hypertonia)
- Altered responsiveness

Abnormal VS’s, Sx i.e. cough, dyspnea, fever, hypoxia

NOT BRUE – out of guideline, manage accordingly

Low Risk Patient – Apply Guideline Suggestions

• SHOULD:
  - Educate caregivers on BRUE.
  - Share decision making to guide further evaluation and disposition.
  - Offer CPR training.
  - Admission not necessary.

• MAY:
  - Obtain pertussis testing, 12-lead EKG.
  - Briefly monitor with ox and serial observations.

LOW RISK BRUE MANAGEMENT RECOMMENDATIONS

• SHOULD NOT:
  - Obtain CBC, blood cultures, LP, BNP, PAO2, ABG, CXR, echocardiogram, EEG, gastroes.
  - Home cardio-respiratory monitoring.
  - Rx acid suppression Tx or Sz medications.

• NEED NOT:
  - Obtain viral respiratory testing, UA, POC glucose, lactate, neuroimaging.
  - Admission solely for cardiorespiratory monitoring.
SUMMARY

- Brief Resolved Unexplained Events – BRUE – is the suggested term to replace ALTE.
- This occurs in infants, short-lived event, and has strict criteria.
- If the patient is determined to be low risk by history and physical exam with no explanation elucidated the guideline suggests minimal testing and no admission.
- This guideline can be applied to the emergency department as most patients will present with such events to that venue.

WELL BEING

- Shanfelter TD, Noseworthy JH.
- Executive leadership and physician well-being: nine organizational strategies to promote engagement and reduce burnout.
- This was a summary of the Mayo Clinic wellness program that is comprehensive and academically rigorous involving a large administrative commitment.

HEALTHCARE SYSTEM CHALLENGES

- Challenges facing healthcare executives:
  - Declining reimbursement.
  - Increasing meaningful use of electronic medical records (EMR).
  - Large IT capital expenditures.
  - Rising clerical burdens for staff.
  - Need to improve efficiency while reducing costs.
  - Quality.
  - Mergers and acquisitions of hospitals threaten organizational survival.

ADMINISTRATIVE RESPONSES

- Easiest response seen in healthcare organizations is to external threats, that is what is typically done by most executives.
- Ignore or do not see internal threats of which employee and physician engagement is the most important and difficult to address.
- Administrations need a physician staff that is nimble, resilient, and invested in the welfare of the hospital or healthcare system but are faced with and do little with the reality of exhausted and burned out physicians.

PHYSICIAN BURNOUT

- Over 50% of physicians experience professional burnout characterized by:
  - Exhaustion.
  - Cynicism.
  - Reduced effectiveness.
- This burnout rate has been rising and is far higher than in any other field of work in the US.
- Emergency physicians are high on the list of burnout specialties, the highest in some surveys.
- “Dr. Singh’s the third E.R. burnout we’ve lost this week.”

CONSEQUENCES OF PHYSICIAN BURNOUT

- Authors state “There is a moral imperative to address burnout in physicians.”
- Burnout leads to drug/alcohol abuse, divorce, suicide.
- Ample research that reduced burnout is good for business as it:
  - Increases patient safety and satisfaction.
  - Reduces malpractice risk, improves test ordering and prescribing habits.
  - Reduces hospital costs particularly to replace a physician (2-3x annual salary) or to increase staff as burned out physicians have reduced efficiency and are less productive.
FACTORS THAT CONTRIBUTE TO BURNOUT

- Mayo administration has described several dimensions that contribute to burnout and engagement:
  - Workload
  - Efficiency
  - Flexibility/Control over work
  - Work-life integration
  - Alignment of individual and organizational values
  - Social support/Community in the workplace
  - Degree of meaning derived from work

HEALTHCARE ORGANIZATION MYTHS

- The authors cite two pervasive myths that never-the-less guide most healthcare organizations approach to physician well-being:
  - The assumption that steps necessary to cultivate physician engagement and well-being will conflict with other organizational objectives.
  - That any effective intervention to reduce burnout will be cost prohibitive.

NINE ORGANIZATIONAL STRATEGIES TO PROMOTE PHYSICIAN WELL-BEING

- Acknowledge and Assess the Problem.
- Harness the Power of Leadership.
- Develop and Implement Targeted Interventions.
- Cultivate Community at Work.
- Use Rewards and Incentives Wisely.
- Align Values and Strengthen Culture.
- Promote Flexibility & Work-Life integration.
- Provide Resources to Promote Resilience and Self-Care.
- Facilitate and Fund Organizational Science.

ACKNOWLEDGE & ASSESS THE PROBLEM

- Acknowledge at the CEO and Board level the overwhelming evidence that well-being important to the long-term viability of the organization.
- Measuring satisfaction with town halls, small group meetings, and annual surveys.
- Aggregate information by work-unit keeping individual anonymity.
- Site specific credible data is key.

HARNESS THE POWER OF LEADERSHIP

- The “right leaders must be selected”.
- 11% variation in burnout and 47% in satisfaction can be explained by aggregate leadership rating.
- Annual evaluations of leaders.
- Leaders must be able to know what motivates individual physicians and facilitate development in that area, i.e. research, teaching, clinical.
- Organizations must have the “courage” to make leadership changes.
DEVELOP & IMPLEMENT TARGETED INTERVENTIONS
- While drivers of burnout are defined the specific way they impact a physician vary by unit, hospital, and specialty.
- Interventions must be relevant at the physician level.
- Mayo Clinic saw their high burnout/low satisfaction units as high-opportunity areas to intensely target to identify local factors and address them.
- Their data supports this approach with improved burnout and satisfaction scores with half the units no longer being classified as high-opportunity.

CULTIVATE COMMUNITY AT WORK
- Peer support has always been a crucial part to address the challenges that physicians face, i.e. errors, malpractice, clinical stressors.
- Can be both formal (specific meetings or groups) or informal (physician dining room).
- Tend to overlook opportunities to celebrate achievements both personal and professional.

USE REWARDS AND INCENTIVES WISELY
- Income is entirely productivity based and variation tends to be narrow.
- Productivity can be increased by shortening time with patients, ordering more tests/procedures, or working more.
- All have obvious drawbacks and are focused on the individual physician.
- Effectiveness of financial incentives on quality is unclear.
- A rewards model that includes self-care and well-being might add a needed dimension to incentives.

ALIGN VALUES & STRENGTHEN CULTURE
- All healthcare systems emphasize providing the best care possible for its patients.
- This focus has to include the milieu in which that care is provided including how staff is supported – happy staff, happy patients.
- Any perceived erosion of core values must be picked up on (annual surveys) and aggressively addressed.

PROVIDE RESOURCES TO PROMOTE RESILIENCE AND SELF-CARE
- Providing resources for physicians is obvious step towards addressing this problem.
- Taking care of one's physical health has been found to improve patient care.

One thing not mentioned in the article is that many healthcare organizations do little more than “here’s a list of things you can access if you feel burnt out” and it is up to the physician to figure it out from there; easily accessible resources would seem to be a better solution if the goal is to improve physician wellness.
FACILITATE & FUND ORGANIZATIONAL SCIENCE

• Developing evidence based strategies to address physician wellness strengthens the program(s) and critically assesses utility and progress.
• Metrics both hospital specific and national allow assessment of how one’s wellness and interventions compare to other healthcare systems.
• Mayo Clinic currently has over 100 peer-reviewed publications regarding their program.
• Stanford Medical Center has just made substantial investments in physician wellness programs based on rigorous data that shows its benefit and return on investment.

SUMMARY

• “Any health care organization that recognized it had a system issue that threatened quality of care, eroded patient satisfaction, and limited access to care would rapidly mobilize resources to address the problem.”
• Burnout is exactly this problem but is it addressed by most hospitals?
• The Mayo Clinic demonstrates that a deliberate, sustained, and comprehensive effort to reduce burnout and improve physician engagement can make a difference in the care provided to its patients, is effective, and not prohibitively expensive.

SUMMARY

• Most hospitals operate under the assumption that burnout and professional satisfaction are the responsibility of the individual physician to either solve on their own or access certain resources that are housed outside the hospital.
• Such strategies ignore the organizational factors that are primary drivers of physician burnout.
• This article summarized one medical’s system approach that was based in a scientifically reproducible and rigorous approach.

UNEXPECTED DELIVERY

• Gupta AG, Adler AO.
• Management of an unexpected delivery in the emergency department.
• This was a review of the handling and resuscitation of a neonate in an unexpected or precipitous delivery in the ED.

Given the fact many institutions have an OB unit there are fewer deliveries in ED’s however, depending on access prenatal care can be difficult to obtain for many patients and for some patients their first physician visit for their pregnancy is delivering in the ED.

UNEXPECTED DELIVERY

• Approximately 90% of deliveries occur without the need for special assistance from physicians.
• In the remaining 10% that require some type of intervention only 1% will need extensive resuscitative measures – but that 1% makes up for all.
• The authors suggest to be prepared, review your equipment, know the resources you have in your hospital and when those resources would be less available.
PREPARATION
- The authors suggest that there are 4 important questions to ask the mother upon ascertaining you might have to deliver the child in the ED:
  - Prenatal care, is the pregnancy known to the patient?
  - How many babies are there if know?
  - Approximate gestational age?
  - Are there any major complications of pregnancy - gestational diabetes or hypertension, concern for fetal growth retardation, maternal infection or fever, prolonged rupture of membranes?

PREPARATION
- Within the article there is a very complete list of equipment both to deliver the child and to resuscitate should that become necessary (I refer you to the article should you want that list).
- Obviously one needs two teams – one to deliver and treat the mother, the other to handle the infant.
- This article focuses entirely on the neonate.

NEWBORN RESUSCITATION
- Immediately upon delivery there are three critical questions that need to be quickly answered and assessed:
  - Term gestation?
  - Good tone?
  - Breathing/crying?
- If the answer to any of those three questions is “NO” immediate measures to resuscitate the newborn must commence without delay.
- If the answer to all three questions is “YES”, relax, let the child stay on the mom’s stomach, wait before clamping the cord, dry and swaddle the child, and allow the mom to hold it.

INITIAL STABILIZATION
- Initial stabilization includes:
  - Moving the child to a radiant warmer so it can be worked on.
  - Warming and drying the infant often stimulates it to become more active.
  - Positional the airway if necessary.
  - Stimulating the infant by flicking the feet or rubbing the back.

HEART RATE
- A key assessment is if the heart rate by the first minute of life is > than 100 and is there adequate respiratory effort (apnea, gasping, hypopnea).
- If the heart rate is < 100 despite adequate ventilations ventilate the newborn with a BVM, FiO2 21% (room air).
- This can progress to endotracheal intubation or ventilation with an LMA of appropriate size.
The authors note that there are challenges to getting an IV started quickly in the best of circumstances. They suggest an IO be placed or use of an umbilical vein catheter.

An umbilical vein IV is my preferred route in the newborn. It is quick, it is easy, and it is the reason I cut the umbilical cord long.

### Umbilical Vein Cannulation

**Special Considerations**

- **Delayed Cord Clamping**
  - Current recommendations for the stable term & preterm newborns is to delay cord clamping for 30-60 seconds.
  - Less intraventricular bleeding, higher BP and blood volume, lower incidence necrotizing enterocolitis, no change in mortality, slight increase in bilirubin.

- **Thermoregulation**
  - Hypothermia increases metabolic demand, increases mortality/morbidity.
  - Warm newborn in radiate warmer, wrap in plastic, warm/dry linens.
  - Goal is 36.5-37.5°C.

### Elements of Neonatal Resuscitation

**Airway:**

- If there are signs of airway obstruction or inadequate ventilation requiring positive pressure ventilation one should also suction out the mouth and pharynx.

- Unnecessary suctioning can induce a vagal response and bradycardia.

- Guidelines NO longer recommend routine intubation in the face of meconium stained amniotic fluid as previously.

**Breathing:**

- If the newborn HR < 100 at 1 minute promptly initiate PPV - BVM with room air 21% oxygen at 40-60/minute (hyperoxia even for a short time is injurious).

- NOTE: pulse oximetry is low for the first 10 minutes of life gradually coming up.

- Frequently reassess pulse, if not coming up or pulse is dropping to < 60/min either intubate or place an LMA in newborns > 34 weeks or > 2 Kg.

- Measure Pox preductal - right arm.

- If chest compressions are initiated it is a 3:1 compression to breath rate.

### Targeted Preductal SpO2 After Birth

<table>
<thead>
<tr>
<th>Target Time</th>
<th>Target SpO2</th>
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<tbody>
<tr>
<td>1 minute</td>
<td>80-85%</td>
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<tr>
<td>2 minutes</td>
<td>65-70%</td>
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<td>3 minutes</td>
<td>70-75%</td>
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<td>4 minutes</td>
<td>75-80%</td>
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<tr>
<td>5 minutes</td>
<td>80-80%</td>
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<tr>
<td>10 minutes</td>
<td>85-95%</td>
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</tbody>
</table>
ELEMENTS OF NEONATAL RESUSCITATION

- Circulation:
  - Assess pulse by auscultation or 3-lead EKG monitor.
  - Goal is > 100/minute, < 60/minute is an indication to initiate resuscitation.
  - CPR using the two-thumb technique is recommended allowing sufficient relaxation to allow the chest wall to rebound after compression.
  - Careful coordination with the person breathing to obtain the 3:1 ratio that is necessary.

- Medications and fluids:
  - Peripheral access can be difficult, use of an IO or umbilical catheter is suggested.
  - For pulse < 60/minute despite CPR and ventilation now up to an FiO2 of 100% epinephrine or a volume expander is suggested.
  - IV epinephrine dose = 0.01 - 0.03 mg/kg of 1:10,000 concentration.
  - Epinephrine via the ETT at 0.05 - 0.1 mg/kg is an alternative if no IV access is available.
  - Crystalloids or blood are suggested volume expanders at 10 cc/kg.

POST-RESUSCITATION CARE

- Thermoregulation – goal is 36.5-37.5°C.
- Therapeutic hypothermia – has been suggested in special circumstances.
- Glucose monitoring – newborns become hypoglycemic rapidly, D10% at 2 cc/kg is the typical maintenance fluid (not NS).
- Premature labs – need to know maternal hepatitis, HIV, syphilis, blood type status.
- Fentanyl 0.5 mg/kg is recommended to both eyes for GC within the first hour of life.
- Vitamin K = 0.5-1 mg IM is recommended in all newborns to prevent early and late vitamin K deficiency bleeding.
- APGAR score = color, HR, irritability, muscle tone, respirations on a 0-2 scoring every 5 minutes until > 7.

SUMMARY

- Most often (90%) of births do not require intervention, only 1% require more advanced procedures.
- Postbirth the newborn is full term, has good tone, is breathing or crying allowing to stay with mother, delay umbilical cord clamping, keep warm, dry, swaddle.
- If no to any of the above start with drying, stimulating, position airway.
- If apnea or HR < 100 start PPV ventilation with room air, place on monitors.
- Ramp up accordingly to preductal Pox.
- Note low initial Pox is normal up to 10 minutes.

- If HR < 100/min despite PPV ventilation consider ETT or LMA intubation.
- If HR < 60/min despite 2 thumb CPR, intubate if not already done so, IV access via peripheral/IO/umbilical vein.
- If HR still below 60/min IV epinephrine 0.01 - 0.03 mg/kg (0.05-0.1 via ETT) of 1:10,000 concentration.
- If ROSC is obtained avoid hypothermia (unless cooling), check glucose, erythromycin ointment to eyes, vitamin K, D10 at 2 cc/kg maintenance fluid, admit to NICU.

HYPEREMESIS GRAVIDARUM

- Treatments for hyperemesis gravidarum and nausea and vomiting in pregnancy – a systematic review.
- JAMA 2006;316:1392-1401.
- A review of the literature (13,075 articles, 222 reviewed, 78 included with 8,930 patients) for this relatively frequent presentation to the ED.
HYPEREMESIS GRAVIDARUM (HG)

- Nausea and vomiting during pregnancy effects 80% of women with the most severe form considered HG in 0.3 - 3% of pregnancies.
- HG is characterized by intractable vomiting, electrolyte abnormalities, weight loss, nutritional deficiencies.
- Typically starts in the 6-8 week period and subsides by 20 weeks.
- Pregnancy is an increased risk of preterm delivery, small for gestational age infants, but no association with congenital abnormalities.

TREATMENT OBJECTIVES

- Treatment focuses on:
  - Relief of symptoms.
  - Preventing serious morbidity i.e., renal impairment, severe weight loss.
  - First-line treatments: OTC agents, lifestyle adjustments, sea bands, high carbohydrate diet, ginger.
  - Second-line treatments: initiated by physicians, antihistamines, IV fluids, electrolyte replacement.
  - Third-line treatments: severe symptoms that failed 1st and 2nd line treatments, includes corticosteroids and enteral feeding (via NGT or IV).

FIRST-LINE TREATMENT FOR HG

- Ginger: as fresh root, capsules, tablets, syrup –
  - Found to be more effective than placebo in 4 RCT regardless of dose or form.
  - In 2 comparison studies vs. vitamin B6, ginger was found to be more effective in one and as effective in the other.
  - Compared to doxylamine-pyridoxine there was no difference between groups.
  - Found to be superior to sea bands.
  - Level A, class IIa recommendation – ginger improves mild vomiting of pregnancy.

SECOND-LINE TREATMENT FOR HG

- Vitamin B6 – Pyridoxine/Doxylamine (P/D) Combination
  - In 3 RCTs, the P/D (10mg/10mg) combination was compared to placebo or ondansetron (Zofran) showed improvement in symptoms in moderate to severe HG.
  - Doxylamine is available as Unisom.
  - Hydroxyzine 25mg BD improved symptoms in 82% of patients compared to placebo.
  - Level A, class IIa recommendation – treatment with hydroxyzine 25mg BD is superior to placebo and is a reasonable alternative to other treatments.

Vitamin B6 lollipops
SECOND-LINE TREATMENT FOR HG

**Dopamine antagonists:**
- Both metoclopramide 10mg IV and promethazine 25mg IV given TID over 24 hours improved symptoms equally.
- Level A, Class IIa recommendation - dopamine antagonists can improve symptoms in moderate to severe HG.

**Serotonin antagonists:**
- 2 RCT's compared ondansetron 4mg with metoclopramide 10mg IV showed improved symptoms with a high rate of adverse reactions with the metoclopramide.
- Level A, class IIa recommendation - serotonin antagonists can reduce symptoms in moderate to severe HG.

THIRD-LINE TREATMENTS FOR HG

**Corticosteroids:**
- In 3 RCT's there was no difference in nausea/vomiting scores between prednisolone vs. placebo, in one study hydrocortisone showed better symptom improvement than metoclopramide.
- Level A, Class IIb recommendation - corticosteroids and treatment with steroids have an unclear benefit and might be considered in severe cases.

**Transdermal clonidine:**
- There is limited evidence for transdermal treatment for severe symptoms of HG and treatment is not well established at this time (Level B, Class IIb recommendation).

SUMMARY

- A review of evidence for treatment of hyperemesis gravidarum produced primarily good evidence with several Class A recommendations.
- For mild symptoms gingko, 5HT3 receptor antagonists and vitamin B6 were all found to reduce symptoms with very low risk of adverse side-effects.
- For moderate to severe symptoms antihistamines (alone or combined with vitamin B6) improved symptoms of HG with some evidence to support the use of psychotherapy, metoclopramide, or promethazine.

FIRE-RELATED INHALATION INJURY

- Sheridan RL.
- Fire-related inhalation injury.
- This was a review by the author of the incidence, prevention, and implications of inhalation injuries in victims of fires.
INCIDENCE

• Inhalation injury has been recognized as a significant contributor to the mortality and morbidity of fire-related injury since the Coconut Grove fire in 1942.

• The pathophysiology is complex and includes thermal injury, chemical exposure, absorbed toxins, loss of ciliary function, endobronchial debris, infection, and long-term sequelae.

![Inside view of the burned out Coconut Grove club with dead bodies being examined – note the lack of burns.](image)

INCIDENCE

• There are ~ 40,000 patients admitted for burns in the US with ~ 2,000 (5%) involving inhalation injury.

• In a study by the US Army in patients with a burn > 20% TBSA, the mortality was 20% greater with a concomitant inhalation injury.

• Injury is complex and may be as the result of direct thermal injury, chemical exposure, immune responses, inhaled toxins, debris in the branches, and secondary infection.

DIRECT THERMAL LOCAL INJURY

• Direct injury due to heated gases or direct flame are typically confined to the upper airway save in burns from pressurized steam.

• Such direct injury can obstruct the airway and early intubation is often necessary.

• Physical exam may include facial burns, soot in the upper airway, and edema of the lips and upper airway.

• Injury below the glottis is usually due to aerosolized chemicals and particles.

INFLAMMATION

• There is an intense inflammatory response to the various things inhaled by a fire victim that create reactive oxygen species, attract inflammatory cells, and trigger the release of inflammatory mediators and cytokines.

• The result is bronchospasm, vasospasm, and shunt with V/Q mismatch, bronchorrhea, and alveolar flooding, with exudate formation, bronchial obstruction by casts, and fluid loss increasing resuscitation fluid requirements.

• ARDS is not an uncommon sequela with all the problems of ventilation.

ANOXIA, TOXINS

• Anoxia – burning fuel in an enclosed space rapidly consumes available oxygen that can lead to hypoxia resulting in anoxic brain or heart injury.

• Carbon monoxide – is a very common by-product of incomplete combustion and often results in high levels in burn victims with levels > 30% symptomatic with weakness, confusion, and levels > 50% usually lethal.

• Cyanide is released with the combustion of a lot of synthetic materials such as plastics in flooring, furniture, or drapery; a clue can be that patients manifest a persistent acidosis despite successful resuscitation.

• Identification of both anoxia and CO is ABG or VBG with co-oximetry and treatment is oxygen by NRB mask, NRB mask + nasal cannula, or NIV (CPAP/BiPAP); rarely is hyperbaric oxygen therapy necessary though cytochrome clearance of CO takes longer than clearance from the blood stream.

• Cyanide is more difficult to diagnose with treatment of just just Hydroxocobalamin alone is highly effective with the Cyanokit going out of favor.
SECONDARY INFECTION

- Endobronchial and alveolar epithelial injury results in mucosal sloughing, debris, ciliary clearance impairment leading to progressive airway occlusion, atelectasis, V/Q mismatch, ARDS, and great conditions for infection.
- Infection is not seen in the ED nor is prophylactic antibiotic treatment recommended but, it is an issue that substantially contributes to mortality and morbidity of inhalational injuries.

**Bronchoscopic view of endobronchial injury and then pneumonia in a burn patient.**

DIAGNOSIS

- There is no good test for inhalational injury and diagnosis is not always as obvious as it would seem but should be suspected with:
  - History of closed space fire.
  - Cutaneous burns of the face, nose, mouth, lips.
  - Singed nasal hairs or soot in the oropharynx or even the airway.
  - Hoarseness or coughing up carbonaceous sputum.
  - Wheezing, stridor, or hypoxia.

**Bronchoscopic view of carbon staining and pallor of the airway**

BRONCHOSCOPY

- Bronchoscopic evaluation substantially increases the ability to diagnose inhalation injury, degree of injury, ability to intubate if necessary, and subsequent course.
- Soot/carbon staining, debris in the airway, casts, ulceration, pallor, and mucosal slough can be seen.
- The access to bronchoscopy is increasing in many ED’s.

**Bronchoscopic views of carbon staining and pallor of the airway.**

IMAGING

- Typically the only radiologic image that is need is a chest X-ray.
- V/Q scanning has been advocated but is clumsy and not readily available particularly in the unstable patient.
- CT scanning has been shown to be promising for stratification and prognosis but does not change the management in the ED.

**CXR does not look that bad however the CT shows great detail of the injury.**

**The chest radiograph is not helpful in this patient for stratification.**
MANAGEMENT

• The spectrum of inhalation injury varies widely.
• Its presence does NOT mandate intubation particularly if the airway is patent particularly if the burn is < 20% TBSA.
• In that case elevation of the head of the bed, humidified oxygen, close observation is indicated.
• Routine labs to include a VBG/ABG with co-oximetry, CXR, IV fluids, and pain medications are standard suggestions.

ED MANAGEMENT

• However, impairment of the airway can be rapid with facial/oropharynx edema making intubation difficult if not impossible.
• Early intubation should be considered (you can always extubate the patient).
• In the face of burns > 20%, unstable vital signs, and obvious burn to the face with evidence of inhalation injury rapid sequence intubation should take place forthwith.

ED MANAGEMENT

• Treatment with 100% oxygen for 6 hours for CO poisoning is a standard treatment.
• Controversy exists for testing for cyanide and/or treating prophylactically.
• Currently in most burn centers cyanide is neither treated or tested for.
• If treatment is considered the simple infusion of hydroxocobalamin is suggested.

SUMMARY

Inhalation injury is suspected:
- take a history, do physical exam.
- tests – CBC, VBG, CXR, BMP

Usual burn care

- Observe, elevate head of bed, humidified O2, monitor closely.
- Determine – airway patency, presence of hoarseness/stridor, facial burns, labored breathing or impaired oxygenation.
- STABLE

MANAGING VENTILATION

• Weingart SD.
• Managing initial mechanical ventilation in the emergency department.
• Well, you have intubated the patient, let us say the burn patient in the previous article, you can then:
  • Turn to the respiratory therapist (if you have one) to set the vent.
  • Or manage the ventilator yourself.
• This article is a primer on ventilation management by one of the leading lights in emergency medicine critical care and is an excellent review of the subject.
LUNG PROTECTIVE STRATEGY

- A lung protective strategy focuses on a low tidal volume (6-8ml/kg) to reduce over distension of the alveoli and subsequent acute lung injury (ALI).
- Make no mistake – ALI can occur in a very short period of time, < 2 hours.
- This concept of ventilation is for the patient without pre-existing lung disease: COPD, asthma – essentially most of the patients you will intubate.

LUNG PROTECTIVE STRATEGY

- **Mode** – assist-control (AC) is the easiest and most common ventilator mode used in the ED, it prevents patient fatigue but offers full ventilatory support, once selected only five other settings need to be considered.
- **Tidal Volume** – for alveolar protection, start at 8 ml/kg predicted body weight, one may decrease the TV depending on other parameters.
- **Inspiratory Flow Rate** – for patient comfort, a normal breath starts with a high flow rate tapering to small flow, decelerating, this is hard for a ventilator to do, a rate of 60 L/min is adequate for most patients.

LUNG PROTECTIVE STRATEGY

- **Respiratory Rate** – for titrating ventilation, PaCO2 is chosen according to the acid-base balance of the patient, rates up to 30-40/min are acceptable and necessary in some patients, a rate of 15-16/min to start results in normocapnia in most patients, ETCO2 often underestimates PaCO2 in patients with a lot of alveolar shunt or dead space, will need ABG.
- **PEEP and FiO2** – are for titrating oxygenation, if the FiO2 is > 50% and continued hypoxia is due to shunt, PEEP reduces shunt by opening up collapsed alveoli and keeping them open, hyperoxia is common post-intubation and rapidly injurious, unless the patient was overtly hypoxic start with an FiO2 of 30-40% and a PEEP of 5 cmH2O, then pull up the ARDSnet FiO2/PEEP table and titrate from there according to the ABG.

LUNG PROTECTIVE STRATEGY

- **Plateau Pressure** – this final parameter looks to protect the alveoli from overdistension and possible rupture, the key parameter is a plateau pressure that is the alveolar pressure after full inspiration and reflects the pressure in the large airways and alveoli, it can be easily measure with any modern ventilator by with an inspiratory pause (ask the respiratory therapist to measure this), the goal is a pressure < 30 cmH2O, if it is > 30 reduce the tidal volume 1-2 ml/kg, TV as low as 4 ml/kg are sometimes necessary.

LUNG PROTECTIVE STRATEGY

- **Obstructive** – in the patient with underlying lung disease, COPD or asthma, a lung protective strategy has be altered to an obstructive one.
- **Bronchospasm** is a problem that a ventilator has difficulty overcoming.
- Intubating and placing a patient on a ventilator is great for getting air in, not so good at getting air out – key goal is to allow sufficient time to exhale.
- **BEST STRATEGY IN THESE PATIENTS IS NOT TO INTUBATE THEM**.
- Very deep sedation is necessary in these patients once intubated, if one has to paralyze try to do so for as short a period of time as possible.

<table>
<thead>
<tr>
<th>FiO2</th>
<th>PEEP</th>
</tr>
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<tbody>
<tr>
<td>0.3</td>
<td>5</td>
</tr>
<tr>
<td>0.4</td>
<td>5-8</td>
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<tr>
<td>0.5</td>
<td>8-10</td>
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<tr>
<td>0.6</td>
<td>10</td>
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<tr>
<td>0.7</td>
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<td>0.8</td>
<td>14</td>
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<tr>
<td>0.9</td>
<td>14-18</td>
</tr>
<tr>
<td>1.0</td>
<td>20-24</td>
</tr>
</tbody>
</table>

ARDSnet FiO2/PEEP Table

Do not bother memorizing this, it is readily available on your computer.
OBSTRUCTIVE STRATEGY

- **Mode** – AC as usual.
- **Tidal volume** – the usual 8 ml/kg predicted body weight as before.
- **Flow** – some experts advise an increased flow, 60-80L/min is sufficient for the majority of these patients.
- **Rate** – this is the primary titratable parameter to allow for extra exhalation time, a reduced rate starting at 8-10/min, permissive hypercapnia is often necessary in these patients.

PEEP and FiO2 – usually an FiO2 of 40% will allow a SpO2 > 88%, higher levels may be necessary, there is no good data to support the use of PEEP in bronchospastic patients in the first hours of ventilation, a PEEP of 0 is recommended.

- **Peak pressure alarm** – peak pressures reflect airway resistance in obstructive large airways and is not translated to the alveoli and so high PP’s do not harm them or the lung parenchyma, the alarm must be increased often to very high levels to prevent the ventilator from terminating a breath prematurely when the alarm pressure has been hit, however plateau pressures should be < 30 cmH2O as usual.

OBSTRUCTIVE STRATEGY

- **Checking for safety and further titration** – watching that the patient has adequate time to exhale involves looking at both the flow/time graph and plateau pressure, the flow/time graph will show if the patient has fully exhaled before the next breath is initiated, or the plateau pressure can be measured and if it is > 30 the issue is not TV but time to exhale, reducing respiratory rate and/or increasing the I:E ratio from its usual 1:2 to 1:3 or 4 will prevent breath stacking.

FLOW/TIME GRAPH shows that the next breath is initiated prior to full exhalation resulting in air trapping or auto-PEEP that will continue to rise resulting in lung overexpansion, pneumothoraces, and hypotension as the heart is squeezed between the lungs.

SUMMARY

- Setting of the ventilator post-intubation can be intimidating but, cannot be abrogated completely to the respiratory therapist.
- For the majority of patients without pre-existing lung disease a lung protective strategy is the choice however, for patients with pre-existing lung disease and or bronchospasm an obstructive strategy is recommended.

**Lung protective strategy**

- **Mode** – assist control
- **TV (alveolar protection)** – start at 8 ml/kg predicted body weight.
- **Respiratory flow rate** (patient comfort) – start 60 L/min.
- **Respiratory rate** (titrating ventilation) – start 15-16/min, titrate to PaCO2.
- **PEEP and FiO2** (titrate oxygenation) – for those patients not hypoxic start PaO2 > 30-40% and PEEP = 3cmH2O titrate according to ARDSnet FiO2/PEEP chart.
- **Alveolar safety** – plateau pressure < 30cmH2O, reduce TV to achieve that pressure.
SUMMARY

• Obstructive strategy:
  • Mode – AC
  • TV – start 8 ml/kg ideal body weight
  • Flow – 60-80 L/min.
  • Respiratory rate – adjust to allow time to exhale, start at 8-10/min.
  • PEEP and FiO2 – start FiO2 at 40%, goal SpO2 = 88%, PEEP = 0.
  • Peak pressure alarm – adjust so inspiration is not cut off by high peak pressures to allow full TV of 8 ml/kg.
  • Safety and further titration – measure either plateau pressure (< 30 cmH2O) and/or flow/time graph to look for breath stacking and full expiration before the next breath is initiated, reduce RR and/or adjust I:E ratio to 1:3 or 1:4 to increase exhalation time.

MESENTERIC ISCHEMIA

• Clair DG, Beach JM.
• Mesenteric ischemia.
• A review of arterial obstruction of the intestinal vessels out of the Cleveland Clinic department of vascular surgery.

TYPES

• Arterial occlusion is the most common type of mesenteric ischemia with both acute and chronic forms:
  • Acute embolic – 40-50% of cases.
  • Acute thrombotic – 20-35%, often thrombosis on an area of atherosclerotic narrowing.
  • Acute dissection or inflammation – < 5%.
  • Chronic form – 90% due to atherosclerosis with treatment usually revascularization.
  • Mesenteric versus thrombosis:
    • 5-15% of cases.
  • Primary idiopathic thrombosis with 90% due to some form of thrombophilia, trauma, or local inflammation such as pancreatitis or diverticulitis.
  • Non-occlusive mesenteric ischemia:
    • Mesenteric bed is a high resistance circuit, vasospasm can account for 5-15% of cases.

ANATOMY

Three main vessels:
- celiac artery
- superior mesenteric
- inferior mesenteric

Interconnections between all three tends to ensure that occlusion of one does not typically lead to catastrophic ischemia.

CAUSES OF ALTERED CIRCULATION

• Once occlusion of an artery occurs vasodilatation is the first response, as continued ischemia continues vasoconstriction results worsening intestinal perfusion even if blood flow returns to normal.
  • Inflammatory response cause vasospasm again worsening the ischemia.

HISTORY AND PHYSICAL EXAM

• Over 70% of patients with mesenteric ischemia are female.
• Atherosclerosis, peripheral vascular disease, diabetes, CAD often accompany such patients.
• The classic “pain out of proportion to exam” is present with many patients, some with epigastric bruits, but many do not have such symptoms.
• Differentiating venous from arterial obstruction can be difficult though venous mesenteric obstruction tends to manifest less acutely and can be associated with a history of cancer, DVT, portal vein thrombosis, inflammatory bowel disease, and thrombophilia.
CHRONIC MESENTERIC ISCHEMIA

• Typically these patients have had ongoing abdominal pain that has defied diagnosis with often extensive past work-ups and even surgery.
• Common symptoms include pain, early satiety, pain ~ 30 minutes after eating, diarrhea, and weight loss is very common that is not common with other entities in the differential such as cholecystitis, irritable bowel, and PUD.
• As many chronic mesenteric ischemia patients are older and smoke cancer needs to be ruled out in the work-up.

WORK-UP

• Common blood studies include a CBC and BMP looking for leukocytosis and metabolic acidosis.
• An elevated lactate often indicates severe ischemia or even irreversible injury.
• Duplex ultrasonography of the mesenteric vessels is highly accurate with a sensitivity of 85-90%.
• CTA has an accuracy of 95-100% and is now the gold standard test both to diagnose the obstruction but can identify a source of emboli, and can show other findings such as bowel wall thickening, mesenteric stranding, pneumatosis, or free air or portal vein gas.

WORK-UP

• Endoscopy is often part of the work-up, sometimes before the patient presents to the ED.
• It is most useful in diagnosing conditions other than ischemia such as PUD, inflammatory bowel disease, cancer.
• However, ischemic changes in the stomach or bowel wall can support the diagnosis of mesenteric ischemia.
• Selective catheter angiography can both define the lesion but also offers the opportunity for thrombolysis, angioplasty with or without stenting, or arterial vasodilation.

ED INITIAL THERAPY

• Fluid and electrolytes management:
  • Fluid loss is often substantial in patients with mesenteric ischemia particularly in more severe cases with severe metabolic acidosis and hyperkalemia are extant.
  • Resuscitation with isotonic fluid and blood is recommended and often in large volumes.
  • Pressors are suggested only as a last resort.
• Anticoagulation with heparin should be initiated early regardless of acute or chronic mesenteric ischemia.
• prophylactic antibiotics are a bit controversial but their use has been associated with improved outcomes (R Flagg) + Cipro, no recommendations were made.
• Surgery consultation is necessary in most cases.

TREATMENT OPTIONS

• The authors go into the various therapy options that in general are not decisions emergency physicians will make including:
  • Endovascular repair – in some studies is 87% successful in restoring perfusion, has a lower risk compared to surgery, and includes thrombolysis.
  • Open repair – most successful in revascularization in proximal obstructions, allows for inspection of the bowel for viability and resection of necrotic segments, short term mortality is reported to be 26-65% often due to co-morbidities.
  • For chronic mesenteric ischemia revascularization is indicated for all such patients.
  • For venous mesenteric ischemia typically anticoagulation both acutely and long term are recommended.
  • For nonocclusive mesenteric ischemia the mortality is 50-83%, local infusions of dilating agents such as papaverine have been suggested along with anticoagulation.
SUMMARY

- Mesenteric ischemia is not uncommon and is seen in 1/1,000 hospital admissions.
- The majority of cases are due to arterial obstruction by emboli or thrombosis in patients with atherosclerotic disease.
- Venous mesenteric ischemia represents a less frequent etiology with chronic mesenteric ischemia and nonocclusive ischemia being less common etiologies.
- Patients at risk include older age and those with pre-existing issues such as atrial fibrillation, CAD, peripheral vascular disease, and thrombophilia.

- Presentation tends to start with acute abdominal pain associated with hypotension and metabolic acidosis.
- The classic description of “pain out of proportion to abdominal exam” is not that uncommon but often the physical exam is non-specific.
- The work-up includes basic labs including a lactate acid that if elevated can suggest catastrophic necrosis and perforation of the bowel.
- The most accessible and best test from the ED is clearly the CTA though duplex ultrasonography is also a reasonably good test but nowhere near the accuracy of a CTA.

SUMMARY

- Fluid resuscitation starts with isotonic fluids, i.e. NS, and often in larger amounts than expected as these patients third space a lot of fluid (might want to consider less acidotic fluids in selected patients).
- Hyperkalemia is common in these patients and often needs treatment beyond just fluids.
- Blood can also be used, particularly in anemic patients.
- Antibiotics seem to help with better outcomes and are suggested.
- Surgery consultation along with interventional radiology should be done early in the course with endovascular procedures being highly effective and less invasive.

OVERVIEW

- Migraines affect 18% of women and 9% of men in the U.S.
- 1.2 million ED visits.
- Can be preceded by an aura that is a reversible neurologic phenomenon that can involve vision, speech, dizziness, or sensory disturbances seen on EEG as a slow wave of depolarization in the brain.
- Similar phenomenon can occur during the headache period but are not considered an aura.

DIAGNOSIS

- Migraine is a clinical diagnosis with no imaging or lab test available.
- The idea it is a vascular headache is not supported by advanced imaging now available.
- It is felt to be a neurologic disorder of dysfunctional sensory processing that results in non-noxious stimuli being perceived as headache, photophobia, phonophobia, and osmophobia (aversion to odors).
- It is a recurrent disorder with attacks lasting 4-72 hours typically a unilateral headache, pulsating quality, moderate to severe intensity, associated with nausea or photo/phonophobia.

MIGRAINES

- Friedman BW.
- Managing migraine.
- Part of the Expert Clinical Management series in Annals in an overview of migraine headaches from a noted expert in the field.
**DIAGNOSTIC CRITERIA**

- At least 5 attacks fulfilling at least one of the following:
  - Headache lasting 4-72 hours if untreated.
  - Headache as at least 2 of following:
    - Unilateral
    - Pulsating
    - Moderate or severe
    - Aggravated by routine activities, avoiding routine activities
  - During headache have at least one of:
    - Nausea/vomiting
    - Photophobia, phonophobia
  - Not attributable to another disorder.

**SCREENING QUESTIONS**

- Compared to expert opinion asking the following questions has a sensitivity of 81% and specificity of 75% for a positive screen of 2 of 3 positive responses:
  - During the last 3 months has the patient had any of the following with their headache?
    - Nauseated during the headache.
    - Bothered by light more than when not having the headache.
    - Has the headache limited work, study, or other activities for at least 1 day.
  - This screening question set has not been evaluated in the ED setting.

**WORK-UP**

- Routine labs or CT/MRI imaging are unlikely to contribute to management.
- This assumes no red flags: thunderclap headache, focal neurologic deficit, fever, trauma, or altered mental status.

**TREATMENT**

- Three classes of medications have assumed a first-line therapy recommendation for migraines:
  - **Antidepressants** – metoclopramide, prochlorperazine, droperidol, haloperidol.
  - **Triptans** – sumatriptan.
  - **NSAIDs** – ketorolac.
- Opiates have a complex relationship with migraines and are used in some 50% of cases however, studies show it is less likely to achieve the therapeutic goal of sustained pain relief (“doctor nothing helps my headache except Dilaudid and I’m allergic to Toradol and I have a high pain tolerance so I need 4mg immediately”).

**TREATMENT**

- The only available parenteral triptan is subcutaneous sumatriptan, 6mg SC.
- Successful treatment has a NNT of 2.5 versus placebo.
- Unpleasant reactions include chest pain, flushing, or worse headache (NNH = 4) with a recurrence rate of 66% within 24 hours.
- For the patients in whom it is effective it dramatically cuts down on the number and severity of the headache particularly if used early in the onset.
TREATMENT
• Intravenous ketorolac is often used for migraines though good data to support its use is not robust.
• Ketamine 0.1mg/kg IV and propofol 30-40mg IV up to 120mg have also been found to be effective but what one should prescribe after the headache is improved is unclear.
• Magnesium has been suggested for treatment but has not consistently shown a benefit.
• IV fluids are often given but its benefit is unclear though unlikely to harm.

DISCHARGE
• For most patients the headache will recur with 2/3's within 24 hours.
• Parenteral dexamethasone (10mg IV) is moderately effective in mitigating the recurrence with a NNT = 9.
• Naproxen 500mg or sumatriptan 100mg with help some patients, not all.
• In more refractory cases beta-blockers or the antiepileptic topiramate (Topamax) might be considered.

SUGGESTED TREATMENT REGIMEN
• Metoclopramide 10mg IV over 15 minutes + diphenhydramine 50mg IV (or prochlorperazine 10mg).
• If not improved repeat antidopaminergic + ketorolac 30mg IV.
• If not improved repeat antidopaminergic for 3rd time + dihydroergotamine 1mg IV over 15 minutes.
• Consider bilateral occipital nerve blocks with bupivacaine 0.5%.
• Opiates as a last resort if no improvement.
• Consider dexamethasone 10mg IV to reduce recurrence.

SUMMARY
• Migraines are extremely common, recur in most cases in the short-term, and often difficult to treat.
• The diagnosis can be more rigorous and a screening set of three questions might be useful in the ED.
• The theory that the etiology of migraines were vascular in nature is not longer thought to be true.
• Work-up with labs or imaging is not recommended assuming none of the red flags associated with the headache.

PEDIATRIC HIPS
• Wagner Neville DM, Zuckerbraun N. Pediatric nontraumatic hip pathology.
• This is a review of the nontraumatic etiologies of hip pain and pathology in children.
PRESENTATION AND EXAMINATION

• Nontraumatic pediatric hip pathology most often presents with hip pain and can be associated with thigh pain, knee pain, pain or inability to bear weight, and an altered antalgic gait.
• Examination begins with the stance of the hip at rest often being in flexion and external rotation.
• Palpation for tenderness over the bony prominences coming down along the thigh and knee.
• ROM is an important aspect of the exam and compared to the contralateral side, limitation to internal rotation indicates a problem with the hip joint itself.
• Including the back in ones exam is also important looking for limitation of motion and tenderness to palpation.

X-RAY IMAGING

• Plain radiographs are almost always ordered though in children younger than 9 y/o only 1% of such images were positive.
• Position of the epiphyses becomes more important in older children with comparison views of the contralateral hip can be particularly important.
• Fractures are uncommonly seen.

ULTRASOUND

• US is used primarily to detect joint effusions and is superior to plain X-rays for that purpose.
• The hip should be positioned in slight abduction and modest external rotation.
• The linear probe is placed in parallel to the long axis of the femoral neck.

MAGNETIC IMAGING

• MRI gives very detailed pictures of the hip and are helpful when other modalities have not shown an etiology.
• It is a more difficult exam to obtain and may require sedation in younger children.
• As such it is not a routine exam and most often reserved in cases where more detail of the soft tissue and joint space might be necessary.

LABS

• Primary indication for labs is when infection or malignancy is suspected.
• CBC, C-reactive protein, and erythrocyte sedimentation rate are the typical labs ordered along with blood cultures is the patient suspected of infection.
• In patients with a history of tick exposure a Lyme titer is suggested.
• Synovial fluid can extremely helpful looking for crystals (uncommon in children), bacteria, and cell counts.

TRANSIENT SYNOVITIS

• TS is a self-limited inflammation and effusion of the hip of unknown etiology that is the most common diagnosis of nontraumatic pediatric hip complaints.
• It is typically unilateral and more common in males with a mean age of 4.7 y/o with a range of 3-8 years old.
• A history of a preceding URI, GI infection, urinary infection, or minor trauma may be elicited, in one study of 383 children 40% had a preceding URI within 2 weeks of presentation with TS.
TRANSIENT SYNOVITIS

• History – acute onset of unilateral hip pain radiating to the groin or knee, unwilling to bear weight or limp, otherwise the child looks well and nontoxic.
• PE – hip is held in flexion, abduction, and external rotation with pain on movement and weight bearing.
• Dx – largely clinical, high likelihood in proper age, no trauma or fever, Sx < 1-2 weeks, and improved movement and gait with a dose of NSAID's is consistent with TS.

TRANSIENT SYNOVITIS

• Work-up – labs and X-rays typically unnecessary, US is often done if an effusion is suspected.
• Clinical course – TS typically resolves in 3-10 days, in a study 60% resolved in 7 days, 100% by 14 days.
• Management – NSAID's, weight bearing per pain tolerance, return for repeat evaluation if develop fever, looks toxic, Sx’s not improving.

LEGG-CALVE-PERTHES DISEASE (LCPD)

• LCPD is an aseptic, noninflammatory, self-limited, idiopathic aseptic necrosis of the capital femoral epiphysis, the devascularization restores itself in 1-2 years.
• Primarily effects children 3-12 years old, 4x more common in males, obesity and hypercoagulability are risk factors, incidence 0.2-19/100,000 children.

LCPD

• History – usually subacute with weeks to months of limp with pain in the hip, groin, thigh, or knee – occasionally a history of minor trauma is reported.
• Exam – limited ROM of the hip with abduction and internal rotation seen first, Trendelenberg gait (leans over the affected leg during ambulation).
• Diagnosis – X-rays establish the aseptic necrosis though early X-rays can be normal as it can take months for the bony changes to be seen, MRI can also be used.
• Acute management – as resolution can take years, permanent arthritis is a common sequelae referral to a pediatric orthopedic surgeon is recommended, in the ED it is primarily diagnosis.

SLIPPED CAPITAL FEMORAL EPIPHYSIS (SCFE)

• SCFE is displacement of the capital femoral epiphysis along the epiphyseal plate felt to be due to the fragile blood supply of that area.
• Incidence – 10/100,000 children, mostly males, mean age 12 y/o, range 10-16 years, obesity is a risk, 6-22% have bilateral disease, 24% go on to develop SCFE in the contralateral hip.
• Atypical presentations outside the usual age range can occur in patients with endocrine diseases such as hypothyroidism or growth hormone deficiency.*

SCFE

• History – subacute presentation of hip pain and limp over months with pain radiating to the groin, thigh, or knee, minor trauma might be reported.
• Knee pain is the prominent symptom in 15-50%.
• Altered gait is usually painful, hip is held in abduction, flexion and external rotation with limited internal rotation and flexion.
• Overall the child is nontoxic.
DIAGNOSIS OF SCFE

- Plain radiographs are the typical test with the use of Klein's line on the AP view of the pelvis so one can view both hips.
- A review of 60 SCFE patients found a low sensitivity of Klein's line with a miss rate of 60% leading to a recommendation of other measurements.
- Measurement of epiphyseal widths medial to Klein's line, a difference of > 2mm between the hips has a 79% sensitivity.
- Other findings include widening and irregularity of the physis, loss the round concavity of the head-neck junction.
- Use of CT, MRI, and US have been described but most patients can be diagnosed by plain radiographs.

ED management – primarily diagnosis and referral to orthopedics for surgery.

SEPTIC ARTHRITIS, OSTEOMYELITIS

- Of the issues so far discussed septic arthritis represents a true emergency.
- Delay in diagnosis and treatment can lead to irreversible destruction of the joint.
- Such infections occur in all age groups with the most common etiology being hematogenous spread from a distant source, less commonly penetrating trauma or spread from an adjacent infection.
- Joints with an intracapsular metaphysis are more susceptible to infection including the hip, knee, shoulder, and ankle.

SEPTIC ARTHRITIS, OSTEOMYELITIS

- History – acute presentation (< 1 week), fever, toxic appearance, with hot, swollen, very painful joint held in the usual position.
- Diagnosis – definitively established with isolation of a pathogen from the site of infection or a positive blood culture in a patient with inflammation of the hip.
- Blood cultures are positive in only 14–30%, joint fluid cultures positive in 30–50% but PCR can increase the positive results by 20–40%.
- Kocher criteria – fever, refusal to bear weight, ESR > 40 mm/hr, WBC > 12K had a 99.6% probability of septic arthritis if all 4 were positive - use of CRP added to the accuracy of these criteria.
- Authors recommend caution with relying too heavily on the Kocher criteria and joint aspiration should take place if the diagnosis is suspected in atypical cases.

SEPTIC ARTHRITIS, OSTEOMYELITIS

- Synovial fluid analysis – cell count, culture, gram stain are standard tests.
- Cell count – a count > 50,000 should be concerning for SA but this is not as hard and fast as one would like with studies showing SA in 48% with a cell count > 50,000 but in 17% in those < 17,000.
- Imaging – plain radiographs are usually normal, US can show an effusion, MRI is both sensitive and specific for SA and can differentiate between other causes of hip pathology including osteomyelitis.
- ED management – diagnosis, stabilization, emergent referral to orthopedics for surgical drainage, antibiotics should be started that address staph/strep and Neisseria in appropriate age patients. K. kingae has now become the most common pathogen in children < 4 y/o.

LYME ARTHRITIS

- There are a few reports of Lyme monoarticular hip arthritis though the authors do not recommend routine testing in patients thought to have TS.
- Lyme disease is most frequent in children 5-15 years old with > 1/3 presenting with signs of disseminated disease, i.e. erythema migrans and relatively brief pain in 1 or more joints.
- As in all hip pathology pain is alleviated with ROM and weight bearing but, in most cases the pain is not so severe as to prevent any weight bearing.
LYME ARTHRITIS
• Diagnosis can be challenging with synovial fluid analysis can mimic findings seen in septic arthritis, i.e. cell counts 25-75,000 being typical.
• Lab tests for Lyme, ELISA with reflex to Western blot, is recommended.
• Treatment is with antibiotics; amoxicillin in children < 8 y/o, otherwise doxycycline.

MALIGNANCY
• Malignancy is an uncommon cause of nontraumatic hip pain in children.
• Osteosarcoma is the most common bone cancer in children followed by Ewing’s sarcoma both have a peak incidence in adolescence, leukemia can cause bone pain with a limp reported in 11.6% of patients with leukemia.
• Symptoms include hip pain, mass, with 25% reporting minor trauma.
• Plain X-rays are the first imaging study as labs are typically normal.
• Treatment in the ED primarily consists of diagnosis and referral.

Erythema migrans of Lyme Disease

SUMMARY
• Nontraumatic hip pathology in children is a relatively common complaint that mostly presents with hip pain and difficulty bearing weight, the common stance in all hip problems is abduction, flexion, external rotation.
• Severe of the child complaining of knee pain as the hip is often the issue.
•Transient synovitis is by far the most common etiology in children 3-8 years old, acute onset, male predominance, who are nontoxic, may have an history of viral type illness preceding hip symptoms, comfortable at rest, the diagnosis is clinical with labs or radiographs typically normal thought US can show an effusion, pain reduction with an NSAID is almost diagnostic, treatment is symptomatic with resolution over 7-10 days in most cases.

SUMMARY
• Legg-Calve-Perthes disease is an idiopathic aseptic necrosis of the hip that revascularizes over 1-2 years, predominantly in males 2-12 years old, subacute onset, obesity and hypercoagulability are risks, plain X-rays are typically diagnostic, Trendelenberg gait can be seen, treatment is referral.
• SCFE is seen in older children until to be more prevalent, 10-16 years old, the capital epiphysis slips off the metaphysis, gait is painful and antalgic, pain can be primarily in the knee, plain X-rays are typically diagnostic with Klein’s line not as diagnostic as once taught, a better measure is comparing the width of the epiphysis that should not be > 2mm difference, treatment is referral to orthopedics for surgery.

SUMMARY
• Hip joint infections are devastating to the joint, child is typically toxic looking and often febrile, hematogenous spread of gram + bacteria is typical, a joint effusion is common and needs aspiration, lab work including some when are necessary along with blood cultures, cell counts are often > 50K but can be < 17K in some cases, treatment is antibiotics and emergent surgery.
• Lyme arthritis is a growing source of hip pain, history of exposure is helpful, erythema migrans rash is classic, ELISA and Western blot testing confirm the diagnosis, treatment is amoxicillin or doxycycline.
• Malignancy is an uncommon source of nontraumatic hip pain but osteosarcoma followed by Ewing sarcoma are the typical tumors, plain X-rays are the first imaging exam with MRI giving greater detail, treatment is referral to orthopedic oncology.

THROMBOLYSIS IN PULMONARY EMBOLISM
• Long B, Kayfman A.
• Current controversies in thrombolytic use in acute pulmonary embolism.
• This is a review article covering the current uses of thrombolytics in PE.
• In a nutshell it is well established with massive PE but not really found to be beneficial in submassive PE—the rub is defining massive and submassive.
OVERVIEW
• PE's are common with a high mortality and morbidity.
• > 100,000 cases/year with an increasing incidence with age to up to 1:300 persons > 80 years old.
• Mortality overall is 17% increasing to 30-50% in massive PE's, mortality as expected increases with increasing age and co-morbidities.
• The AHA classifies acute PE's into nonmassive, submassive, and massive based on vital signs, signs of shock, and instability.
• The definitions are key as they guide therapy with thrombolysis as an established treatment.

CLASSIFICATION
• Prior classifications were anatomy based but current ones focus on hemodynamic stability:
  • Massive — pulseless, persistent bradycardia < 40, shock or sustained hypotension < 90 for > 15 minutes (or > 40% reduction from baseline) not due to dysrhythmia, hypovolemia, sepsis, or LV dysfunction
  • Submassive — with evidence of cardiac stress including RV dysfunction or EF with elevated BNP, RV dilatation on US (RV diameter/LV diameter > 0.9), BNP elevation, new RBBB on EKG, anteroseptal ST elevation or T-wave inversion.
  • Nonmassive = no signs of clinical instability or hemodynamic compromise.

TREATMENT
• The rational for thrombolytics is that the right side of the heart does not tolerate the increase in resistance though the pulmonary circuit for very long and the increase in pulmonary hypertension and RV strain are the leading cause of death if the patient survives the initial insult.
• Thrombolytic contraindications include prior ICH, known cerebrovascular disease, malignant intracranial lesion, stroke within 3 months, recent surgery on brain or spinal cord, or recent CHI or facial trauma with fracture or intracerebral injury.

RECOMMENDATIONS — CARDIAC ARREST, MASSIVE
• Thrombolysis in cardiac arrest — CTA is not feasible, US can demonstrate RV dysfunction, consideration should be given to systemic thrombolytics (most available), catheter-directed thrombolytics, or surgical embolectomy.*
• Thrombolytics in massive PE — thrombolysis is advised in massive PE with several trials showing improved mortality and morbidity with a decrease in the risk of death or recurrent PE of 9.4-19%, NNT = 10, NNH = 8.

RECOMMENDATIONS - SUBMASSIVE
• Results for thrombolysis in submassive PE overall and in a number of studies and meta-analyses has been discouraging to say the least.
• Varying outcomes and definitions muddy the water but, overall no benefit in mortality has been demonstrated.
• MOPPET-3 — no mortality benefit over placebo group.
• PATH — what reduction in mortality was found by increase mortality from bleeding.
• TOPCAT — did show a better result with thrombolysis but only had 83 patients.
• Meta-analysis — several done, NNT = 18 that was not significant in patients > 65, NNT = 59, overall mortality benefit was 1.12%, several other meta-analyses found similar results though there was an added high level of heterogeneity and differing definitions.
• Bottomline — results of thrombolysis in submassive PE does not consistent benefit and considering the risk of adverse events makes it difficult to endorse its use.

CATHETER-DIRECTED THROMBOLYSIS
• Catheter-directed infusion of a thrombolytic with ultrasound assistance has the enticing attractions of using much less drug, fewer bleeding complications, being able to observe the results.
• There is not a huge amount of literature on this methodology and in the SEATTLE II trial including 31 massive and 119 submassive PE's treatment decreased RV dilatation, reduced pulmonary HTN, decreased clot burden, with only 1 major bleed and no ICH (impact on mortality was not reported).
• It requires rapid access to interventional radiology that may not be widely available in many EDs.
SHARED DECISION MAKING

* The authors comment that shared decision making is a strategy for engaging the patient and/or family in a therapy that has substantial risks.
* This is typically not going to be feasible in a cardiac arrest and/or massive PE when the patient is unstable.
* For submassive PEs where the data is less clear one might engage the patient and family in discussing the risks of bleeding, ICH, and the potential for benefit before undergoing treatment.

SUMMARY

* In the ED for patients who have suffered a cardiac arrest due to a suspected PE all organizations (including ACEP, AHA, ACCP, etc.) support the empiric use of thrombolytics – alteplase 100mg given as a 10mg bolus and infusion of the rest.
* In a massive PE with unstable vital signs, shock, where there are US signs of PE coupled with the potential for rapid deterioration the use of thrombolytics is recommended by systemic infusion, catheter-directed, or surgery as a consideration if available.
* With a submassive PE and relatively stable patient in a patient with no prior lung disease and good pulmonary reserve the use of thrombolytics is not well supported.
* However in a patient with a submassive PE and prior lung disease and/or multiple co-morbidities there might be some benefit to thrombolysis recognizing the increased risk of bleeding, shared decision making might be of benefit in such a case.

NEW-ONSET SEIZURES

* Gavvala JR, Schuele SU.
* New-onset seizure in adults and adolescents: a review.
* JAMA 2016;316:2657-2668.
* This was a literature review of articles from 1976-2016 on the topic from physicians from Baylor and Northwestern.

OVERVIEW

* ~8-10% of the population will experience a seizure at some point in their life with 2-3% going on to develop epilepsy.
* In the ED the first step is to distinguish an actual seizure from mimics such as TIA, migraine, syncope, or factitious events.
* A common definition of epilepsy is 2 unprovoked seizures > 24 hours apart though more current definitions include 1 unprovoked Sz with a probability of further seizures of at least 60% – that seems more the providence of a neurologist.

CLINICAL PRESENTATION

* The diagnosis is largely clinical based on history, often the actual event goes unobserved.
* Seizures can be classified as generalized (bilateral neural networks) or focal (involving one lobe or hemisphere).
* The differential is broad including migraine, TIA, movement disorders, sleep disorders, or psychogenic.
* Recent studies put the incidence at 23-61/100,000/year for a single unprovoked seizure.

RISK FACTORS FOR EPILEPSY

* Risk factors for epilepsy include:
  * Family history of seizures.
  * Sleep deprivation.
  * Medications that lower seizure threshold – clozapine, cephalosporins, fluoroquinolones, bupropian, tramadol to mention commons ones.
  * Metabolic derangements and toxin exposures.
  * Child development or birth complications.
  * Brain injury – infection, traumatic, CNS diseases, prior neurosurgery.
HISTORY AND PHYSICAL

- Diagnosis remains largely clinical so a history of aura, focal seizures, lack of recall of the event, prior staring spells or myoclonic jerks out of sleep, or psychiatric issues in addition to risk factors can bolster or eliminate the diagnosis.
- PE demonstrating a post-ictal state, oral injury (biting the tongue), bruises from a fall, back pain c/w compression fracture suggest a seizure.
- The presence of asterixis or nuchal rigidity suggests a systemic metabolic or infectious disorder.
- Skin findings of neurofibromatosis, tuberous sclerosis, or Sturge-Weber syndrome can suggest etiologies as well.

WORK-UP

- Brain imaging – with a 1st seizure neuroimaging should be obtained starting usually with a plain head CT but, certain lesions are not well seen on CT.
- MRI is a better test to pick up more subtle findings such as a low grade glioma and other brain malformations, an epilepsy specific MRI protocol is suggested.
- EEG – can be safely delayed in most cases unless with a new onset Sz the patient does not return to baseline in 30-60 minutes, waxing/waning LOC, or structural lesion that does not explain the symptoms should be admitted for EEG and EEG monitoring.

LABS

- A chemistry panel looking for electrolyte abnormality such as hyponatremia or hypoglycemia is reasonable along with a drug screen looking for intoxication – this is relevant in ~4% of patients.
- Prolactin levels are elevated after a seizure if measured 10-20 minutes after the event, however its low sensitivity and low NPV make it insufficient to diagnose psychogenic or non-epilepsy etiologies.
- LP – unless there is a concern for CNS infection or subarachnoid hemorrhage no study has shown the systematic utility of a LP in the seizure work-up.

RISK OF RECURRENCE

- For patients with a new-onset Sz the risk of recurrence is 35% within 5 years that increases to 75% on patients who have a 2nd event.
- For patients with demonstrable structural brain lesions and/or EEG abnormalities have a much higher recurrence rate.
- In patients whose seizure is trauma related the 5-year recurrence rate is 29-48%.
- If the seizure occurs at night there is a higher incidence of recurrence compared to patients where the seizure occurs during the day – 33% vs. 54%.

TREATMENT

- The majority of patients, two thirds, do not require anti-seizure medications to be started from the ED.
- Anti-epileptics are divided into broad and narrow spectrum agents the choice is complex and depends on a number of factors including the presumed etiology of the seizure, sex, time to therapeutic onset, IV and PO formulations.
- First line monotherapy agents include fosphenytoin, valproate, and levetiracetam – consultation with a neurologist might be prudent to decide which agent to start.
- Duration of therapy is also a complex decision with 59% of patients remaining seizure free after 2 years of treatment and no recurrent Szs.

SUMMARY

- Seizures are not an uncommon presentation to the ED with a number of etiologies including primary epilepsy, seizures due to structural brain lesions, CNS infection, or trauma.
- Diagnosis is primarily clinical and measurement of risk factors can be helpful.
- Exam may demonstrate a post-ictal state, oral injury, and bruising from falls.
- Evidence of infection including fever, nuchal rigidity, or asterixis can point to infection or toxic etiologies.
SUMMARY

Work-up in the ED would include neuroimaging for first time seizures (CT or MRI, unless contraindicated), and drug screens – if only if infection or a late CNS bleed is suspected.

Most patients do not require EEG from the ED save in selected patients who do not regain their baseline CNS status within 60 minutes.

Most ED patients do not need to be started on anti-seizure medications, if medications are started a neurology consultation is helpful and typically a broad spectrum agent is chosen.

CLASS OF EVIDENCE AND RECOMMENDATION LEVELS

- **Level A** – generally accepted principals reflecting a high degree of clinical certainty based at least 1 study with Class of Evidence I level or multiple Class of Evidence II studies.
- **Level B** – recommendations of moderate clinical certainty based on evidence from 1 or more Class II or strong consensus of Class III studies.
- **Level C** – recommendations based only on Class III studies and/or expert opinion.

QUESTION 1

- In adult patients with suspected TIA, are there clinical decision rules that can identify patients at very low short-term risk for stroke who can safely be discharged from the ED?
  - No Level A or C recommendations.
  - Level B – do not rely on current existing risk stratification instruments to identify TIA patients who can be safely discharged from the ED including age, BP, clinical features, duration, DM, or the ABCD2 score.

QUESTION 1 - RISK STRATIFICATION

- 378 articles, 72 selected, 34 applied to the question.
- Pretest probability for the short-term risk of stroke can be estimated in 3 general ways:
  - Objective criteria such as risk stratification tool.
  - Clinical gestalt.
  - Extrapolation from studies reporting on post-TIA stroke in similar populations.
- The ABCD2 score is the most commonly cited score used for TIA.
- In the 2009 guidelines on TIA by the AHA/ASA was that the ABCD2 score be used to decide hospital admission for those with a score > 3, a score of 0-2 if work-up could not be completed within 2 days, or other evidence of event caused by focal ischemia.

ABCD2 SCORE

<table>
<thead>
<tr>
<th>RISK FACTOR</th>
<th>POINTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt; 60</td>
<td>1</td>
</tr>
<tr>
<td>BP &gt; 140/90 mmHg</td>
<td>1</td>
</tr>
<tr>
<td>Clinical features – unilateral weakness</td>
<td>2</td>
</tr>
<tr>
<td>Clinical features – language disturbance without weakness</td>
<td>1</td>
</tr>
<tr>
<td>Diabetes</td>
<td>1</td>
</tr>
<tr>
<td>Duration &gt; 60 minutes</td>
<td>2</td>
</tr>
<tr>
<td>Duration 10-90 minutes</td>
<td>1</td>
</tr>
<tr>
<td>Duration &lt; 10 minutes</td>
<td>0</td>
</tr>
</tbody>
</table>

A score < 4 identified 34% of patients with a low risk of stroke occurring in 1% and 1.2% of patients in 2 and 7 days.
QUESTION 1 – RISK STRATIFICATION

• The authors found the largest studies with the highest Class of Evidence did not show that posttest probability of 2-7 day risk of stroke was sufficiently identified by the ABCD2 score.
• The literature supports two findings:
  - The ABCD2 score does not sufficiently identify the short-term risk of stroke when used alone as a risk-stratification instrument.
  - No other risk-stratification instrument demonstrated a sufficient ability to do the same.

QUESTION 2 – IMAGING DELAY

• In adult patients with suspected TIA, what imaging can be safely delayed from the initial ED workup?
  - No Level A or B recommendations.
  - Level C – The safety of delaying neuroimaging is unknown and it is reasonable, when MRI is not available, that a non-contrast head CT be part of the initial ED workup, though it should not be used to identify patients at high risk for a short-term stroke.

QUESTION 2 – IMAGING DELAY

• 441 articles, 85 selected, 13 used to address the question.
  - The primary goal of neuroimaging is to look for serious TIA mimics such as intracranial bleeding or mass.
  - A secondary goal would be to identify those patients at high risk for a stroke in 2-7 days.
  - There are 3 imaging modalities – CT, MRI, carotid vessel imaging used for TIA.

QUESTION 2 – HEAD CT

• In 2 Class II studies from Germany and Italy where all ~1,700 patients with TIA symptoms underwent a head CT.
  - Both studies found that a new infarct seen on HCT was not associated with another ischemic stroke in 2-7 days and could not predict those patients at short-term risk.
  - However, a Canadian study came to the opposite conclusion from 2,000 patients from 8 medical centers with TIA’s and an HCT within 24 hours.
  - Given the mix of data conclusions one cannot select a group of low-risk TIA patients in the ED in whom a head CT could be delayed.

QUESTION 2 – MRI, CAROTID IMAGING

• The authors state, “when feasible, . . . Obtain MRI with diffusion-weighted imaging (DWI) to identify patients at high short-term risk for stroke”.
• They further state regarding carotid imaging, “When feasible, . . . Obtain cervical vascular imaging (e.g. carotid ultrasonography, CTA, or MRA)
• Data could not select patients in the ED in whom such imaging could be delayed or low-risk of post-TIA stroke.

QUESTION 2 – IMAGING CONCLUSIONS

• A summary of this questions results in 3 key findings:
  1. Patients with a suspected TIA is likely that an initial non-contrast HCT will identify some patients with serious alternative diagnoses; however there is no evidence evaluating the safety of delaying neuroimaging in the ED.
  2. Initial non-contrast MRI appears reliably predict early stroke in suspected TIA patients.
  3. Both DWI-MRI and cervical vascular imaging are needed absent true TIA risk for stroke in patients with suspected TIA.
**QUESTION 3 – CAROTID US**

- In adult patients with suspected TIA, is carotid ultrasonography as accurate as neck CTA or MRA in identifying severe carotid stenosis?
- No Level A or B recommendations.
- Level C – In adult patients, carotid ultrasonography may be used to exclude severe carotid stenosis because it has accuracy similar to that of CTA or MRA.

**QUESTION 4 – ED DIAGNOSTIC PROTOCOLS**

- In adult patients with suspected TIA, can a rapid ED-based diagnostic protocol safely identify patients at short-term risk for stroke?
- No Level A or C recommendations.
- Level B – In adults with a suspected TIA without high-risk conditions*, a rapid ED-based protocol may be used to evaluate patients at short-term risk for stroke.

* High risk conditions: abnormal HCT, suspected embolic source including AF/cardiomyopathy/valve disease, known carotid stenosis, previous large strokes, and crescendo TIA.

**SUMMARY**

- The ACEP Clinical for patients with a suspected TIA had 4 recommendations that either Level B or C, no Level A recommendations could be established.
  1. There are no risk stratification decision rules that adequately identify patients at very low risk for short-term stroke, including those with the ABCD2 risk.
  2. The safety of delaying imaging from the ED on patients who come to the best imaging site available and would benefit from imaging should be included to identify high-risk patients.
  3. Carotid Doppler ultrasound is as good as MRA or CTA to identify severe stenosis.
  4. An accelerated observation TIA protocol for patients without high-risk conditions that includes carotid imaging, echocardiography, cardiac monitoring, and serial evaluations is safe and effective to identify patients at short-term risk for post-TIA stroke.

**AS ALWAYS – WINSTON (THE OFFICIAL MASCOT OF MCEP) HOPES THAT YOU HAVE A WONDERFUL DAY HERE IN THE WINTER WONDERLAND OF NORTHERN MICHIGAN.**