THE
2025-26

Emergency Medicine & Acute Care UPDATE

A 20-hour Course focused on Key Clinical Topics in Emergency Medicine and Acute Care

American Educational Institute, Inc.

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David R. Victor, JD
CEO

Dear Registrant:

You practice in one of the most demanding and unpredictable environments in medicine. While staying clinically current is essential, it is just as critical to refine your ability to recognize subtle presentations, avoid diagnostic pitfalls, and act decisively when seconds matter. ***The 2025–26 Emergency Medicine and Acute Care Update*** has been designed with that mission in mind.

In this course you will receive 20 hours of instruction from national experts in emergency medicine, pediatrics, radiology, critical care, and related specialties. Their lectures address the full spectrum of acute care challenges, including pediatric rashes, high-risk spinal conditions, abdominal CT interpretation, pediatric trauma management, stroke mimics, non-ACS chest pain, alcohol withdrawal, febrile infants, musculoskeletal injuries, and pediatric cardiac emergencies. You will also explore topics such as acute dizziness and BPPV, pediatric resuscitation, diagnostic error reduction, and best practices in imaging and communication.

To help you assess your comprehension, we have provided brief self-evaluations that may be taken either before or after the corresponding presentations. These tests are included in this syllabus and can be easily identified by the black edges of the pages on which they appear.

As always, I am eager for your feedback. Please take the time to complete the evaluation questions presented on screen for each lecture, and feel free to reach out directly to our faculty with questions or comments.

Finally, I encourage you to take advantage of the diversity of your colleagues enrolled in this course. Emergency physicians, primary care providers, physician assistants, nurse practitioners, and nurses all bring unique perspectives. Discussing these complex issues with peers from different disciplines will enrich your learning experience and broaden your approach to acute care.

Thank you for your participation, and please accept my best wishes for a safe, enjoyable, and rewarding course.

Cordially,
American Educational Institute, Inc

David R. Victor, Esq.
CEO

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COURSE OBJECTIVES

After completing ***The 2025-26 Emergency Medicine and Acute Care Update*** you should have acquired the knowledge that will better enable you to better:

- Recognize and manage **common and high-risk pediatric rashes** with an emphasis on accurate diagnosis, clinical context, and appropriate treatment strategies.
- Recognize and evaluate red flags in patients with **acute non-traumatic back or neck pain** that may indicate spinal cord or cauda equina compression.
- Identify **critical abdominal CT findings** to improve rapid decision-making in emergency situations.
- Improve recognition and management of sepsis and septic shock to optimize patient outcomes.
- Understand strategies for the **assessment and management of pediatric trauma**, with emphasis on imaging, airway management, and injury-specific considerations.
- Improve diagnostic accuracy by identifying **atypical stroke presentations** and distinguishing them from stroke mimics.
- Improve diagnostic accuracy for **non-ACS life-threatening causes of chest pain** using clinical strategies and imaging.
- Identify risk factors, clinical features, and evidence-based management strategies for **alcohol withdrawal** in the ED setting.
- Understand how to apply current evidence-based strategies to **evaluate and manage febrile infants and children** in the emergency setting.
- Identify uncommon but critical causes of **acute generalized weakness** and outline appropriate diagnostic and management strategies.
- Develop strategies to rapidly evaluate and manage sudden deterioration in critically ill patients.
- Recognize **high-risk musculoskeletal injuries** that are commonly missed in the emergency department and understand how to avoid associated diagnostic pitfalls.
- Recognize and manage **critical pediatric cardiac presentations**, including congenital heart disease, myocarditis, chest pain, syncope, and arrhythmias.
- Identify common pitfalls in **medical documentation** and apply best practices to reduce malpractice risk and improve patient care.
- Understand how to use a symptom-based, bedside assessment approach to differentiate benign from dangerous causes of **acute dizziness**.
- Identify the clinical features of **BPPV** and apply bedside diagnostic and therapeutic maneuvers to effectively manage it in the emergency setting.
- Improve understanding of best practices in **pediatric resuscitation** to manage critical illness and trauma across age groups.
- Evaluate evidence-based strategies for airway management, pharmacologic interventions, and post-arrest care in patients with **cardiac arrest**.
- Identify common contributors to **diagnostic error** and apply strategies to reduce preventable mistakes in clinical decision-making.
- Improve diagnostic accuracy and patient outcomes by **selecting appropriate imaging** and communicating effectively with radiology in the emergency setting.

All learning objectives above address IOM/ACGME core competencies.

THE 2025-26

Emergency Medicine & Acute Care UPDATE

FACULTY DISCLOSURES

The individuals listed below have control over the content of ***The 2025-26 Emergency Medicine and Acute Care Update***. None of them have a financial relationship with an ineligible company.

David R. Victor, Esq., CEO, American Educational Institute
Billy J. Allen, president, American Educational Institute
Michael P. Zintsmaster, MD, clinical content director
Brian S. Fuehrlein, MD, PhD, faculty member
Emily Rose, MD, FAAP, FAAEM, FACEP, faculty member
Jonathan A. Edlow, MD, faculty member
Robert J. Tubbs, MD, faculty member
Alexander D. Grushky, MD, faculty member
Michael W. Donnino, MD, faculty member
Kevin Klauer, DO, EJD, faculty member

All relevant financial relationships have been mitigated prior to the start of this activity in accordance with ACCME standards.

FACULTY

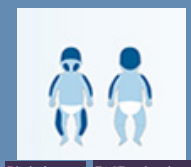
Emily Rose, MD, FAAP, FAAEM, FACEP

Emily Rose, MD, FAAP, FAAEM, FACEP, of Los Angeles, California, is the Assistant Dean for Career Advising in the Office of Student Affairs at Keck School of Medicine of the University of Southern California and Associate Professor (Educational Scholar) of Clinical Emergency Medicine and Medical Education at Los Angeles General Medical Center. A native South Dakotan, she trained in Emergency Medicine and was chief resident at Los Angeles County/ USC Medical Center and completed a Pediatric Emergency Medicine fellowship at Loma Linda University. She has been core Emergency Medicine faculty at LAC/USC Medical Center since 2010 where she continues to care for both sick/injured children and adults. Dr. Rose is a prolific educator with over 100 publications and over 1000 invited presentations locally, nationally, and internationally. Dr. Rose has been awarded several education awards including multiple LAC/USC faculty of the year awards, outstanding teaching performance, and the Honorable Mention Outstanding Speaker of the Year for the American College of Emergency Physician Scientific Assembly. She has been course director for several national Pediatric Emergency Medicine courses and has published two textbooks--one on life-threatening rashes and a practical clinical textbook for pediatric emergencies for EM-trained providers.

You may contact Dr. Rose with your questions or comments at emilyros@usc.edu.

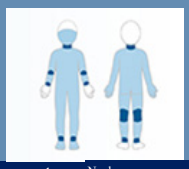
Pediatric Rashes You Can't Miss: Diagnosis, Management, and Pitfalls
Emily Rose, MD, FAAP, FAAEM, FACEP

Eczema in Children



Birth-2 years — FACE and scalp EXTENSOR surfaces

Pruritic, red, weeping or scaly and crusted lesions



2yrs-puberty — Neck, Flexural surfaces, Lichenified plaques

Localized with lichenification

Eczema Treatment

Eliminate Exacerbating Factors

HYDRATE Skin (thick creams/ointments)

Control Skin Inflammation

What is the treatment?

A. Acyclovir
B. Mupirocin
C. Frequent Bathing
D. Silvadene
E. Tar Baths

What is the treatment?

A. Acyclovir
B. Mupirocin
C. Frequent Bathing
D. Silvadene
E. Tar Baths

Eczema Herpeticum

Eczema Molluscatum

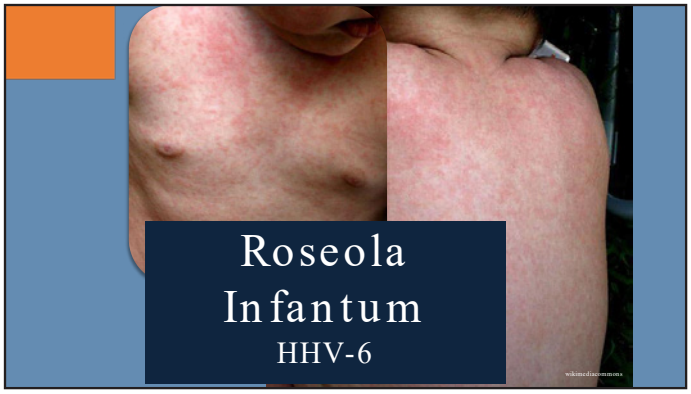

Eczema Coxsackium

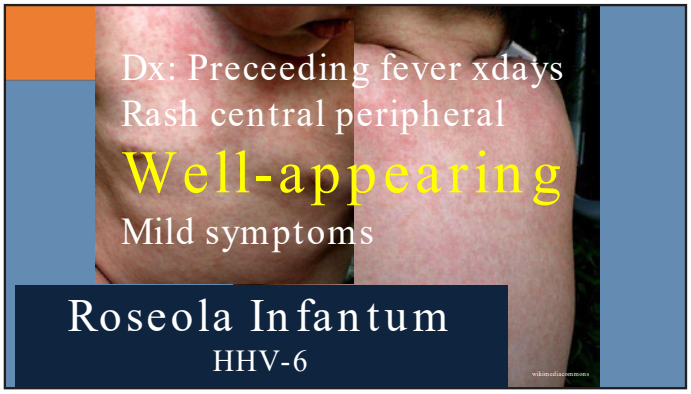

Viral Exanthem


Viral Exanthems:
Macular
Papular
Vesicular
Urticarial
Petechial…
Widespread
Localized


Slapped Cheek
Fifth Disease
Parvovirus B19

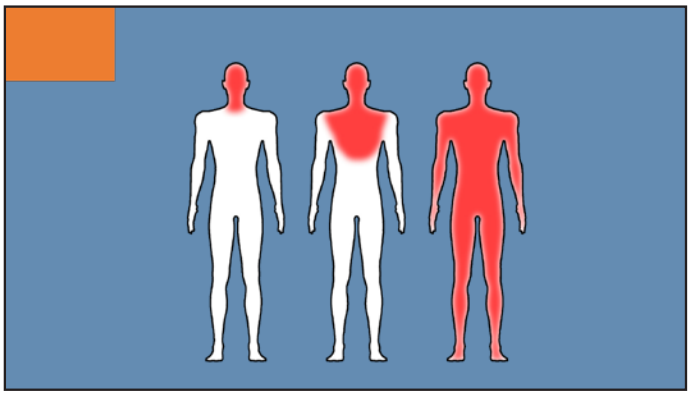

Pregnancy
Sickle Cell Disease


Roseola
Infantum
HHV-6

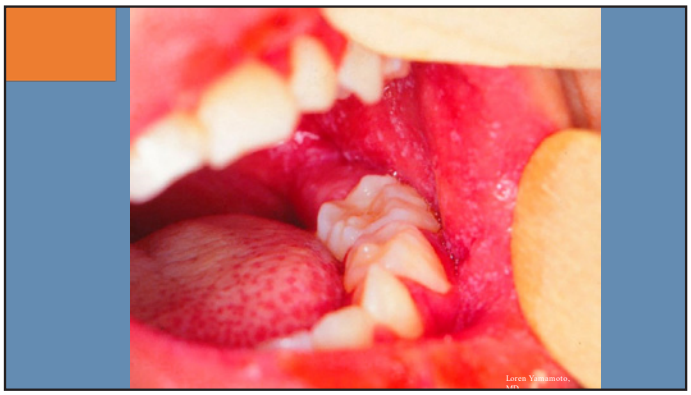

Dx: Preceeding fever xdays
Rash central peripheral
Well-appearing
Mild symptoms
Roseola Infantum
HHV-6

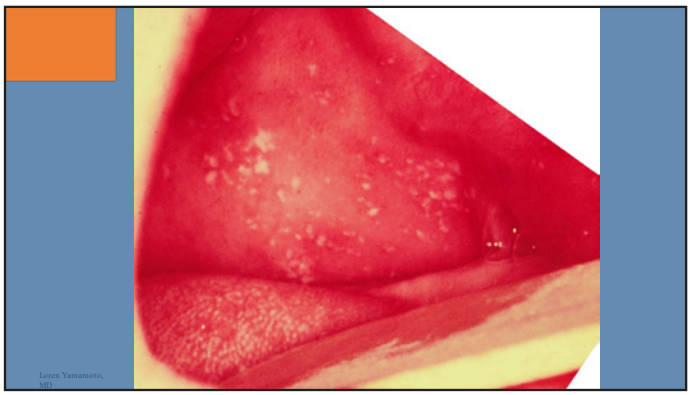

Head/Neck
Trunk/Extremities

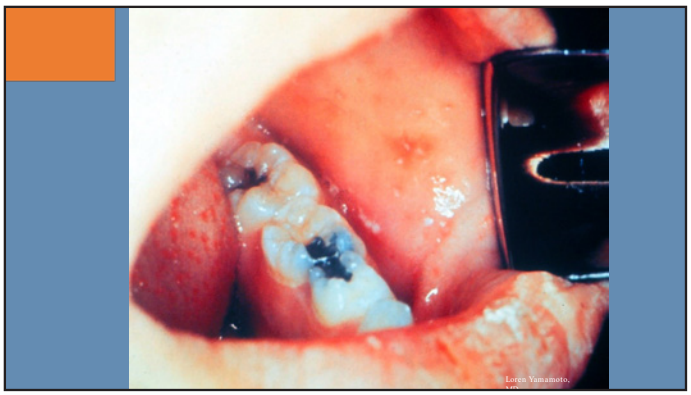

Head/Neck
Trunk/Extremities
Egg dripping down

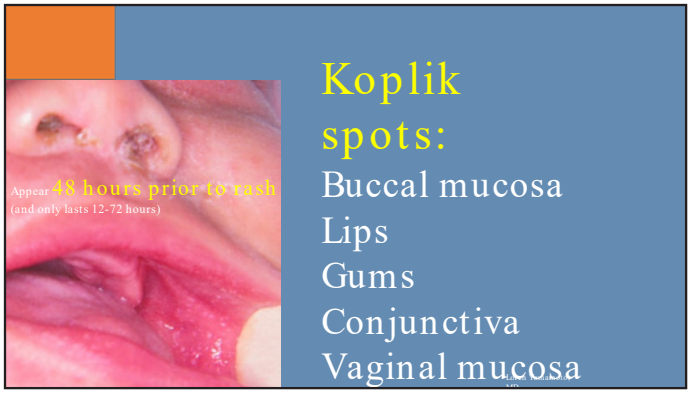



Enanthem

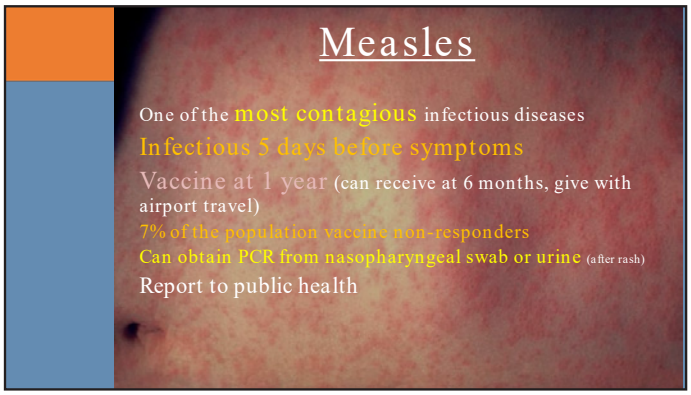




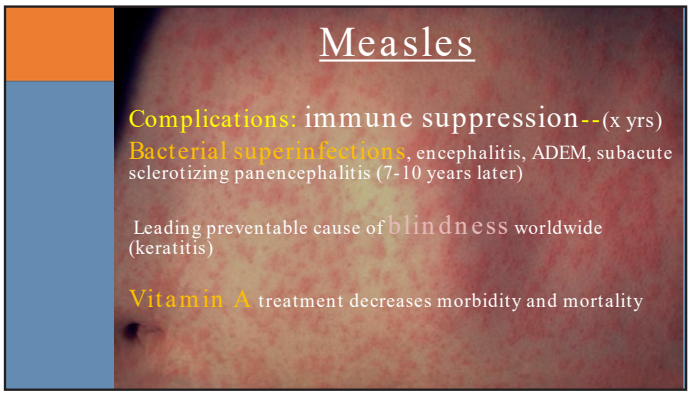



Koplik spots:
Buccal mucosa
Lips
Gums
Conjunctiva
Vaginal mucosa
Appear 48 hours prior to rash
(and only lasts 12-72 hours)


Measles
Rash head → trunk
Conjunctivitis
Koplik spots
Kawasaki mimic


Measles
One of the most contagious infectious diseases
Infectious 5 days before symptoms
Vaccine at 1 year (can receive at 6 months, give with airport travel)
7% of the population vaccine non-responders
Can obtain PCR from nasopharyngeal swab or urine (after rash)
Report to public health


Measles
Vaccine at 12 months
Can be given as early as 6 months
Booster at 4-5 years
Can be given 1 month after first shot
1 in 1000 die


Measles
Complications: immune suppression--(x yrs)
Bacterial superinfections, encephalitis, ADEM, subacute sclerotizing panencephalitis (7-10 years later)
Leading preventable cause of blindness worldwide (keratitis)
Vitamin A treatment decreases morbidity and mortality

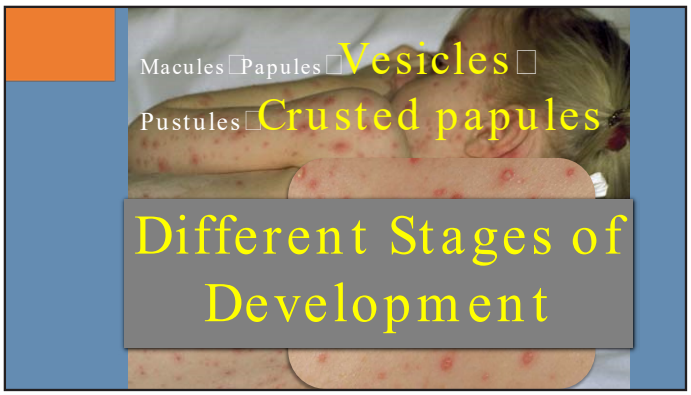
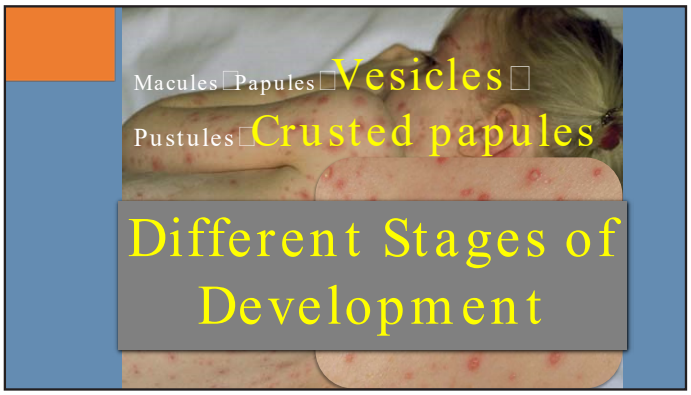

Macules → Papules → Vesicles →
Pustules → Crusted papules
Different Stages of Development

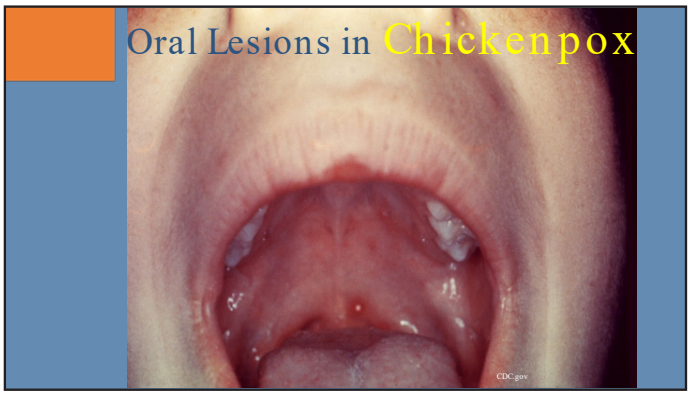

Photo courtesy Michaela Cribb


Photo courtesy Michaela Cribb


Varicella
(Chickenpox)
Photo courtesy Michaela Cribb

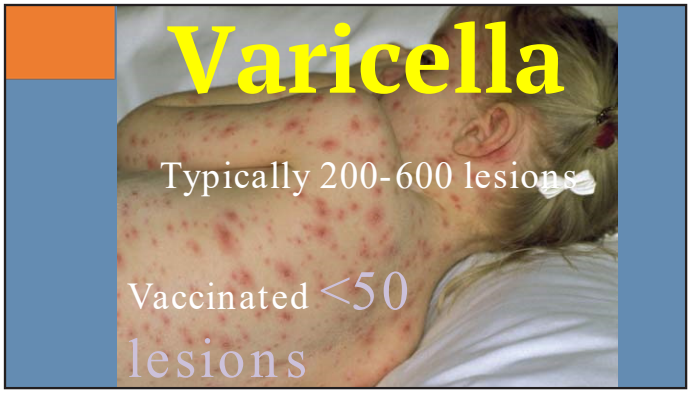

Macules Papules Vesicles
Pustules Crusted papules
Different Stages of Development

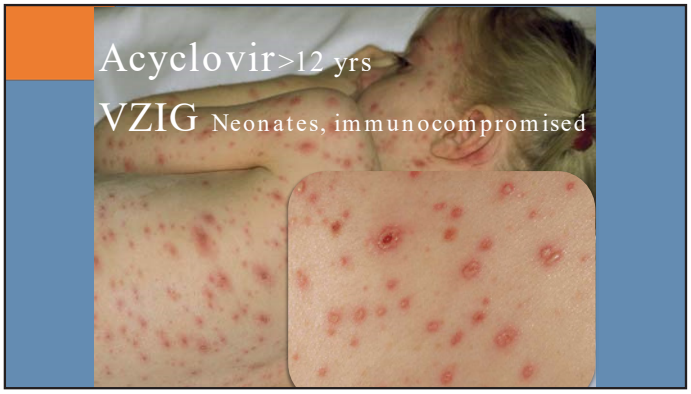

Bullous Varicella
Photo courtesy Dr. Stanley Inkelis, MD


Oral Lesions in Chickenpox
CDC.gov


Varicella
Vaccination at 12 months


Varicella
Can Recur!
Even in Vaccinated


Varicella
Typically 200-600 lesions
Vaccinated <50 lesions


Acyclovir>12 yrs
VZIG Neonates, immunocompromised


Varicella
Zoster


Varicella
Zoster
Reactivation
Prodromal pain common


Varicella
Zoster
Treatment:
Acyclovir/valacyclovir

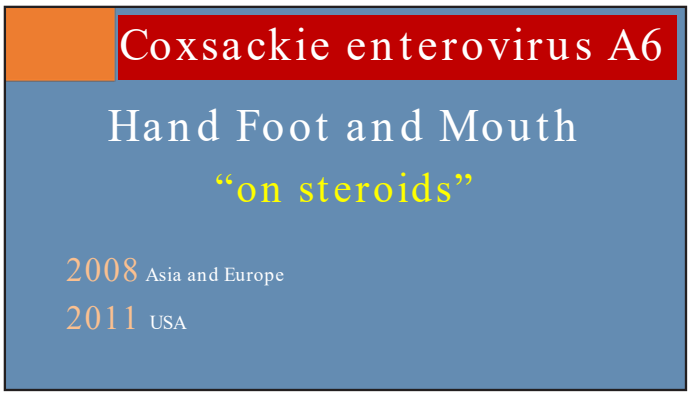

Gingivostomatitis
2/2 Herpes


Herpetic Whitlow


Herpangina


Hand Foot Mouth


Hand
Foot
Mouth


Coxsackie enterovirus A6
Hand Foot and Mouth
"on steroids"
2008 Asia and Europe
2011 USA


Feder HM Jr, Bennett N, Modlin JF.
Atypical hand, foot, and mouth disease:
A vesiculobullous eruption caused by Coxsackie virus A6. Lancet Infect Dis. Jan 2014;14(1):83-6
Coxsackie enterovirus A6


A
B
C
D

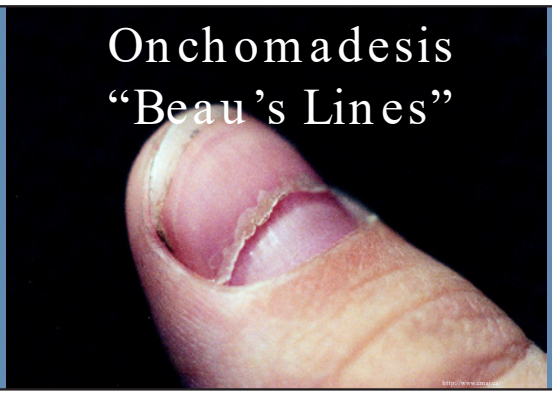

Banta J, Lenz B, Pawlak M, et al. Notes from the Field: Outbreak of Hand, Foot, and Mouth Disease Caused by Coxsackievirus A6 Among Basic Military Trainees — Texas, 2015. MMWR Morb Mortal Wkly Rep 2016;65:678–680
A
B
C
D


Photo courtesy of Dr. Richard Bracken


Photo courtesy of Dr. Richard Bracken


Photo courtesy of Dr. Nicholas

Onchomadesis "Beau's Lines"

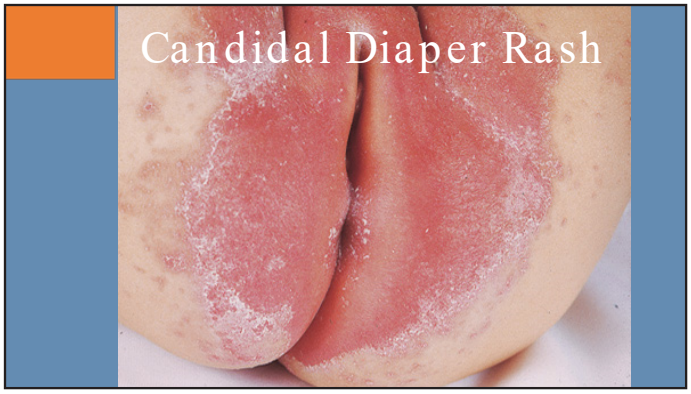


Diaper Dermatitis

Diaper Rash

Diaper Rash Rx

Minimize "wet time"
Bathe regularly
Give baby "air time" after bath
Barrier cream (zinc oxide, petrolatum jelly, aquaphor)


Candidal Diaper Rash

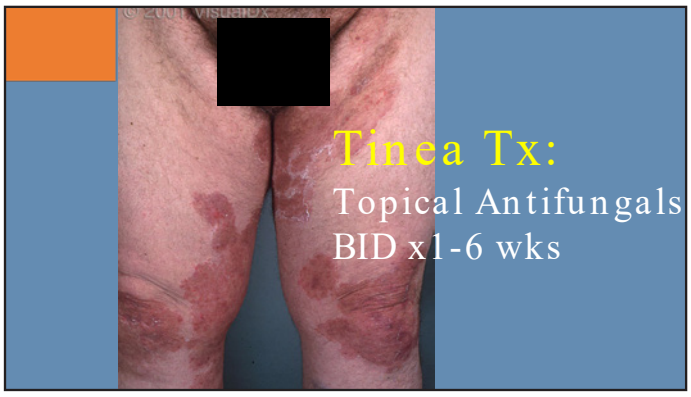

Candidal Diaper Rash
Topical nystatin, miconazole, clotrimazole


DermNet.com, Hardin MD


Tinea Corporis
DermNet.com, Hardin MD


Tinea Corporis


© 2012 VisualDx


Tinea Tx:
Topical Antifungals
BID x1-6 wks

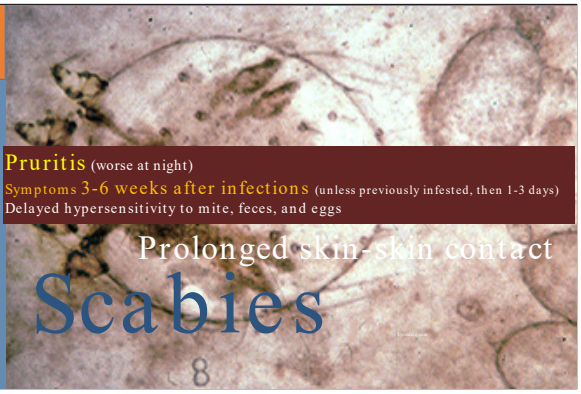

Tinea Capitis
ORAL antifungals


© 2008 VisualDx


Scabies


Scabies
Consider in
ANY
Pruritic Rash


Pruritis (worse at night)
Symptoms 3-6 weeks after infections (unless previously infested, then 1-3 days)
Delayed hypersensitivity to mite, feces, and eggs
Scabies



Scabies Home Treatment

Transmission prolonged skin to skin contact
mites survive off host 24-36 hours
Treat close contacts simultaneously
Wash clothing/bedding used in prior 3 days in hot water
Quarantine items (pillows, stuffies) for 72 hours

Scabies Treatment

Topical Permethrin 5%: massage from neck to soles (under nails)
Include head in young children (spare eyes/mouth)
30 g for one adult one time
Reapply in 1-2 weeks
Safe for use in >2 mo old, pregnant/lactating

Oral ivermectin 200 mcg/kg single oral dose
Second treatment in 1-2 weeks (IMPORTANT)
Ideal for nursing home outbreaks

Scabies Treatment

Topical Permethrin 5%: massage from neck to soles (under nails)
Include head in young children (spare eyes/mouth)
30 g for one adult one time
Reapply in 1-2 weeks
Safe for use in >2 mo old, pregnant/lactating

Pruritis persists x wks

Oral ivermectin 200 mcg/kg single oral dose
Second treatment in 1-2 weeks (IMPORTANT)
Ideal for nursing home outbreaks


Photo courtesy Dr. Dina Seif Radwan


Photo courtesy Dr. Dina Seif Radwan

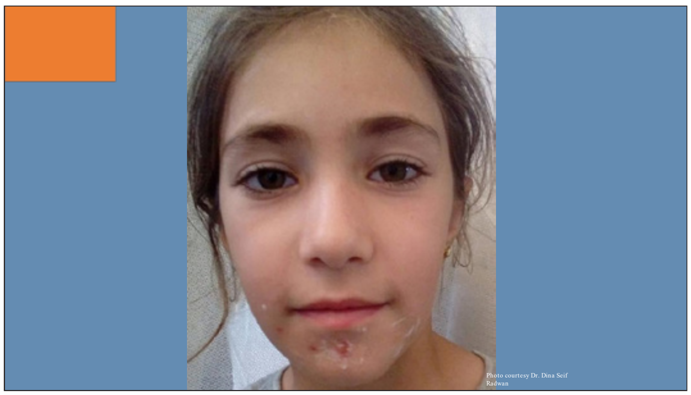


Photo courtesy Dr. Dina Seif Radwan


Photo courtesy Dr. Dina Seif Radwan


Photo courtesy Dr. Dina Seif Radwan

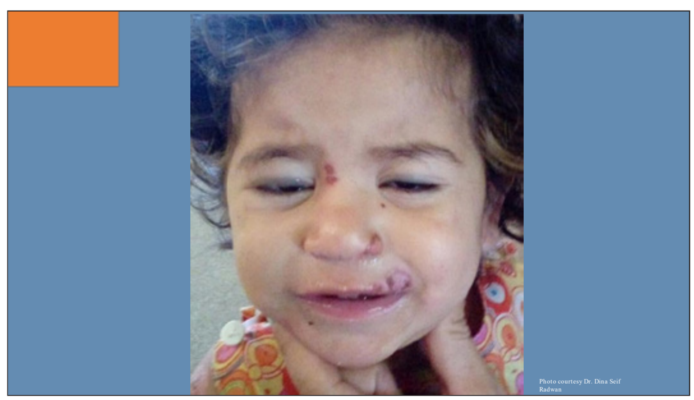


Photo courtesy Dr. Dina Seif Radwan


medscape.com

Impetigo

Face/
Hands

Papules
Pustules
Crust

Impetigo

Staph/
Strep

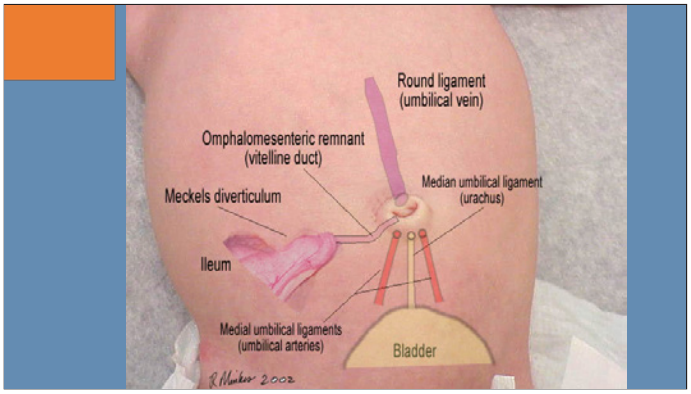
Topical
Mupirocin

Staphyloccal Scalded Skin Syndrome

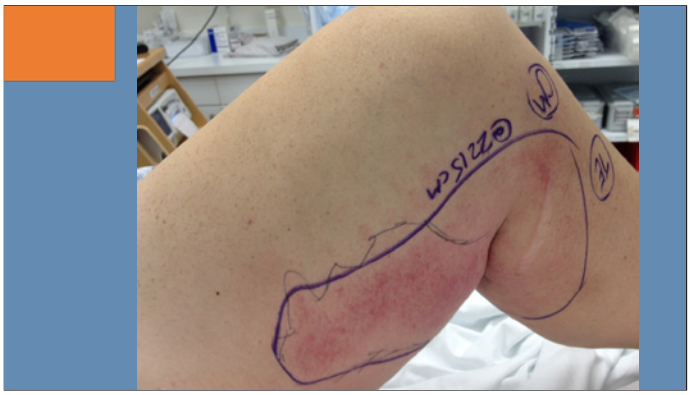
Treatment:

IVF
ABX
Wound care


Omphalitis

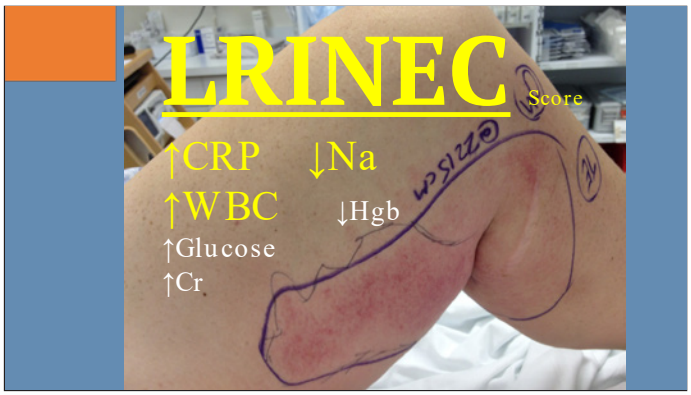

Round ligament
(umbilical vein)
Omphalomesenteric remnant
(vitelline duct)
Meckels diverticulum
Median umbilical ligament
(urachus)
Ileum
Medial umbilical ligaments
(umbilical arteries)
Bladder

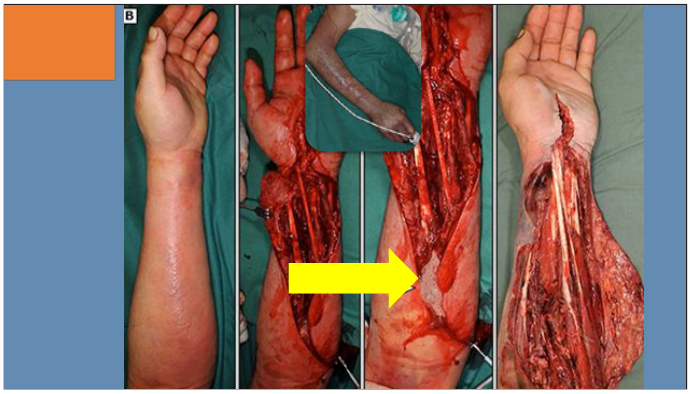

SEPSIS
PERITONITIS
NECROTIZING
FASCIITIS

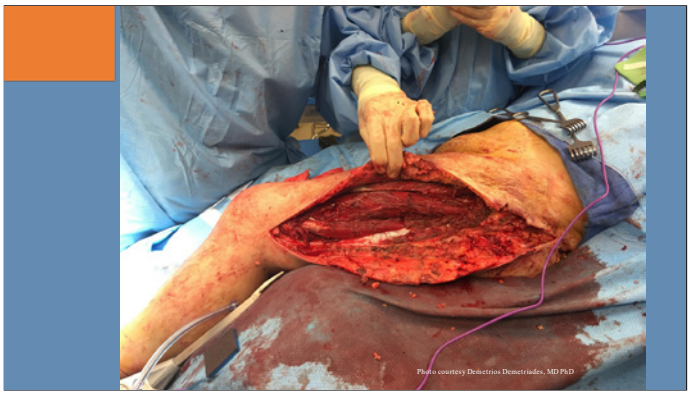



Only
50%
Febrile


Index of
Suscpicion


LRINEC Score
↑CRP ↓Na
↑WBC ↓Hgb
↑Glucose
↑Cr





Necrotizing Fasciitis

Rapid Progression
Pain out of Proportion
Subcutaneous Gas

SURGICAL EMERGENCY

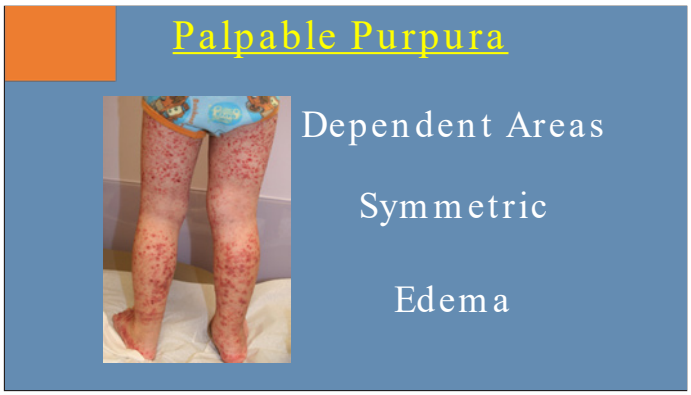

Henoch Schonlein Purpura


HSP Rash
Often Initially
Macular,
Urticarial

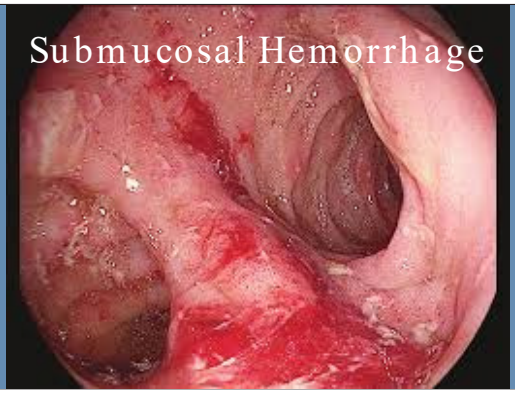

Hemorrhagic
Bullae

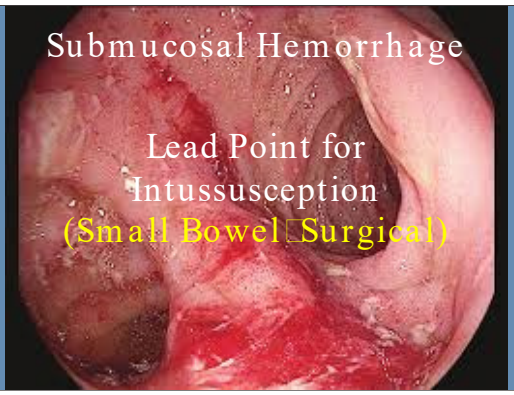

GU Pain/
Swelling
May be
ONLY
Presenting
Symptom


Henoch Schönlein Purpura
1. Palpable Purpura
2. Arthralgias/Arthritis (50-84%)
3. Abdominal Pain (50%)
4. Renal Disease (20-50%)


Palpable Purpura
Dependent Areas
Symmetric
Edema


Arthalgias/Arthritis
1-4 Joints
Hips, Knees,
Ankles





Testing in HSP?

Urine Dip in ALL patients

Serum Creatinine In all adults; If urine abnormal

CBC if atypical presentation, uncertain diagnosis

HSP Treatment

NSAIDs for pain

Steroids: decrease abdominal pain/arthritis but DO NOT impact outcome

HSP Follow Up

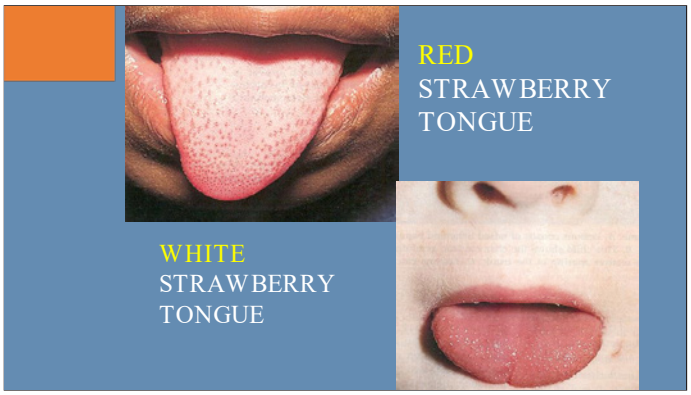
ALL need **PMD** (may develop renal sequelae)

Abnormal urine/normal Cr nephrologist follow up

Abnormal Cr nephrology consult

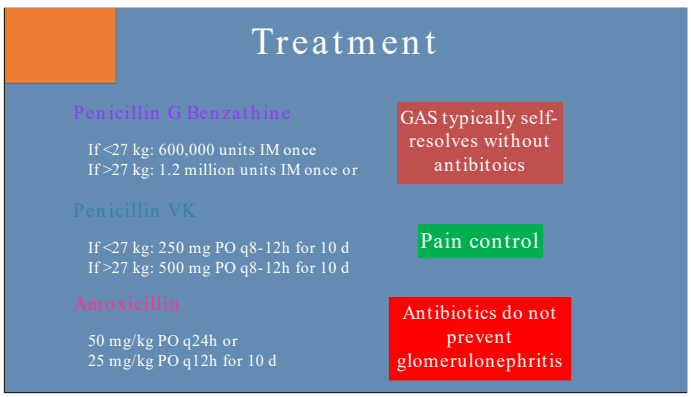

Pastia's Lines

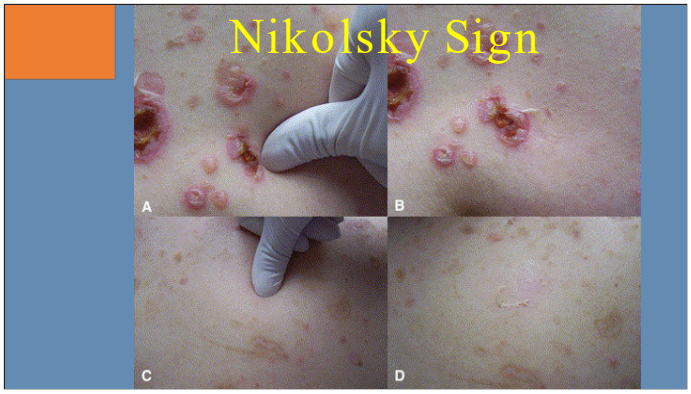

Scarlet
Fever


RED
STRAWBERRY
TONGUE
WHITE
STRAWBERRY
TONGUE


Desquamation


Scarlet Fever
Tx: Penicillin


Treatment
Penicillin G Benzathine
If <27 kg: 600,000 units IM once
If >27 kg: 1.2 million units IM once or
Penicillin VK
If <27 kg: 250 mg PO q8-12h for 10 d
If >27 kg: 500 mg PO q8-12h for 10 d
Amoxicillin
50 mg/kg PO q24h or
25 mg/kg PO q12h for 10 d
GAS typically self-resolves without antibitoics
Pain control
Antibiotics do not prevent glomerulonephritis

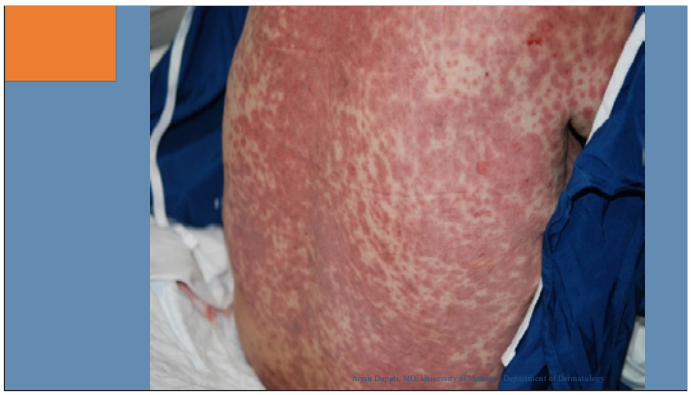

Nikolsky Sign
A
B
C
D


Stevens Johnson Syndrome


Photo courtesy Neil Rifenbark, MD


Photo courtesy Neil Rifenbark, MD


Arjun Dupati, MD; University of Michigan Department of Dermatology

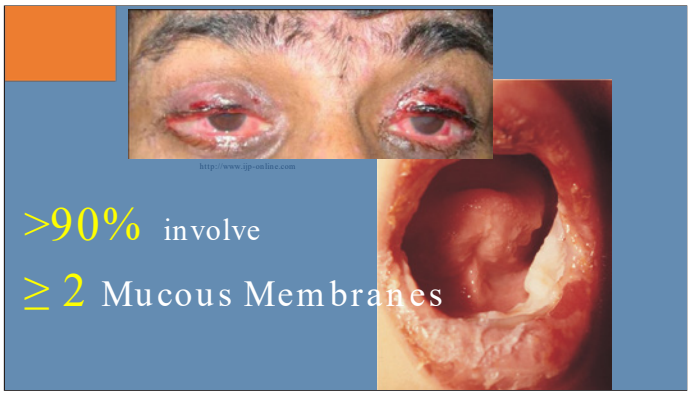

Arjun Dupati, MD; University of Michigan Department of Dermatology

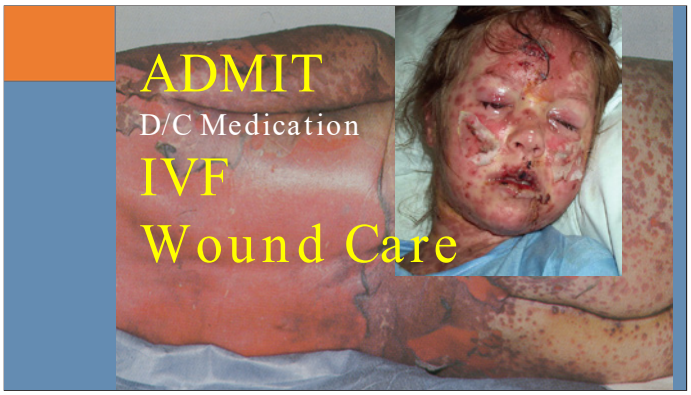

Relative sparing of distal extremities
Arjun Dupati, MD; University of Michigan Department of Dermatology


Toxic
Epidermal
Necrolysis


SJS
<10%
TBSA
>30%
TEN


Photo credit: Tracie Pearson, MD University of Michigan Department of Dermatology


>90% involve
≥ 2 Mucous Membranes


ADMIT
D/C Medication
IVF
Wound Care

Fever x5 days

+ 4/5 criteria:

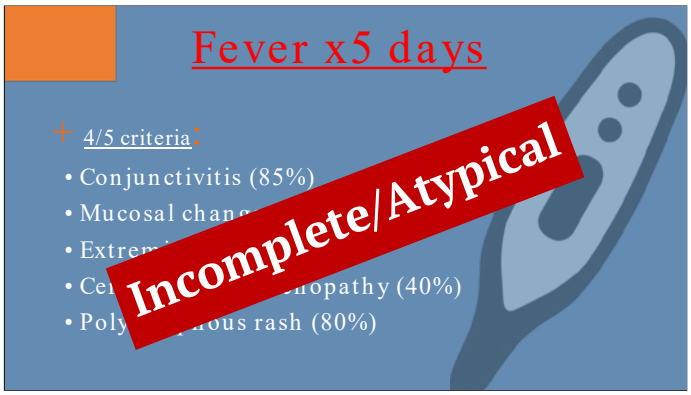
- Conjunctivitis (85%)
- Mucosal changes (90%)
- Extremity changes (75%)
- Cervical lymphadenopathy (40%)
- Polymorphous rash (80%)


Conjunctivitis
Limbic-Sparing

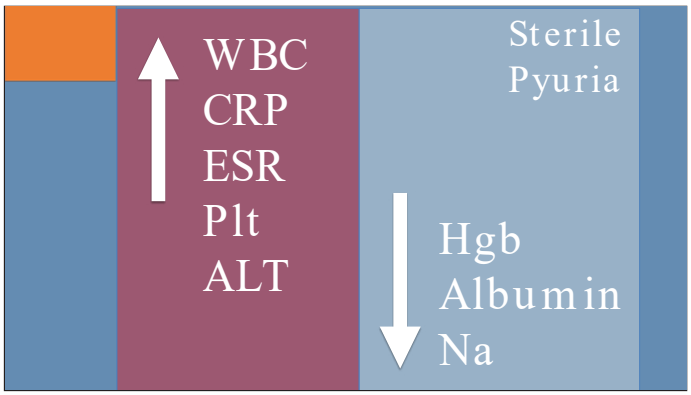

Mucositis

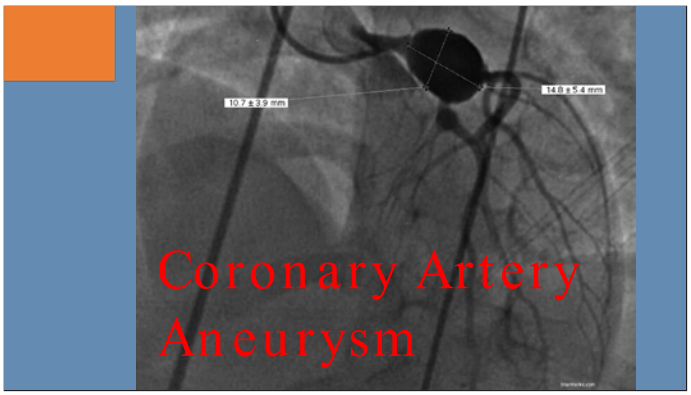

LAD
Unilateral

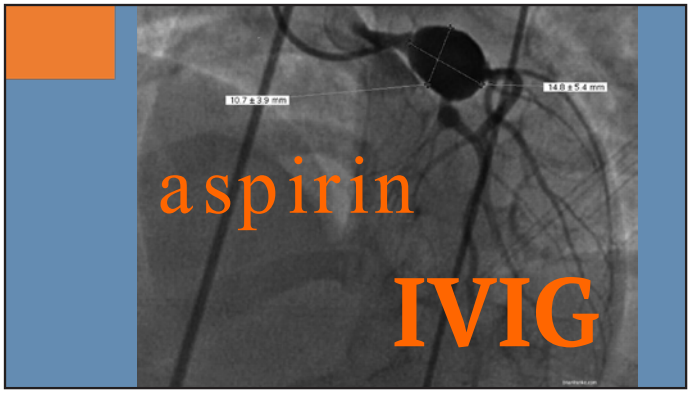

1st week
2nd-3rd week

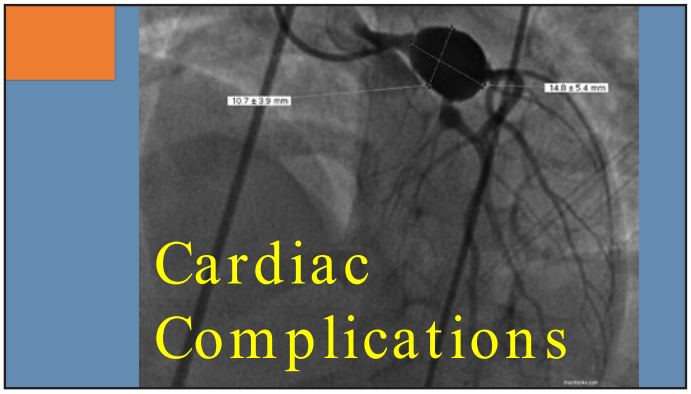

Fever x5 days
+ 4/5 criteria:
• Conjunctivitis (85%)
• Mucosal chang
• Extremi
• Ce lopathy (40%)
• Poly ous rash (80%)
Incomplete/Atypical

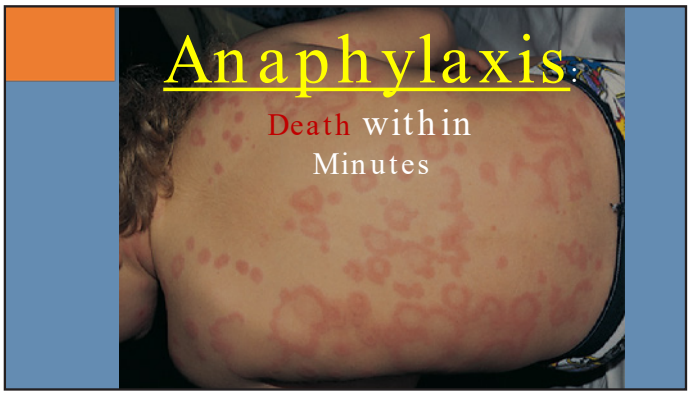

Incomplete Kawasaki
LESS criteria
fever <5 days
intechopen.com

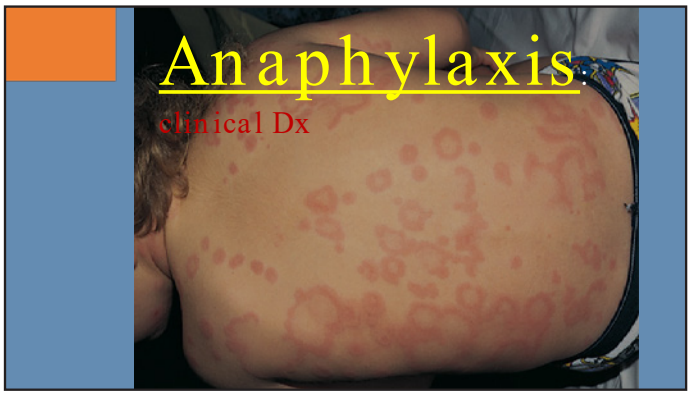

WBC
CRP
ESR
Plt
ALT
Sterile Pyuria
Hgb
Albumin
Na

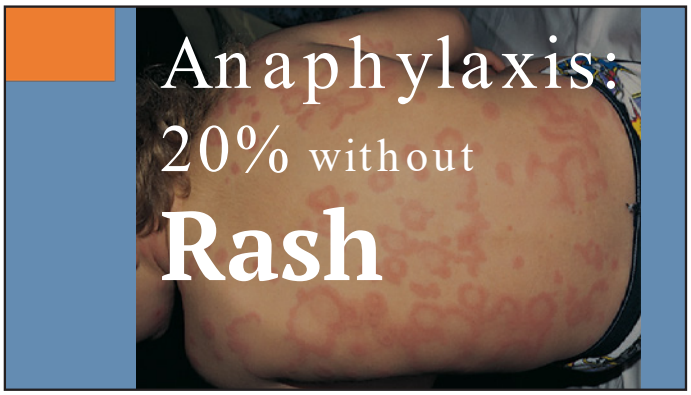

Coronary Artery Aneurysm


aspirin
IVIG


Cardiac
Complications


Anaphylaxis:
Death within
Minutes


Anaphylaxis:
clinical Dx

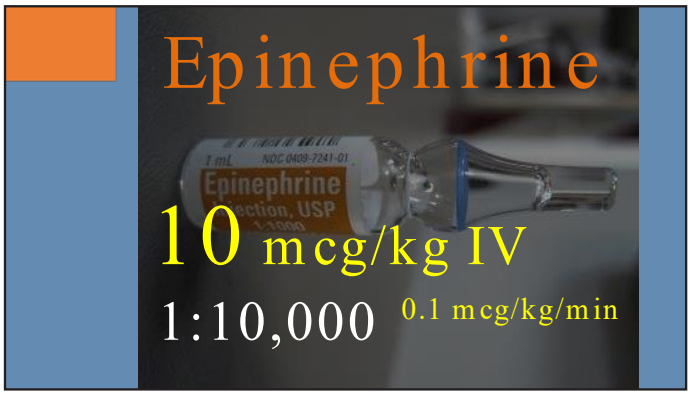

Anaphylaxis:
20% without
Rash


Skin/Mucosa
Respiratory
Gastrointestinal
Cardiovascular


Epinephrine
Epinephrine
Injection, USP
THE Treatment


Epinephrine
0.01 mg/kg
1:1000 IM


Epinephrine
0.15 mg IM
0.3 mg IM


Epinephrine
10 mcg/kg IV
1:10,000
0.1 mcg/kg/min


Resource Recommendation
• Visual Dx


Thank you!
emilyros@usc.edu
@EmilyRoseMD1

SELF EVALUATION

Pediatric Rashes You Can't Miss: Diagnosis, Management, and Pitfalls

1. A child comes in with a diffuse rash and fever. Which of the following makes this most concerning for Kawasaki Disease?

a. Association of fever with a rash
b. Rash with mucosal ulcers
c. Rash with vomiting, diarrhea and dry lips
d. Mild rash but association with persistent fever, toxic appearance, conjunctival injection, and dry, cracked lips.

2. A child presents with fever, conjunctivitis, and rash. Because of their vaccination history and potential exposure, you are concerned about measles. The mother asks when measles is most contagious. You respond:

a. After rash onset
b. When mucosal findings are present
c. 5 days before symptoms appear
d. 5 days after symptoms appear

3. Which of the following is true regarding Varicella:

a. Vaccination routinely occurs at 6 months
b. Lesions are in the same stage of development
c. Reactivation is uncommon
d. Disease may occur despite vaccination

4. A 4-year-old child presents with a "slapped cheek" appearance followed by a lacy, reticular rash on the trunk. Which virus is the most likely cause?

a. Measles virus
b. Parvovirus B19
c. Rubella virus
d. Human herpesvirus 6

5. A 6-month-old infant presents with high fever for 3 days that resolves abruptly, followed by the onset of a pink, maculopapular rash on the trunk and neck. What is the most likely diagnosis?

a. Roseola (exanthem subitum)
b. Rubella
c. Kawasaki disease
d. Scarlet fever

6. A 7-year-old presents with fever, cough, conjunctivitis, and Koplik spots, followed by a cephalocaudal spreading rash. What is the most likely diagnosis?

a. Rubella
b. Scarlet fever
c. Measles (rubeola)
d. Erythema infectiosum

7. Which of the following is a hallmark feature of atopic dermatitis in infants?

a. Target lesions on the palms and soles
b. Vesicular rash in the diaper area
c. Erythematous patches on the cheeks and extensor surfaces
d. Petechial rash on the lower limbs

8. Which of the following conditions is most associated with a vesicular rash in various stages of healing on the face and trunk?

a. Measles
b. Varicella (chickenpox)
c. Impetigo
d. Molluscum contagiosum

Answer Key: 1. D, 2. C, 3. D, 4. B, 5. A, 6. C, 7. C, 8. B

FACULTY

Jonathan A. Edlow, MD

Jonathan A. Edlow, MD, of Lancaster, Virginia, is a Professor of Medicine and Emergency Medicine at Harvard Medical School. He lectures nationally and internationally on topics such as stroke, TIA, subarachnoid hemorrhage, and various causes of weakness and dizziness. He is an internationally renowned expert on neurological emergencies. Dr. Edlow's specialty areas include ED quality assurance, ED physical layout and facilities redesign, physician professional development, and the creation and implementation of clinical practice guidelines. For the past 15 years, Dr. Edlow has contributed to the international development of emergency medicine in a variety of countries, participating in educational, quality assessment, and emergency care systems consulting projects. Practicing medicine since 1981, he is board-certified in both emergency medicine and internal medicine. In addition to being well-published in peer reviewed literature, Dr. Edlow has written a book on stroke, as well as two award-winning books for the lay public; one about the history of Lyme disease titled "Bull's Eye: unraveling the medical mystery of Lyme disease," and the second titled "The Deadly Dinner Party: 15 true medical detective stories."

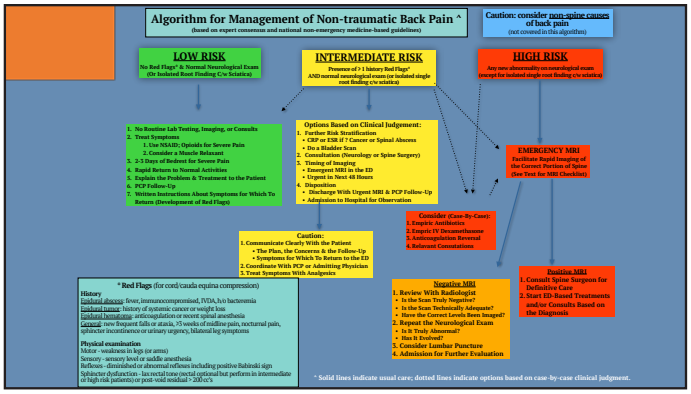
You may contact Dr. Edlow with your questions or comments at jedlow@bidmc.harvard.edu.

Acute Atraumatic Neck and Back Pain

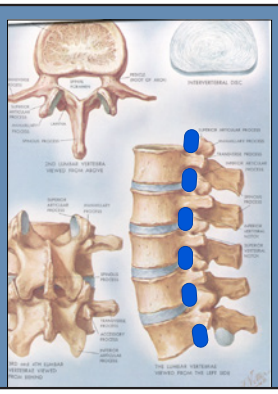
Jonathan A. Edlow, MD

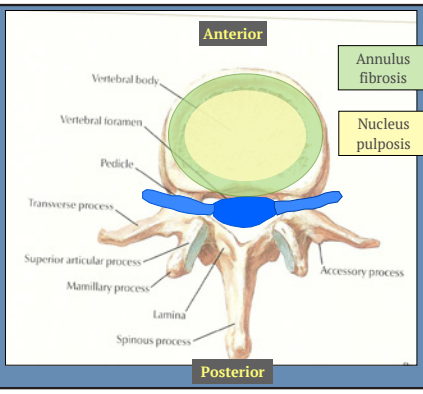
Objectives

- Learn to use an ED **clinical pathway** for evaluation of patients presenting with acute non-traumatic neck and back pain in order to reduce the misdiagnosis
- Use history and physical examination to identify **RED FLAGS** that suggest the possibility of spinal cord or cauda equina compression
- Develop strategies to facilitate a **targeted & rapid diagnostic work-up** and treatment for patients with true compression

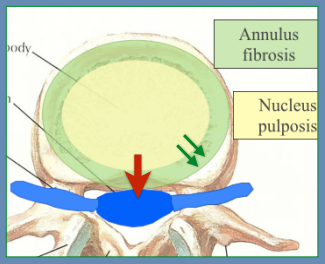


BONY ANATOMY





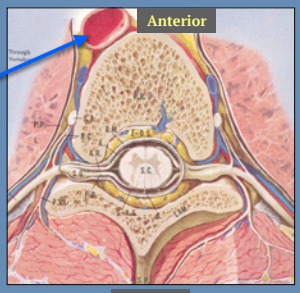
Disc Disease
Postero-Lateral Versus Central Herniation

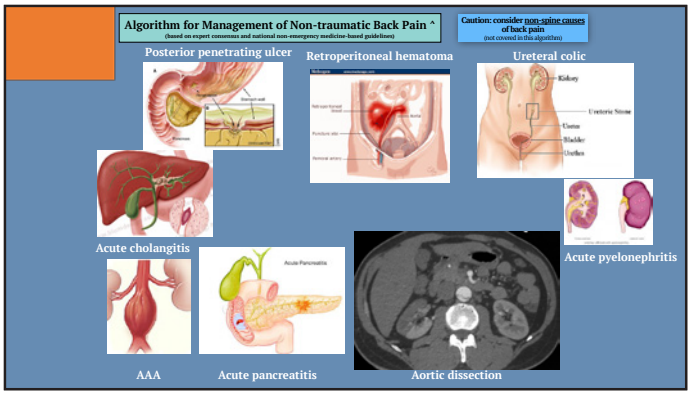


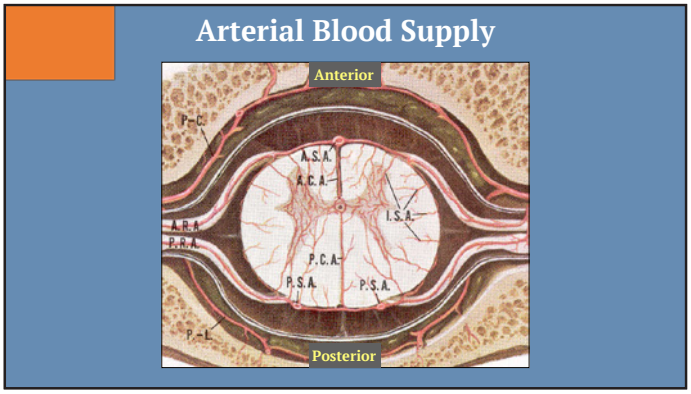
Cord in the Spinal Canal

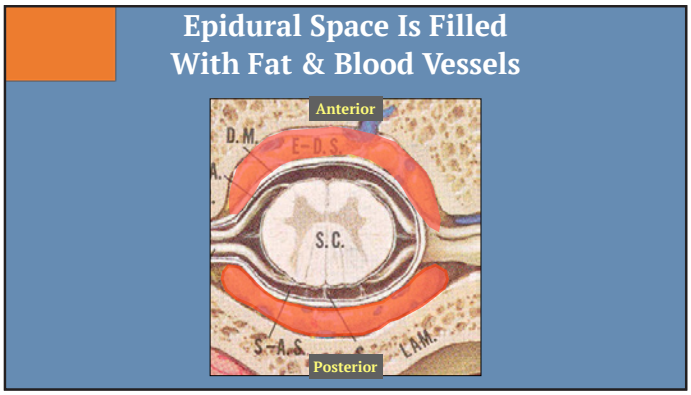
Don't forget about the aorta

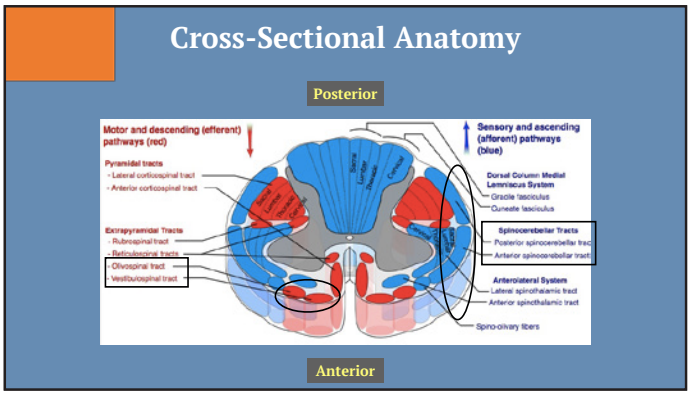
(and other non-spine causes of back pain)

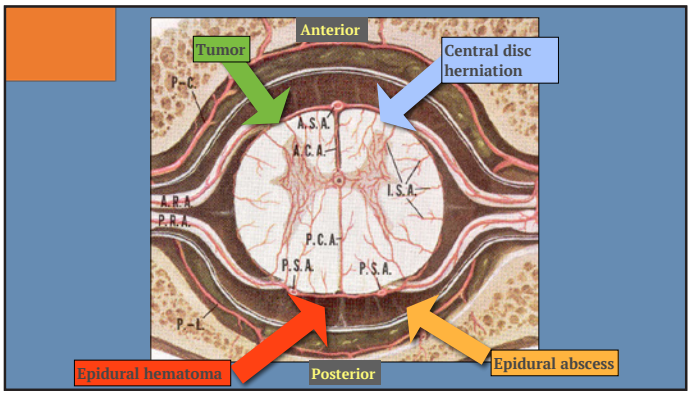


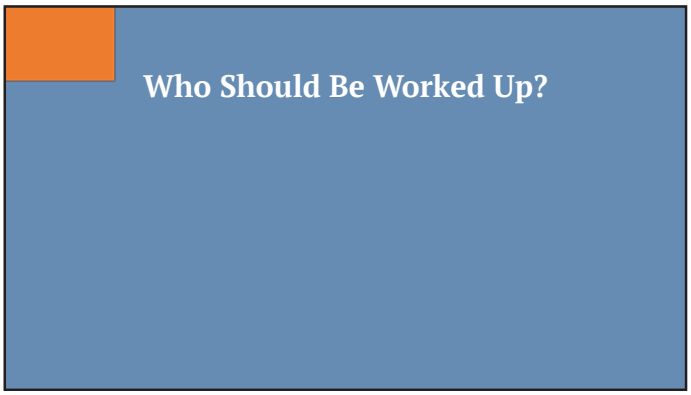

Algorithm for Management of Non-traumatic Back Pain ^
Caution: consider non-spine causes of back pain
Posterior penetrating ulcer
Retroperitoneal hematoma
Ureteral colic
Acute cholangitis
Acute pyelonephritis
AAA
Acute pancreatitis
Aortic dissection

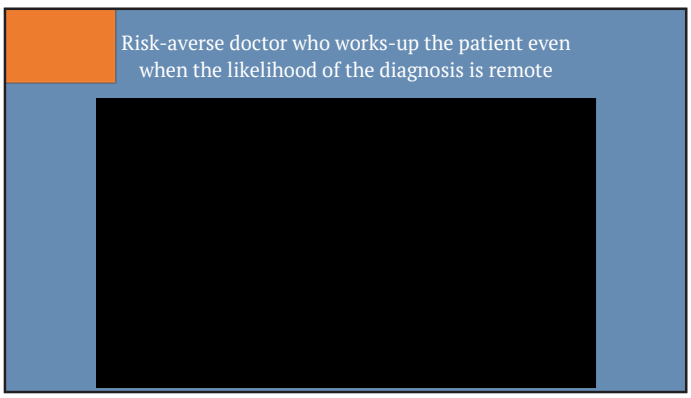

Arterial Blood Supply
Anterior
P-C.
A.S.A.
A.C.A.
I.S.A.
A.R.A.
P.R.A.
P.C.A.
P.S.A.
P.S.A.
P-L
Posterior

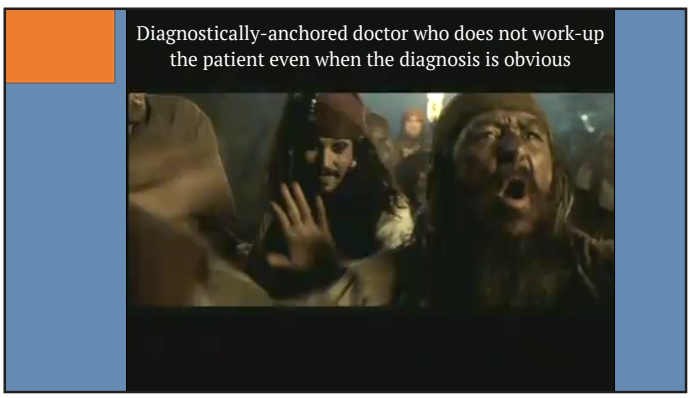

Epidural Space Is Filled With Fat & Blood Vessels
Anterior
D.M.
E-D.S.
S.C.
S-A.S.
L.A.M.
Posterior

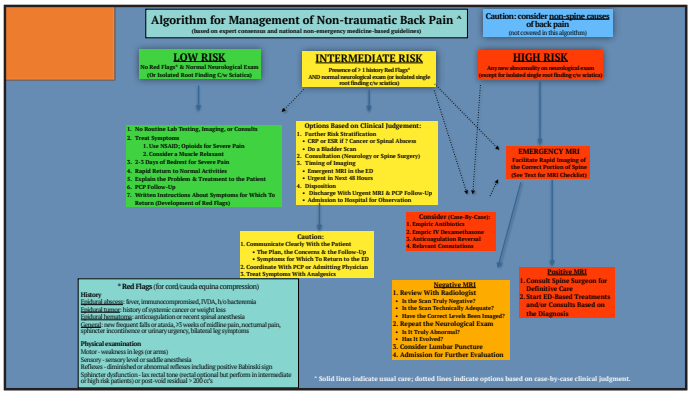

Cross-Sectional Anatomy
Posterior
Motor and descending (efferent) pathways (red)
Sensory and ascending (afferent) pathways (blue)
Pyramidal tracts
Lateral corticospinal tract
Anterior corticospinal tract
Dorsal Column Medial Lemniscus System
Gracile fasciculus
Cuneate fasciculus
Extrapyramidal Tracts
Rubrospinal tract
Reticulospinal tracts
Olivospinal tract
Vestibulospinal tract
Spinocerebellar Tracts
Posterior spinocerebellar tract
Anterior spinocerebellar tract
Anterolateral System
Lateral spinothalamic tract
Anterior spinothalamic tract
Spino-olivary fibers
Anterior

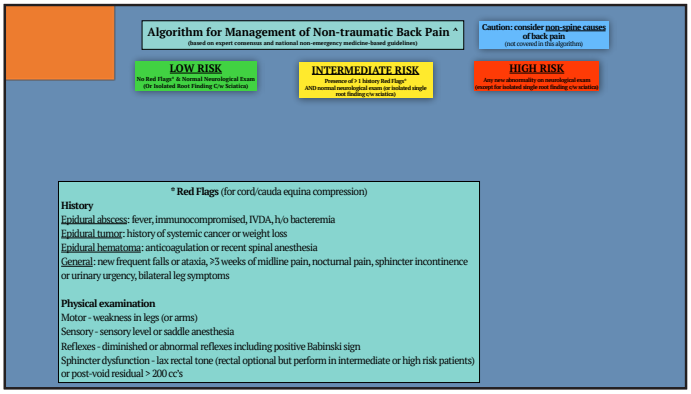

Anterior
Tumor
Central disc herniation
P-C.
A.S.A.
A.C.A.
I.S.A.
A.R.A.
P.R.A.
P.C.A.
P.S.A.
P.S.A.
P-L
Epidural hematoma
Posterior
Epidural abscess


Who Should Be Worked Up?


Risk-averse doctor who works-up the patient even when the likelihood of the diagnosis is remote


Diagnostically-anchored doctor who does not work-up the patient even when the diagnosis is obvious





Consultation, Testing (Usually MRI), & Timing of the Testing Is Driven by RED FLAGS Identified by History and Physical Examination

As a general rule, a patient's neurological outcome is a function of their status at the time of diagnosis

RED FLAGS
what is the evidence base?

- Overall, weak, but the best validated are:
  - History of cancer
  - Corticosteroid use (fracture)
  - Anticoagulant use
  - New physical findings including new ataxia & difficulty walking
- History findings suggest risk (of neurological problem) whereas hard physical findings that suggest presence (of neurological problem)
- Groups of red flags may perform better than single ones and one must factor in the context

RED FLAGS

History - specific diagnoses

Epidural tumor - h/o systemic cancer, weight loss

Epidural abscess - unexplained fever, immunocompromised state, IVDA, h/o recent bacteremia or other active infection

Epidural hematoma - anticoagulation, recent spinal anesthesia

RED FLAGS

History - general factors

New frequent falls and/or difficulty walking

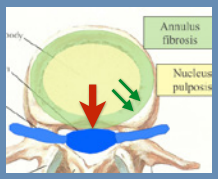
Duration ≥3 weeks of midline back pain

Pain wakes patient from sleep

Sphincter incontinence and urinary urgency

Lhermitte's sign (electric shock-like sensation shooting down spine & limbs on neck flexion)

Bilateral leg symptoms



RED FLAGS

Physical Examination

Weakness in legs (or arms)

Sensory level

New ataxia (even without demonstrable motor or sensory signs)

Diminished or abnormal reflexes

Sphincter signs

- Full bladder (Bladder scan for pre- and post-void residual)
- Test saddle anesthesia in all patients
- In intermediate or high-risk patients, do rectal exam

RED FLAGS & "WHITE" FLAGS

There is NO SINGLE symptom or sign that has high sensitivity for cauda equina syndrome

There is also the concept of WHITE FLAGS - symptoms or signs that indicate (and have a high specificity for) a complete cauda equina syndrome for which treatment is very unlikely to help

Todd; 2017; British J NSurg

Dangerous Diagnoses Without Red Flags

- Cauda equina
  - Sphincter symptoms may not be present
  - Saddle anesthesia may preceed lower leg/foot signs
- Cancer
  - ~ 20% of cases, the vertebral metastasis is the presenting symptom
- Abscess
  - Only ~ 66% have fever at presentation
  - Only 10% have the classic triad (fever, back pain & neurological deficit)
- Hematoma
  - 30% have no identifiable reason or risk factor for bleeding

CAUTION: These patients are very difficult to diagnose at first visit; clear discharge instructions/follow-up care mitigate this Beware diagnostic anchoring

Diagnostic Anchoring

Algorithm for Management of Non-traumatic Back Pain ^
(based on expert consensus and national non-emergency medicine-based guidelines)

Caution: consider non-spine causes of back pain
(not covered in this algorithm)

LOW RISK
No Red Flags* & Normal Neurological Exam
(Or Isolated Root Finding C/w Sciatica)

1. No Routine Lab Testing, Imaging, or Consults
2. Treat Symptoms
   - Use NSAID, Opioids for Severe Pain
   - Consider a Muscle Relaxant
3. 2-3 Days of Bedrest for Severe Pain
4. Rapid Return to Normal Activities
5. Explain the Problem & Treatment to the Patient
6. PCP Follow-Up
7. Written Instructions About Symptoms for Which To Return (Development of Red Flags)

* **Red Flags** (for cord/cauda equina compression)
History
Epidural abscess: fever, immunocompromised, IVDA, h/o bacteremia
Epidural tumor: history of systemic cancer or weight loss
Epidural hematoma: anticoagulation or recent spinal anesthesia
Cauda: new frequent falls or ataxia, >3 weeks of midline pain, nocturnal pain, sphincter incontinence or urinary urgency, bilateral leg symptoms
Physical examination
Motor - weakness in legs (or arms)
Sensory - sensory level or saddle anesthesia
Reflexes - diminished or abnormal reflexes including positive Babinski sign
Sphincter dysfunction - lax rectal tone (rectal optional but perform in intermediate or high risk patients) or post-void residual > 200 cc's

^ Solid lines indicate usual care; dotted lines indicate options based on case-by-case clinical judgment.

Why no imaging?

No routine imaging recommended by national (primary care) guidelines
There is a very high rate of false-positive findings with MRI
Studies of MRI vs plain films shows no advantage to MRI (and ↑ costs,↑ surgery)

What medications and for how long?

How long to limit activity level?

What correlates with patient satisfaction?

Table 2: Age-specific prevalence estimates of degenerative spine imaging findings in asymptomatic patients[a]

	Age (yr)						
Imaging Finding	20	30	40	50	60	70	80
Disk degeneration	37%	52%	68%	80%	88%	93%	96%
Disk signal loss	17%	33%	54%	73%	86%	94%	97%
Disk height loss	24%	34%	45%	56%	67%	76%	84%
Disk bulge	30%	40%	50%	60%	69%	77%	84%
Disk protrusion	29%	31%	33%	36%	38%	40%	43%
Annular fissure	19%	20%	22%	23%	25%	27%	29%
Facet degeneration	4%	9%	18%	32%	50%	69%	83%
Spondylolisthesis	3%	5%	8%	14%	23%	35%	50%

Brinkikji; 2015; AJNR

Why no imaging?

No routine imaging recommended by national (primary care) guidelines
There is a very high rate of false-positive findings with MRI
Studies of MRI vs plain films shows no advantage to MRI (and ↑ costs,↑ surgery)

What medications and for how long?

Guidelines (non-EM) recommend acetaminophen/NSAIDs, +/- muscle relaxer
Adding cyclobenzaprine or oxycodone no better than naproxen (Friedman 2015, JAMA) and naproxen alone = naproxyn + diazepam (Friedman 2017 AnnEM)
My practice: 5-7d of a NSAID with 1-2d of a narcotic & muscle relaxer (case-by-case), and ? a short course of steroids if radicular pain

How long to limit activity level?

Data show that 3d of decreased activity leads to faster resumption of normal activities than longer "prescriptions" for decreased activity
Later, abdominal strengthening exercises and hamstring stretching helps

What correlates with patient satisfaction?

The North Carolina Back Pain Project . . .

North Carolina Back Pain Project

- 208 random doctors (PCPs, HMO PCPs), orthopedists, chiropractors
- Enrolled ~ 1600 consecutive patients with acute low back pain
- 6 month outcomes (functional status, patient satisfaction) by phone

Direct health-care dollars spent
Orthopedists/chiropractors >> PCPs >> HMO PCPs

Amount of imaging performed
Orthopedists/chiropractors >> PCPs >> HMO PCPs

Clinical outcomes ? → **Identical**

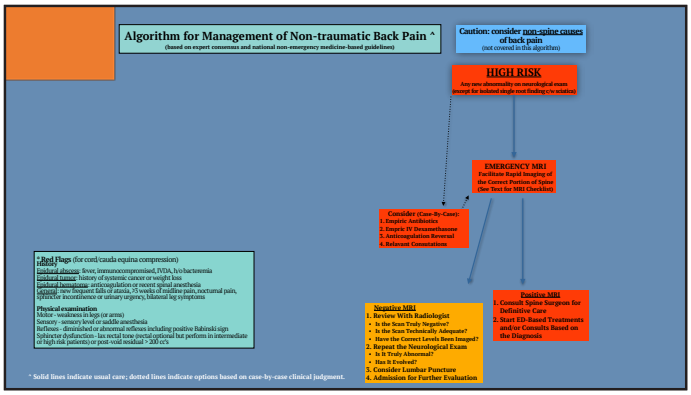
Carey; 1995; NEJM; 333: 913-17

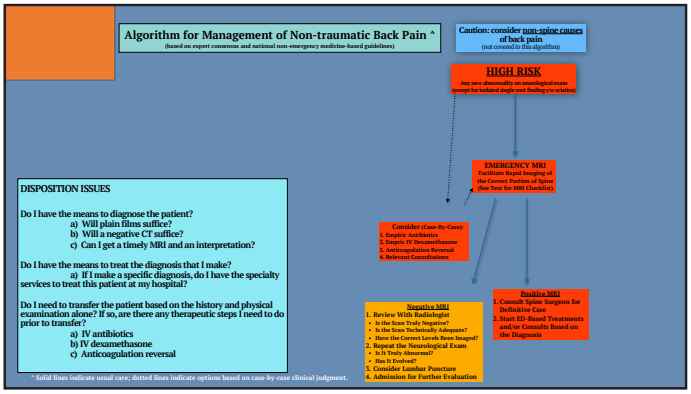
What Correlated With Patient Satisfaction?

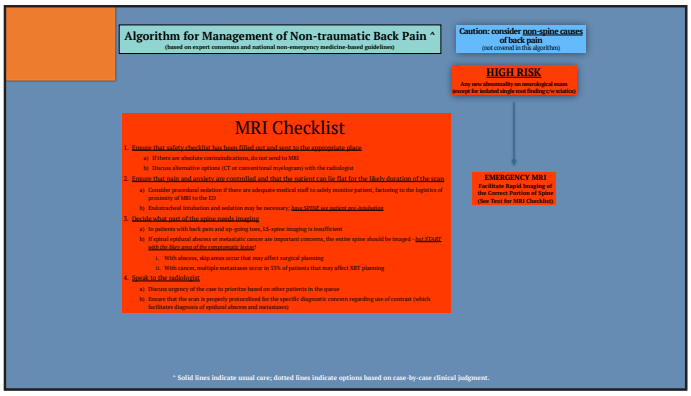
Overall, higher satisfaction with chiropractors than with physicians, but which specific elements of care were important?

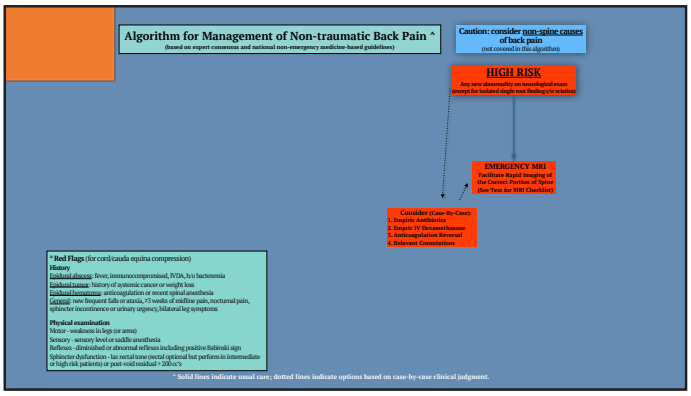
Perception that a detailed history was taken
Perception that a complete physical exam was done
Perception that the doctor clearly explained their thinking

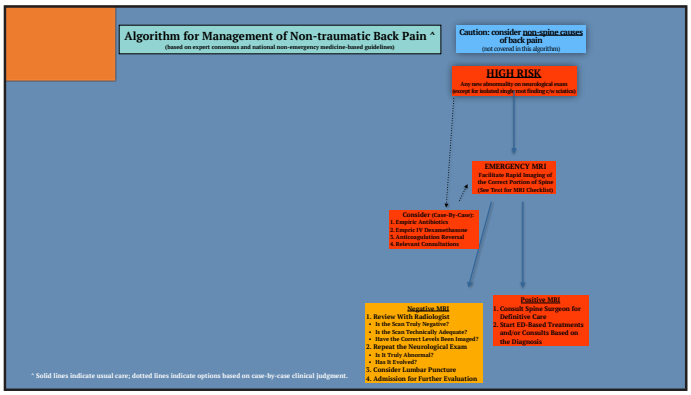
What was not important?
Imaging tests











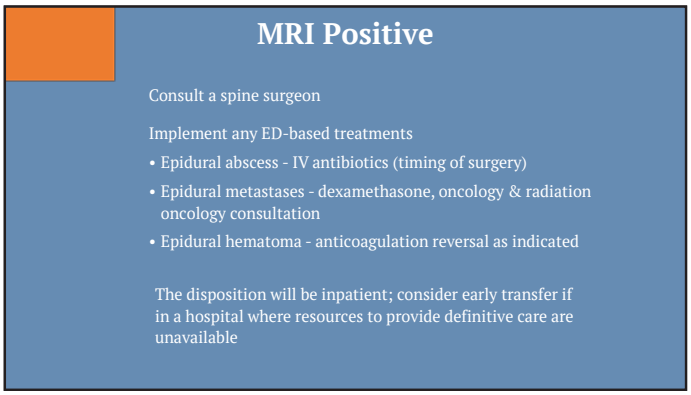
MRI Positive

Consult a spine surgeon

Implement any ED-based treatments

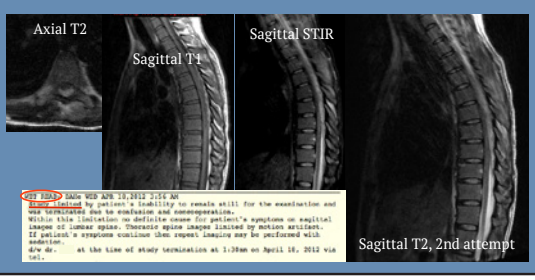
- Epidural abscess - IV antibiotics (timing of surgery)
- Epidural metastases - dexamethasone, oncology & radiation oncology consultation
- Epidural hematoma - anticoagulation reversal as indicated

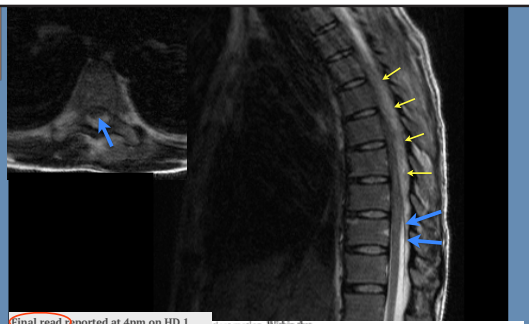
The disposition will be inpatient; consider early transfer if in a hospital where resources to provide definitive care are unavailable



MRI Negative

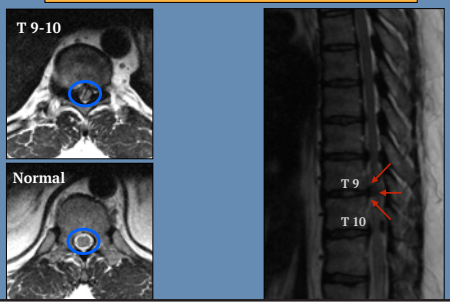
Review the MRI with neurology and/or radiology; is it truly negative? Is it an adequate study? Don't assume a limited study is a negative one!





MRI negative (negative LS spine MRI 3d ago at OSH)

Has the correct portion of the spine been imaged?

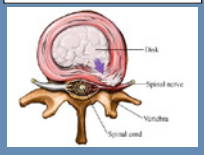


Cauda Equina Syndrome

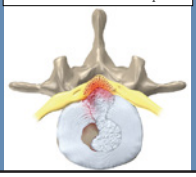
- The spinal cord stops at ~ L1
- There is no spinal cord in the L-S spine, (only nerve roots floating in the CSF)

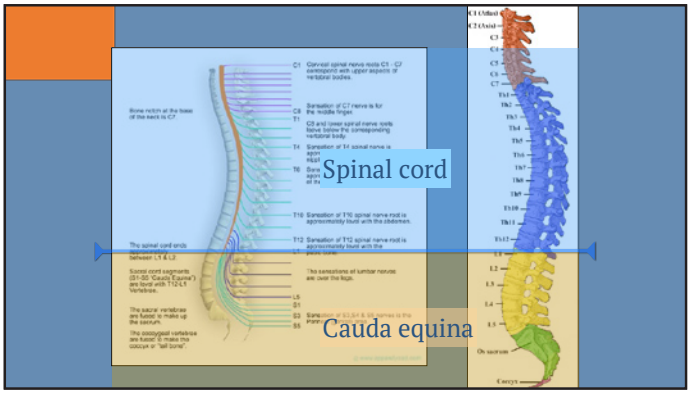
- Typical sciatica causes unilateral symptoms of a single root; in CES, multiple roots are involved & symptoms are often bilateral (but can be asymmetric)
  - Decreased reflexes (LMN), downgoing toes
  - Sphincter symptoms are common
  - **Bilateral** sciatica = CES till proven otherwise

Posterolateral HNP: sciatica



Central HNP: cauda equina





Clinical Implications

Don't miss cord lesion by ordering a L-S spine MRI (this is akin to to ordering a chest CTA to rule out appendicitis - wrong body part)

Don't miss cauda equina lesion without examining the S2-4 roots. Even without leg findings (L3/4-S1). Check saddle anesthesia and PVR +/- rectal (S2-4 roots), to avoid **missing** some cauda equina patients - incomplete exam

A back pain patient with a + **Babinski** sign has an UMN lesion (cord or higher). In a back pain patient with a cauda equina lesion, a LMN lesion, the reflexes are decreased. Test the toes in all back pain patients

MRI Negative

Re-examine the patient; verify the findings and ensure they have not evolved!

Did you do the right study?

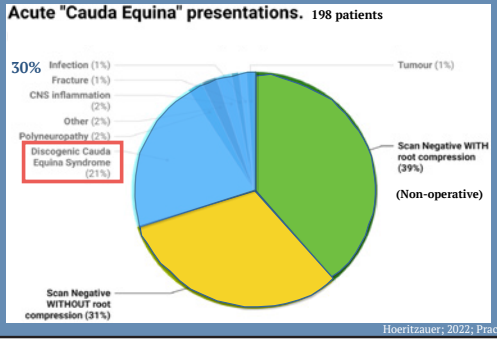
Consider LP (? transverse myelitis, Guillain-Barré).

Consider other studies (Is this ischemic? Could it be an aortic dissection?)

Be very cautious diagnosing conversion reaction (a diagnosis of exclusion) in the ED

Discuss with consultants; likely admit (to neurology) for further studies & observation and diagnostic testing.

"Scan-Negative" Cauda Equina Syndrome



Hoeritzauer; 2022; Pract Neurol

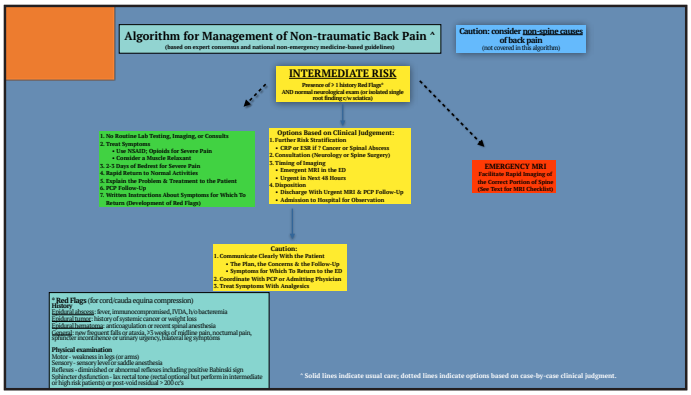
Check List for Identifiable Neurological Causes

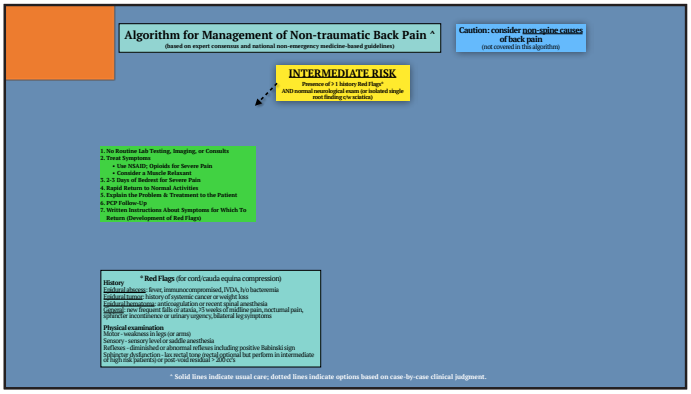
ED evaluation

- No back pain (rare inflammatory myelopathy)
- Is there is a sensory level (review scan with radiologist for missed pathology)
- Is there recent genital ulceration? (HSV-polyneuritis)

In-patient evaluation

- Does urinary retention persist despite adequate analgesia & resolution of constipation over 72 hours
- Is there progressive weakness, reflex loss, or perineal pain (review scan)



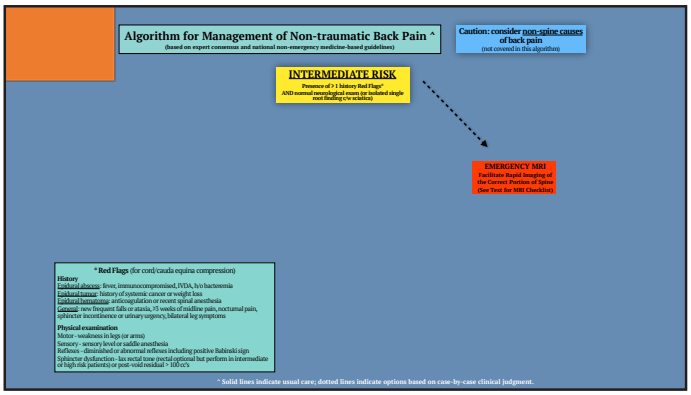


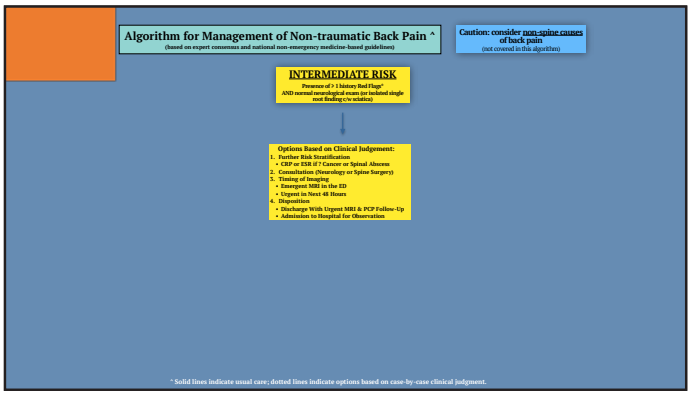
Patient who is HIV positive (RED FLAG) who is compliant on HAART therapy, had a recent non-detectable viral load and normal CD4 count, who has lumbar back pain after exercise. The physical exam is normal. He is followed closely by a PCP.

Otherwise healthy patient who has a few days of vague diffuse low back pain in the setting of a fever of 101 degrees (RED FLAG) associated with symptoms/signs of a specific localized infection.

Patient who has low back pain who urinated while attempting to get to bathroom (RED FLAG) but on detailed history, the issue is that the pain slowed the person down getting to the bathroom and not that they truly lost control of urination - CHECK the PVR!! (or that a patient is constipated while taking narcotics but has good sphincter tone).

Patient with low back pain on warfarin (RED FLAG) with a good story/ exam for sciatica and an INR of 2.2.





CRP & ESR
Inflammatory Markers

No systematic prospective studies in a group of "all-comers" of ED patients with back pain

Most studies are of relatively small numbers of patients with a specific diagnosis (e.g., SEA, tumor)

- For SEA and tumor, CRP & ESR are very sensitive (≥95%)
- Sensitivity for disc & hematoma not clear (but likely lower)

Data are mixed about if CRP or ESR is better than the other

The use of inflammatory markers are incorporated into most other guidelines

Scan the bladder !

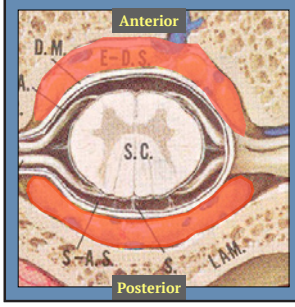
- Normal PVR = zero
- How, what and when to measure?
  - Pre-void volume ("does patient sense a full bladder?")
  - Ultrasound & bladder scans are highly accurate
  - Get the PVR = "***post-void***" so coordinate with nursing & patient
- What should the threshold be?
  - ***Pre-void > 500 cc's (if painless) likely CES***
  - Post-void > 200 cc's 43% likely to have CES
  - Post-void < 200 cc's has a NPV of 97%

Venkatesan; 2019; Spine

Katzouraki; 2020; BoneJointJ

Todd; 2021; BrJNsurg

BEWARE the Thoracic Cord

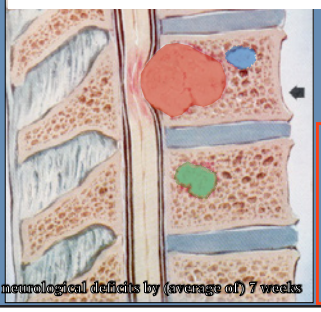


- The epidural space is smallest in the thoracic canal
- Metastatic tumor is more commonly located here (70% of MESCC is in T-spine)
- Disc disease is less common
- Therefore, bad diagnoses are more likely and there is less room for error in patients with thoracic lesions

Evolution of Neurological Signs/Symptoms . . .

. . . is unpredictable

Epidural Metastases
Pathophysiology*

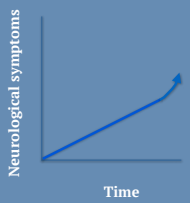


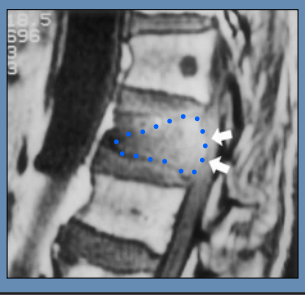
- Usually hematogenous spread to vertebral bodies
- Expansion in vertebrae
- Gradual cord compression

- BUT:
- Distortion of vessels can cause acute cord edema or infarction
- Acute pathological fracture can cause abrupt cord compression or vascular distortion and ischemia

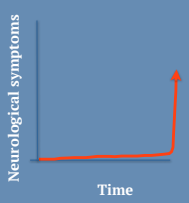
*Pain precedes neurological deficits by (average of) 7 weeks

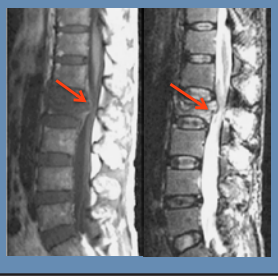
Gradual Onset Cord Compression From Metastatic Disease





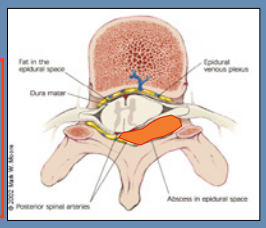
Sudden Cord Compression From Pathologic Fracture of L2





Epidural Abscess
Mechanisms of Cord Damage

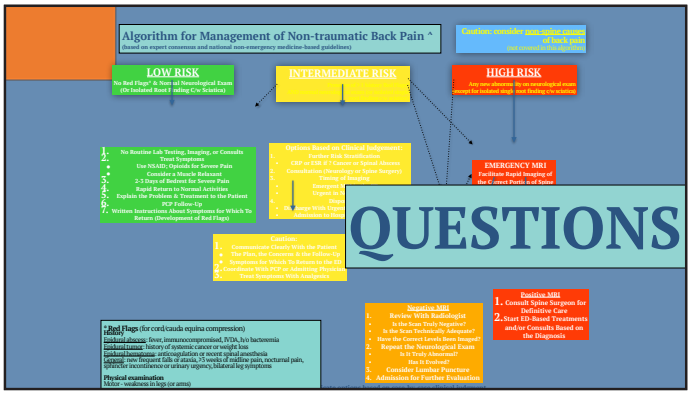
- Mechanical compression
- Mechanical distortion of the arteries causing cord ischemia or infarction
- Septic arteritis or thrombophlebitis leading to cord congestion or infarction



Algorithm for Management of Non-traumatic Back Pain

(based on expert consensus and national non-emergency medicine-based guidelines)

LOW RISK	INTERMEDIATE RISK	HIGH RISK
No Red Flags* & Normal Neurological Exam (Or Isolated Root Finding C/w Sciatica)	Presence of > 1 history Red Flags* AND normal neurological exam (or isolated single root finding c/w sciatica)	Any new abnormality on neurological exam (except for isolated single root finding c/w sciatica)
Vast majority of patients. No testing beyond careful clinical evaluation. Discharge with treatment, careful instructions to patients & family, and PCP follow-up.	Relatively few patients. Here you need to use your judgment (bearing in mind the differential diagnosis & natural history) to decide on next steps, their timing and coordination.	These are the rare patients with abnormal physical exams. Here you need to move really fast to get imaging, start treatments and coordinate with consultants to preserve function.



SELF EVALUATION

Acute Atraumatic Neck and Back Pain

True/False

1. The most common cause of spinal cord or cauda equina compression is a herniated disc.

2. Patients with low-risk back pain defined as no red flags by history and an exam that is either normal or consistent with a single nerve root finding consistent with sciatica do not need lab work or spine imaging.

3. Bilateral leg weakness is not a red flag for a serious cause of back pain.

4. Back pain due to a vertebral metastasis is rare in a patient without a prior history of cancer.

5. In patients with new back pain and a new clear finding on neurological exam of the lower extremities, spine MRI is the best diagnostic test.

6. A bladder scan is a non-invasive way to help make an early diagnosis of spinal cord or cauda equina pathology.

Answer Key: 1. T, 2. T, 3. F, 4. F, 5. T, 6. T

FACULTY

Robert J. Tubbs, MD

Robert J. Tubbs, MD, of Providence, Rhode Island, is an Associate Professor and clinician educator in the Department of Emergency Medicine at Brown University's Warren Alpert Medical School. He serves as Director of the Emergency Radiology elective for the Emergency Medicine Residency Program and has been recognized repeatedly for excellence in clinical teaching, earning awards such as the University Emergency Medicine Foundation "Excellence in Clinical Teaching" (2016) and the national ACEP Faculty Teaching Award. Board certified in Emergency Medicine, he is also an attending physician at Rhode Island Hospital, The Miriam Hospital, and Hasbro Children's Hospital, where he combines front line patient care with educational leadership and innovative work in emergency radiology and medical simulation.

You may contact Dr. Tubbs with your comments or questions at tubbsrj@gmail.com.

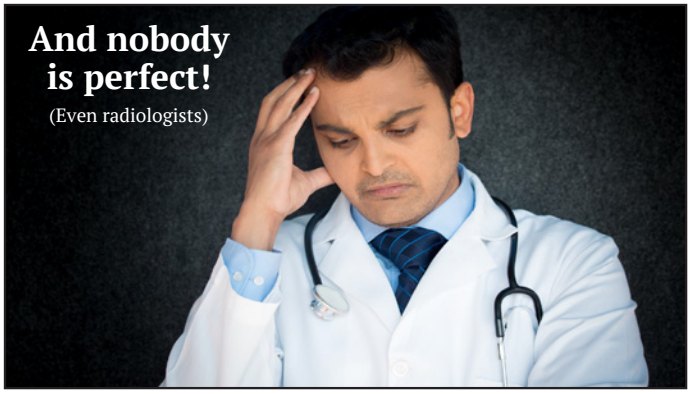
"My Stomach Hurts": Unlocking the Black Box of Emergency Abdominal Imaging

Robert Tubbs, MD

So why do we have to know how to read these?

Isn't that why we have radiologists?

Sometimes the minutes count!



You need to know when to hit the panic button

Objectives

- Explore a practical method to interpret emergent abdominal CT scans
- Use illustrative cases to examine CT findings of life-threating abdominal diagnoses
- Review CT findings of common ED diagnoses

Let's review some general abdominal imaging guidelines and terms

Abdominal CT: IV contrast

IV Contrast Indications:

- Trauma
- Vascular pathology (aorta, mesenteric ischemia, organ infarction)
- Infectious/Inflammatory pathology (diverticulitis, appendicitis)

Abdominal CT: Oral contrast

Majority of CTs DO NOT require oral contrast!!

- Review of studies showing oral contrast is not necessary in routine ED CTs (Aycok *Emerg Med News* 3/18)

Exceptions include:

- CT Esophageal Perforation Protocol
- Some Penetrating Trauma (flank)
- Gastric Bypass Protocols

What does the average CT get you?

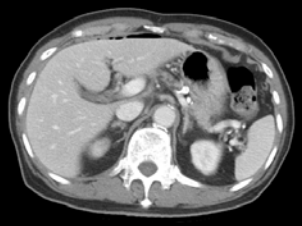
Most commonly CTs of the abdomen/pelvis performed in the ED are going to be obtained in the portal venous phase

Provides the best balance of solid organ, bowel, and vascular enhancement

Venous Phase CT

Good for look for:

- Appendicitis
- Diverticulitis
- Colitis
- Pancreatitis
- Bowel obstruction
- Renal/Splenic Infarcts
- Pyelonephritis



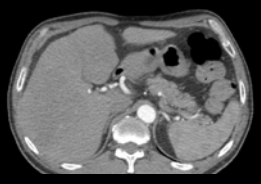
Arterial Phase CT

Obtained earlier after injection, and is designed to highlight the vasculature

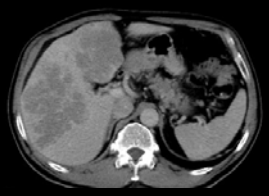
Helpful to evaluate for:

- Aortic dissection
- Branch vessel issues such as SMA or celiac thrombus/dissection

Why it matters



Arterial Phase

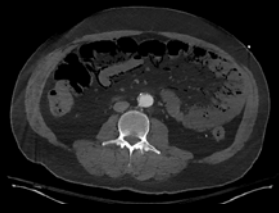


Venous Phase

Multi-phasic CT

Some diagnoses benefit from having CTs obtained in various phases of contrast

Multi-phasic CT



Mesenteric Ischemia:

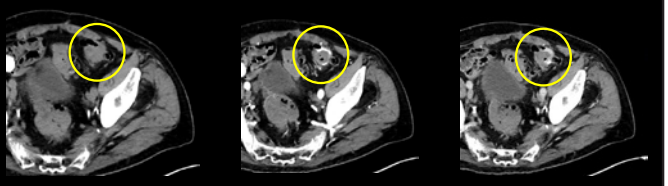
1. Arterial phase to assess proximal vasculature for occlusion
2. Portal venous phase to assess bowel wall perfusion and for other potential causes of pain

Multi-phasic CT

Acute GI Bleeding:

1. Non-contrast CT to establish baseline bowel contents
2. Arterial phase to assess for active arterial extravasation of contrast
3. Venous phase to assess for redistribution

GI Bleed CT



Non-contrast | Arterial Phase | Venous Phase

Let's look at a few definitions

What is "Stranding"?

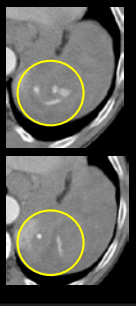
- Soft tissue stranding refers to the presence of fluid within adipose tissue
- Normal fat appears dark grey
- When there is abnormal fluid within the fat it appears as increased haziness or streaking
- Often occurs due to the presence of inflammatory fluid such as in infections, but may also be seen with other conditions such as ascites or anasarca

Soft Tissue Stranding

Normal kidney with normal retroperitoneal fat and clean tissue planes

Edematous kidney with fluid in the fat = Stranding

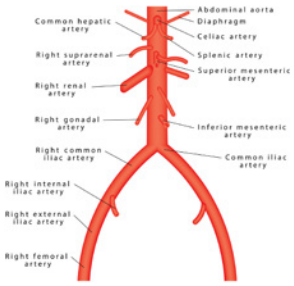
What about a **blush?**



Areas of hyperdensity outside the expected vasculature c/w active extravasation of IV contrast

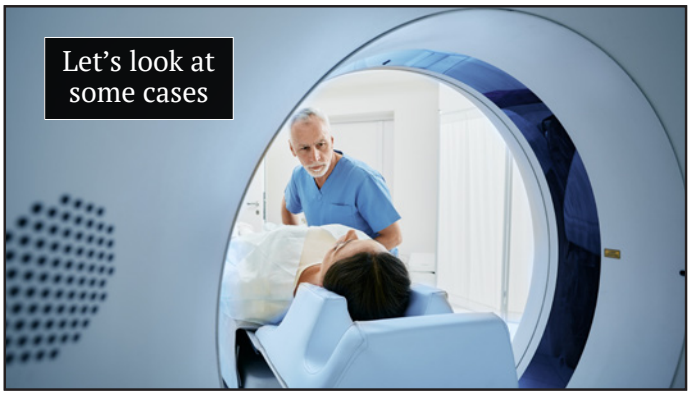
So how do we read these things?

Start with the Vasculature

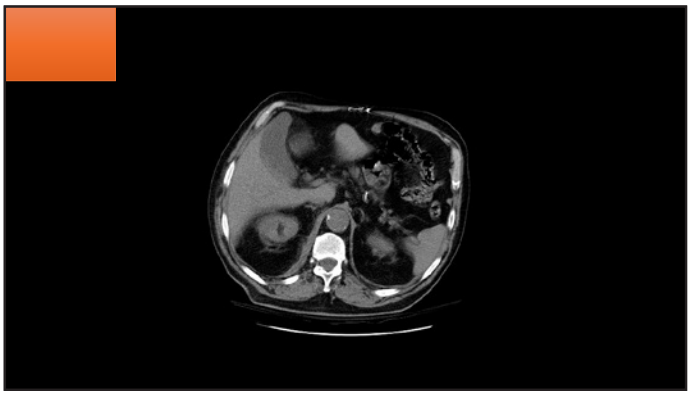


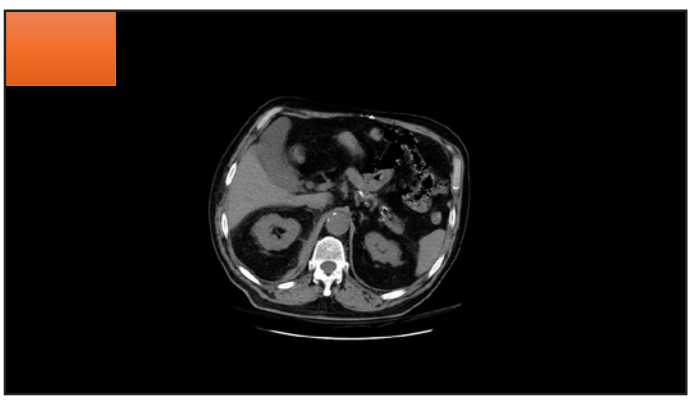
Move on to the Solid Organs

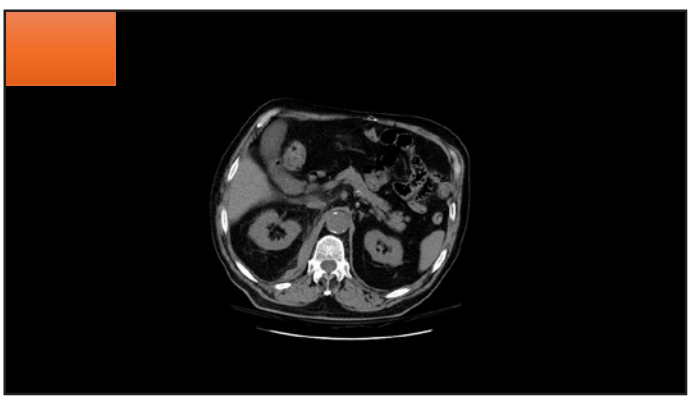
Finish up with the Intestines

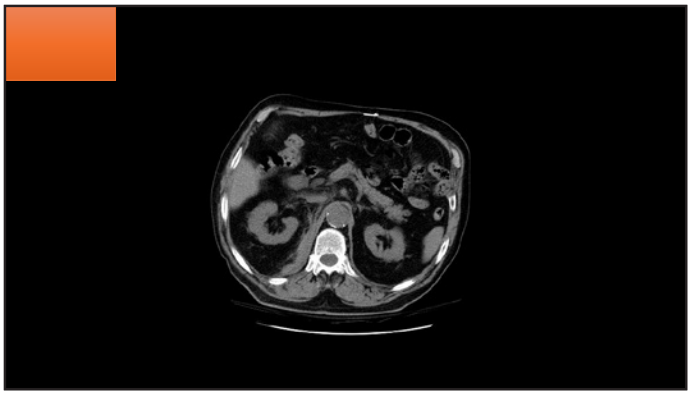


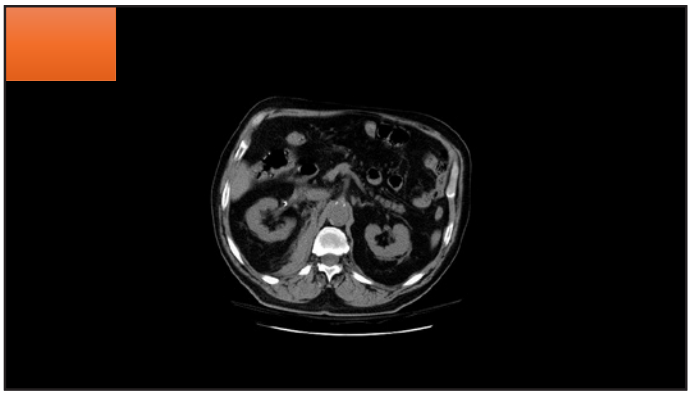
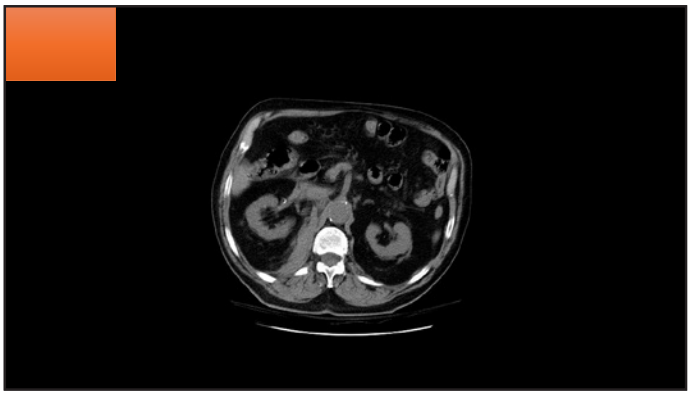
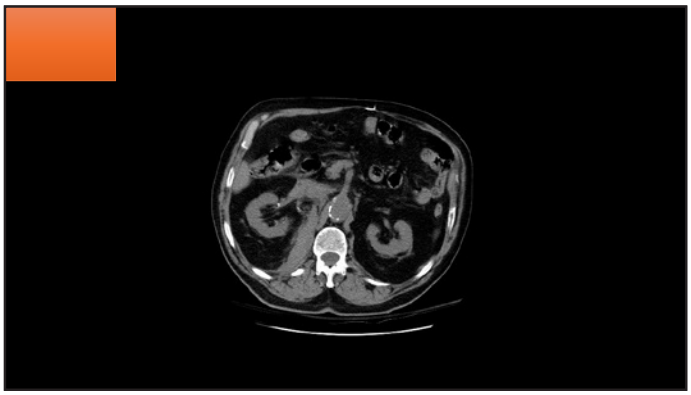
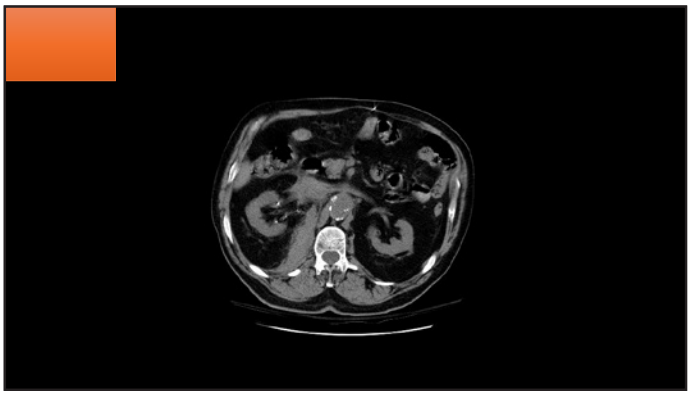
65 y.o. male with flank pain

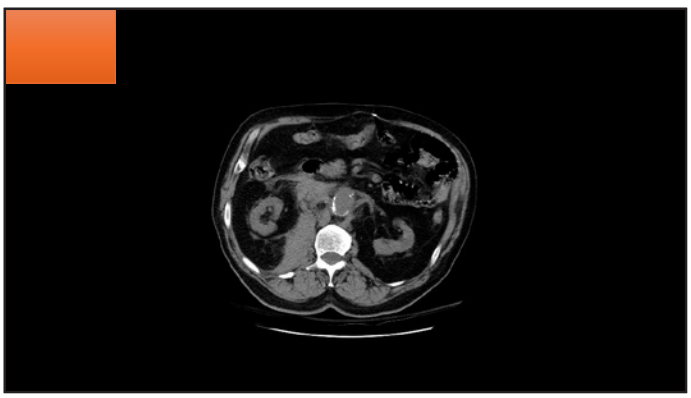


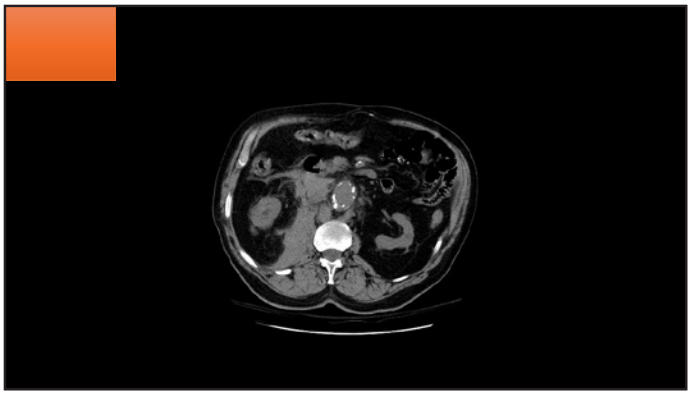
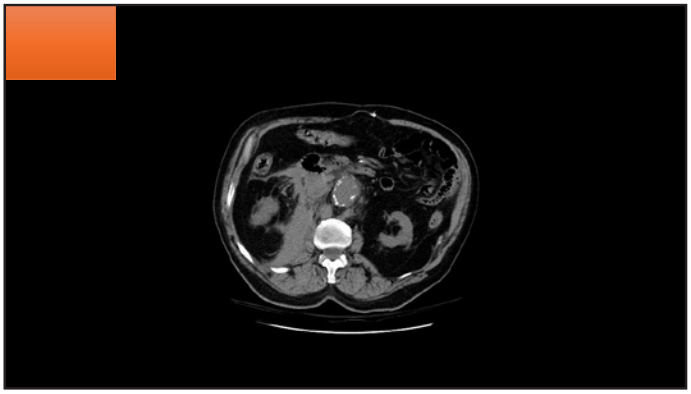
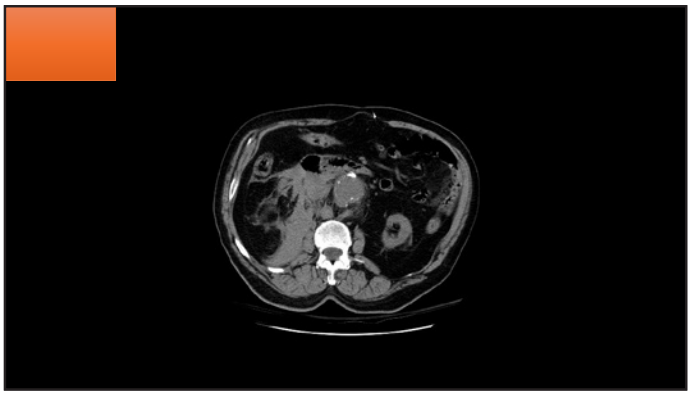
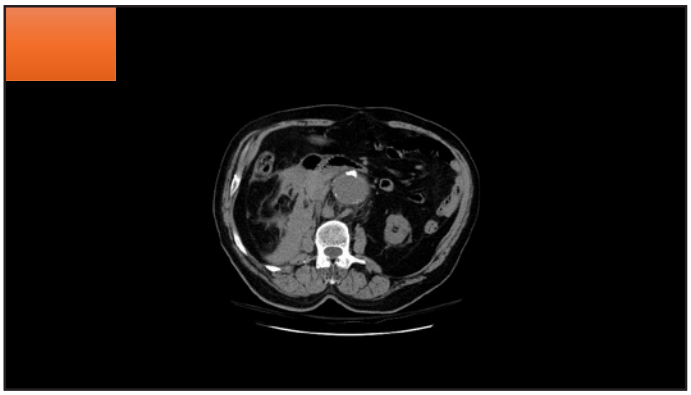


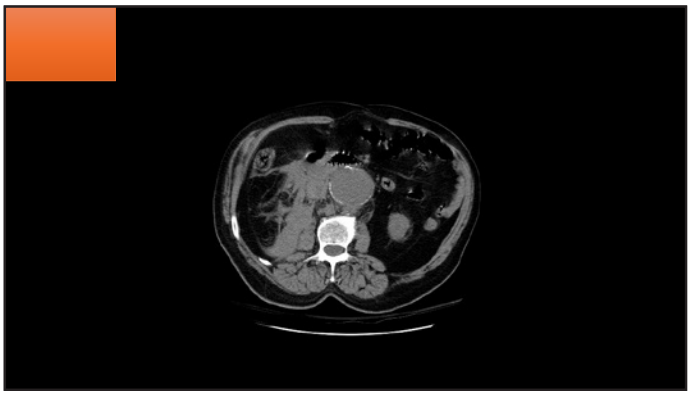
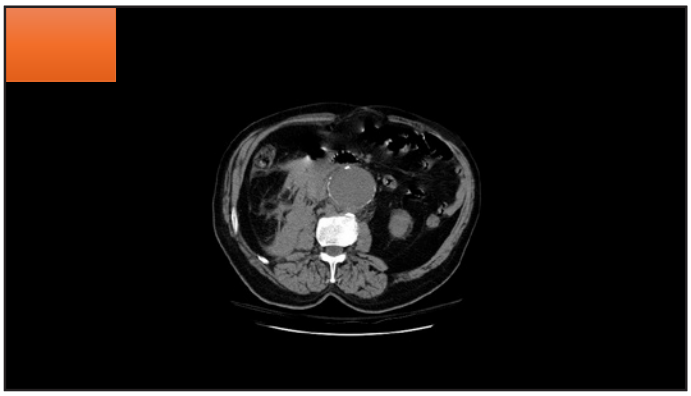
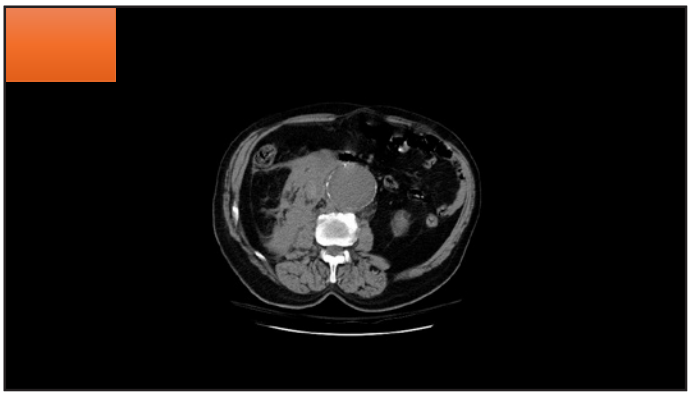


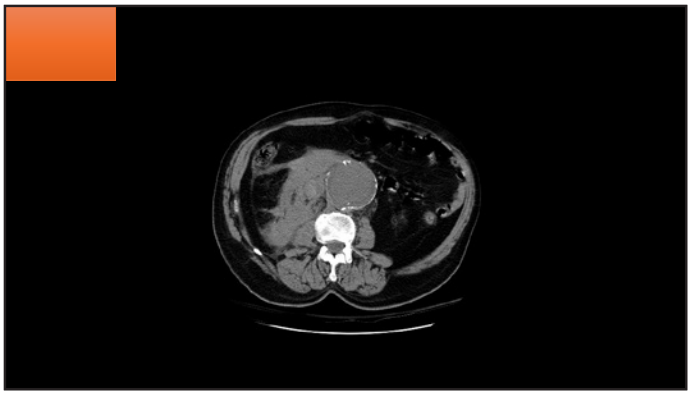


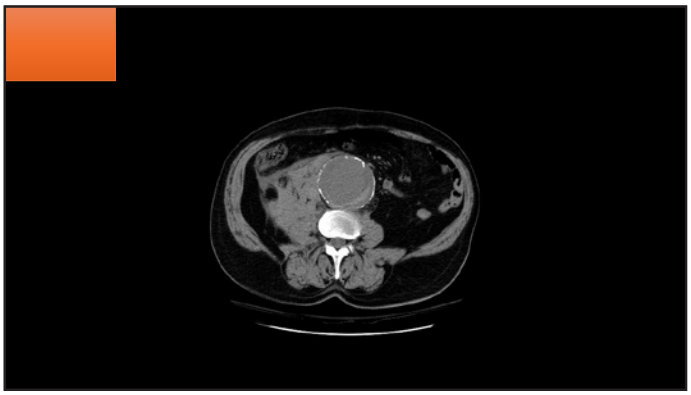


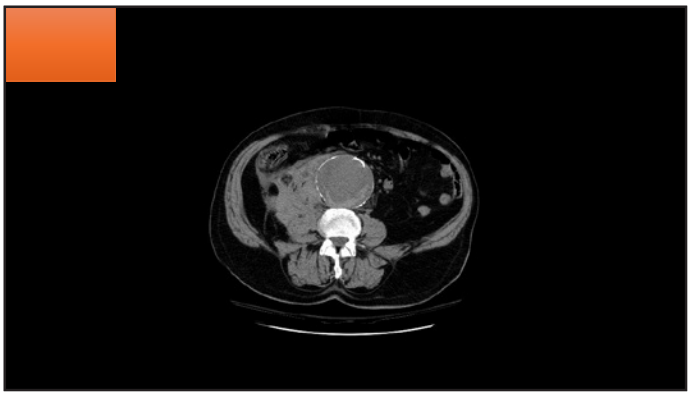





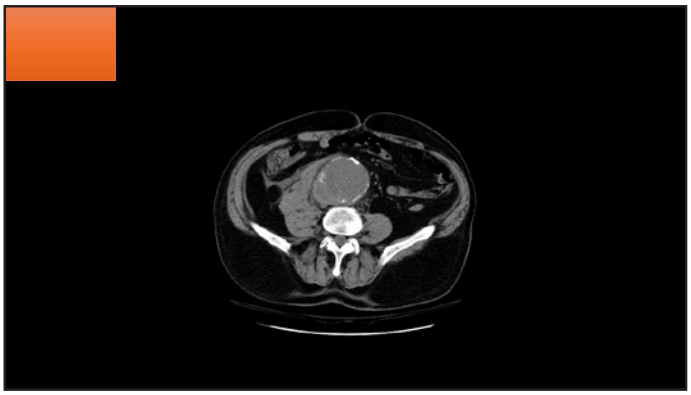


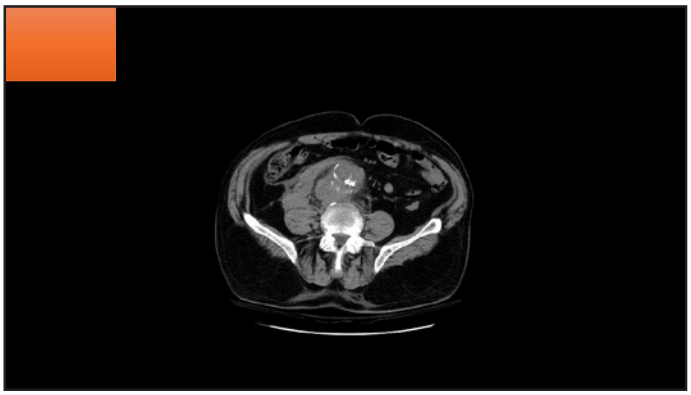
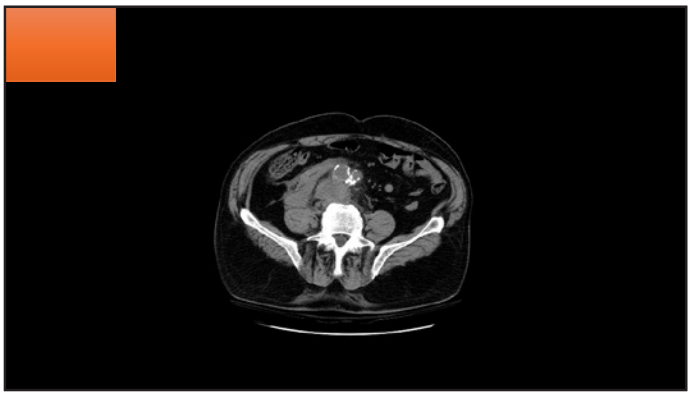
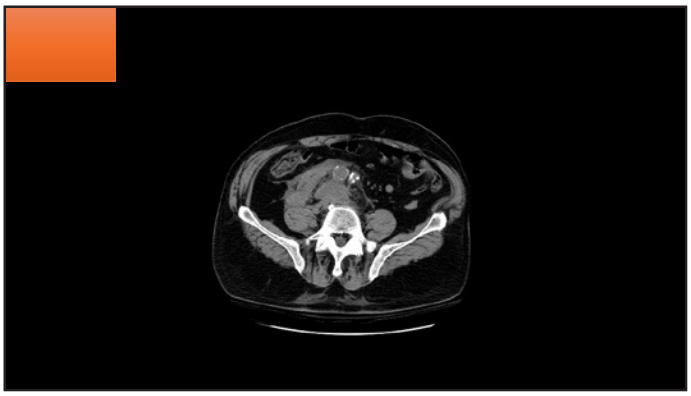


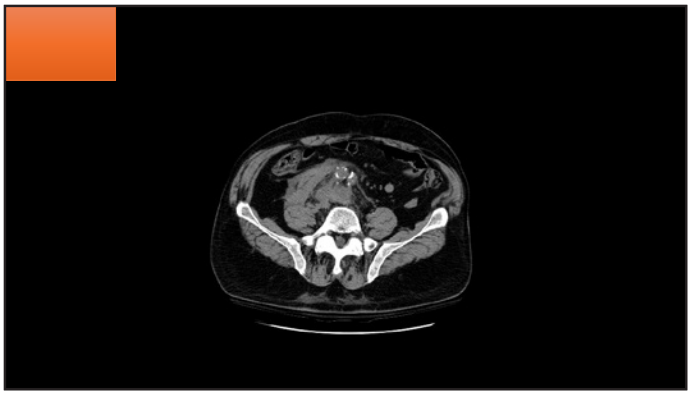



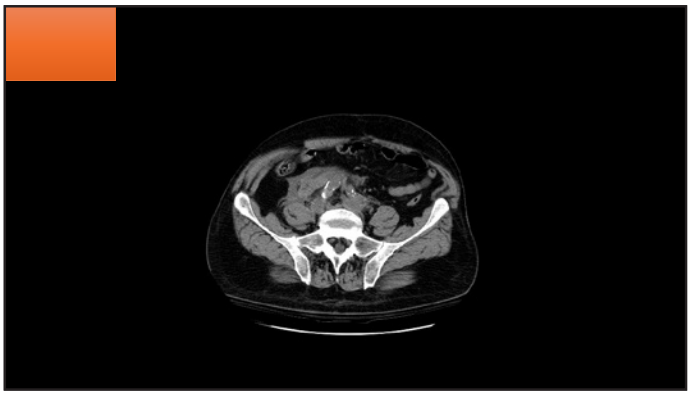


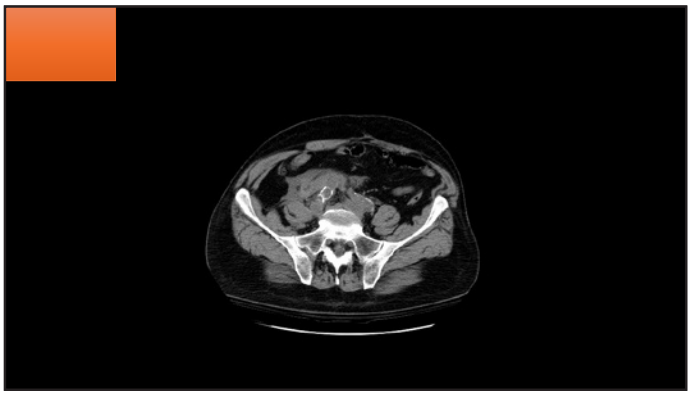


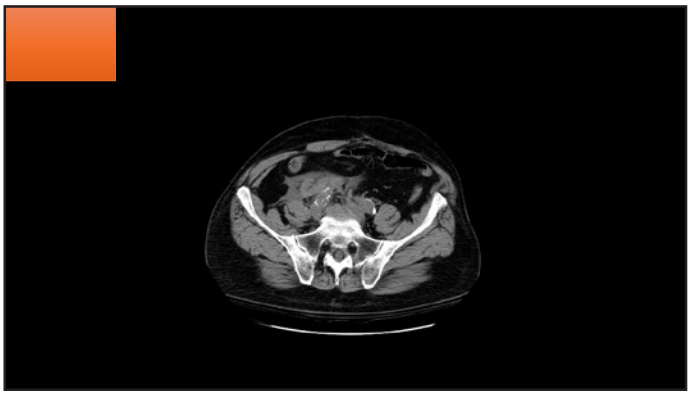





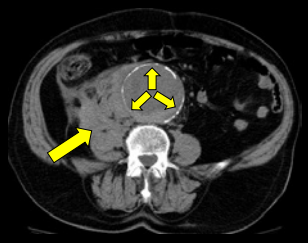






Oh My!!

Abdominal Aortic Aneurysm



Large calcified aneurysm

Soft tissue density in the retroperitoneum c/w rupture

No contrast necessary!

IV contrast is not needed for the *detection* of AAA

Surgeons will usually want it to evaluate extent and for organ/vessel involvement

"Tennis Ball Sign"

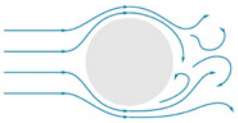
You may need IV contrast if the non-con scan is negative

Abdominal Aortic Aneurysm

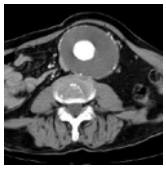
Very often see low-density material around the wall of the aneurysm

This is known as *mural thrombus*

Mural Thrombus



Turbulent flow leads to build-up of thrombus



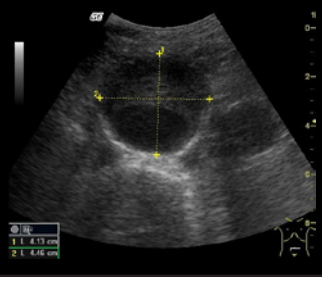
It can become quite large

What about ultrasound?

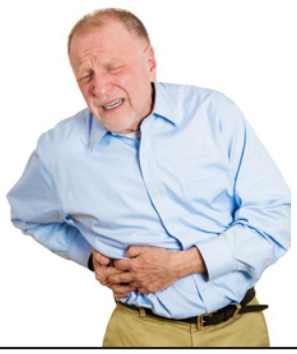
Great test for the detection of *presence* of a AAA

Sensitivity 94-97%
Specificity 97-99%

Ultrasound is NOT adequate to determine rupture



62 y.o. male with sudden-onset flank pain

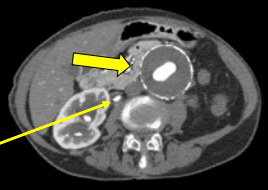


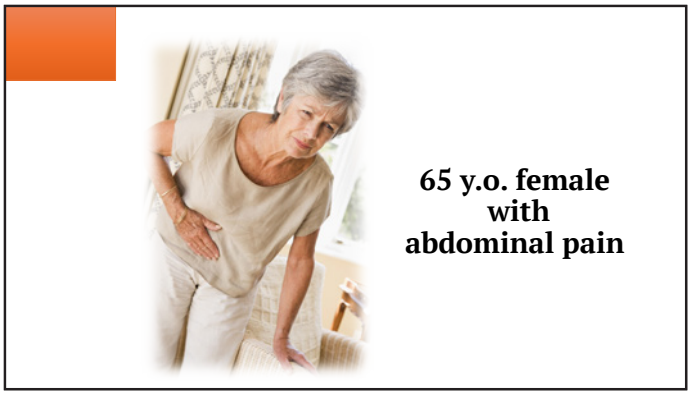
Sometimes people are just unlucky

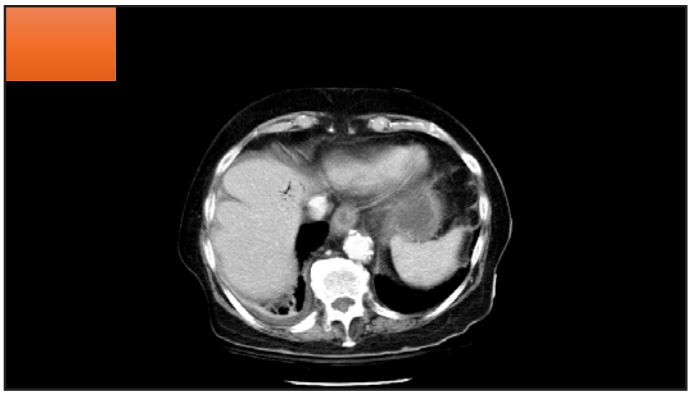
Large calcified AAA with mural thrombus

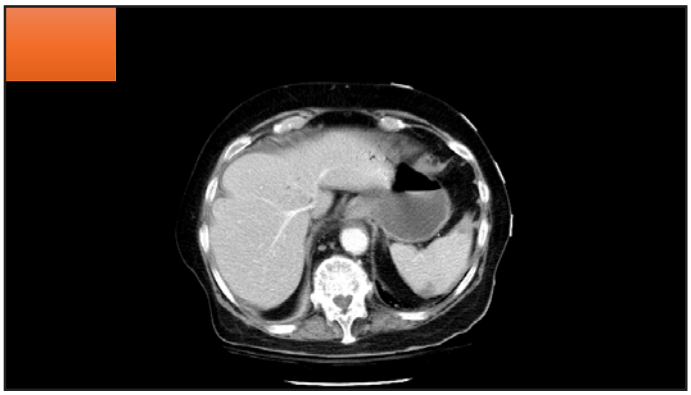
and

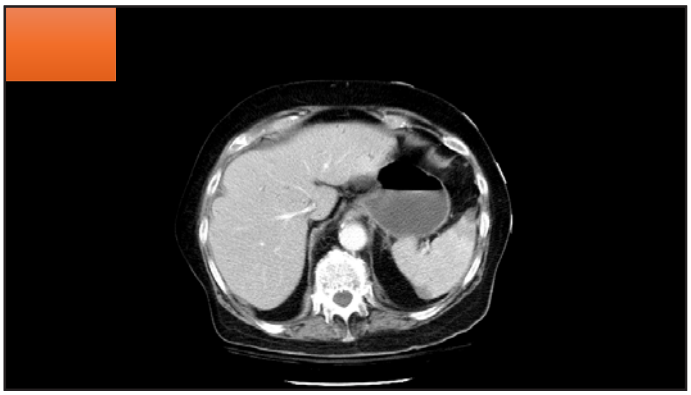
A large proximal ureteral stone



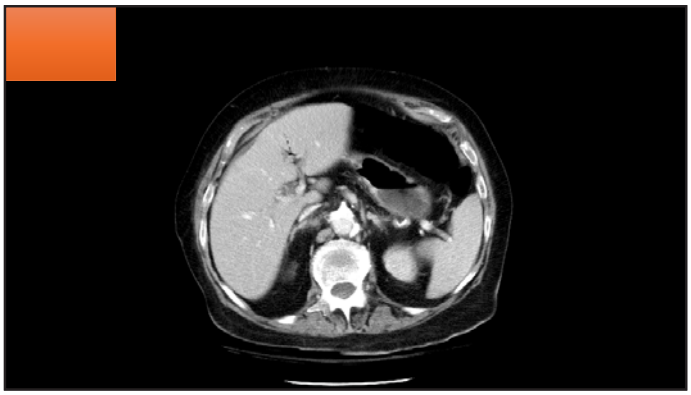
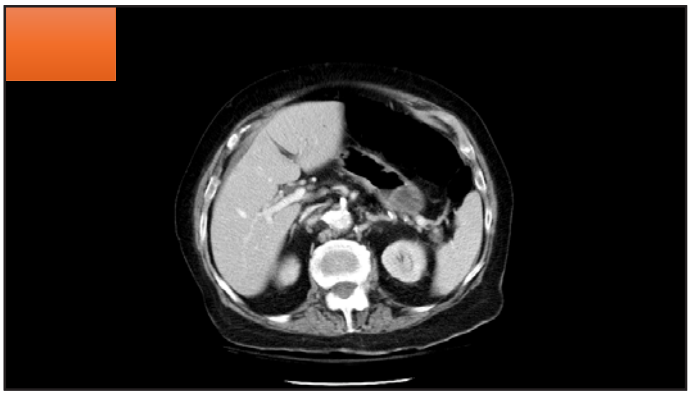

65 y.o. female
with
abdominal pain

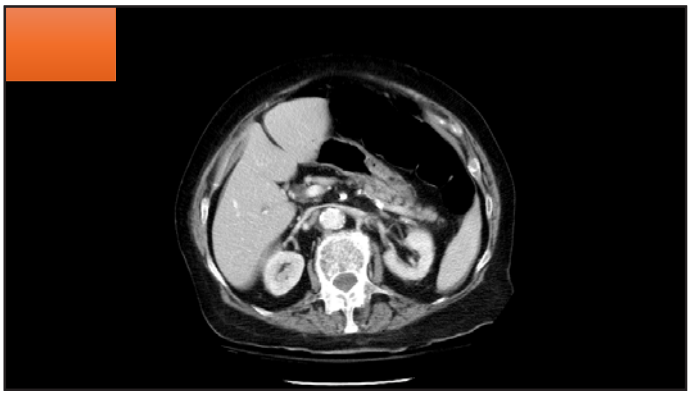
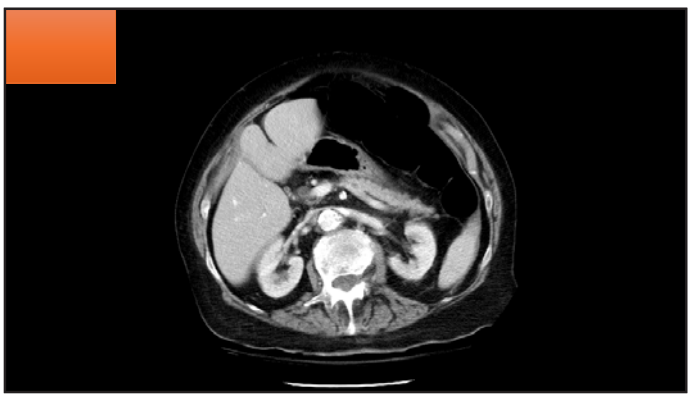




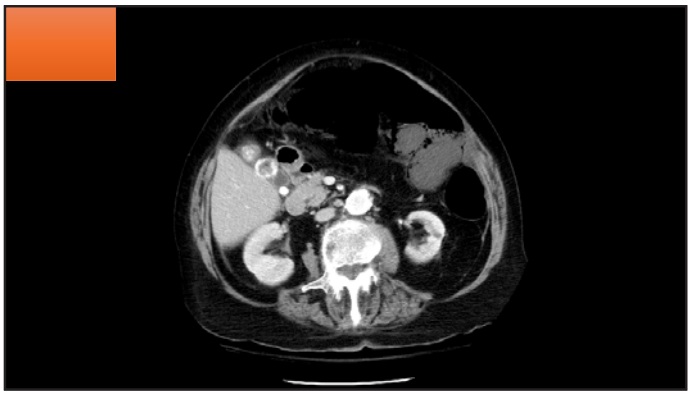


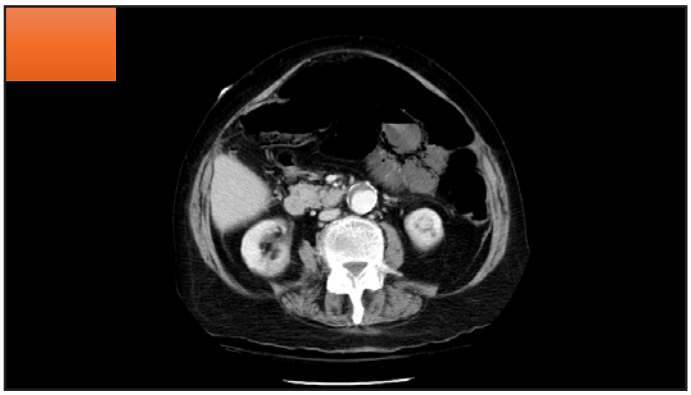
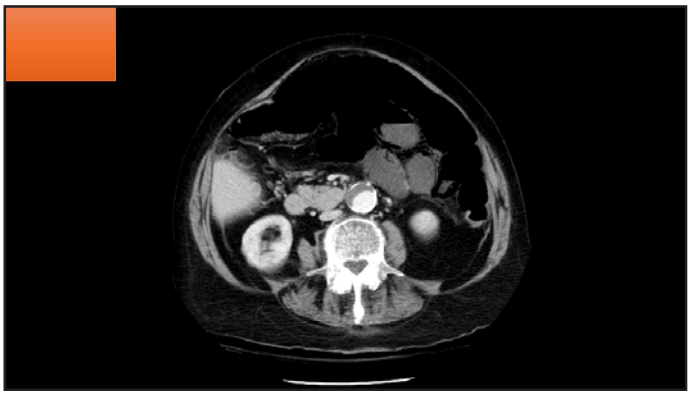


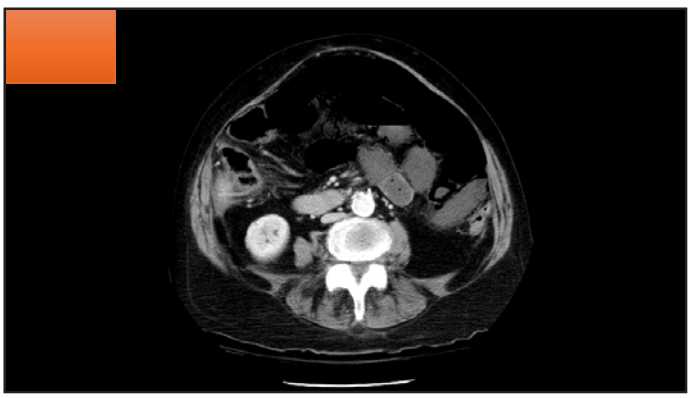





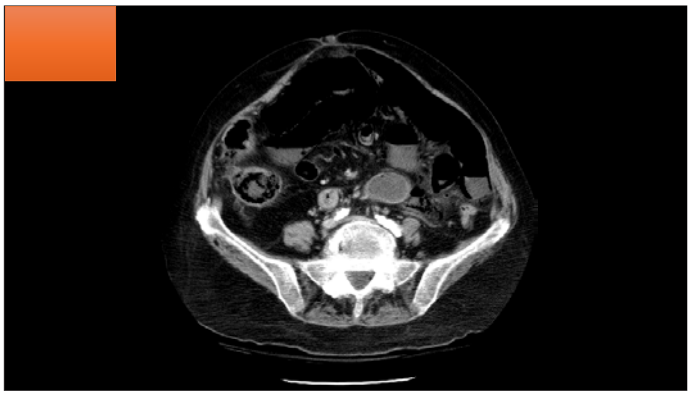
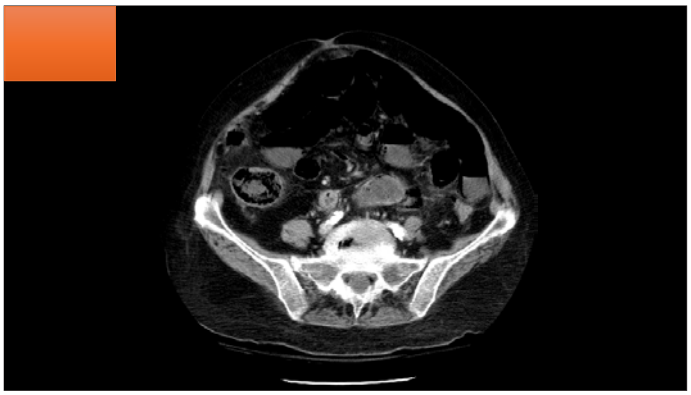



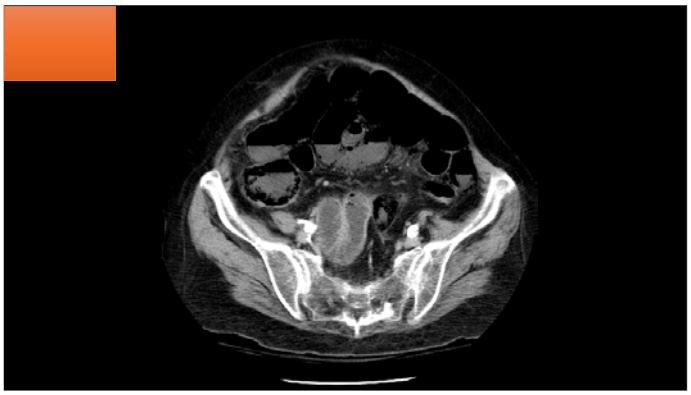


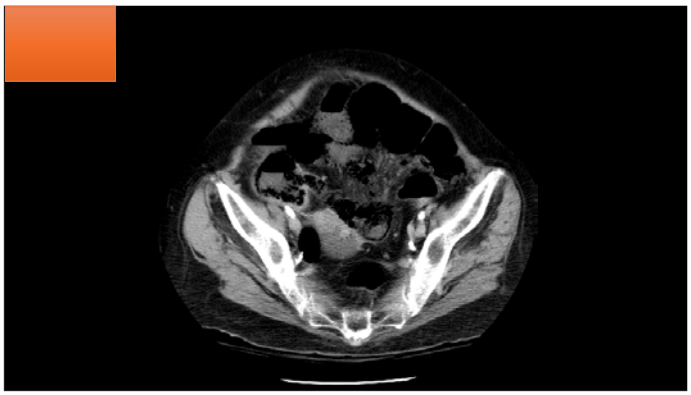


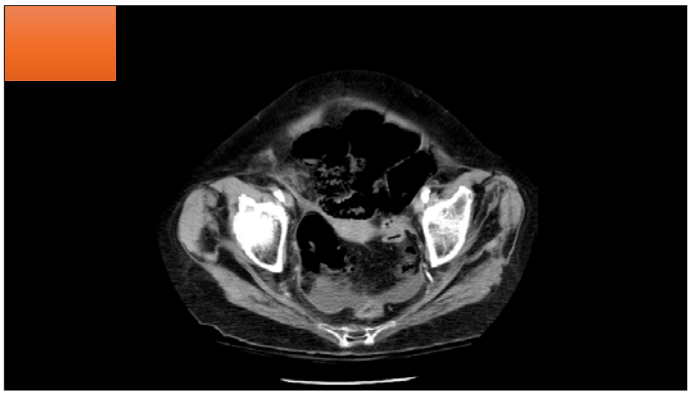


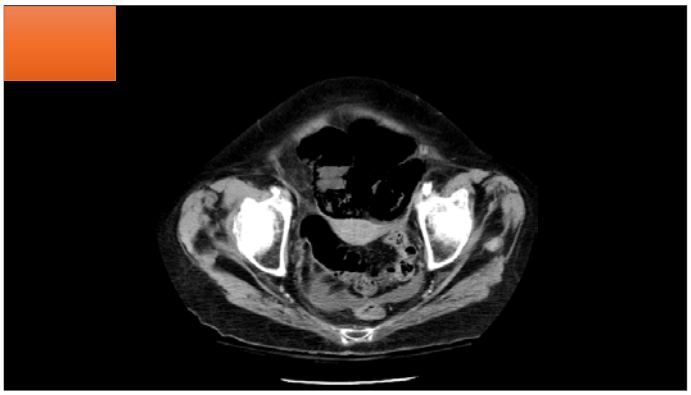



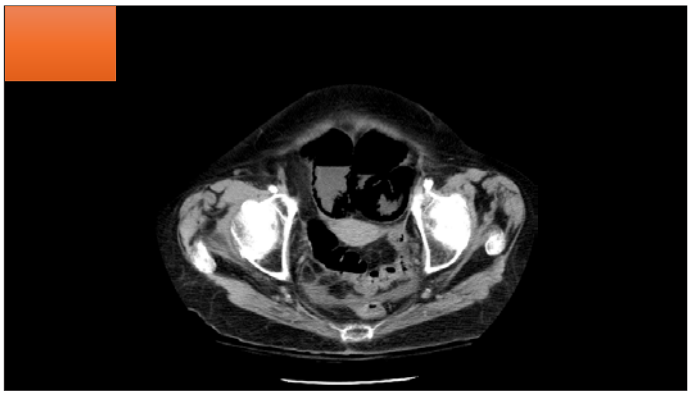


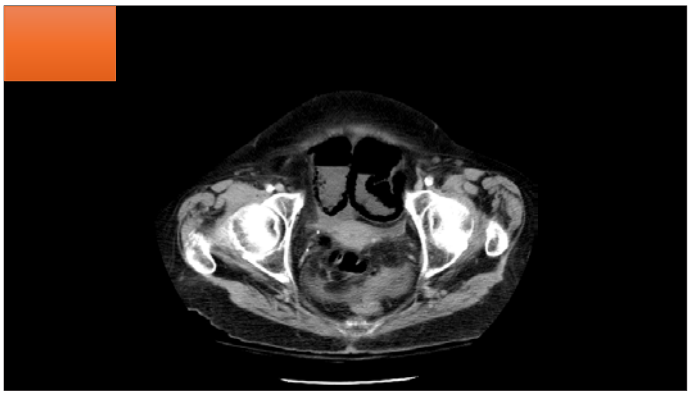





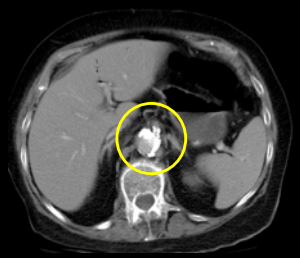




There's…**A LOT**…going on with this one

Let's break it down

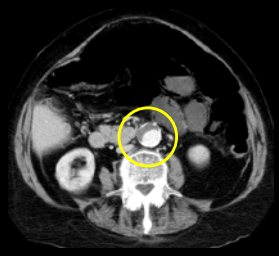
Look at the vasculature



Significant vascular disease in the aorta

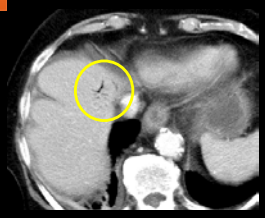
Very tight take-off of the celiac axis and SMA

Look at the vasculature



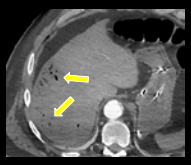
Aneurysmal dilation of the aorta with mural thrombus

Next look at the solid organs

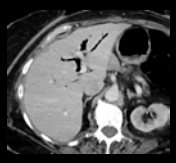


Small areas of gas within the liver c/w portal venous gas

Portal Venous Gas vs Biliary Air



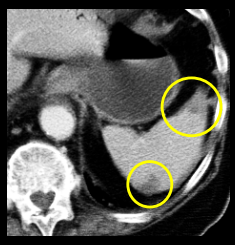
Portal venous gas



Pneumobilia

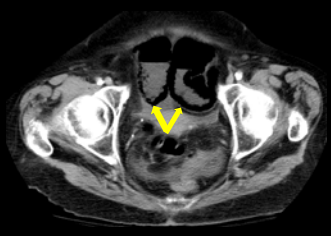
Portal venous gas goes to the periphery, biliary air stays central

Let's look at the spleen



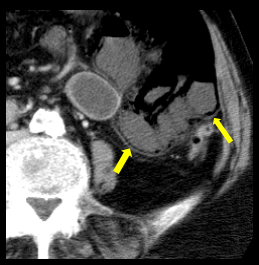
Hypodense areas in the spleen concerning for infarction

Look carefully at the Small Bowel



There are areas of *non-dependent* air in the small bowel concerning for pneumatosis

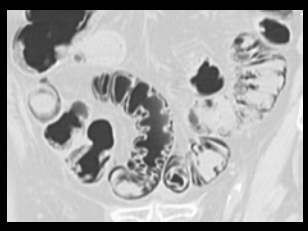
Pneumatosis Intestinalis



Normally air rises in liquid stool (*dependent air*)

With ischemic bowel, see air trapped within the wall in a non-dependent location

Pro Tip: Use lung windows to look for air where it doesn't belong



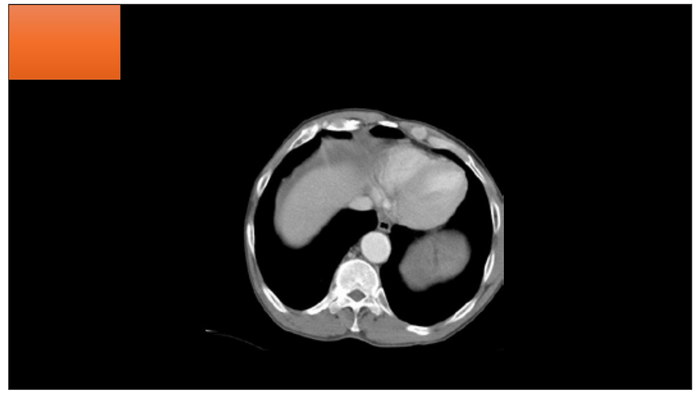
Putting it all together in this case

65 y.o. woman with significant underlying vascular disease

She developed acute atrial fibrillation and threw emboli to her mesenteric vessels

She developed acute mesenteric ischemia as well as splenic infarcts


23 y.o. male
in MVA




Progressive Wavelet Level: Full

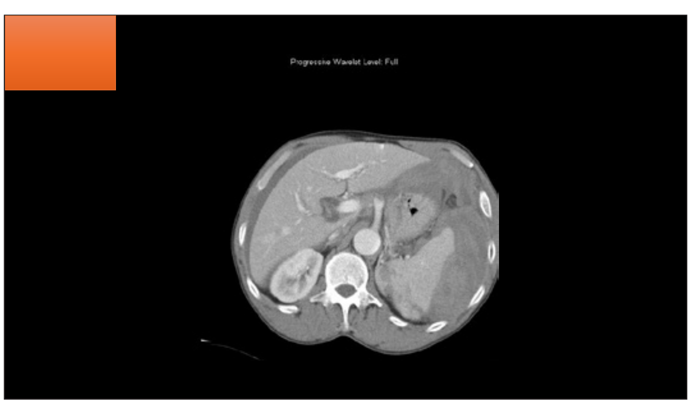

Progressive Wavelet Level: Full


Progressive Wavelet Level: Full

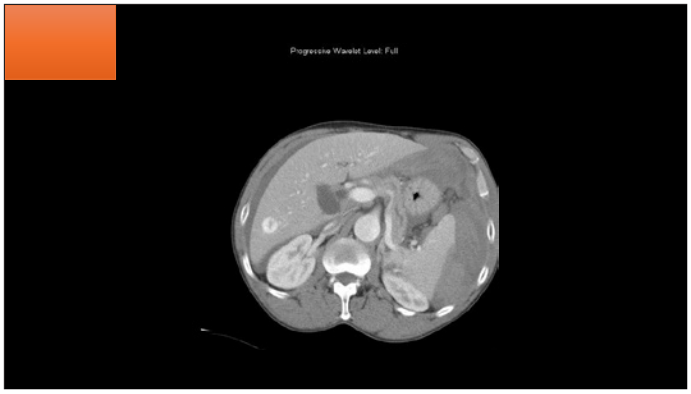

Progressive Wavelet Level: Full


Progressive Wavelet Level: Full


Progressive Wavelet Level: Full


Progressive Wavelet Level: Full


Progressive Wavelet Level: Full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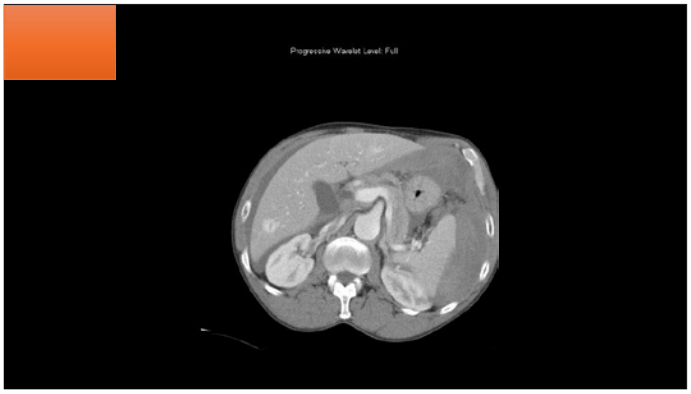
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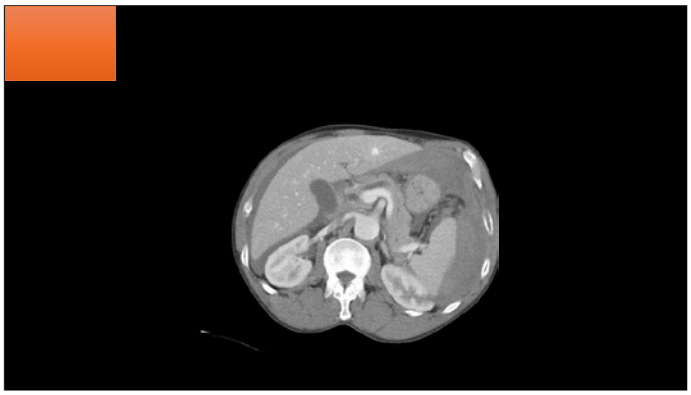
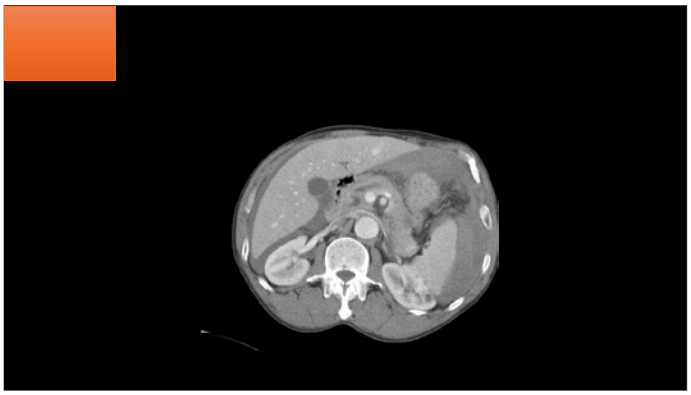





Progressive Wavelet Level: Full


Progressive Wavelet Level: Full


Progressive Wavelet Level: Full

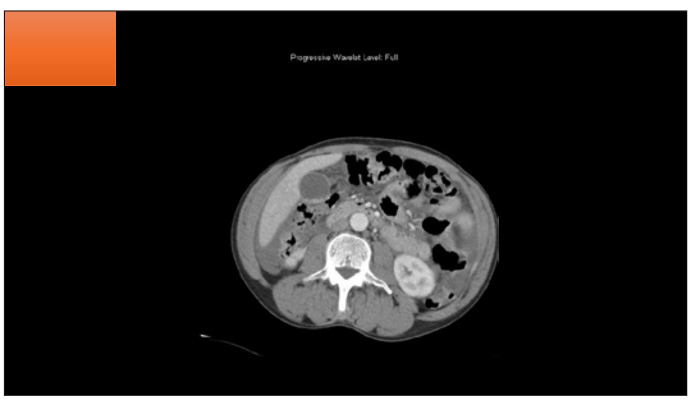

Progressive Wavelet Level: Full


Progressive Wavelet Level: Full


Progressive Wavelet Level: Full

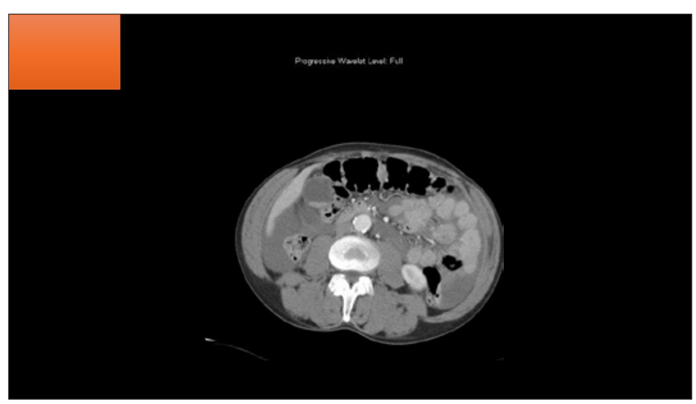

Progressive Wavelet Level: Full


Progressive Wavelet Level: Full


Progressive Wavelet Level: Full


Progressive Wavelet Level: Full


Progressive Wavelet Level: Full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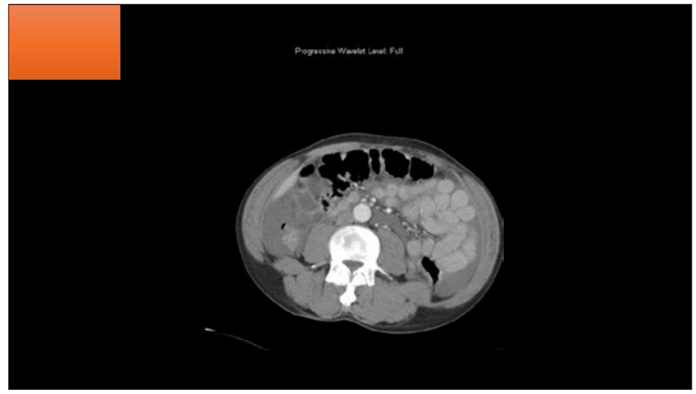
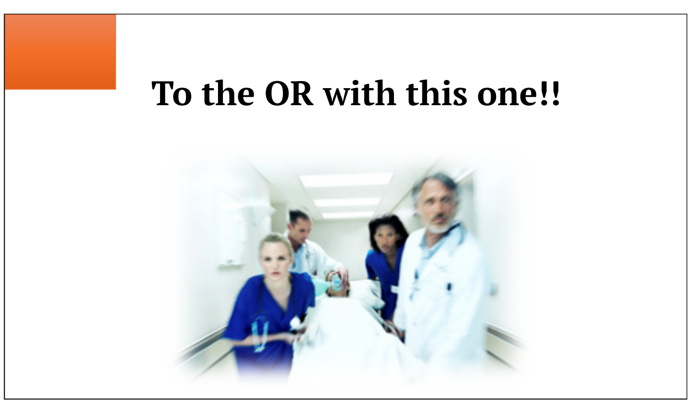
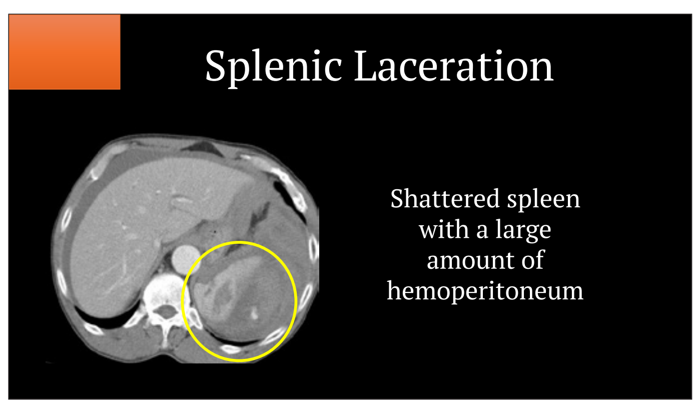
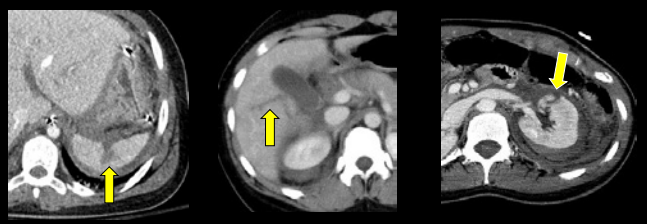



To the OR with this one!!


Splenic Laceration
Shattered spleen with a large amount of hemoperitoneum

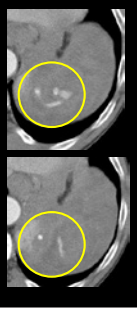
Other examples of solid organ laceration



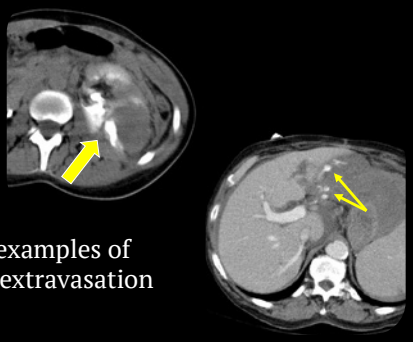
You need to use contrast in trauma!!

You can't adequately evaluate the solid organs without it

Evaluate for a **blush**

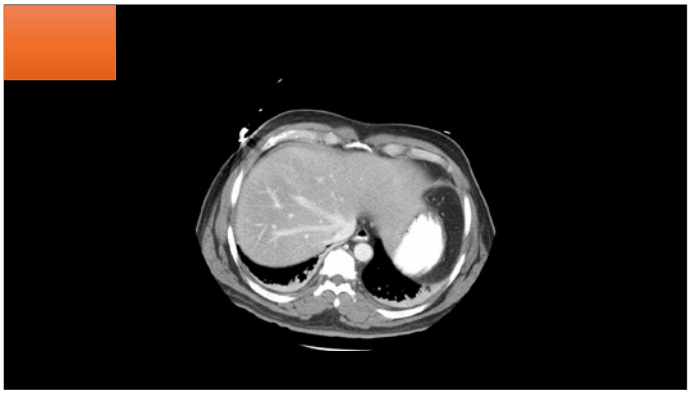


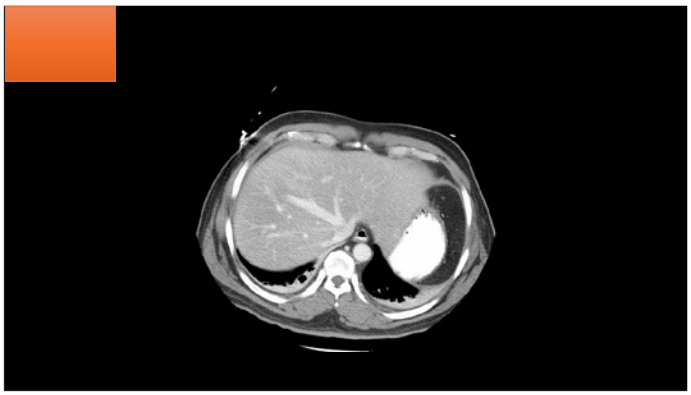
Areas of hyperdensity outside the expected vasculature c/w active extravasation of contrast

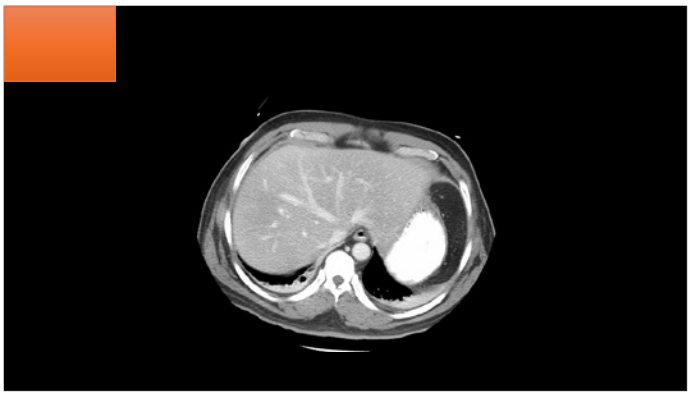


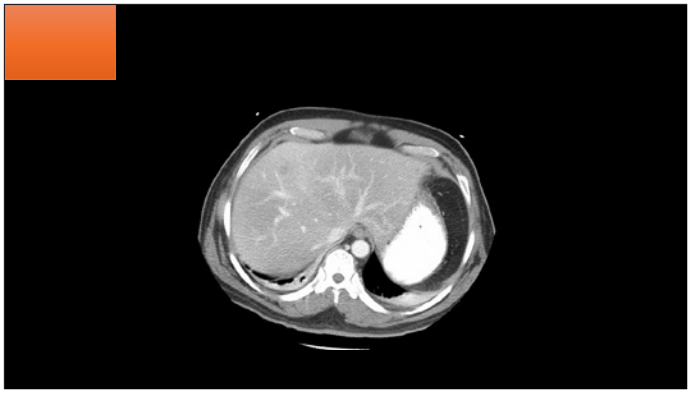
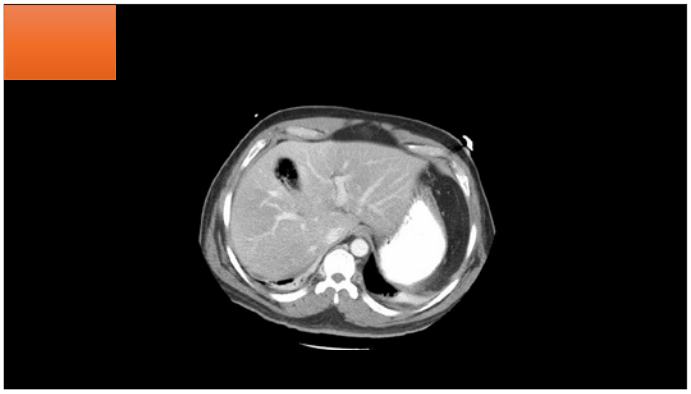
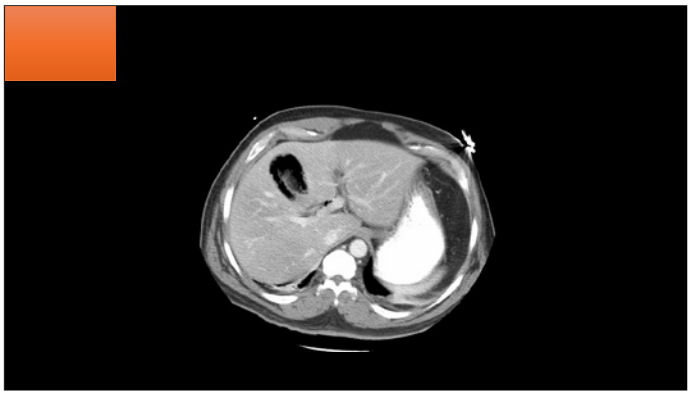
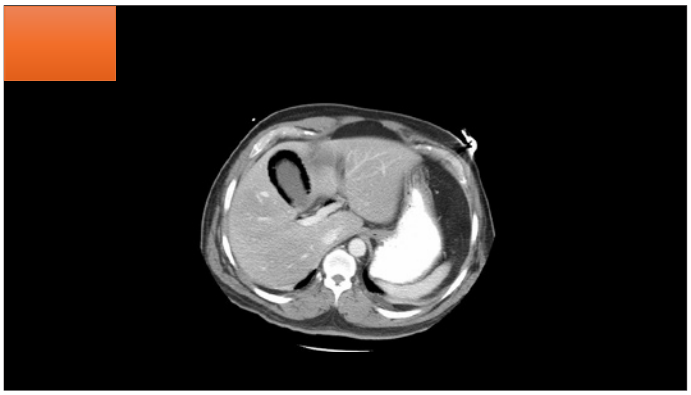
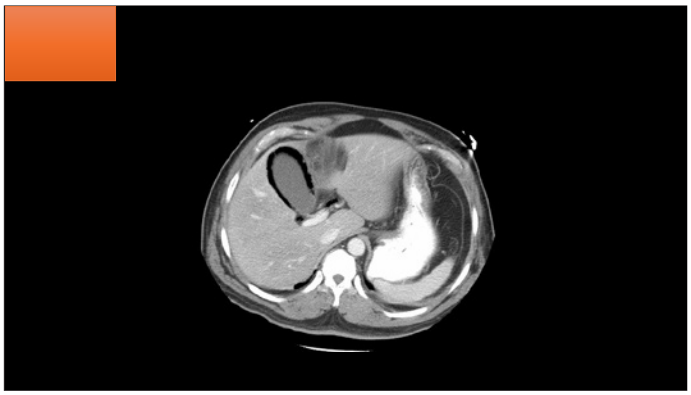
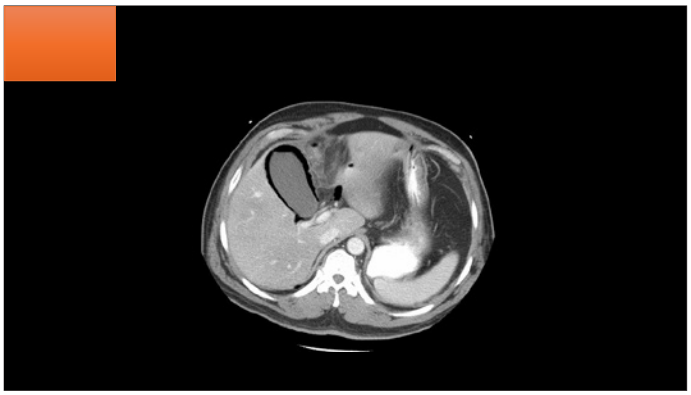
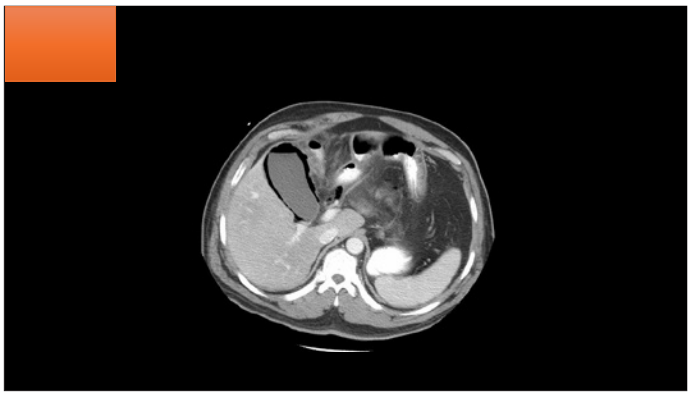
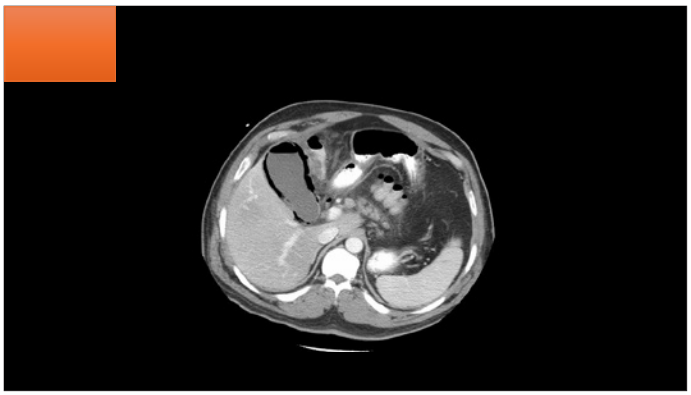
Other examples of contrast extravasation

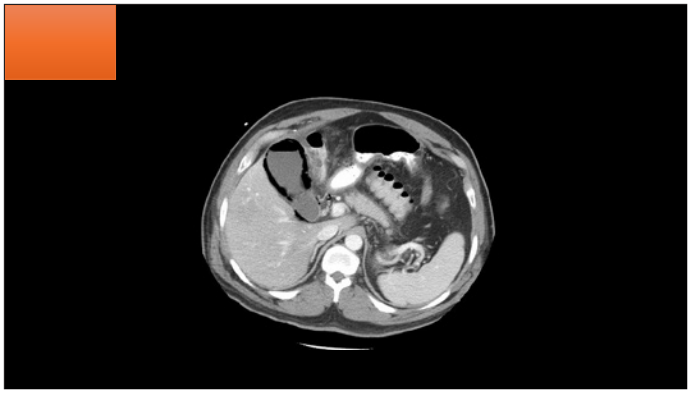
72 y.o. male with abdominal pain

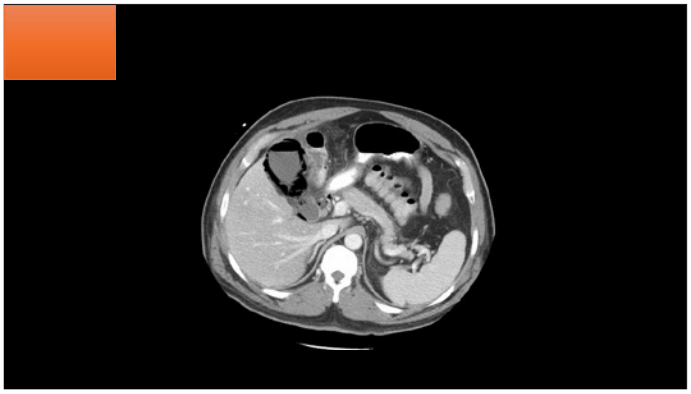


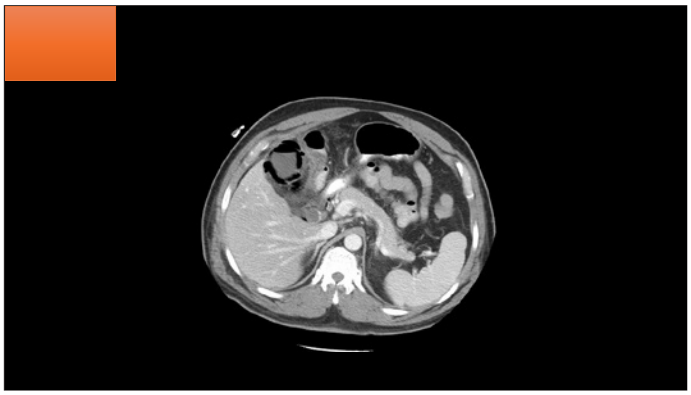


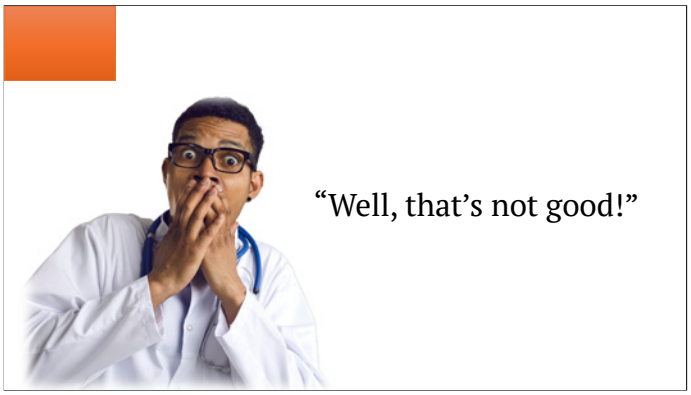


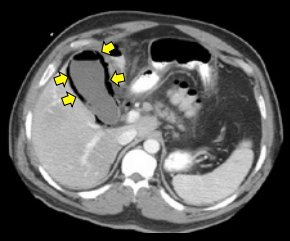








Emphysematous Cholecystitis



Rare form of gangrenous cholecystitis

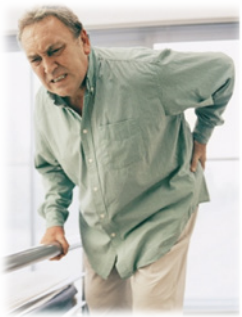
True surgical emergency

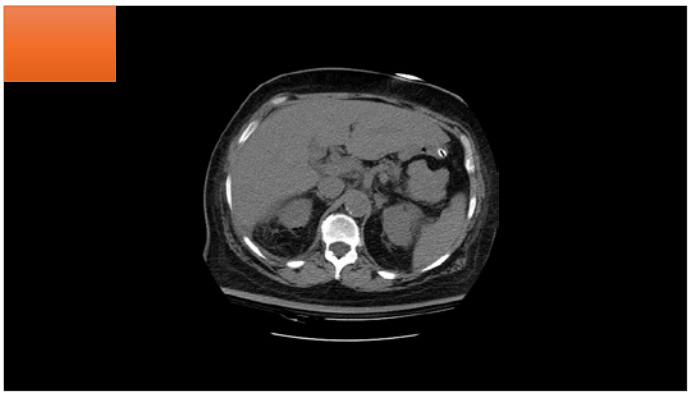
Emphysematous Cholecystitis

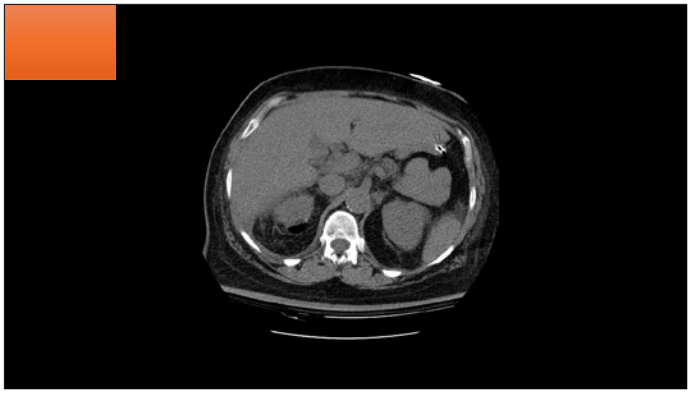
Almost always in diabetics

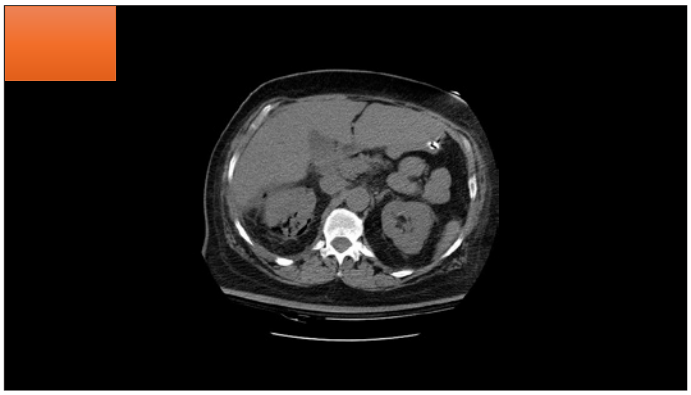
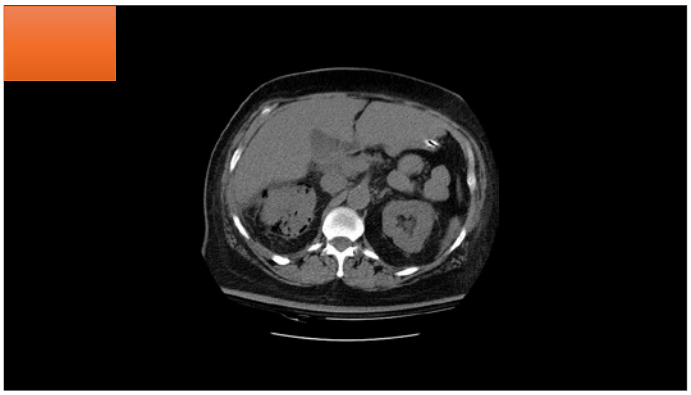
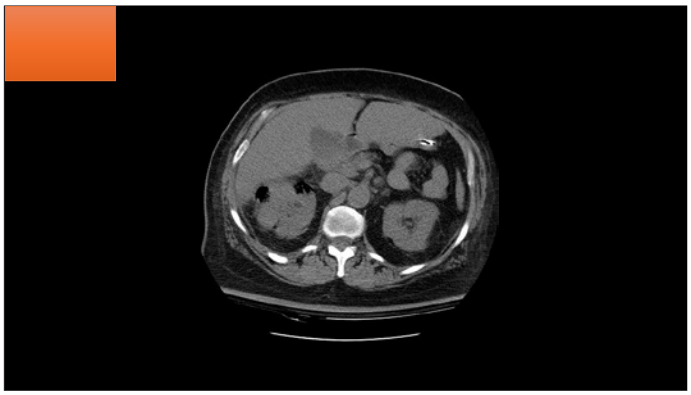
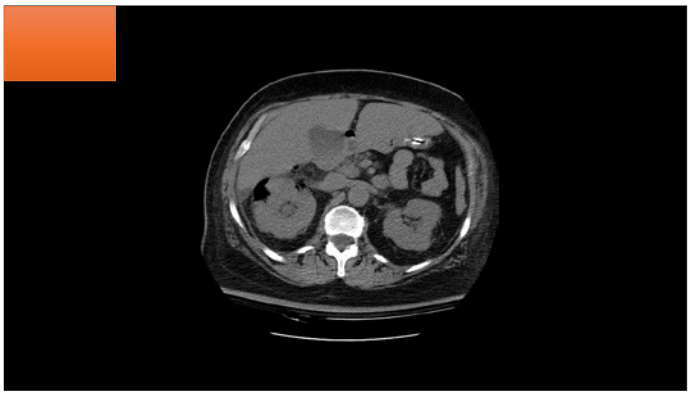
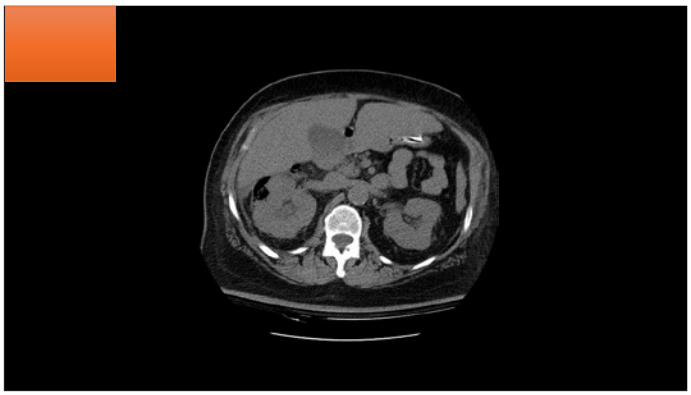
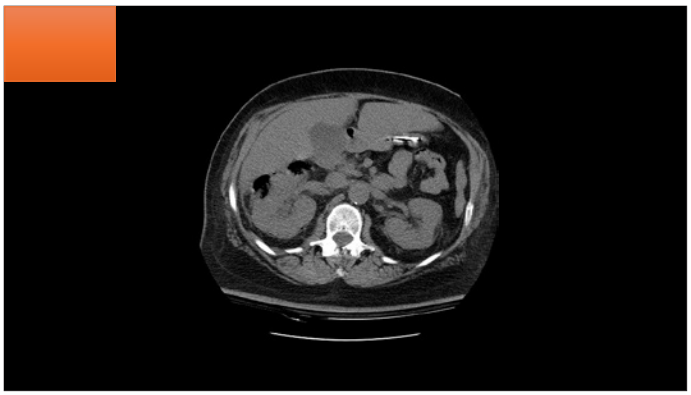
They may not have much tenderness

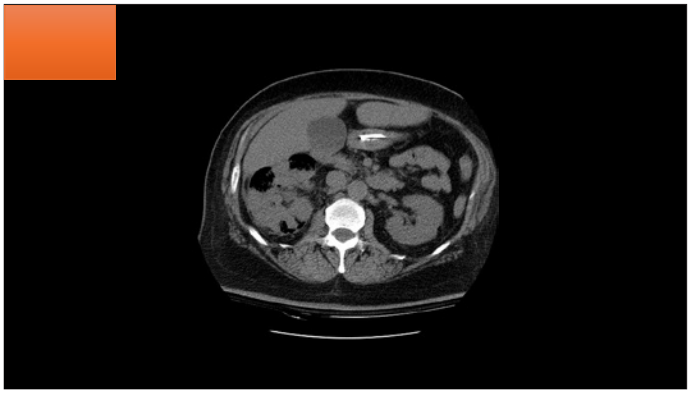
69 y.o. male with flank pain

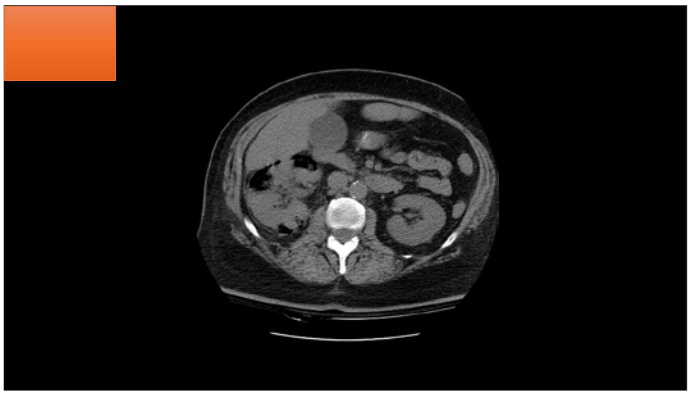




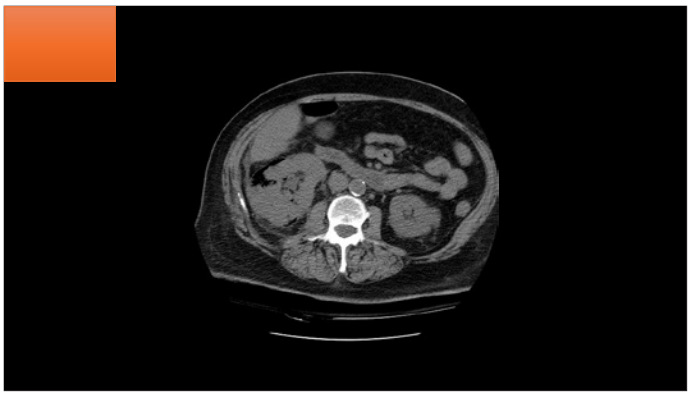


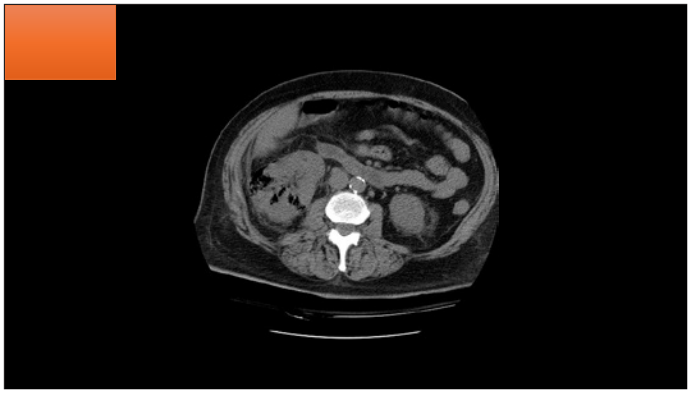







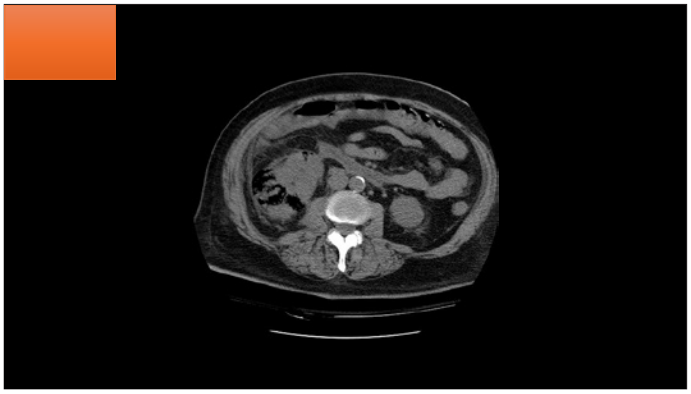



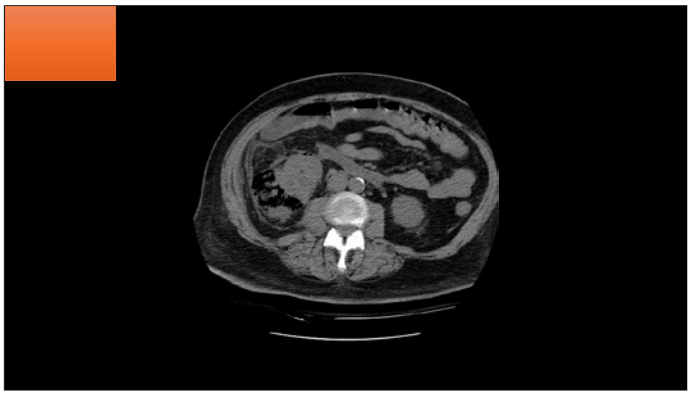


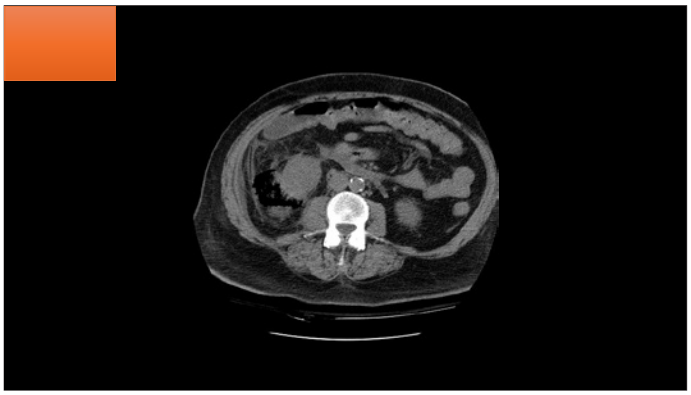


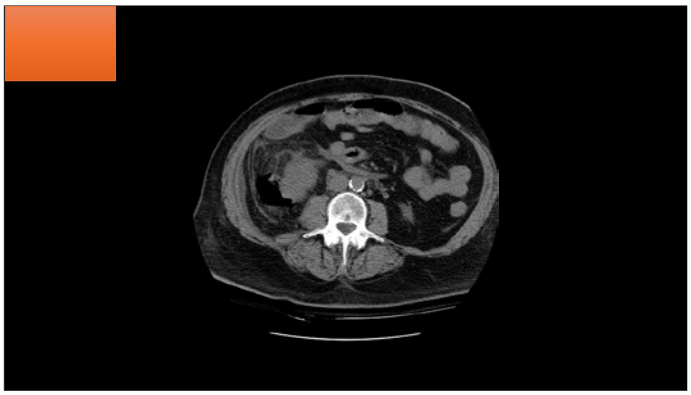


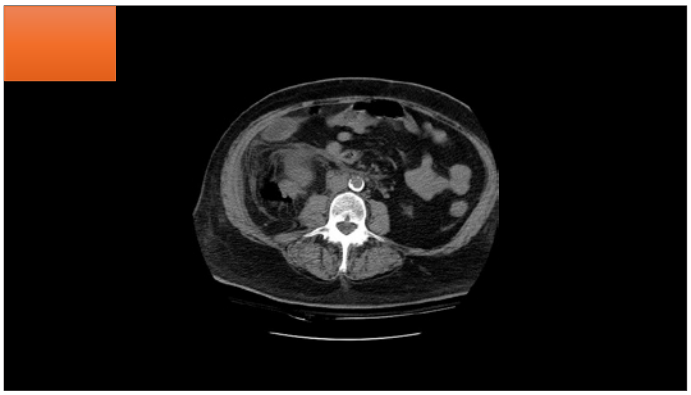


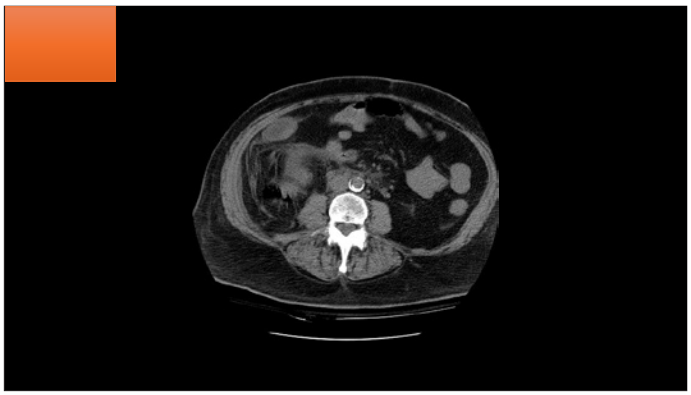






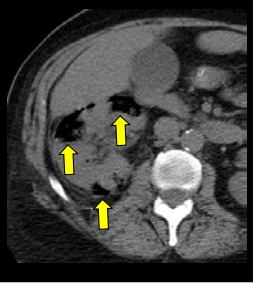







"Well, that's not good either!"

Emphysematous Pyelonephritis



Rare form of gangrenous pyelonephritis

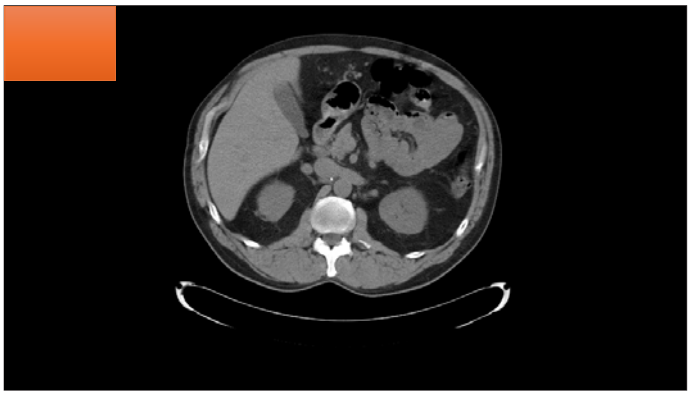
True urologic emergency

Often requires nephrectomy for source control

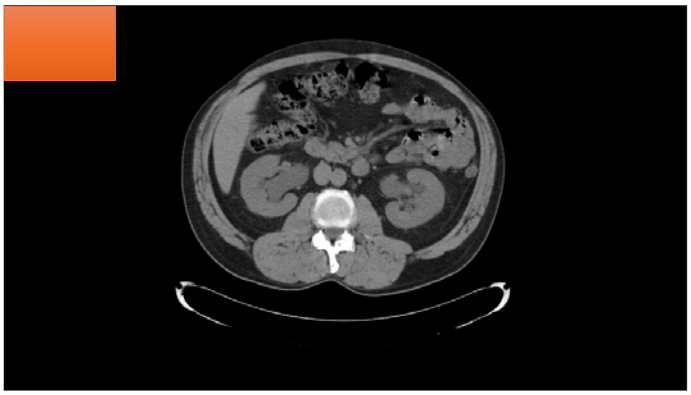
Emphysematous Pyelonephritis

Almost always in diabetics

42 y.o. with bilateral flank pain





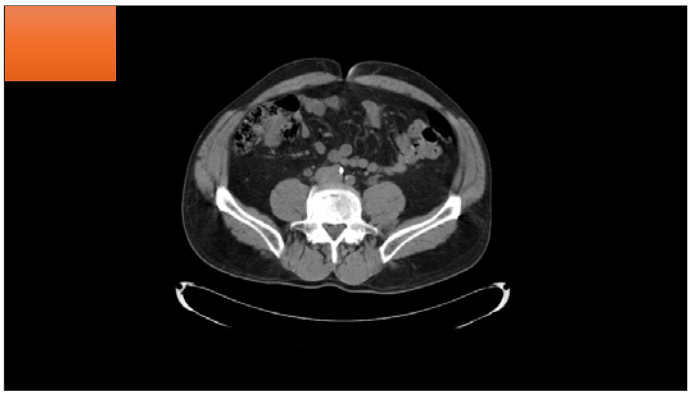
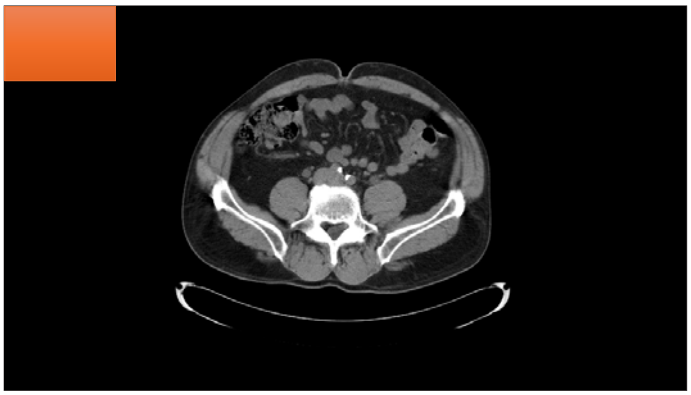
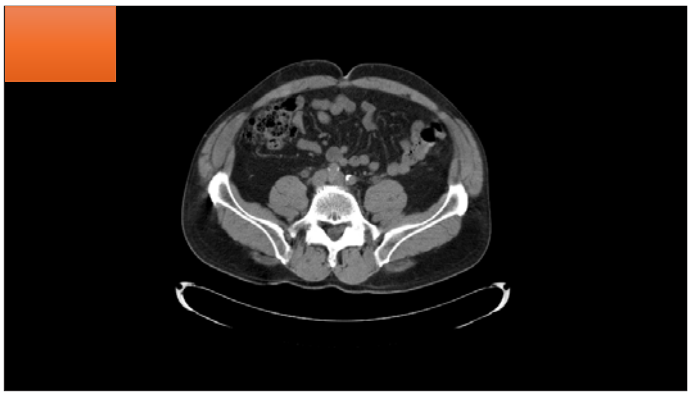
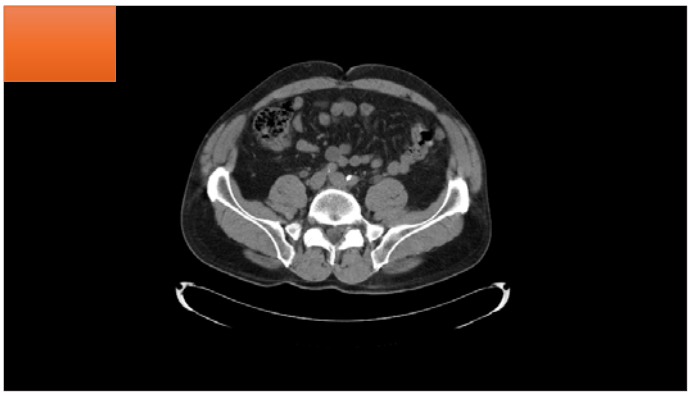



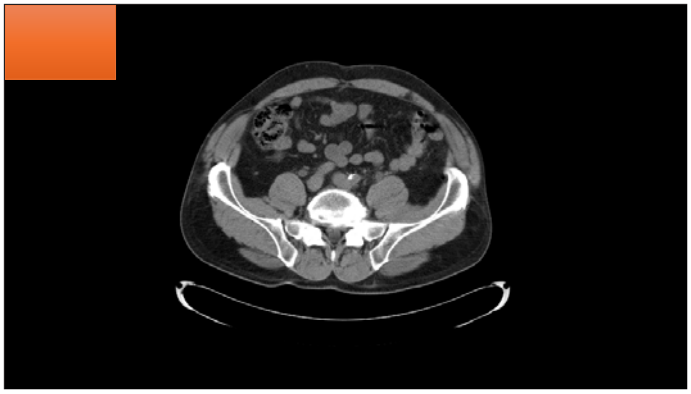
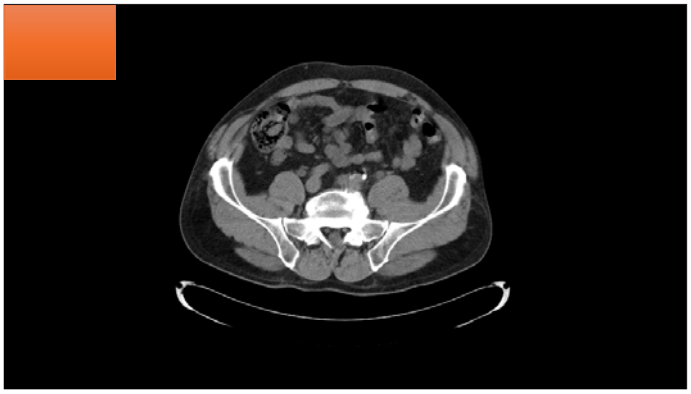
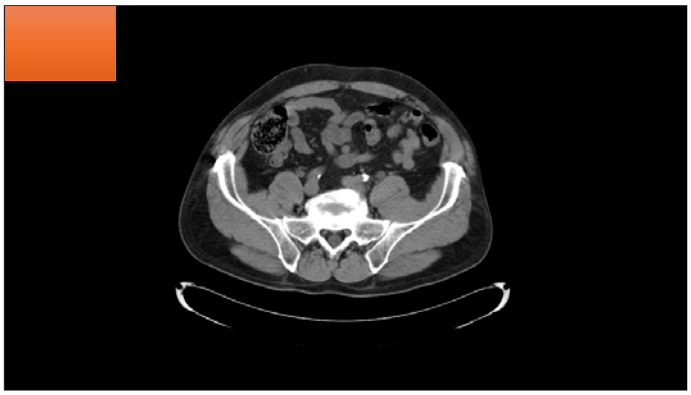
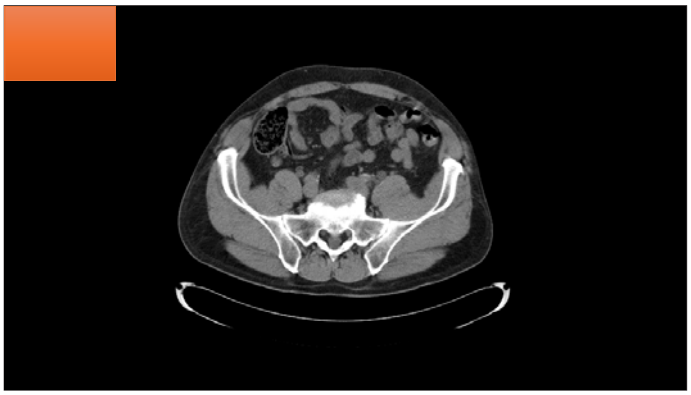
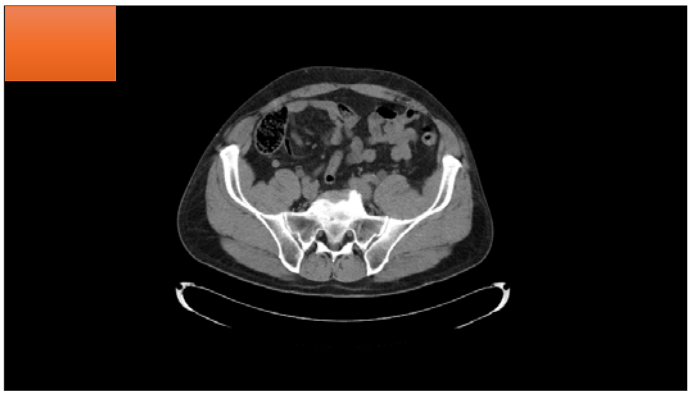
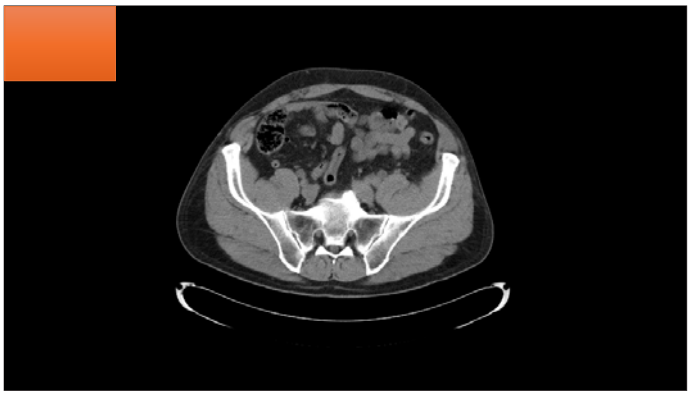
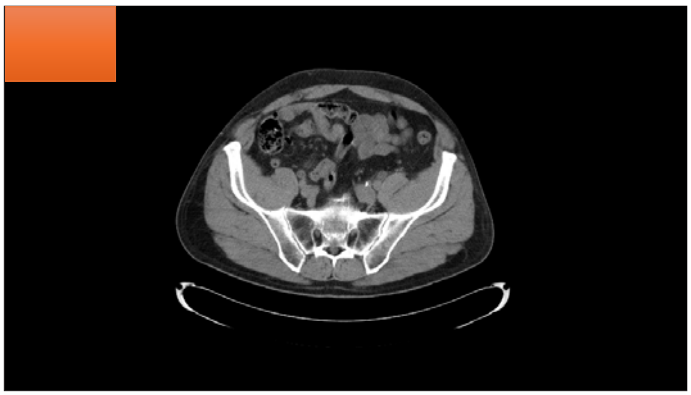
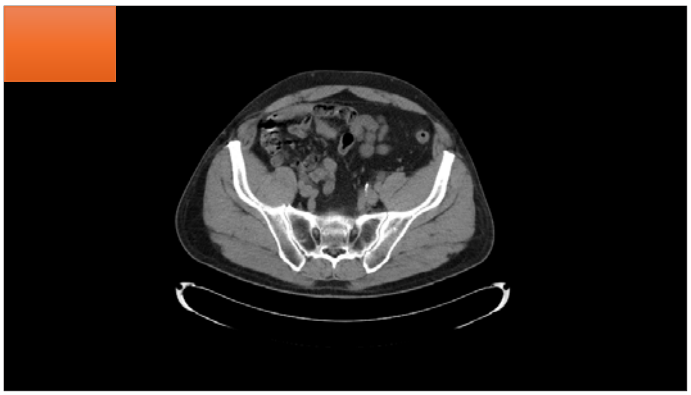


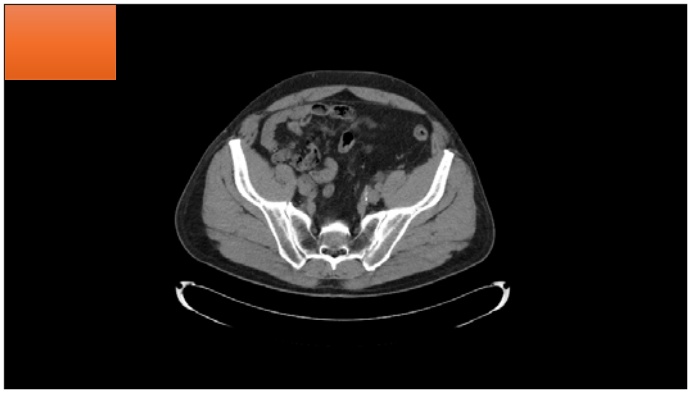
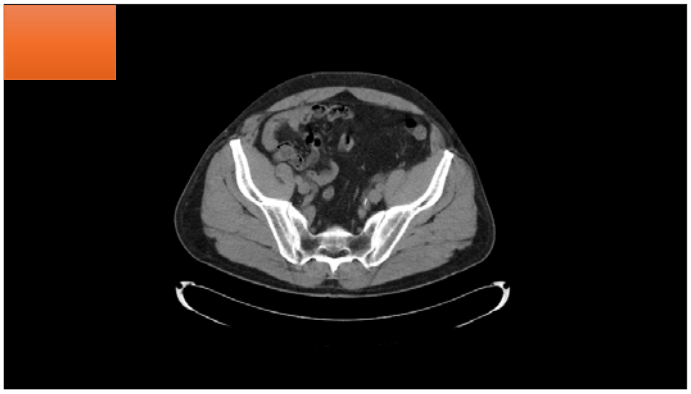
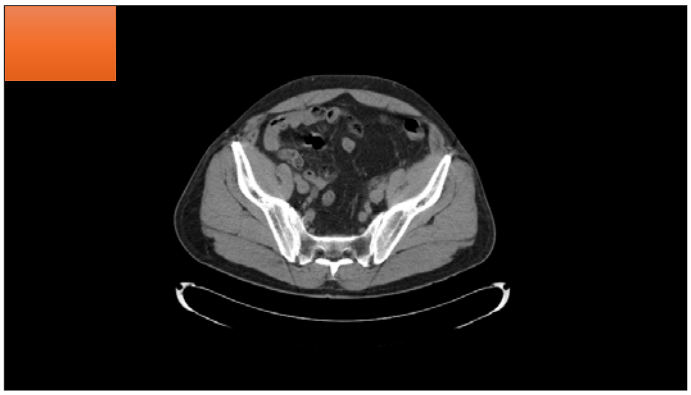
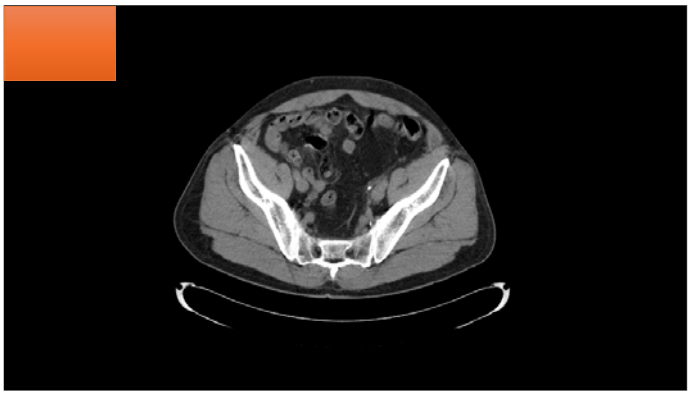
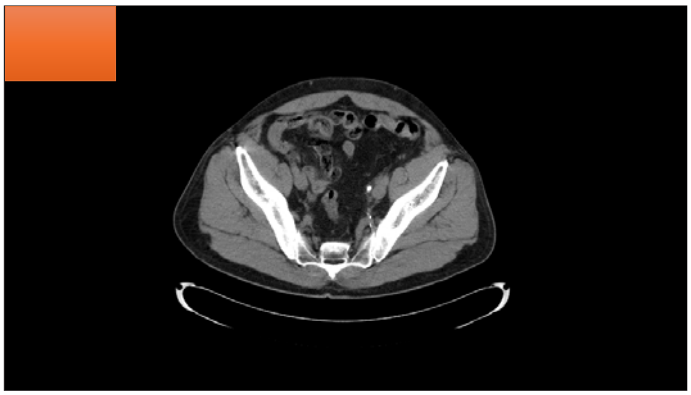
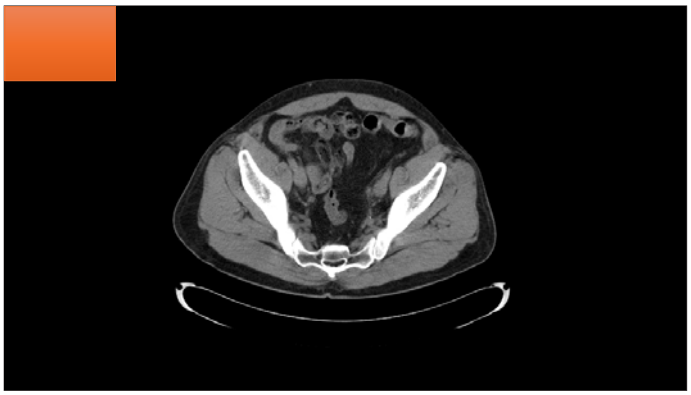
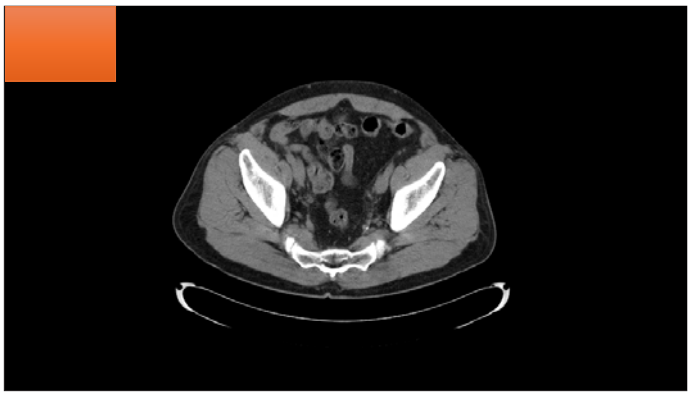
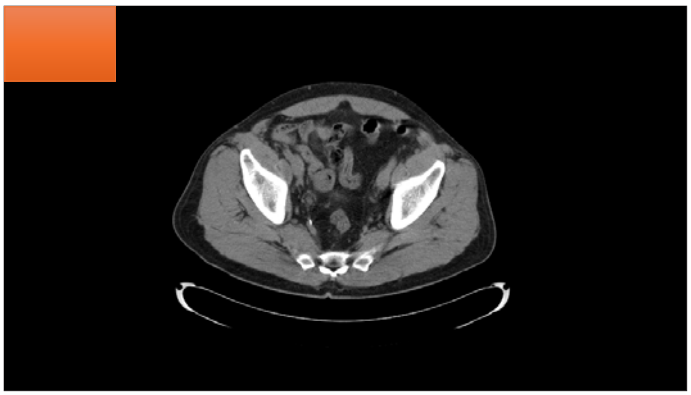

Definitely a stone in the left ureter, but why is the right so dilated as well?

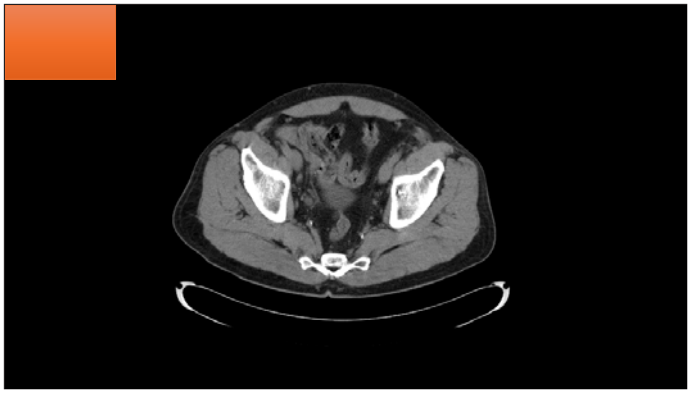


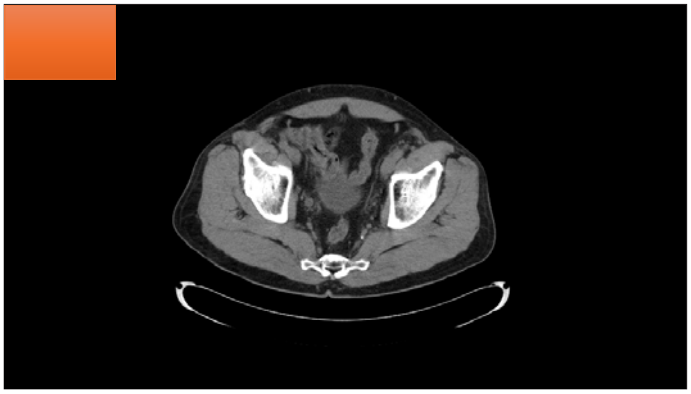


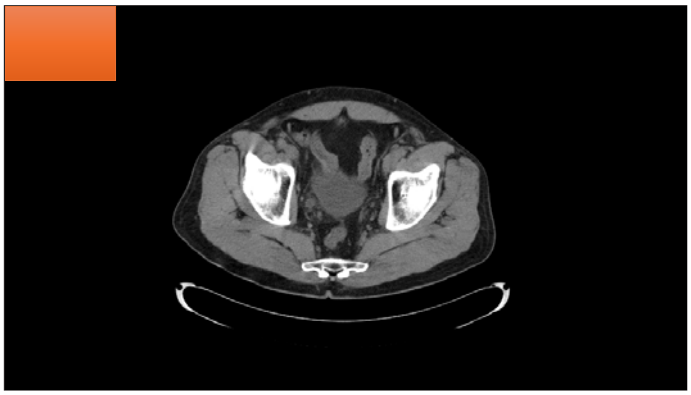





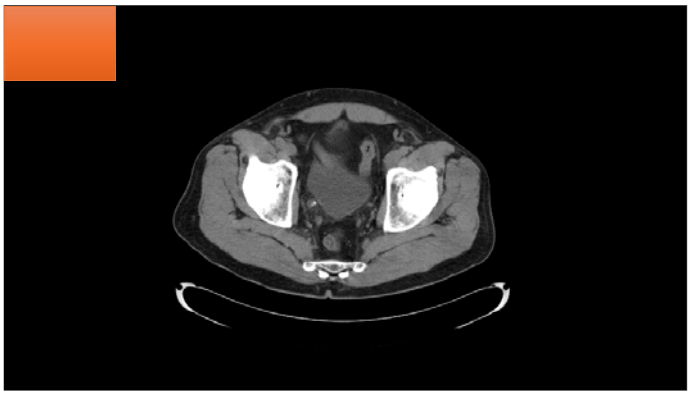









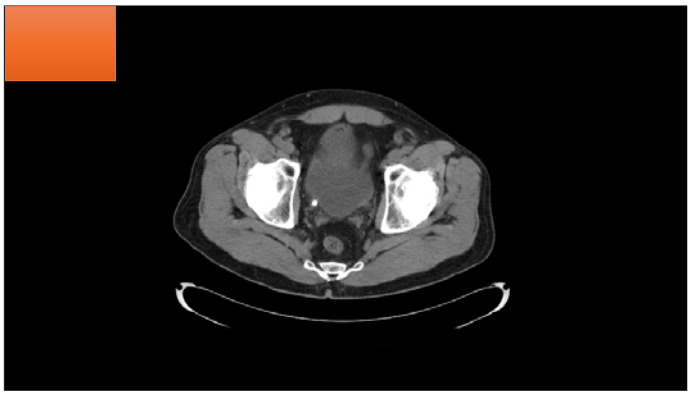









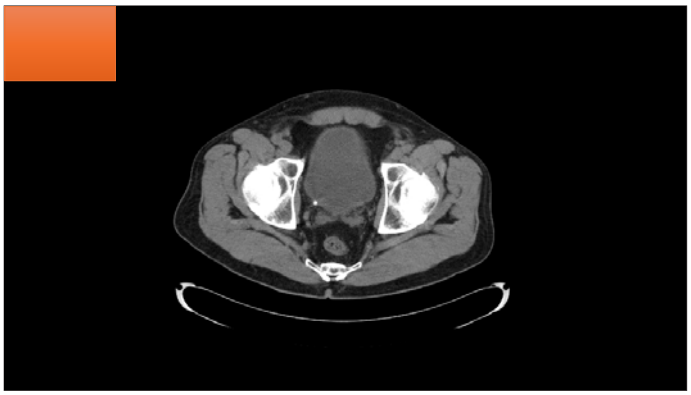






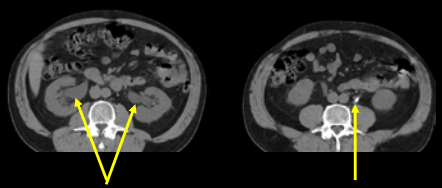






Complete urinary obstruction

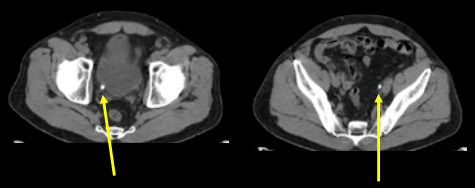
Complete urinary obstruction



Bilateral hydroureter

Proximal left utereter stone

Complete urinary obstruction

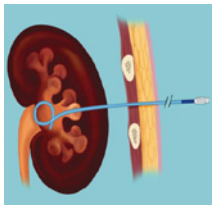


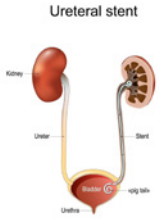
Distal right ureter stone

Distal left ureter stone

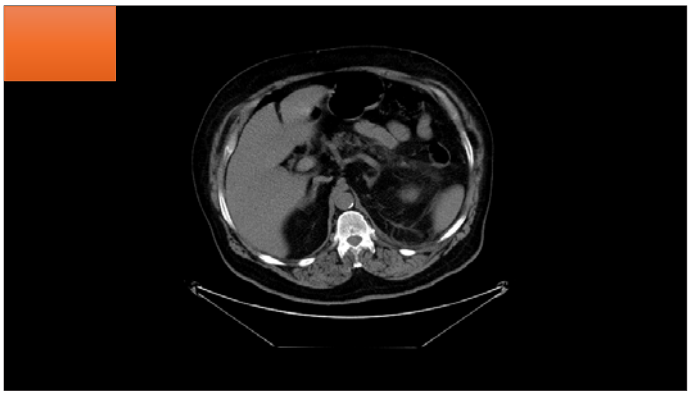
Need to carefully follow the ureters throughout their course

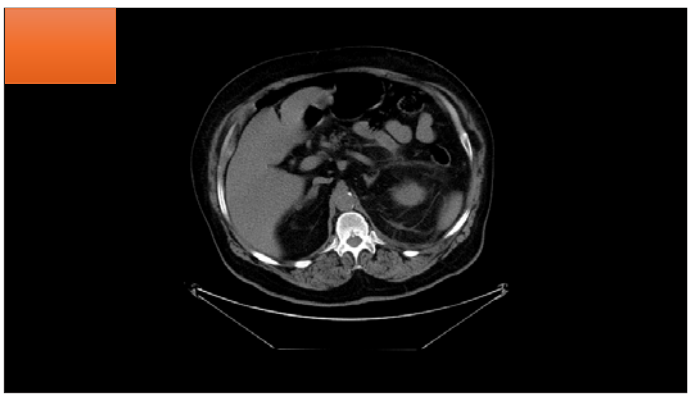
Bilateral obstruction needs emergent decompression

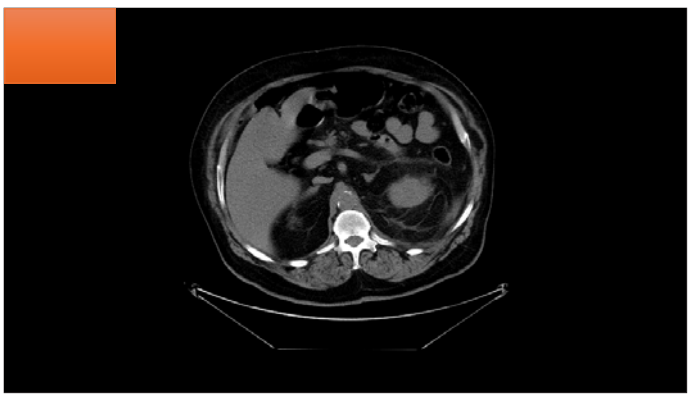


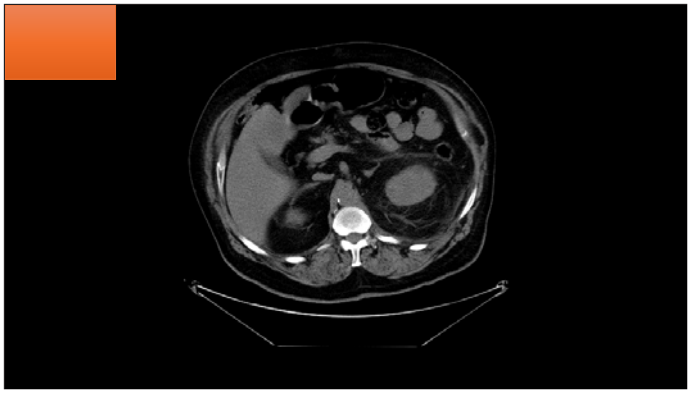
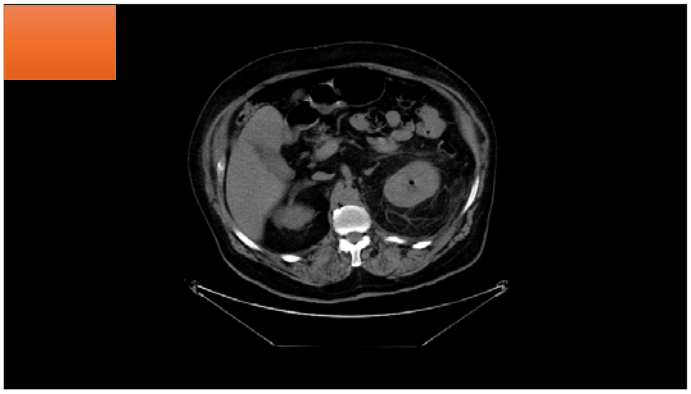


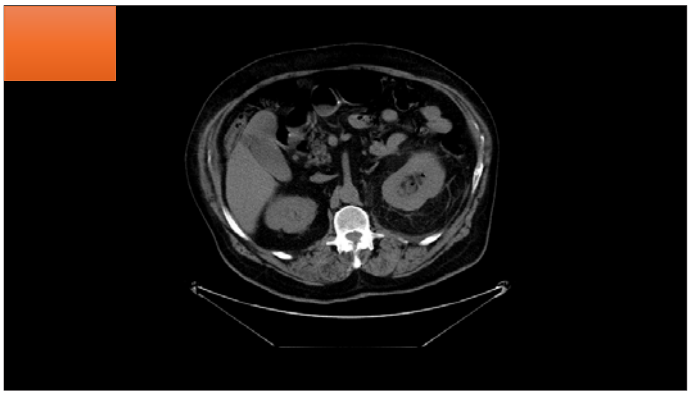
May be due to stones, but more commonly due to pelvic masses, prostatic hypertrophy, or radiation scarring

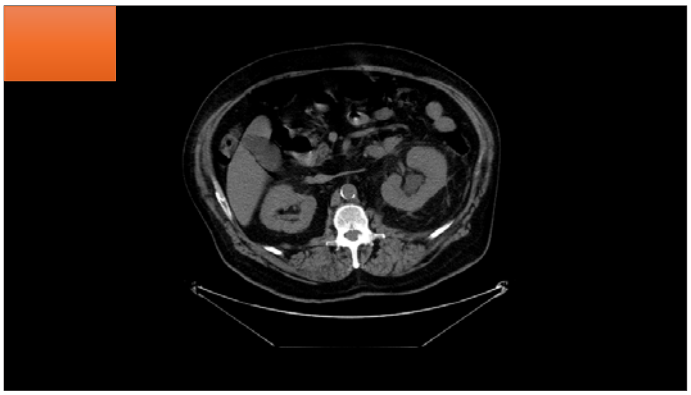
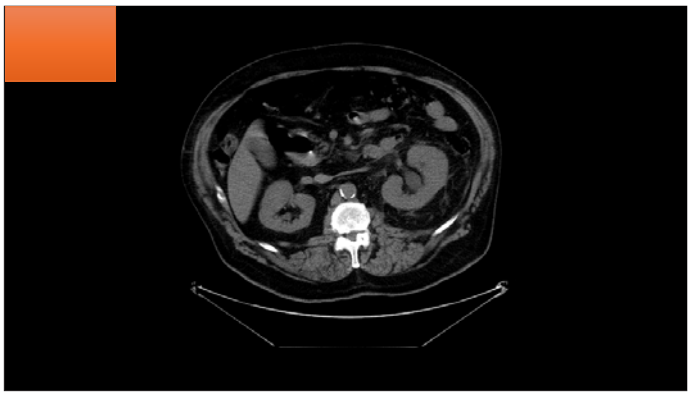
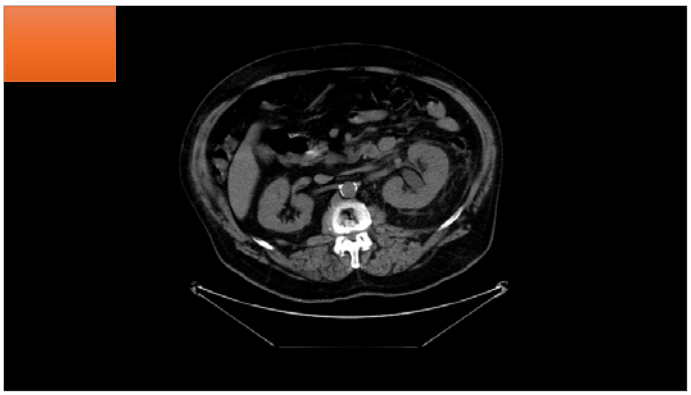


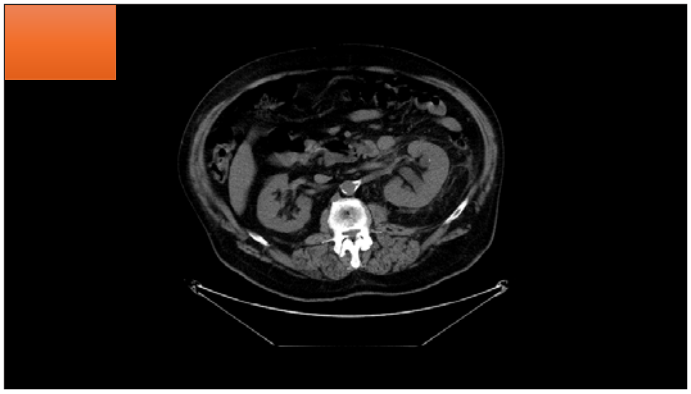


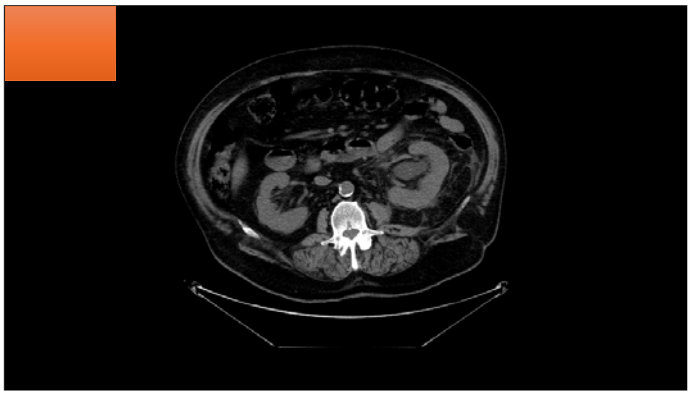
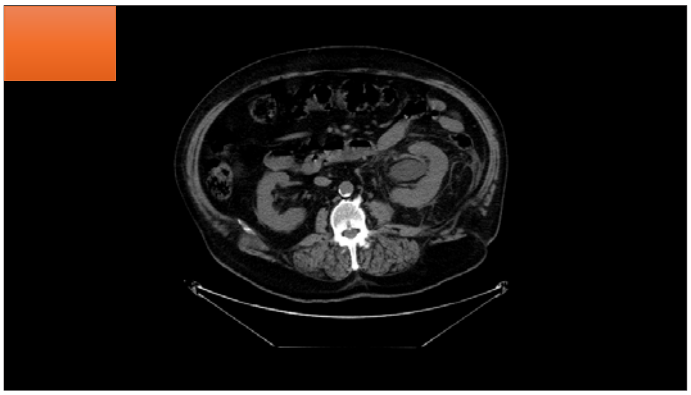
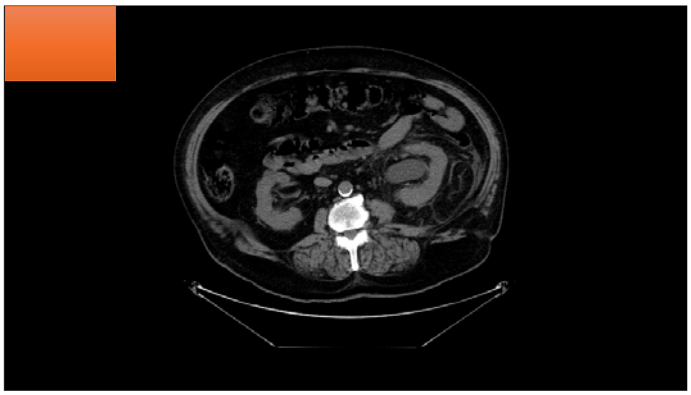
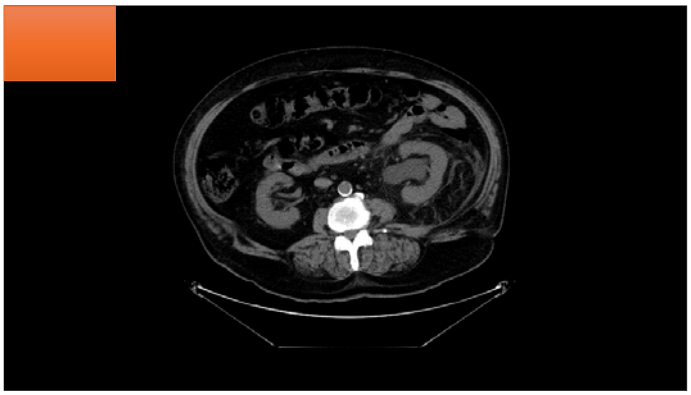
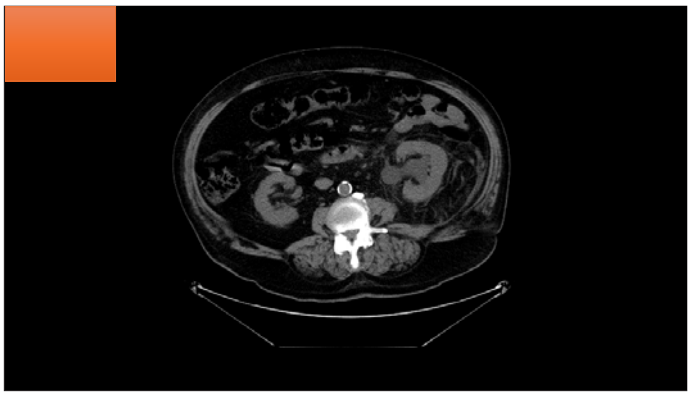
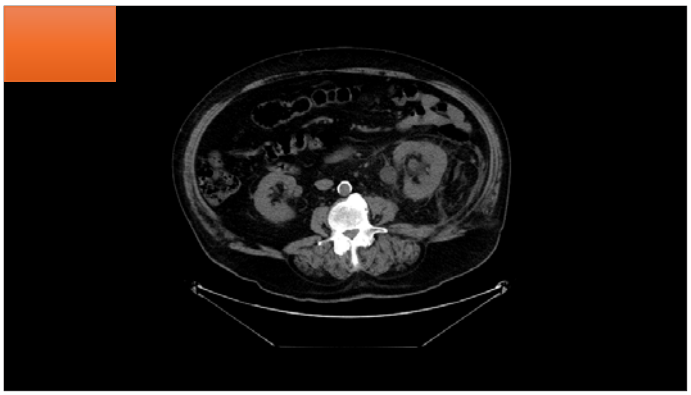


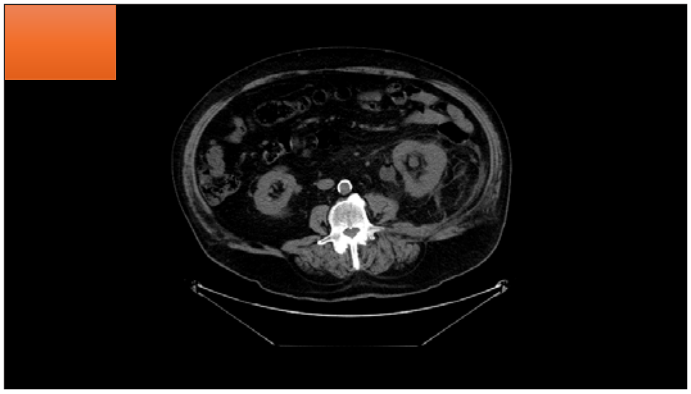
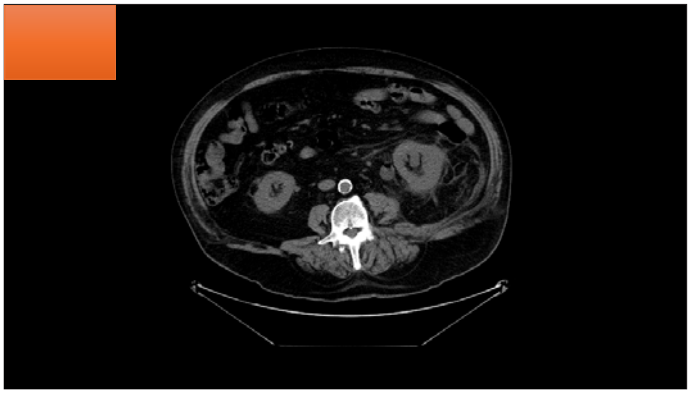
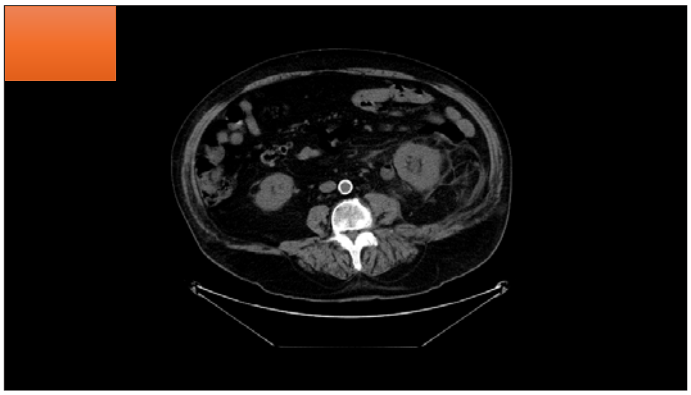
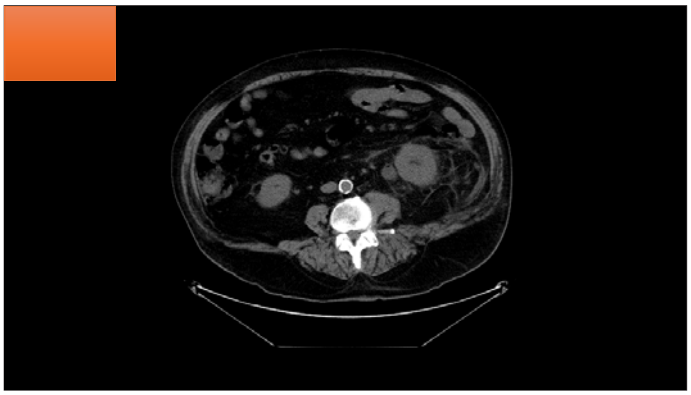
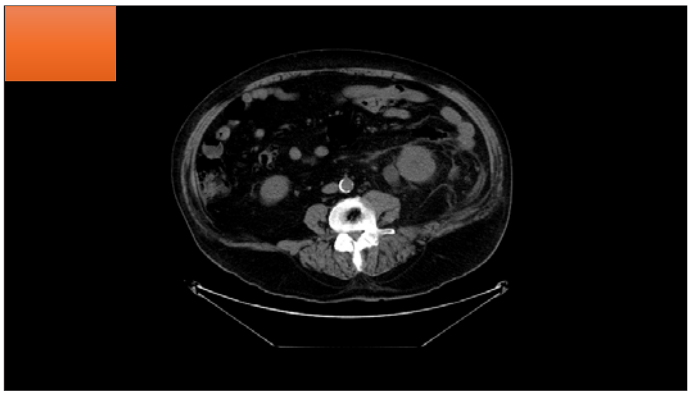
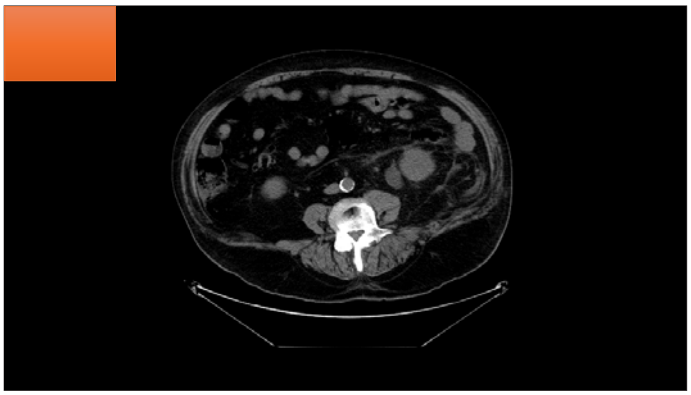
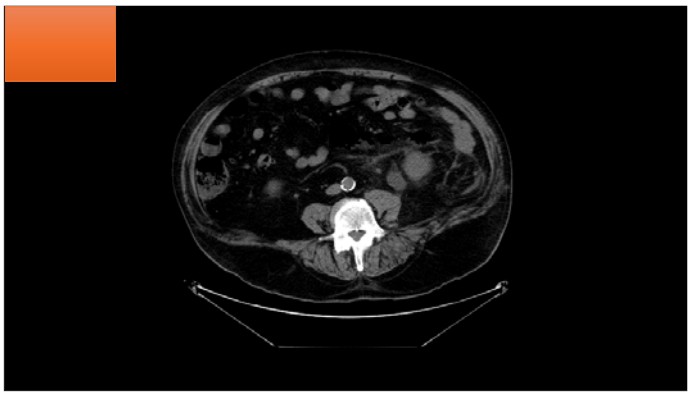
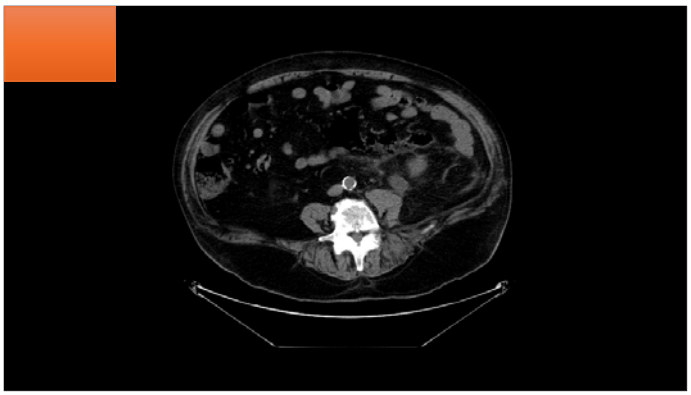



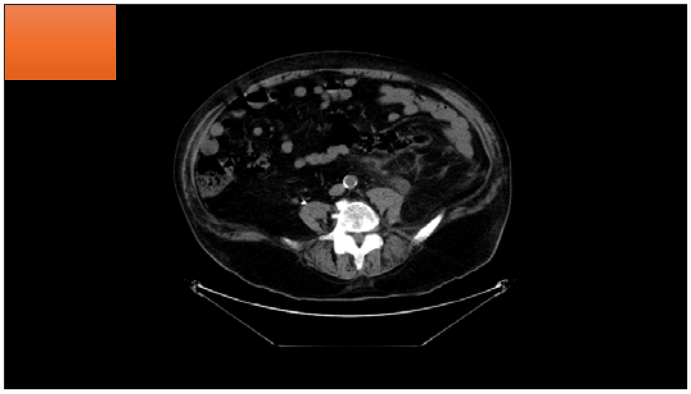
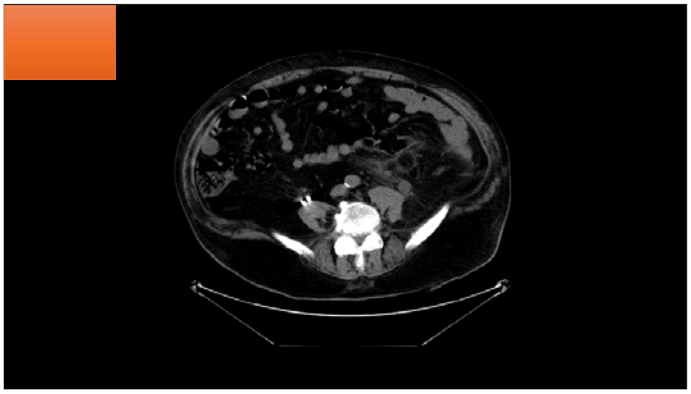
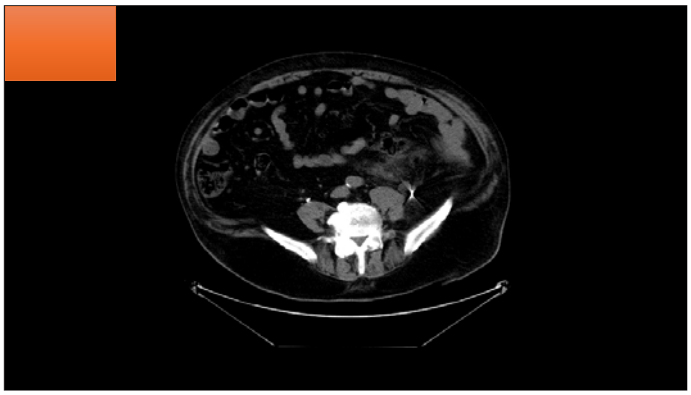
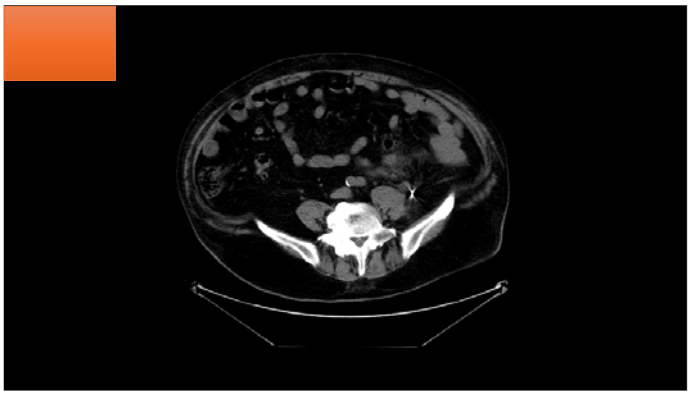
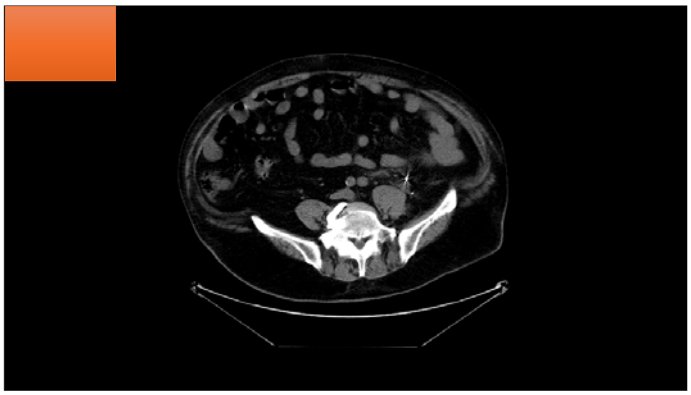
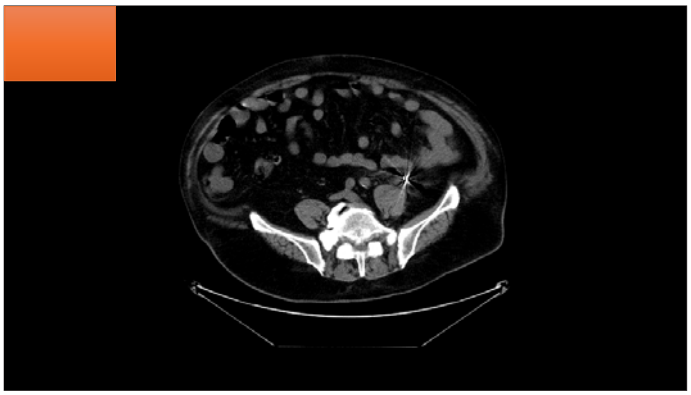
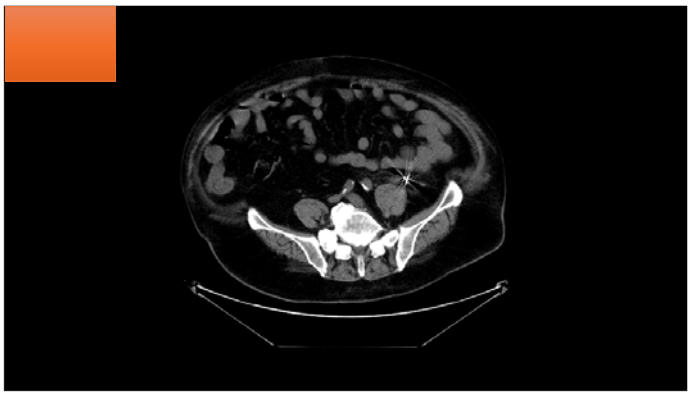
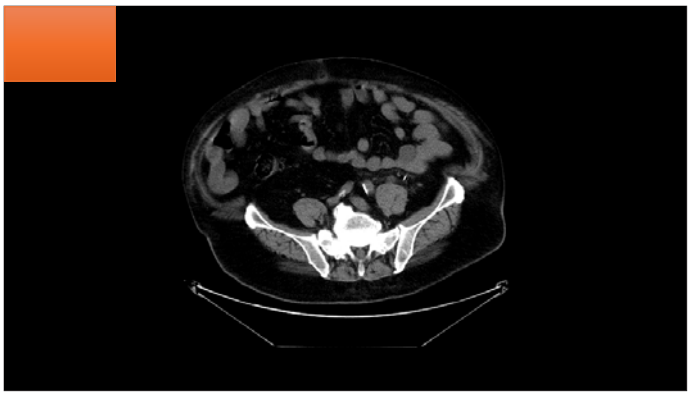


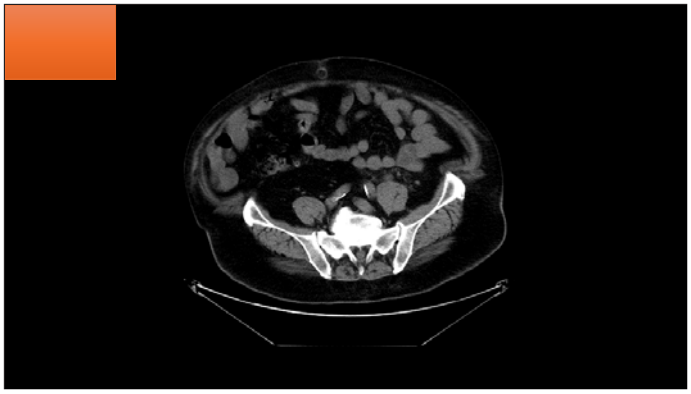
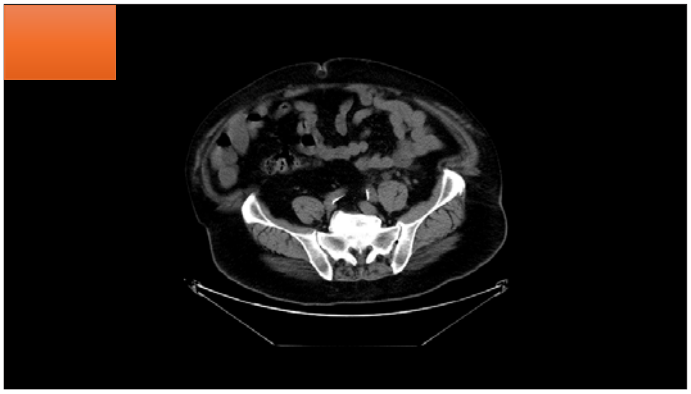
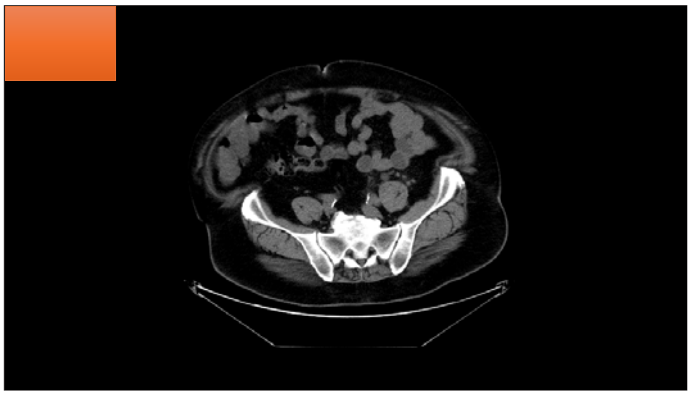
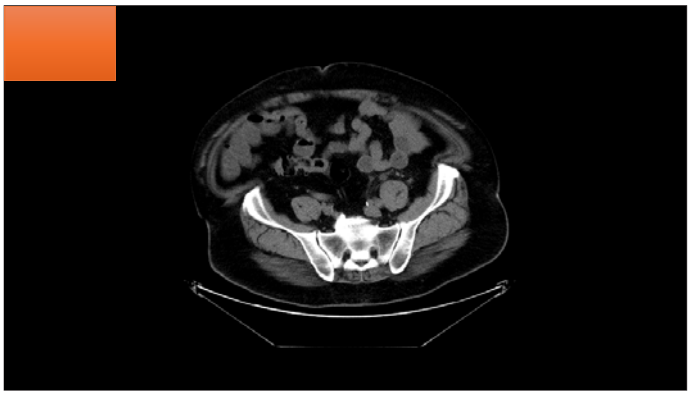
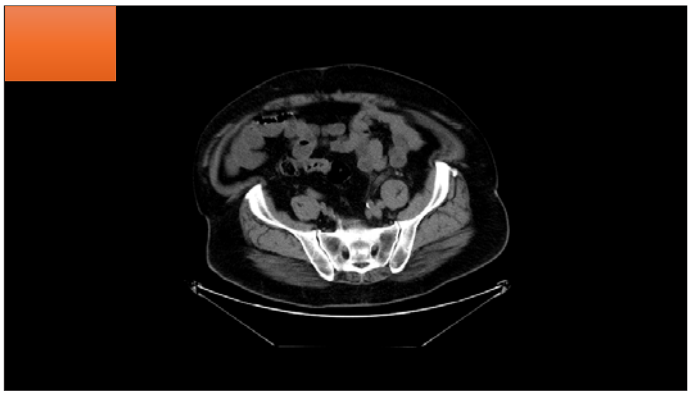
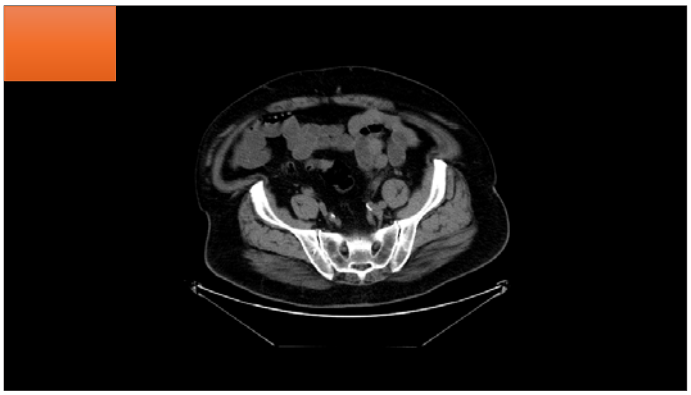
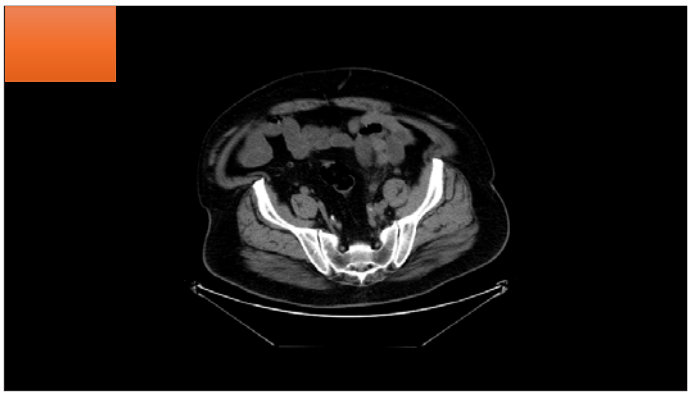
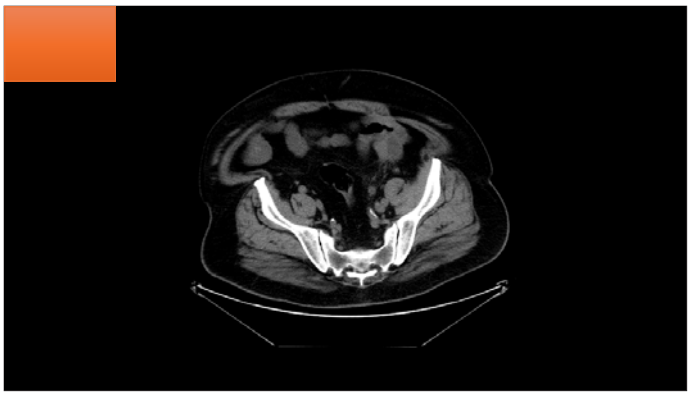




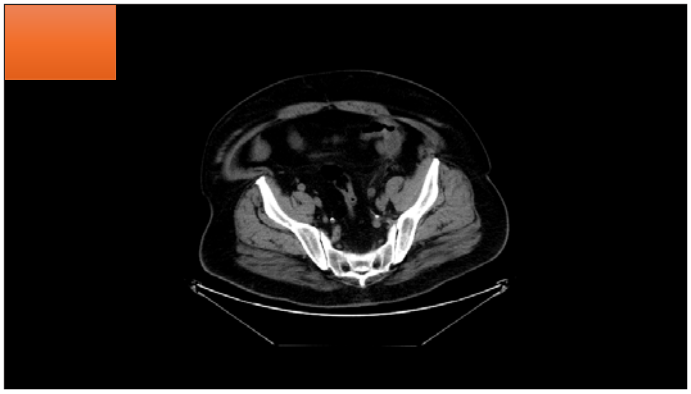
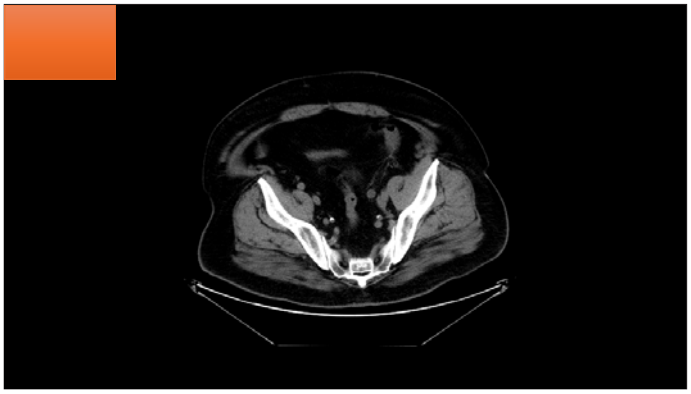
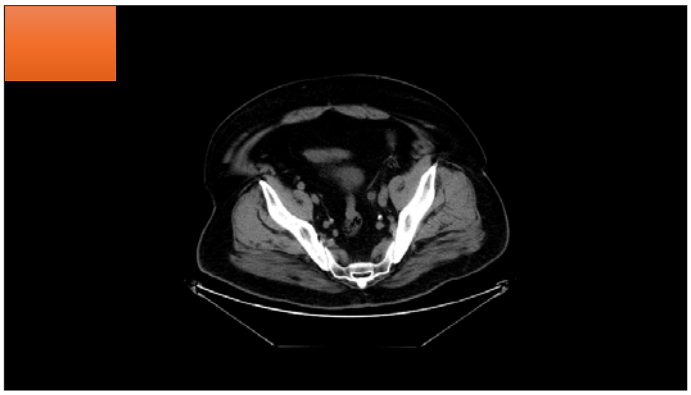
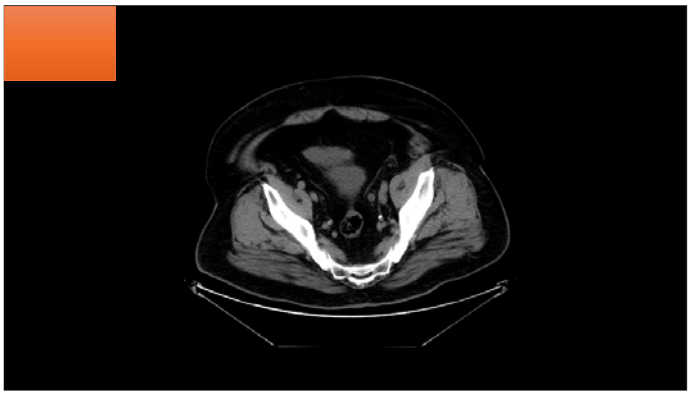
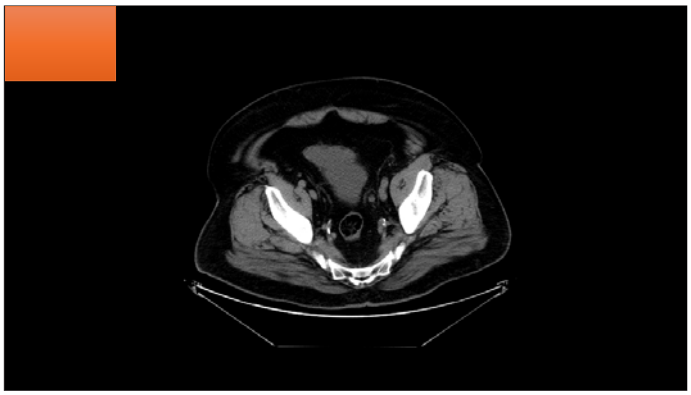
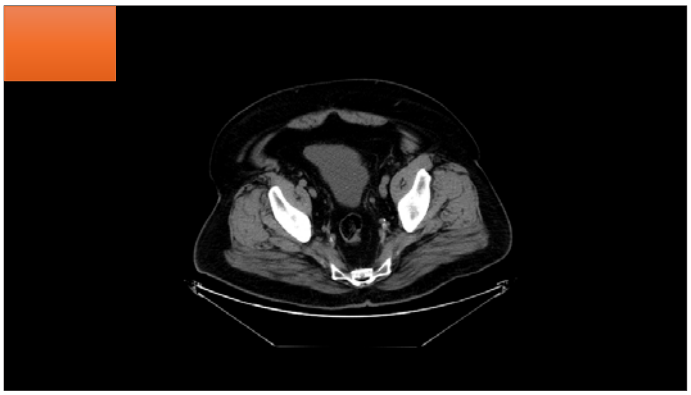
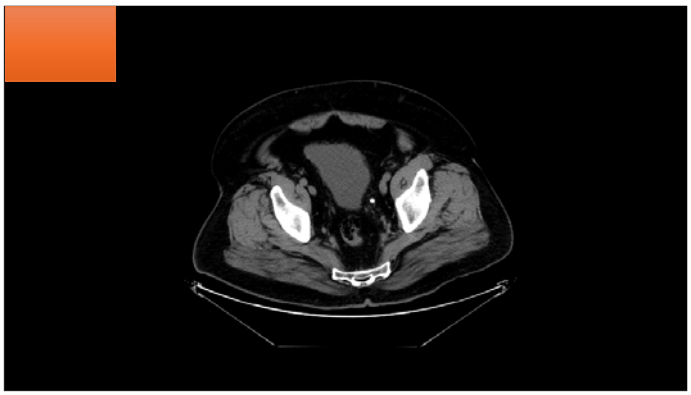
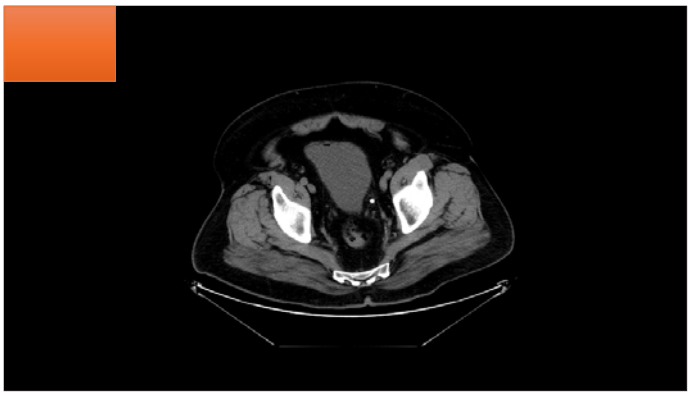


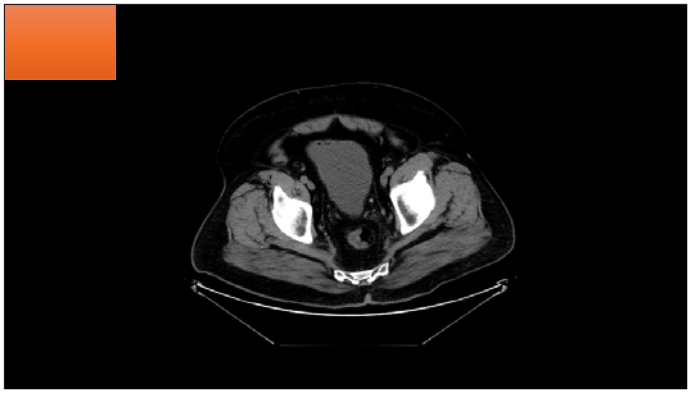







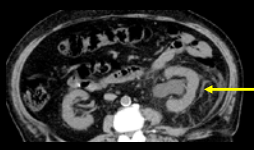











Looks like another kidney stone?

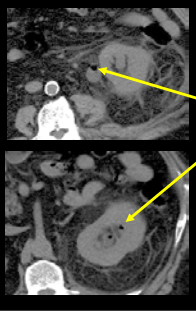


Hydroureteronephrosis with perinephric stranding

Distal ureteral stone

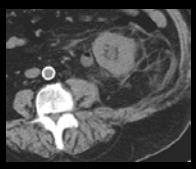
Let's take a closer look

Emphysematous *pyelitis*



Air within the ureter and the collecting system

Differentiate between emphysematous *pyelitis* and *pyelonephritis*



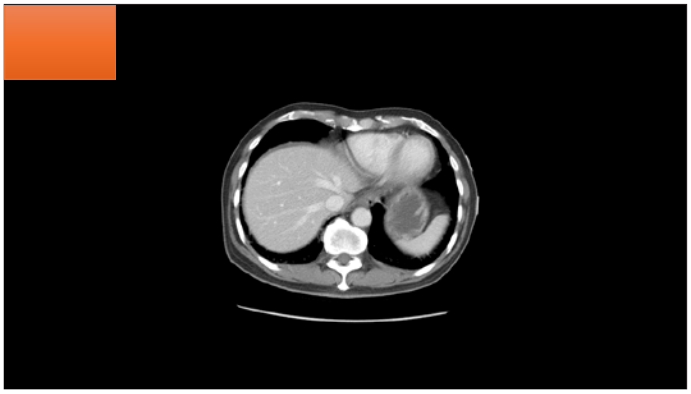
Air confined to the collecting system

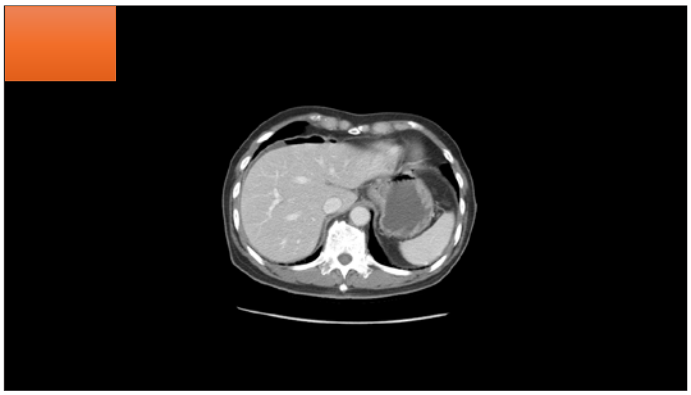
Air in the renal parenchyma

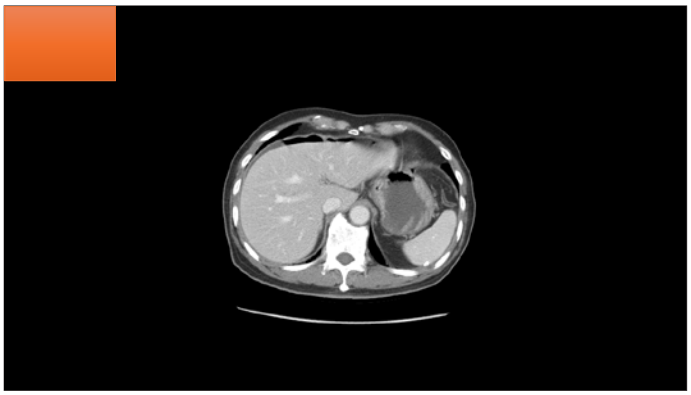
The clock is ticking…
Consult urology early!!

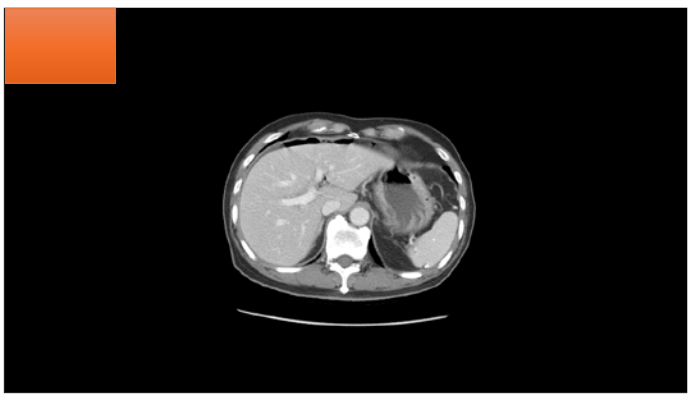
Don't forget to use lung windows to look for air

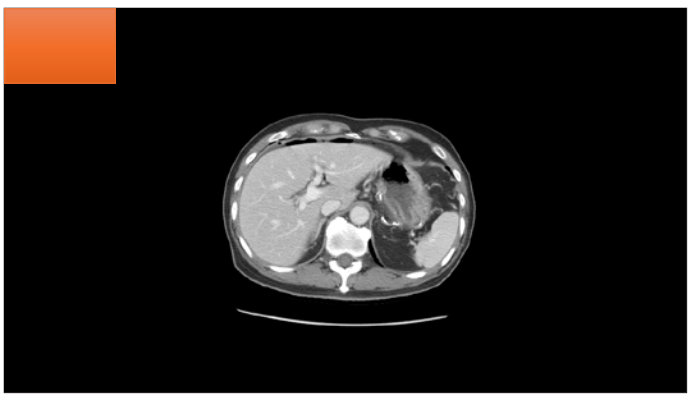
34 y.o. male with sudden onset abdominal pain

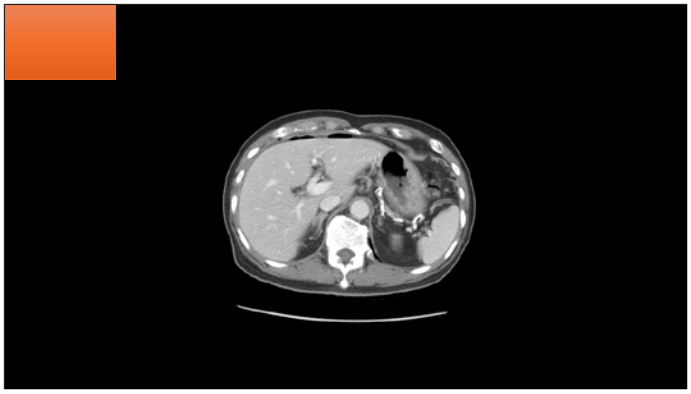
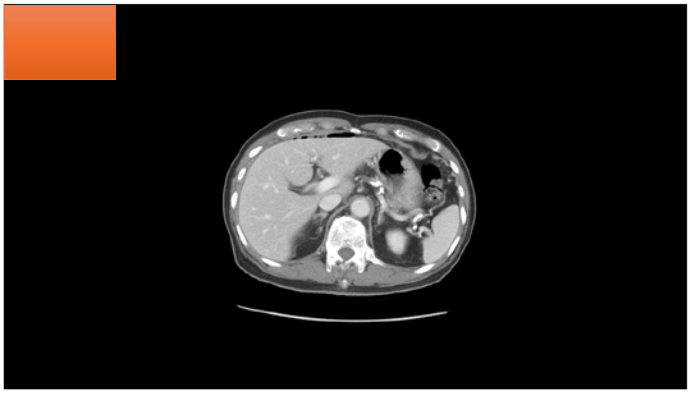
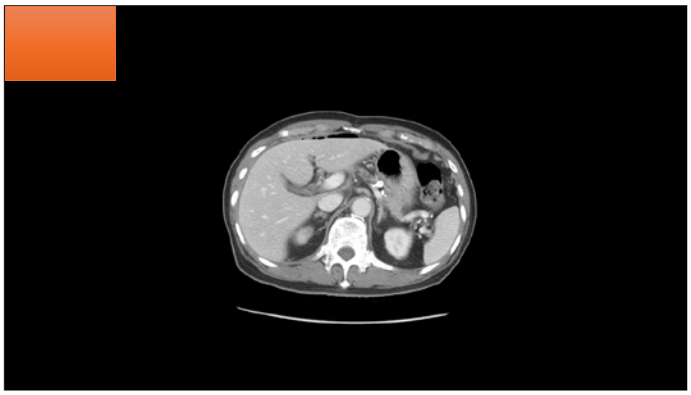
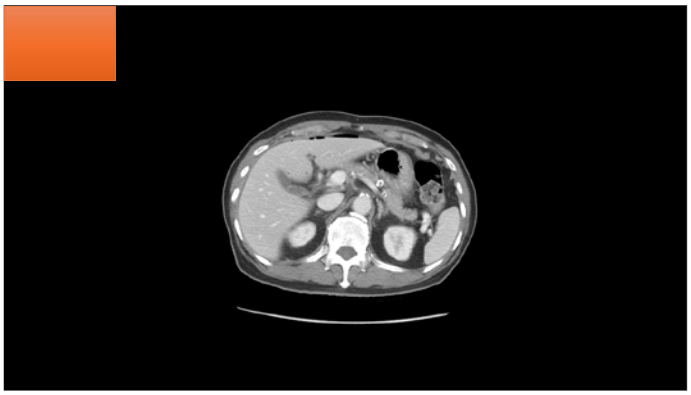
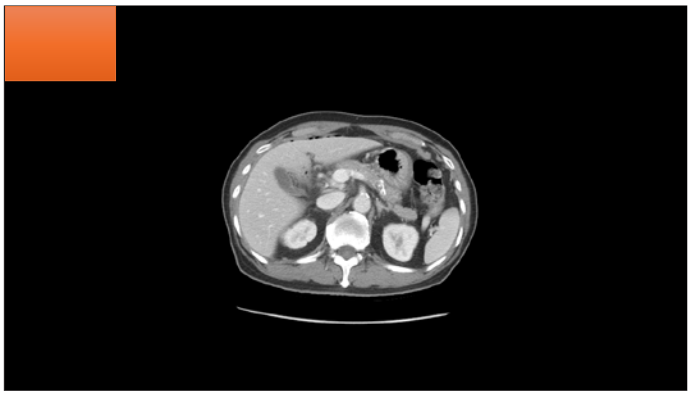
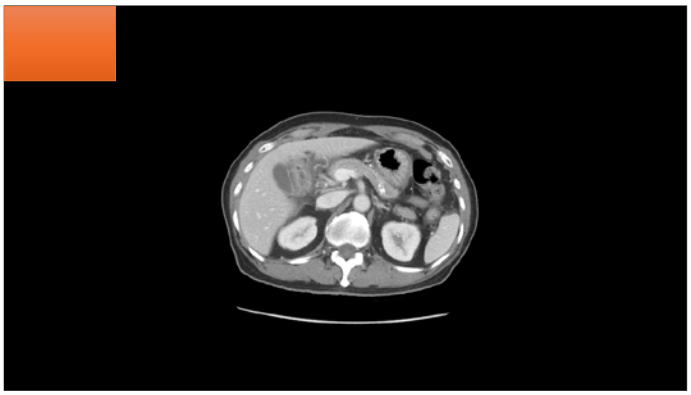
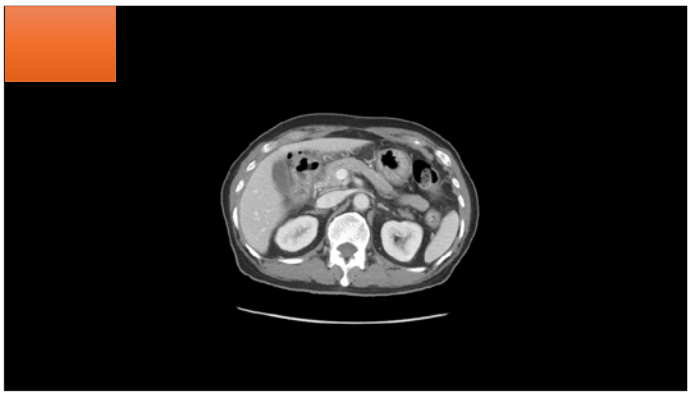
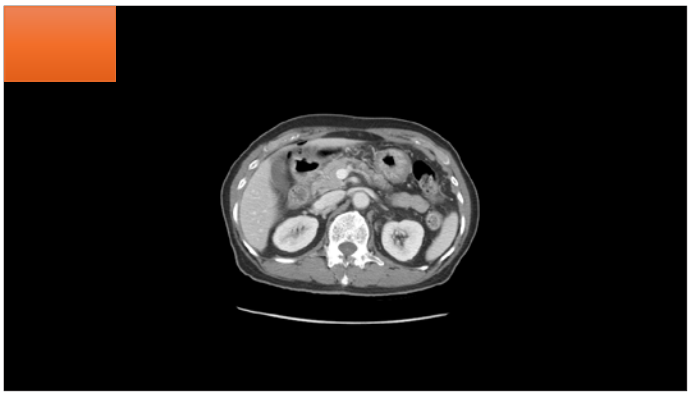


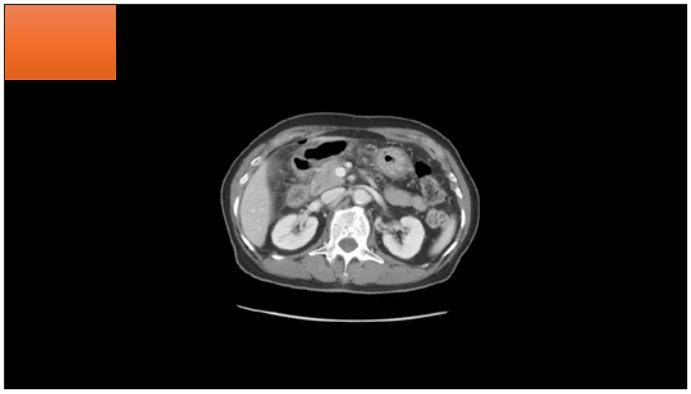


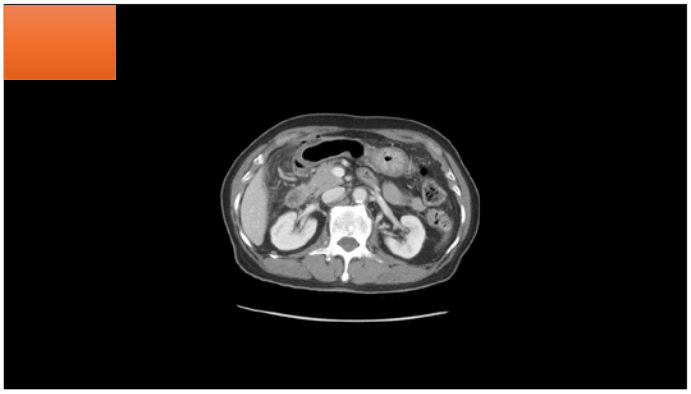


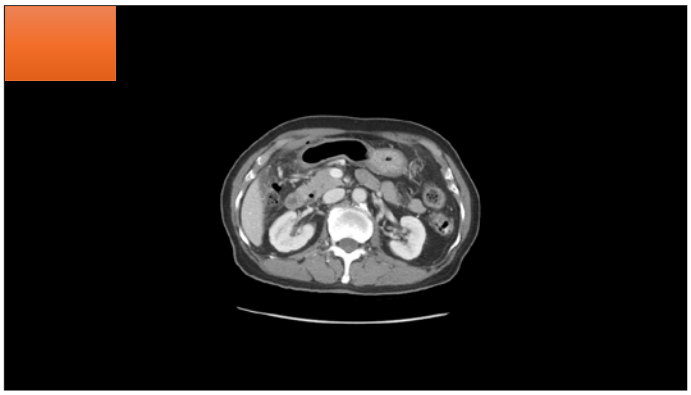


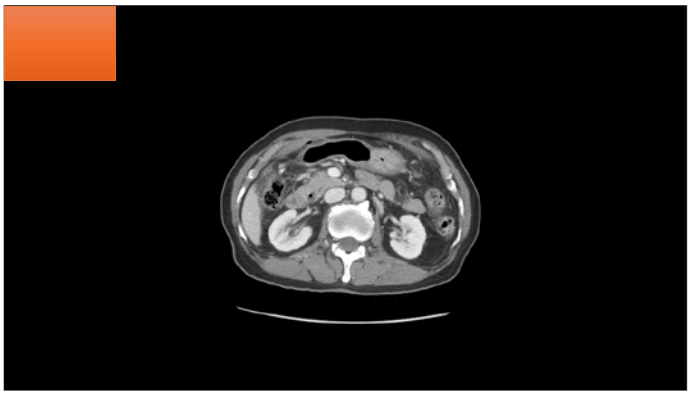


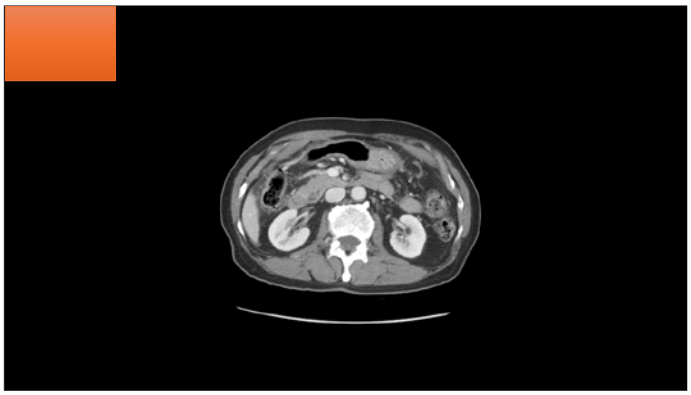









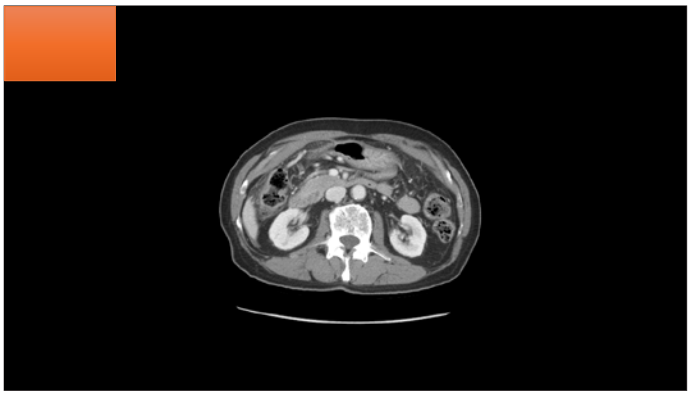


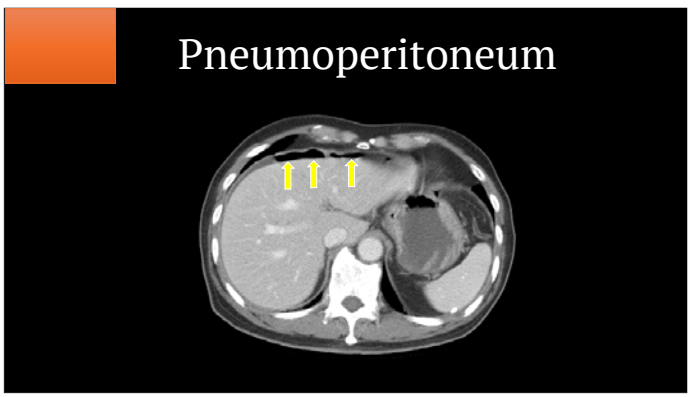


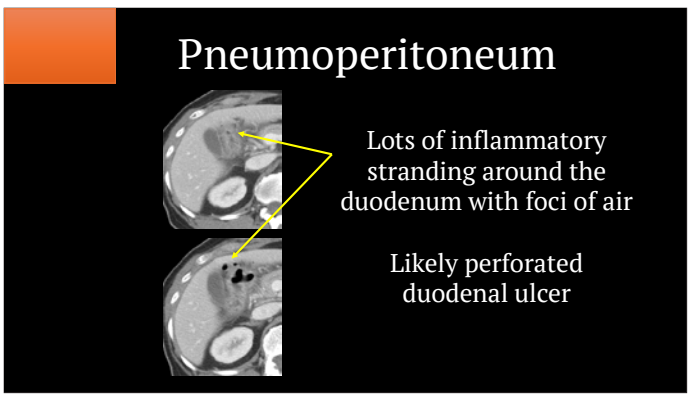




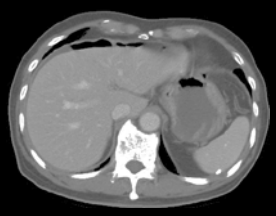





Pneumoperitoneum

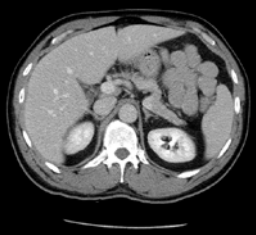

Pneumoperitoneum
Lots of inflammatory stranding around the duodenum with foci of air
Likely perforated duodenal ulcer

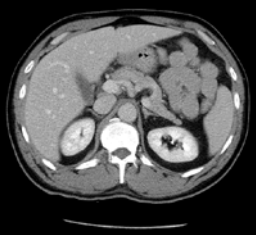
Don't forget the Lung Windows

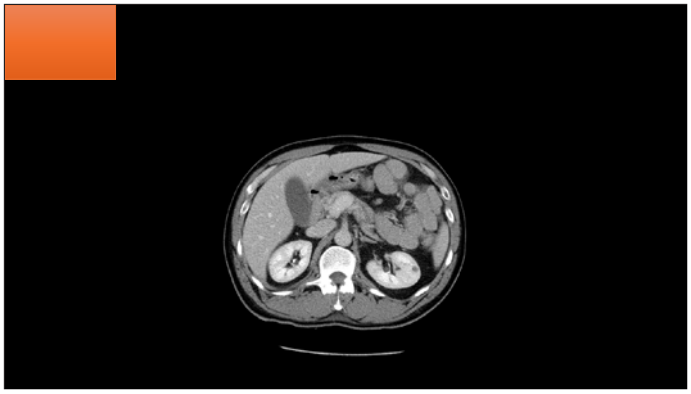
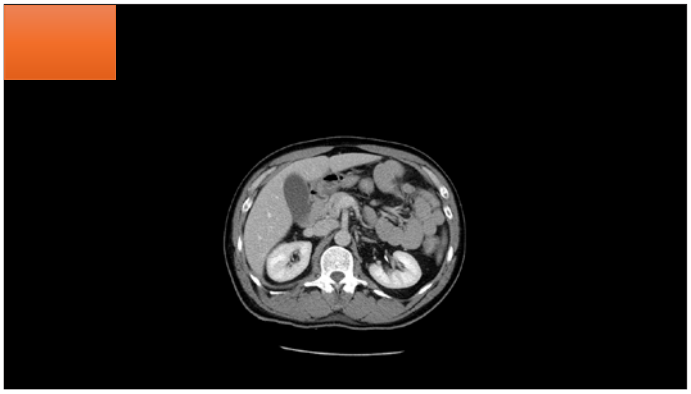
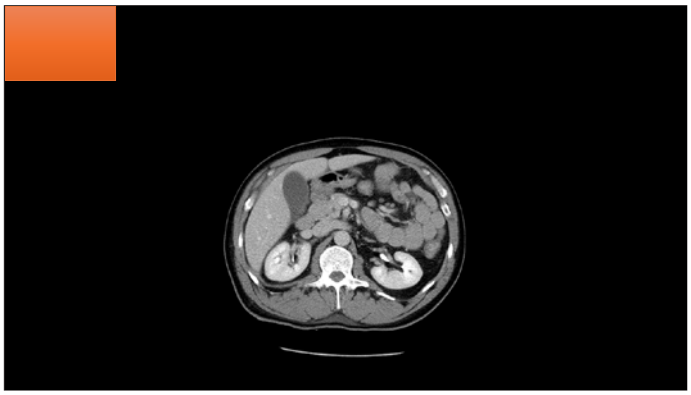
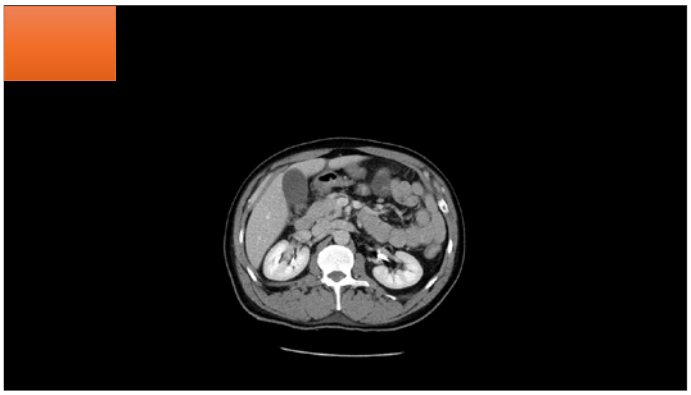
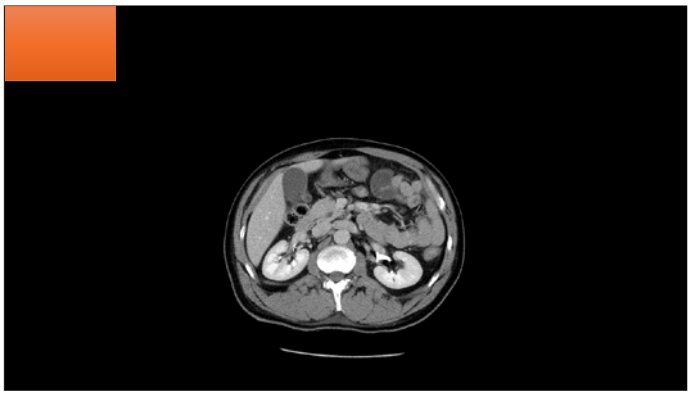
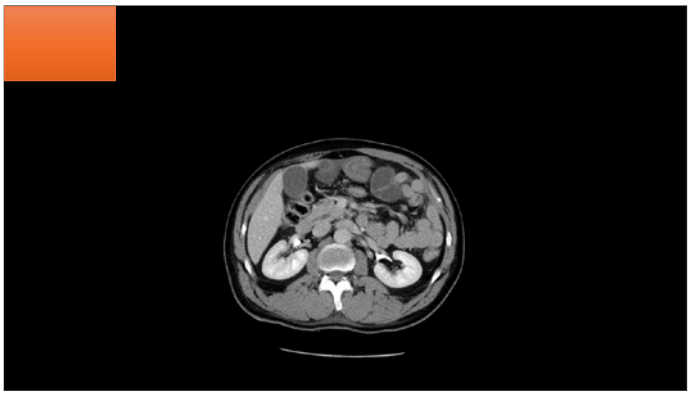
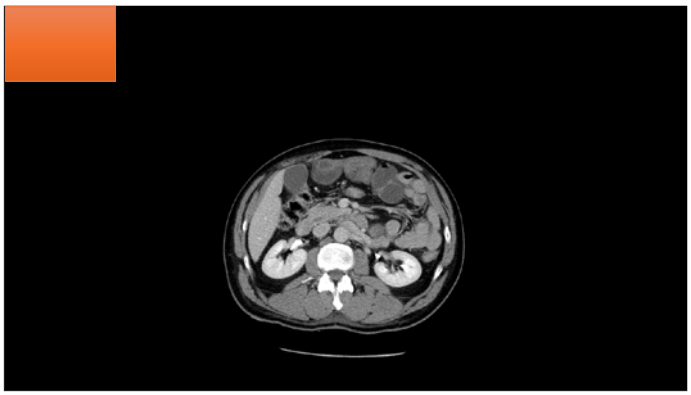
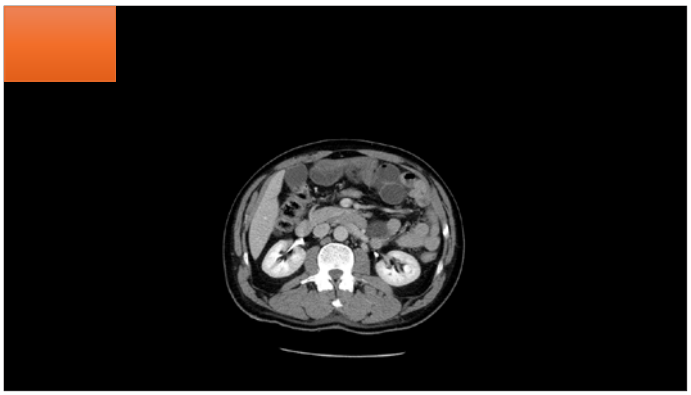


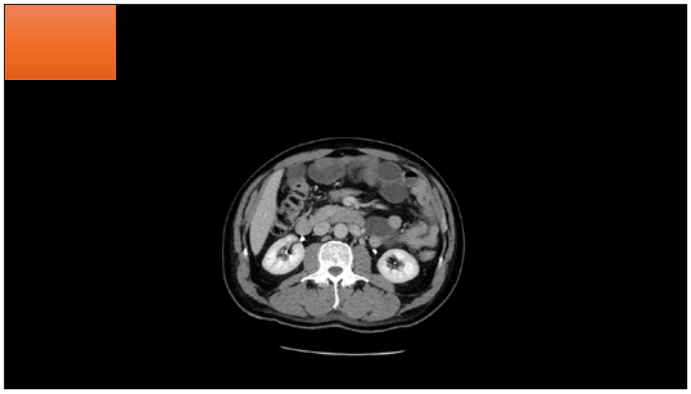
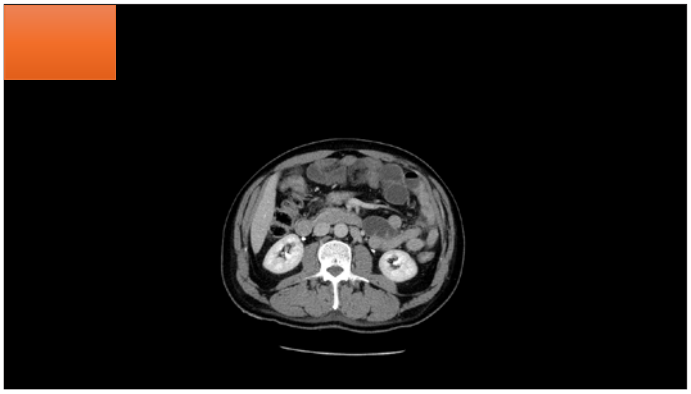
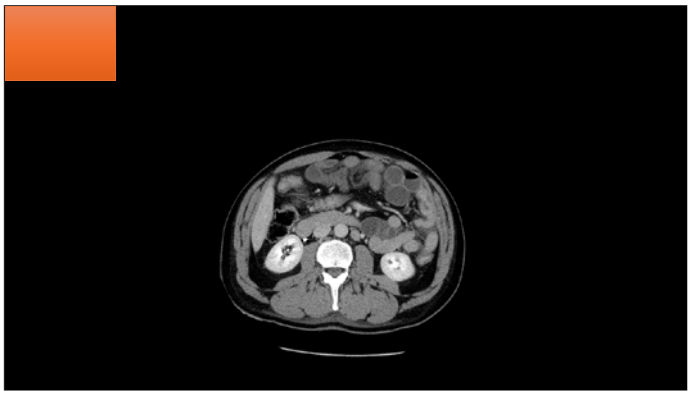
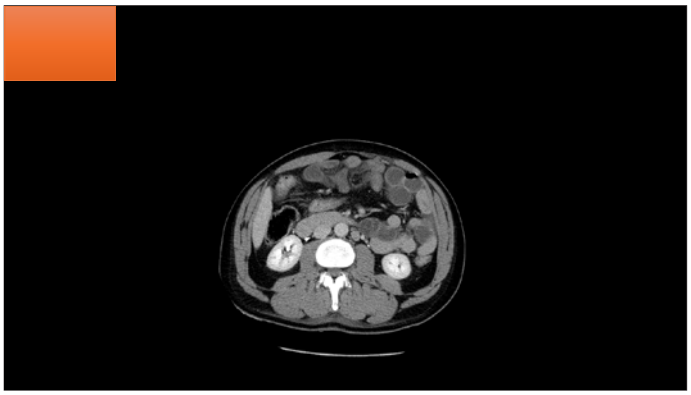
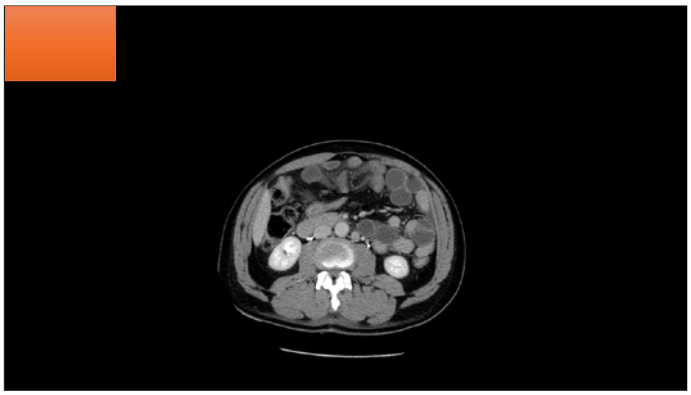
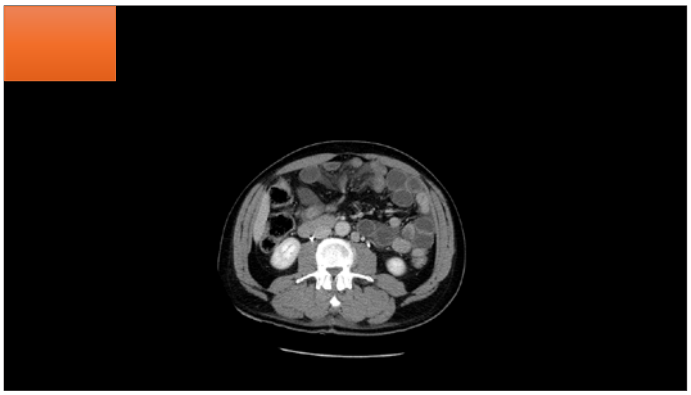
CT may detect as little as 5 mL of air!!

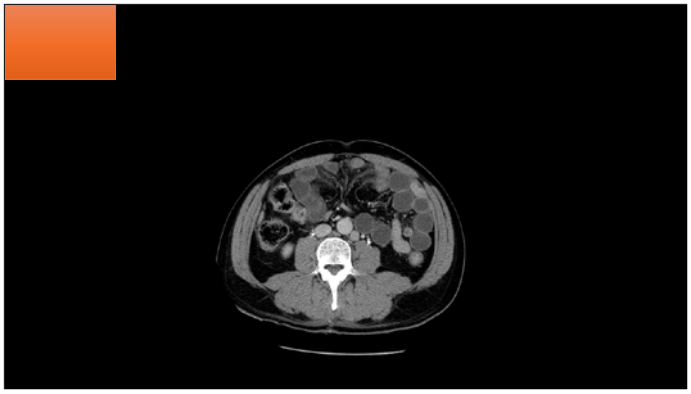
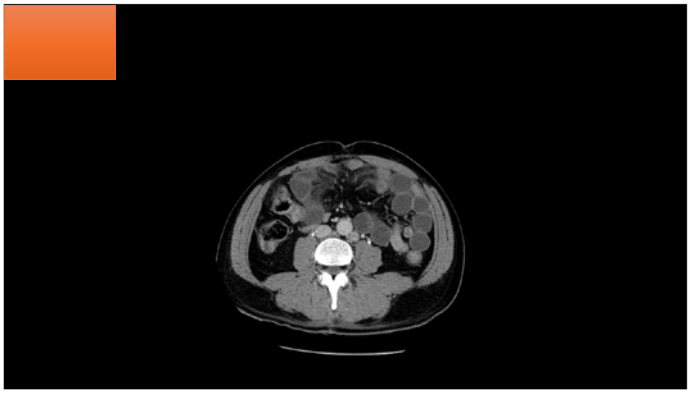
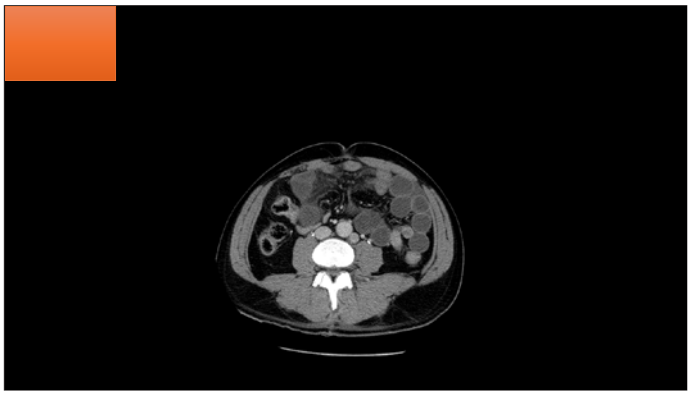
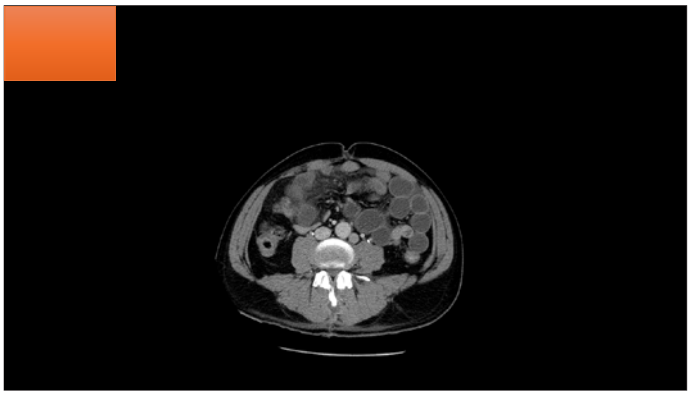
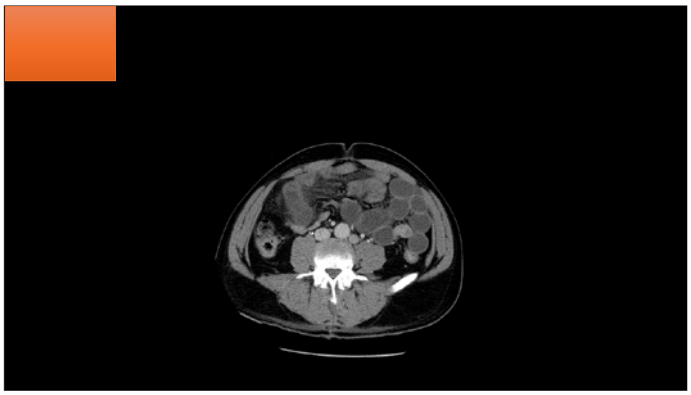
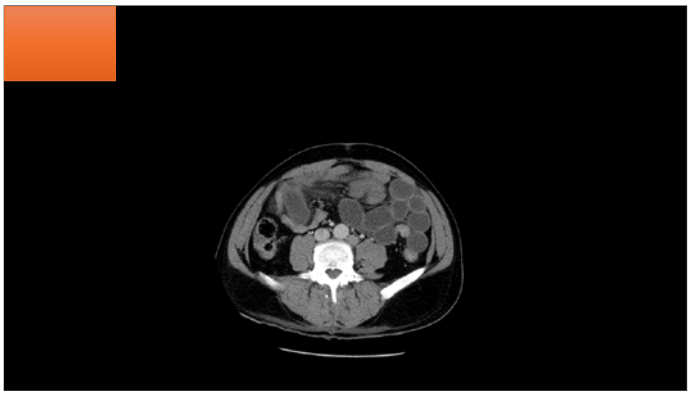
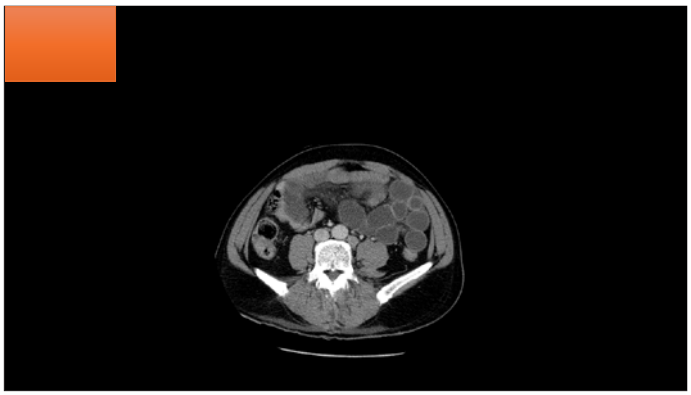
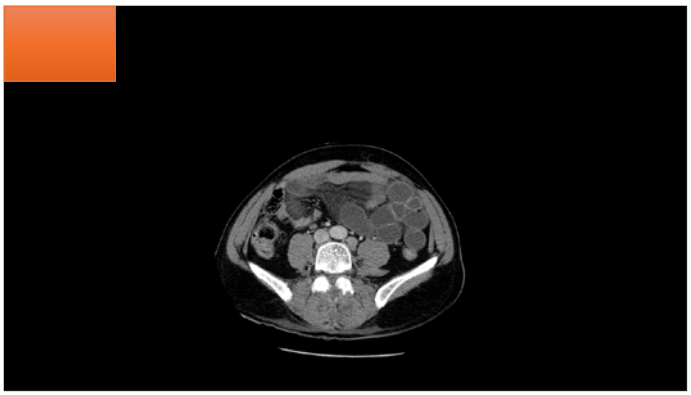
45 y.o. male with severe abdominal pain and vomiting

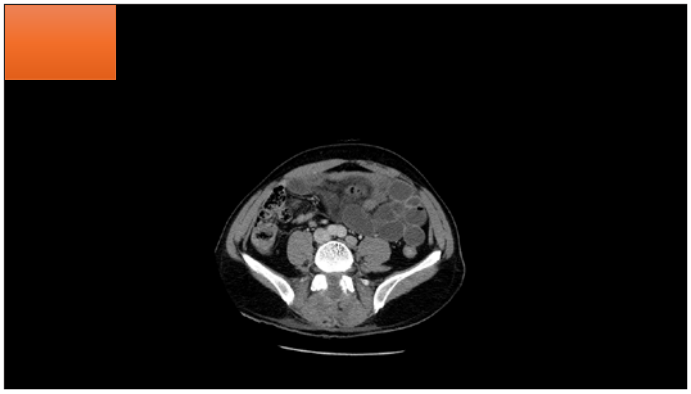
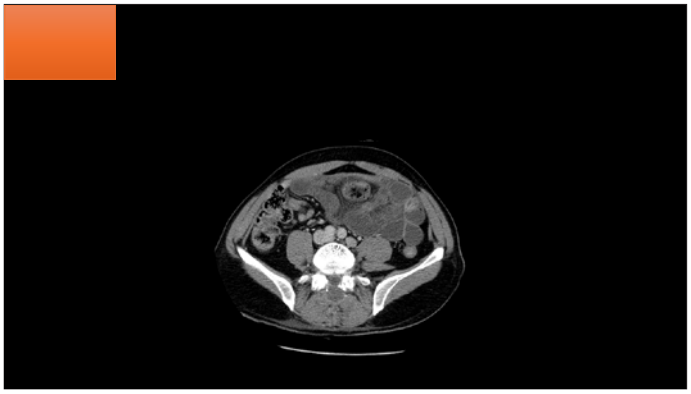
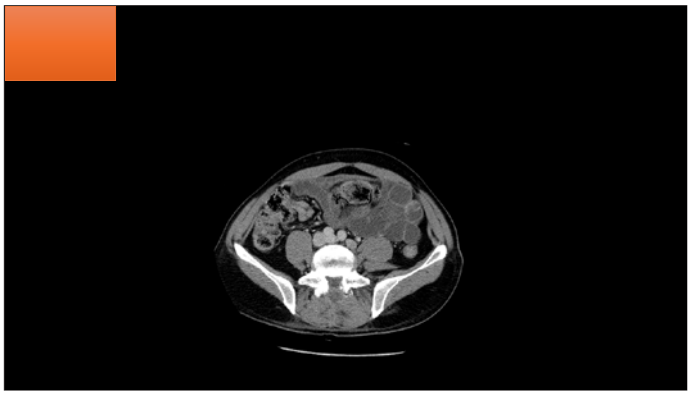
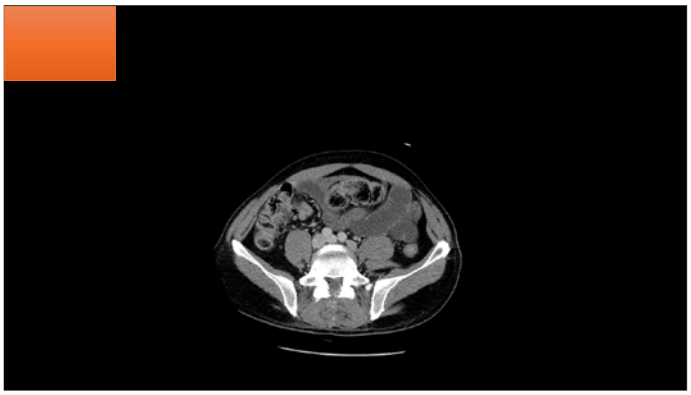
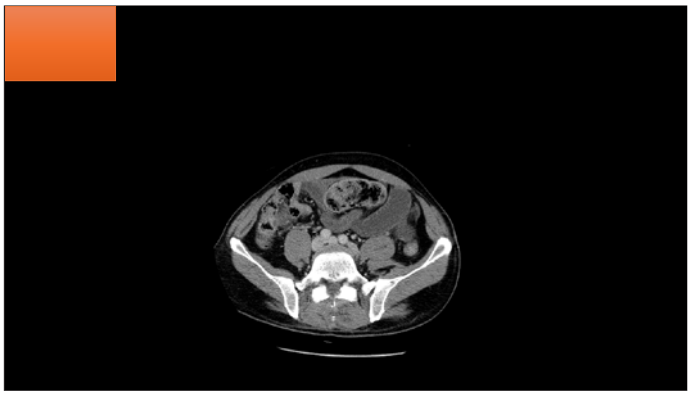
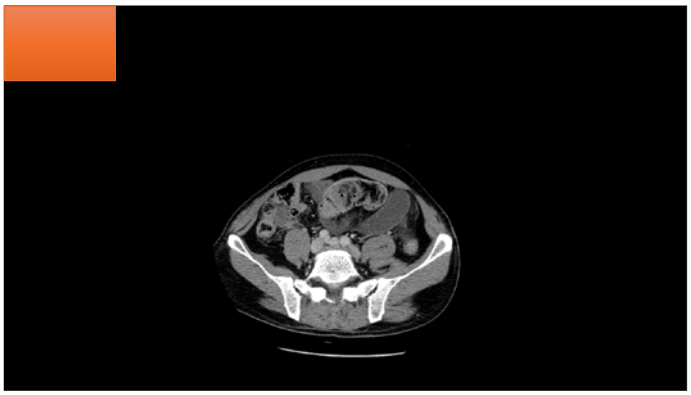
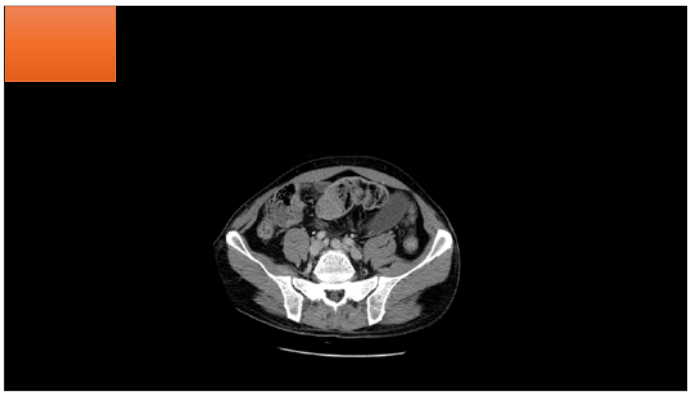
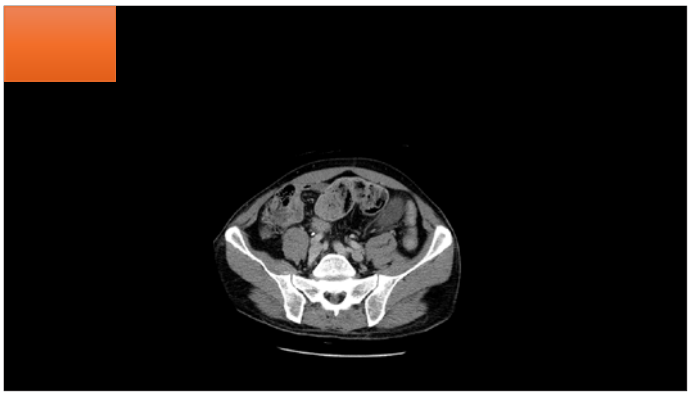




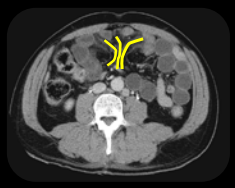







Something's not quite right here…

Closed Loop Obstruction



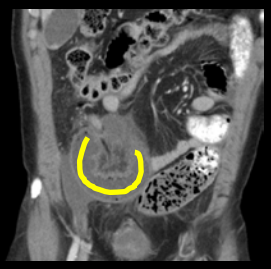
Often due to internal hernias or adhesions

Look for mesenteric vessels converging together

Closed Loop Obstruction

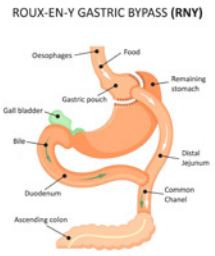
Look for isolated section of dilated small bowel with decompression *proximally* and *distally*

C-shaped loop on the coronal view is very suggestive

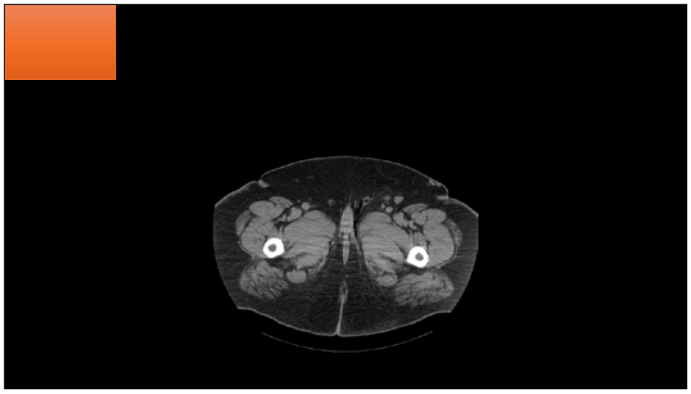
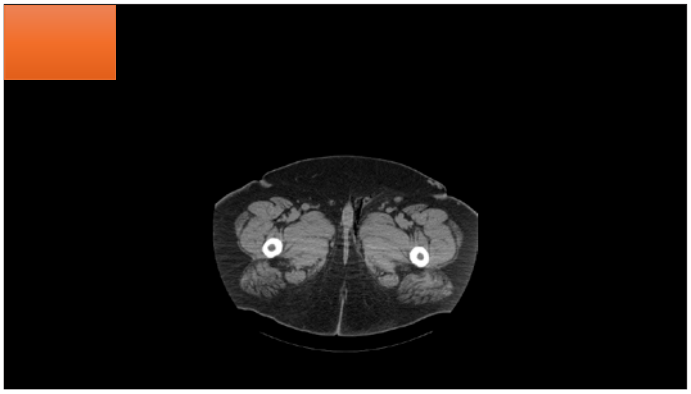
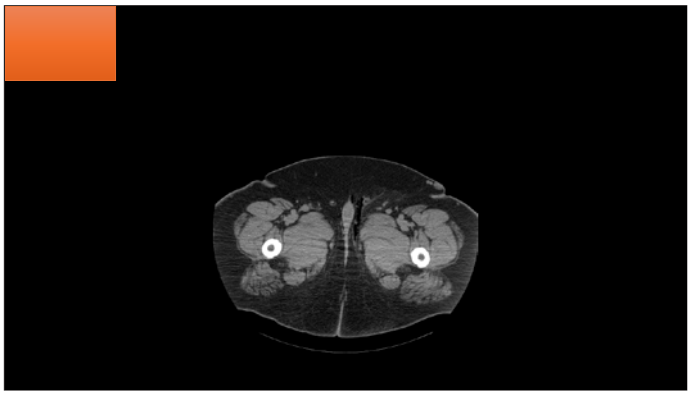
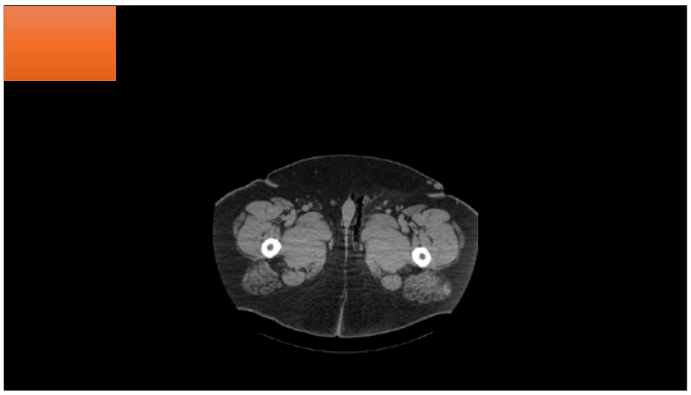
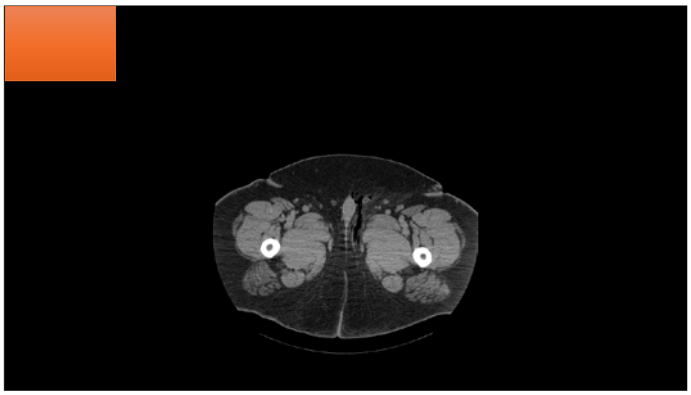
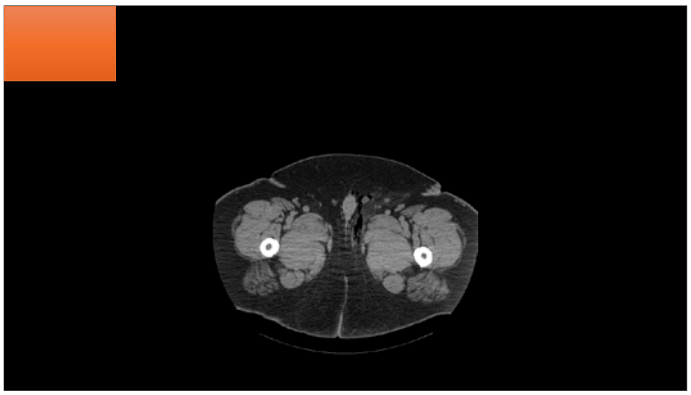
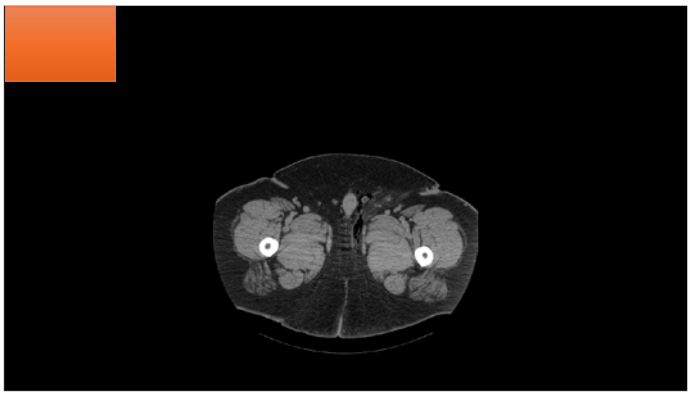
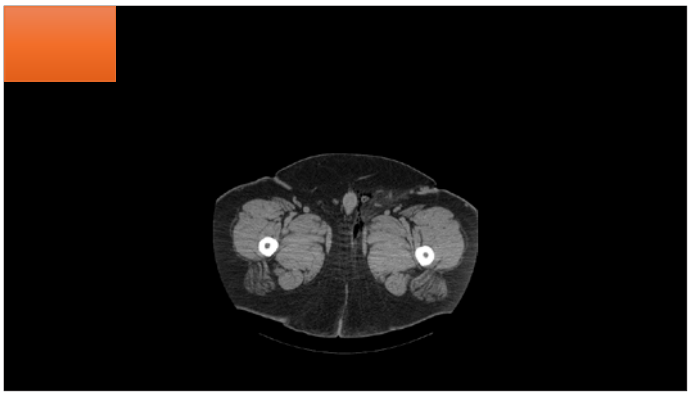


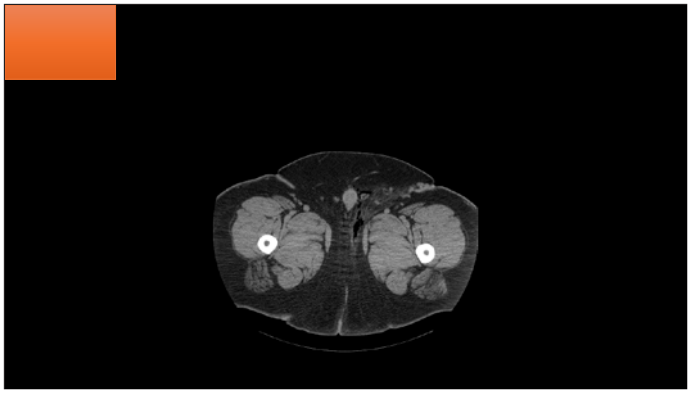
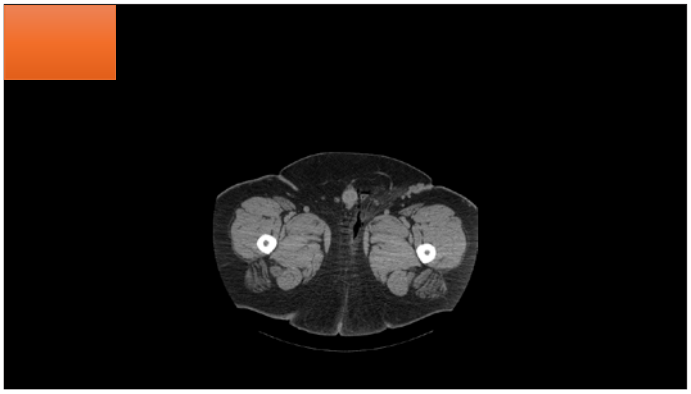
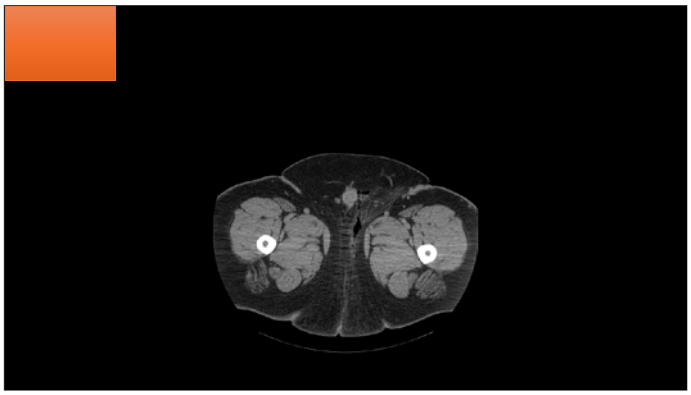
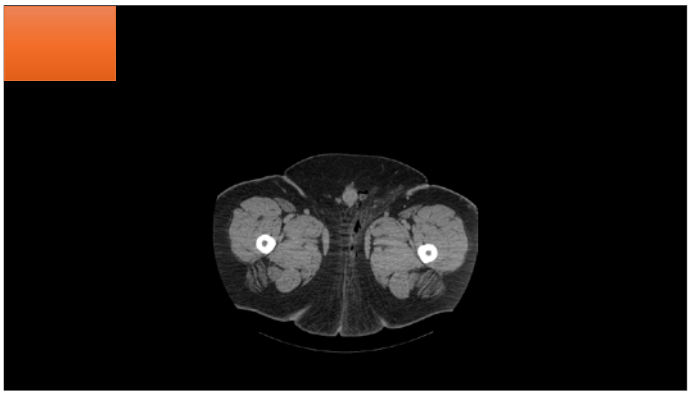
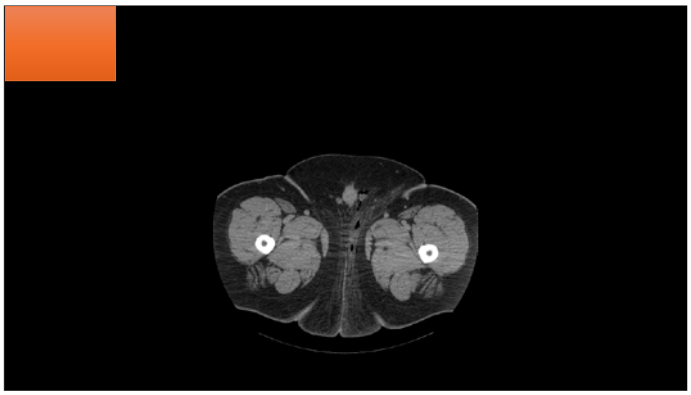
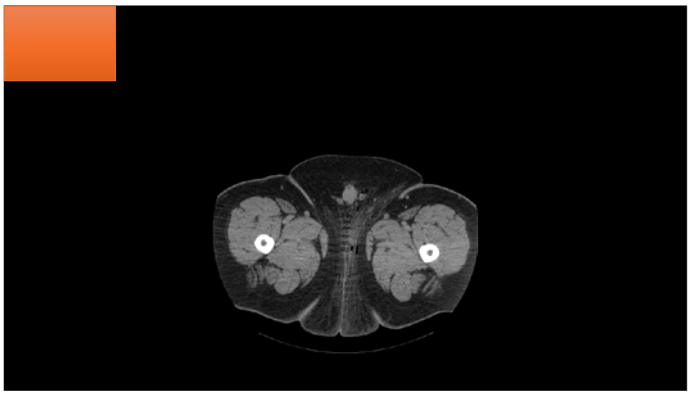
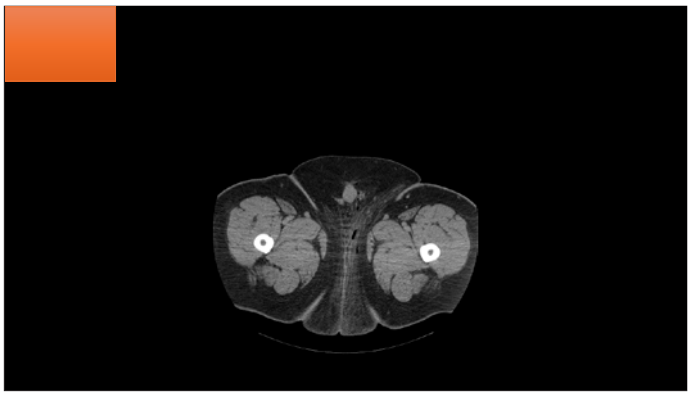
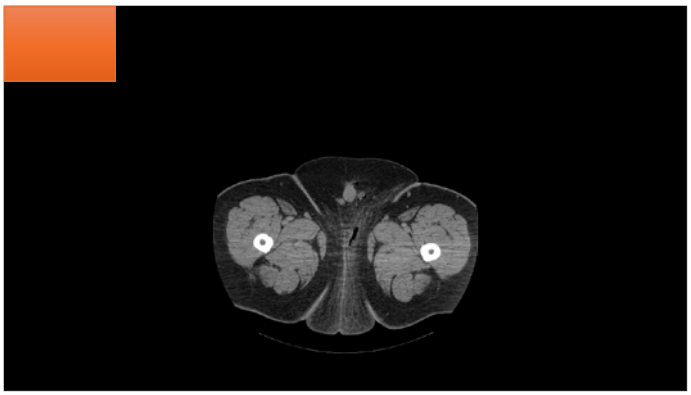
These are a true surgical emergencies!!

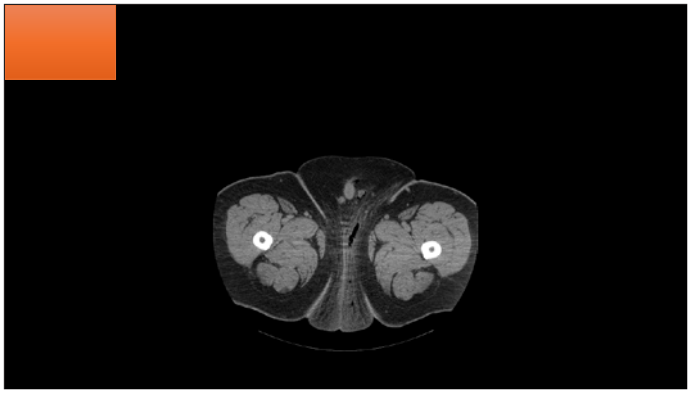
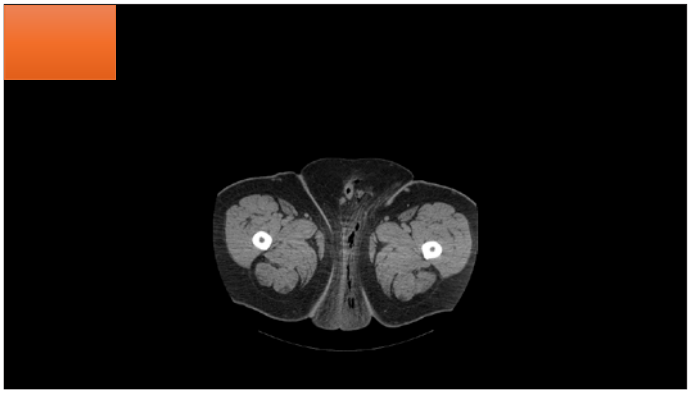
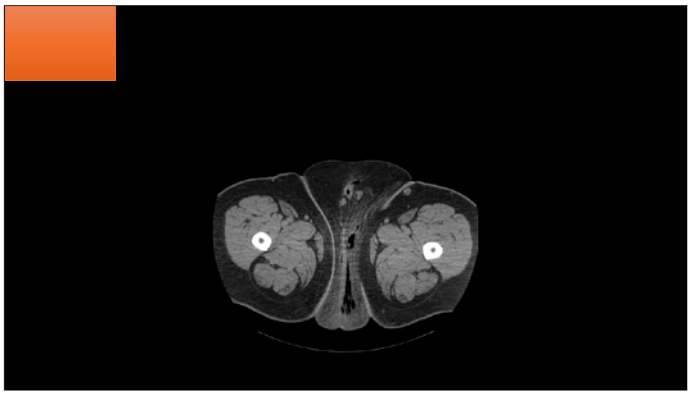
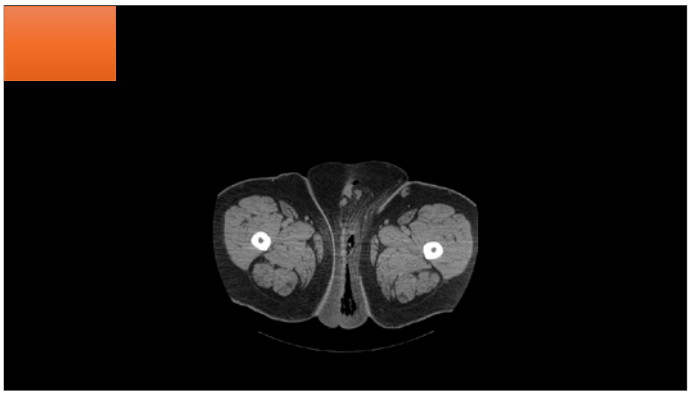
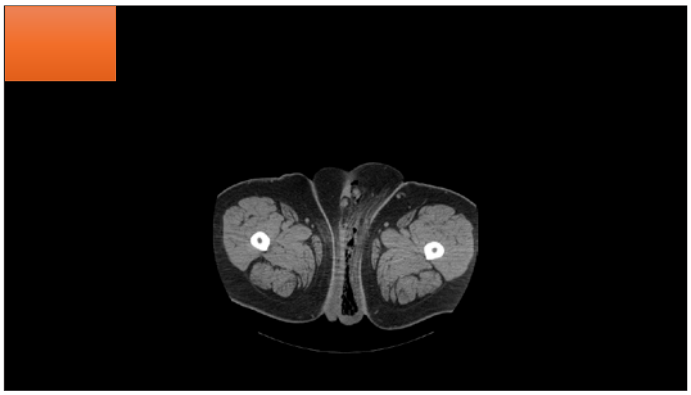
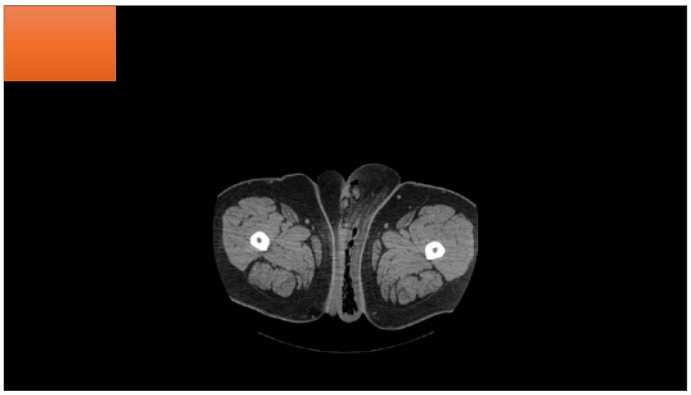
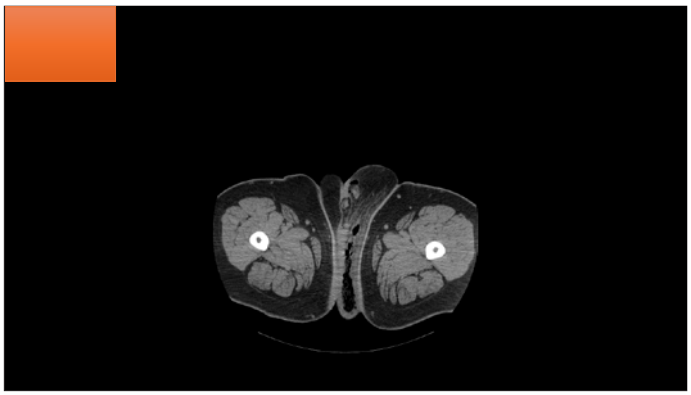
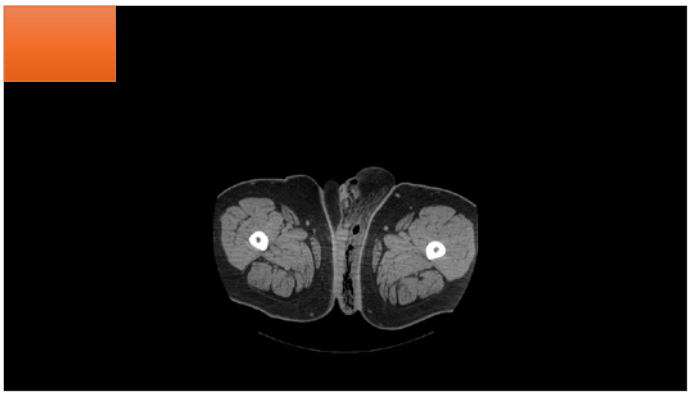
Suspect it in gastric bypass patients

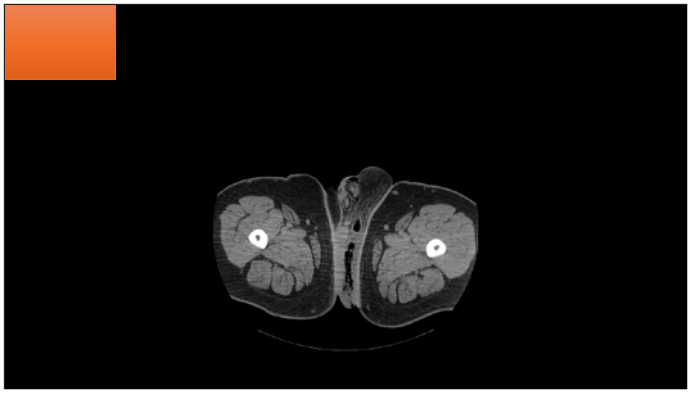
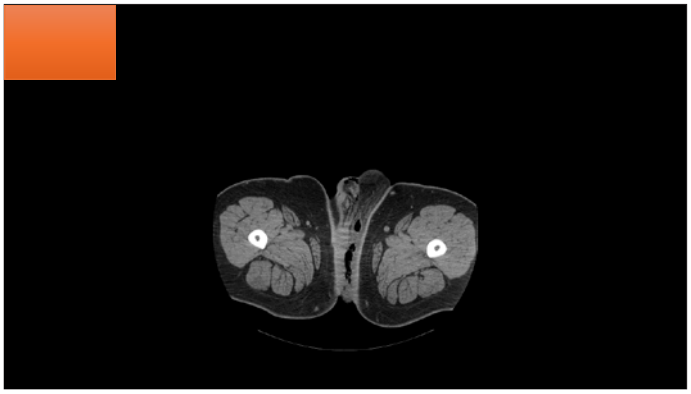
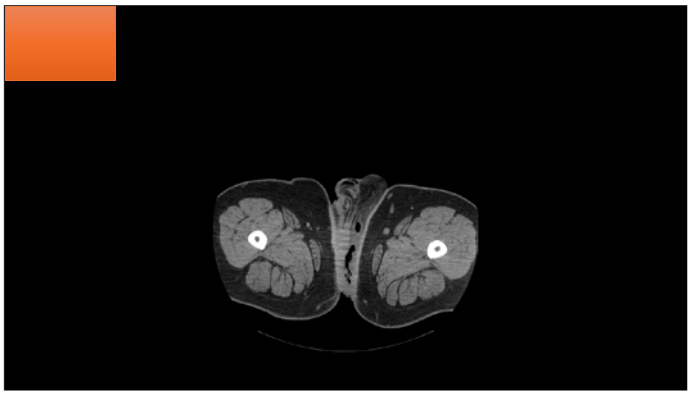
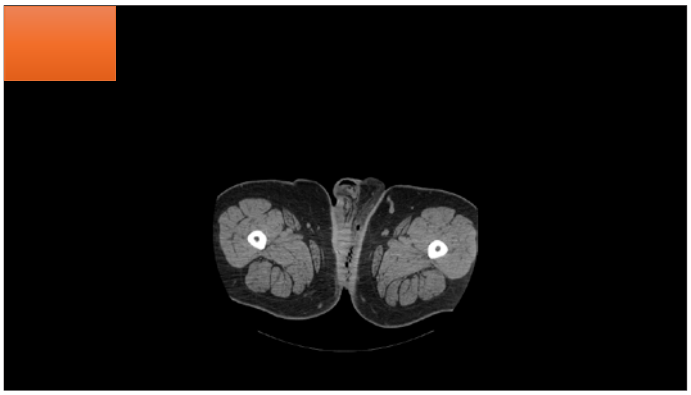
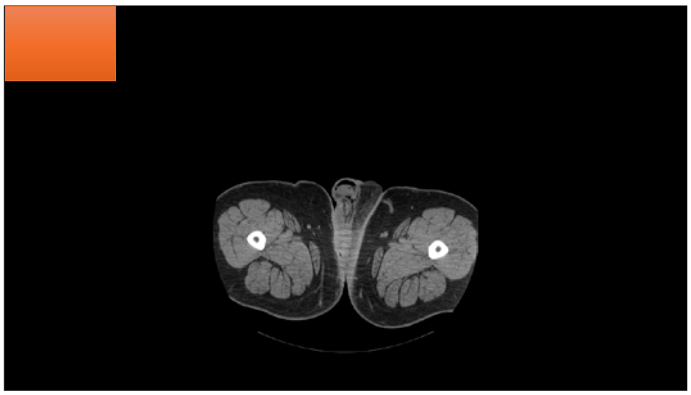


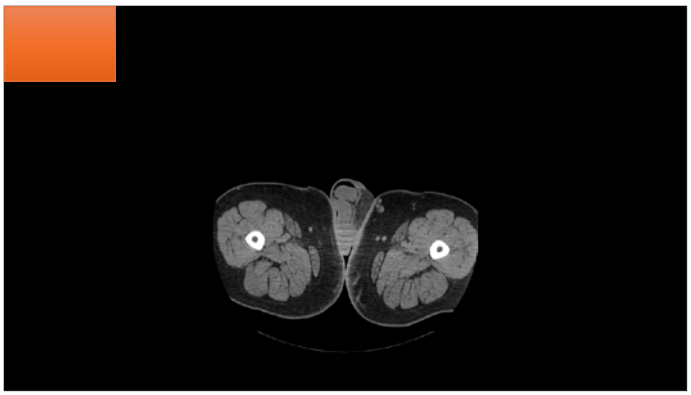
52 y.o. male with pelvic pain

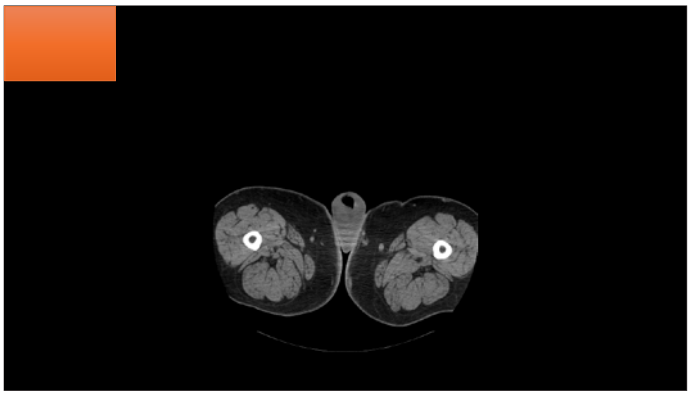









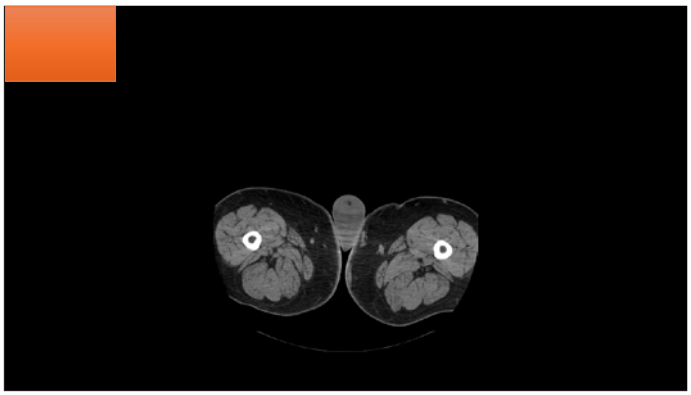









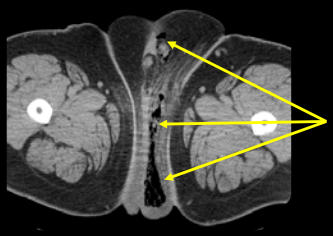










Fournier's Gangrene



Extensive soft tissue gas and stranding in the perineum

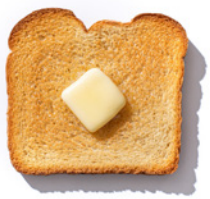
Almost always in diabetics

But… it may be their initial presentation

The minutes count!!

Fascial destruction of
2-3 cm/hr have been reported!!!

Now that we've totally scared you, let's look at some common ED cases



RUQ Pain

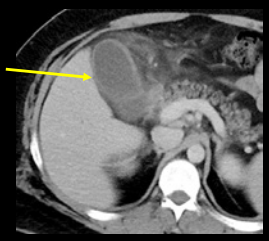
US is the preferred initial method for assessing for acute cholecystitis

However CT is not a bad test to assess for it:
Sensitivity 70-100%

Acute Cholecystitis - CT

May see gallbladder wall thickening, pericholecystic fluid, or surrounding inflammatory stranding

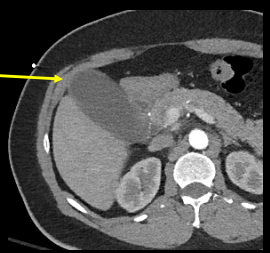
May miss up to 50% of gallstones



Tensile Gallbladder Fundus Sign

The GB is so distended that it bulges into and distorts the abdominal wall

75% sensitive
95% specific for acute cholecystitis

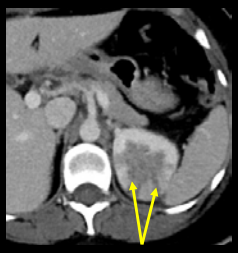


Flank Pain – Kidney Stone vs Pyelo

The classic teaching is that if looking for a kidney stone you should do a non-contrast CT

There are ways to evaluate for stone vs infection with contrast on board

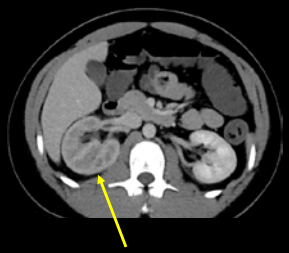
Striated Nephrogram



Alternating areas of high and low attenuation in the kidney on contrast CT

Often seen in pyelonephritis

Delayed Nephrogram



Decreased attenuation on contrast CT due to ureteral obstruction

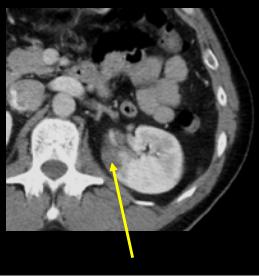
Often seen with obstructing stones, but can be seen with any cause of unilateral obstruction

Ureteral Enhancement

Also see a delayed nephrogram here, but note that the wall of the ureter on this side is enhancing

This may be seen with infection (ureteritis) or with urothelial cancers

Renal Infarct



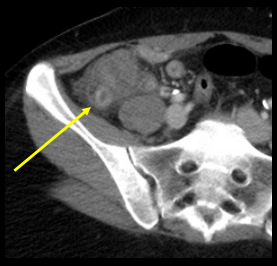
Decreased attenuation on contrast CT due to renal infarction

Often wedge-shaped areas of hypoperfusion

Can present exactly like renal colic

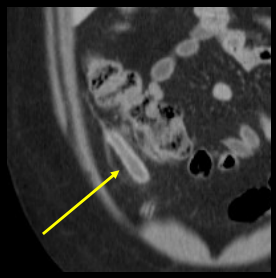
RLQ Pain

Appendicitis



Dilated, hyperemic appendix with surrounding soft-tissue stranding

Appendicitis

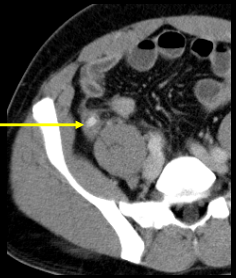


Dilated, hyperemic appendix with surrounding soft-tissue stranding

Appendicitis

Dilated, hyperemic appendix with surrounding soft-tissue stranding

Appendicolith (increases the chance of perforation)



LLQ Pain

Diverticulitis

Multiple diverticuli

Soft-tissue inflammatory stranding around the diverticuli

Diverticulitis with perforation

Soft-tissue inflammatory stranding around diverticuli with foci of extra-luminal gas

Epiploic Appendagitis

Thin high-density ring adjacent to colon with surrounding soft-tissue stranding

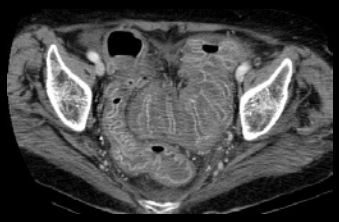
Torsed or thrombosed epiploic appendage

Epiploic Appendagitis

Rectosigmoid most common, but can occur anywhere along the colon

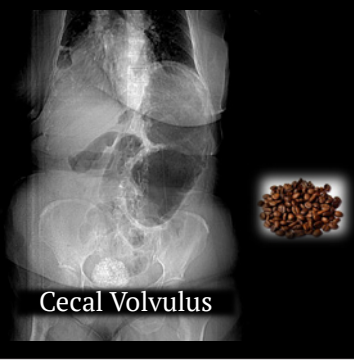
Self-limited ischemic/inflammatory process

Colitis



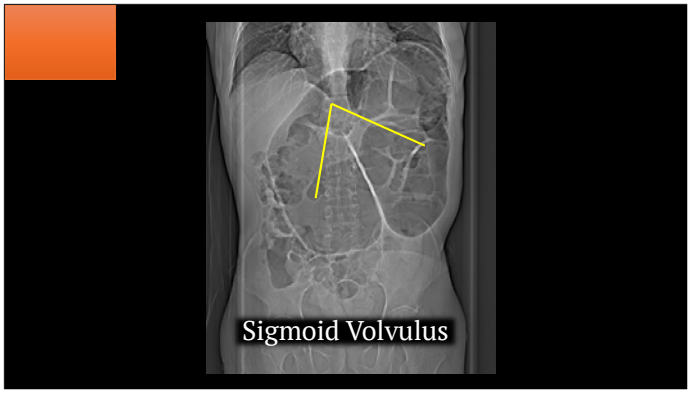
Diffuse edema and hyperemia of the colon

This case was *c. diff* colitis



Cecal Volvulus

Cecal Volvulus



Summary

- Focus on what's going to kill them – Start with the vasculature, then solid organs, then intestines
- Don't forget to make a pass on lung windows to look for air
- Look carefully for non-dependent air in pneumatosis

Summary

- You need IV contrast in trauma
- Assess carefully for closed loop obstruction – this is a surgical emergency
- Fournier's gangrene is another surgical emergency – if suspicious don't wait for a formal read on the CT

SELF EVALUATION

“My Stomach Hurts”: Unlocking the Black Box of Emergency Abdominal Imaging

1. T/F - Abdominal ultrasound has high sensitivity for detection of retroperitoneal rupture of an abdominal aortic aneurysm.

2. Emphysematous cholecystitis occurs most commonly in patients with which of the following underlying medical comorbidity:

a. Active malignancy
b. Cirrhosis
c. Diabetes mellitus
d. Hepatitis C
e. Primary sclerosing cholangitis

3. A striated nephrogram is a classic CT finding in which of the following diagnoses?

a. Pyelonephritis
b. Renal colic
c. Urinary obstruction
d. Renal infarct
e. Contrast-induced nephropathy

4. Management of epiploic appendagitis includes which of the following:

a. Emergent surgical consultation
b. Broad-spectrum antibiotics
c. Anti-inflammatory medications
d. Interventional radiology guided drainage

5. Patients with which of the following conditions are at high risk of developing a closed loop bowel obstruction?

a. Crohn’s Disease
b. S/p Roux-en-Y Gastric bypass
c. Ischemic colitis
d. Clostridium difficile colitis

Answer Key: 1. F, 2. C, 3. A, 4. C, 5. B

Michael W. Donnino, MD

Michael W. Donnino, MD, of Suffolk County, Massachusetts, is a Professor of Emergency Medicine at Harvard Medical School, and an attending physician in the Department of Emergency Medicine and Division of Pulmonary/Critical Care Medicine at Beth Israel Deaconess Medical Center. His clinical practice spans both the intensive care unit as well as the emergency department. Dr. Donnino was the first person in the country to train in a 6-year combined emergency medicine, internal medicine, and critical care program leading to board certification in all three areas. Dr. Donnino is also the Director of the Center for Resuscitation Science and Director of Critical Care in Emergency Medicine. Dr. Donnino's research interests include clinical trials and clinical investigations in septic shock and cardiac arrest. In the field of cardiac arrest, Dr. Donnino is the lead investigator of a multi-center clinical trial evaluating the use of steroids in post-arrest shock and a multi-center trial evaluating thiamine as a metabolic resuscitator in septic shock. Other notable studies include an investigation in microcirculatory dysfunction post-arrest, an evaluation of the human metabolome in sepsis and post-cardiac arrest, and severity of illness scoring systems in post-cardiac arrest patients. Dr. Donnino is a member of the Advanced Cardiac Life Support (ACLS) Subcommittee at the American Heart Association and co-author of sections of the 2010 ACLS guidelines.

You may contact Dr. Donnino with your comments or questions at mdonnino@bidmc.harvard.edu.

Diagnosis and Management of Sepsis and Septic Shock

Michael W. Donnino, MD

Disclosures

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- Research Grants: NIH, Foundational, Philanthropy, Day Zero Diagnostics

Objectives

To review pitfalls in the diagnosis of sepsis and septic shock

To evaluate treatments for patients with sepsis and septic shock

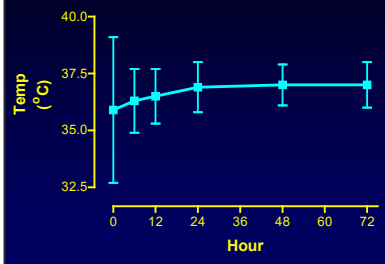
Principles of Sepsis Diagnosis

1) Identify sepsis
2) Identify shock and tissue perfusion

Temperature

Average: 99.5° C

NORMAL: 33%



Inadequacy of temperature and white blood cell count in predicting bacteremia in patients with infection. Seigel T., Cocchi MN, Salciccioli J, Donnino M (2010) JEM

White Blood Cell Count

289 Patients with blood culture positive sepsis

52% had NORMAL White Cell Count

Inadequacy of temperature and white blood cell count in predicting bacteremia in patients with infection. Seigel T., Cocchi MN, Salciccioli J, Donnino M (2010) JEM

Pitfalls in Laboratory Findings

Acute Myocardial Infarction: Troponin

Congestive Heart Failure: Brain Natriuretic Peptide (BNP)

What about Sepsis? ?

Pitfalls in Laboratory Findings

Sepsis: Troponin elevation in 15-69% [1]

Sepsis: BNP elevation in 40% in the ED and upwards of 70% within 24 hours[2]

1. Spies C., Serum Cardiac Troponin T as a Prognostic Marker in Early Sepsis. CHEST 1998; 113:1055-63
2. Donnino MW., et al. B-Type Natriuretic Peptide in Severe Sepsis and Septic Shock. Acad Emerg Med 2005; 12:S1

Clinical Case

PITFALL: Differential Shows 58% BANDS

of weakness.

- PE: T 35.9 P 136 BP 139/70 RR 26
- WBC: 9.2
- ECG: A. Fibrillation with RVR

Diagnosis: New-onset Atrial Fibrillation

Urinalysis: E. Coli

Blood Cultures: E. Coli

Clinical Review & Education

Special Communication | CARING FOR THE CRITICALLY ILL PATIENT

The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3)

Sepsis – New Definitions

Sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection

New Definition = Suspected infection and SOFA ≥ 2

Baseline SOFA score assumed to be zero in patients not known to have preexisting organ dysfunction.

Rationale: SOFA score = 2 reflects = 10% mortality

Singer JAMA 2016

SOFA Score

System	Score 0	1	2	3	4
Respiration					
PaO_2/FO_2, mm Hg (kPa)	≥400 (53.3)	<400 (53.3)	<300 (40)	<200 (26.7) with respiratory support	<100 (13.3) with respiratory support
Coagulation					
Platelets, $\times 10^3/\mu L$	≥150	<150	<100	<50	<20
Liver					
Bilirubin, mg/dL (μmol/L)	<1.2 (20)	1.2-1.9 (20-32)	2.0-5.9 (33-101)	6.0-11.9 (102-204)	>12.0 (204)
Cardiovascular	MAP ≥70 mm Hg	MAP <70 mm Hg	Dopamine <5 or dobutamine (any dose)[b]	Dopamine 5.1-15 or epinephrine ≤0.1 or norepinephrine ≤0.1[b]	Dopamine >15 or epinephrine >0.1 or norepinephrine >0.1[b]
Central nervous system					
Glasgow Coma Scale score[c]	15	13-14	10-12	6-9	<6
Renal					
Creatinine, mg/dL (μmol/L)	<1.2 (110)	1.2-1.9 (110-170)	2.0-3.4 (171-299)	3.5-4.9 (300-440)	>5.0 (440)
Urine output, mL/d				<500	<200

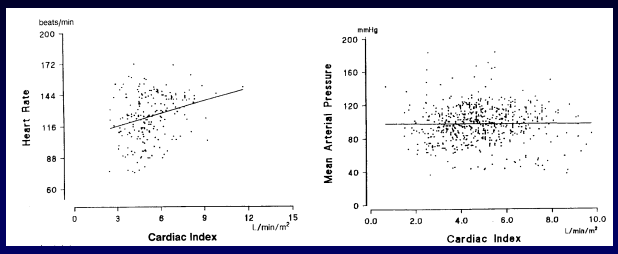
Can qSOFA Help?

- SBP < 100
- RR > 22
- Altered Mental Status

SHOCK = Inadequate Tissue Perfusion

Surrogate for Tissue Perfusion = Blood Pressure

Unreliability of Heart Rate and Blood Presssure



Wo C., Shoemaker W., Appel P. et al. Unreliability of blood pressure and heart rate to evaluate output in emergency resuscitation and critical illness. Critical Care Medicine 1993; 21:218-223

Detecting Hypoperfusion With Lactic Acid Levels

Lactic Acid Level (mmol/l)	Mortality (n/total)	Mortality (%)
0 -2.5	43/877	4.9 %
2.6 – 3.9	24/267	9.0 %
4.0 or greater	38/134	28.0 %

Howell M, Donnino M, Clardy P, Talmor D., Shapiro NI Occult Hypoperfusion in Emergency Department Patients with Infection. Intensive Care Medicine 2007; 33(11):1892-9

Clinical Case

PITFALL 1: Failure to Check Diff. (Bands = 58%)

of weakness.

- PE: T 35.9 P 136 BP 139/70 RR 26
- WBC: 9.2

PITFALL #2: Failure to Check Perfusion (Lactate = 12.0 mmol/dl)

Plan:

- Cardizem (Rate Slows to 100)
- Admit to Cardiology

Outcome: Death within 12 hours

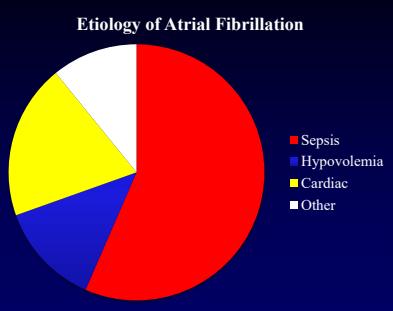
Bottom Line in Shock Assessment

1- Blood pressure is not adequate in early stages and therefore a "perfusion exam" and ancillary measures of perfusion (i.e., lactate) are key

2- Conversely, lactate is not perfect and is not always elevated in overt shock/hypotension

Perfusion Exam: mucous membranes, skin temperature, skin coloring (? mottling), respiratory rate

Pro-tip: Unstable Atrial Fibrillation

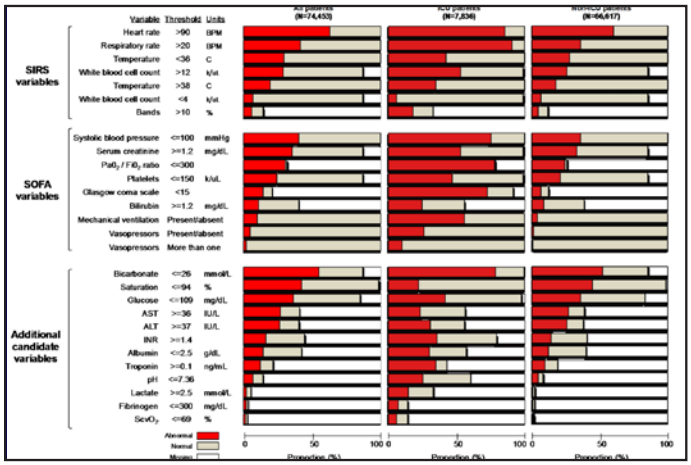


Cochran K, Larniczak R, Cocchi M, Donnino M. Etiology of Unstable Atrial Fibrillation with Type and Effectiveness of Intervention. (Unpublished Data)

The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3)

Septic Shock (old): Vasopressors after adequate fluid

Septic Shock (new): Vasopressors PLUS Lactate > 2



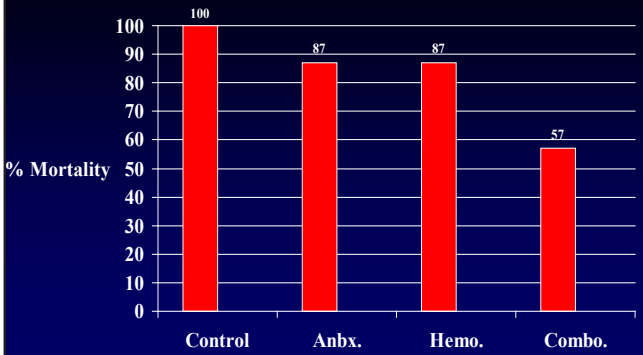
So What Should I Do?

1) Research versus Clinical Definition

2) How should we approach?

Sepsis and Septic Shock Treatment

Antibiotic and Hemodynamic Support



Natanson C, et al. Antibiotics versus cardiovascular support in a canine model of human septic shock. Am J Physiol. 1990 Nov;259(5 Pt 2):H1440-7.

Treatment

1) Source Identification and Control
2) Early, empiric antibiotics
3) Resuscitation to maintain perfusion
4) Adjunctive support (ie., steroids)
5) Avoiding secondary hits (nosocomial infection, etc..)

What Type of Shock is Septic Shock?

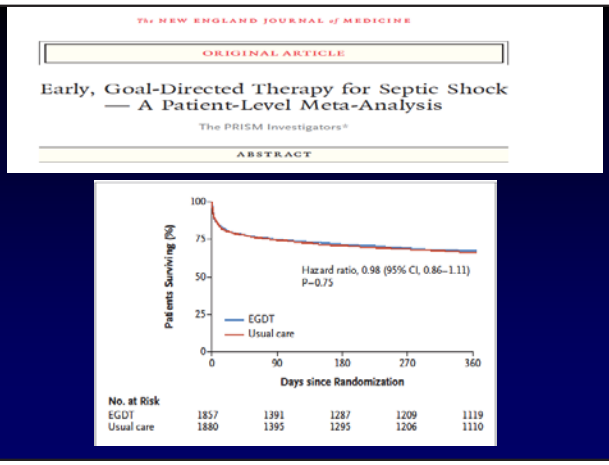
1) Hypovolemic
2) Distributive
3) Cardiogenic

What Type of Shock is Septic Shock?

1) Hypovolemic
2) Distributive
3) Cardiogenic (rare)
4) Any/all of the above

Principles of Sepsis Resuscitation

1) Restore Preload (Fluids)
2) Maintain Afterload (Vasopressors)
3) Evaluate for rare myocardial suppression (Inotropes), especially if not responding to initial treatment



Principles of Sepsis Resuscitation

1) Restore Preload (Fluids)
2) Maintain Afterload (Vasopressors)
3) Evaluate for rare myocardial suppression (Inotropes), especially if not responding to initial treatment

Case

54 year old female history of lupus who presents in respiratory distress and fever. Immediately required intubation and found to have pneumonia (CXR, copious dark green sputum from ETT)

Pt is hypotensive (60/40) and tachycardic and receives a total of 6 liters of fluid as well as broad spectrum antibiotics and stress-dose steroids. Despite the IVFs, she requires:

1- Norepinephrine 0.4 mcg/kg/min

2- Phenylephrine 5 mcg/kg/min

3- Vasopressin 0.04 IU/min

Current Exam…

T: 95 P 64 BP 80/60 (despite vasopressors as shown)

RR 16 Sat: 96% Fio2: .4

Gen: Intubated, sedated

HEENT: slightly dry mucous membranes

Chest: Crackles bilaterally

Heart: S1 S2 reg

Abd: Soft NT ND

Ext: cool, clammy

Other Data…

- CVP: 16 mmHg
- Urine output: 5 cc/hour

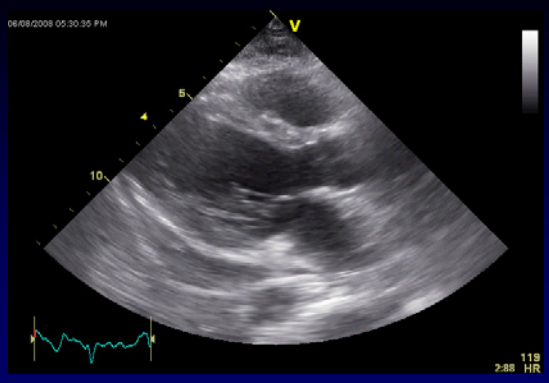
- WBC: 17,000 bands 34%
- BUN: 60 Cr: 2.3
- INR: 2.1 PTT 56
- ALT/ALT: 1006/ 900
- Lactate: 8.7 mmol/dl

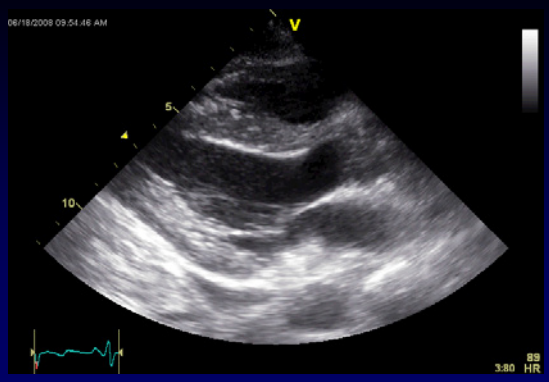
What are you going to do?

1) Provide additional 1 liter fluid bolus and reassess blood pressure, urine output, and lactate
2) Add dopamine drip
3) Arrange family meeting for discussion on goals of care
4) Continue current therapy and reassess in 2-3 hours
5) Seek additional data

More Data…

1) $Scvo_2$ = 52%
2) ECHO: Global hypokinesis (EF=25-30%)
3) Central venous pCO2 = 60, Arterial pCO2 = 45 with gradient = 15
4) CI = 1.2





Pro-Tip for Pacemakers

1) If in septic shock with hypoperfusion AND is pacemaker dependent, increase pacemaker rate

2) Monitor to see if pacemaker rate increase improves perfusion parameters

3) Easy fix, often overlooked (and cardiology sometimes is reluctant to support so need to demonstrate the need)

How Much Fluids Should I Give?

- Depends on overall picture and phase of disease
- Is the patient still hypoperfused? (physical exam, urine output, lactate, vital signs)
- What do my preload surrogates tell me? (filling pressures, fluid challenge, dynamic measurements)
- What are the patient and disease characteristics?

Consider Multiple End-points of Resuscitation

- Heart Rate
- Blood Pressure
- Physical exam (cool skin, mottling)
- Urine output
- Lactic Acidosis/Lactate Clearance
- Filling Pressures (ie., CVP)
- Cardiac Output/Index
- Echocardiography
- Venous-arterial pCO_2 difference
- Central or Mixed Venous Saturation

Each endpoint has advantages and disadvantages, so you need to consider multiple endpoints…

For example, can have good urine output but high lactate….

Or, may have normal lactate but low urine output

- Lactic Acidosis/Lactate Clearance
- Filling Pressures (ie., CVP or PCWP)
- Cardiac Output/Index
- Echocardiography
- Venous-arterial pCO_2 difference
- Central or Mixed Venous Saturation

So, what do we know about lactate?

- Lactate is a marker for hypoperfusion
- Lactate change over time is associated with outcome
- However, there are other causes of lactate elevation besides hypoperfusion
- Persistently elevated lactate can help one step back to assess if perfusion issue and next steps but not always knee-jerk more fluids

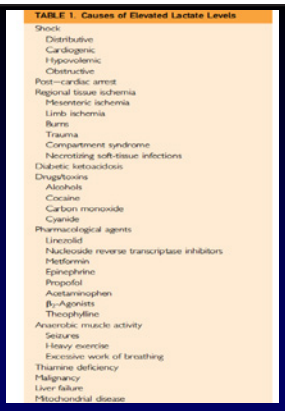


TABLE 1. Causes of Elevated Lactate Levels

- Shock
  - Distributive
  - Cardiogenic
  - Hypovolemic
  - Obstructive
- Post–cardiac arrest
- Regional tissue ischemia
  - Mesenteric ischemia
  - Limb ischemia
  - Burns
  - Trauma
  - Compartment syndrome
  - Necrotizing soft-tissue infections
- Diabetic ketoacidosis
- Drugs/toxins
  - Alcohols
  - Cocaine
  - Carbon monoxide
  - Cyanide
- Pharmacological agents
  - Linezolid
  - Nucleoside reverse transcriptase inhibitors
  - Metformin
  - Epinephrine
  - Propofol
  - Acetaminophen
  - β_2-Agonists
  - Theophylline
- Anaerobic muscle activity
  - Seizures
  - Heavy exercise
  - Excessive work of breathing
- Thiamine deficiency
- Malignancy
- Liver failure
- Mitochondrial disease

Andersen L, Andersen LW, Mackenhauer J, Roberts JC, Berg KM, Cocchi MN, Donnino MW. Etiology and Therapeutic Approach to Elevated Lactate Levels. Mayo Clinic Proceedings (2013)

But Wait, Should I Give Fluids or Vasopressors?

- Isn't there a study that determined do I give fluids or vasopressors?

But Wait, Should I Give Fluids or Vasopressors?

- Isn't there a study that determined do I give fluids or vasopressors?

(Early Restrictive or Liberal Fluid Management for Sepsis-Induced Hypotension, Shapiro, NEJM, 2023)

But Wait, Should I Give Fluids or Vasopressors?

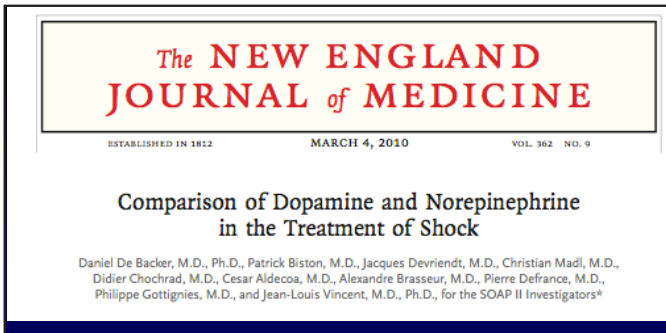
- Fluids vs. Pressors when answer is typical "it depends" and not a dichotomous decision (i.e., often we use both).
- Clinicians could defer enrollment if they felt one or the other (or both should be given)
- Challenge of fixed-treatment groups in clinical trials with titratable care

Some Vasopressor Choices

ALPHA

Phenylephrine
Norepinephrine
Epinephrine
Dopamine

BETA



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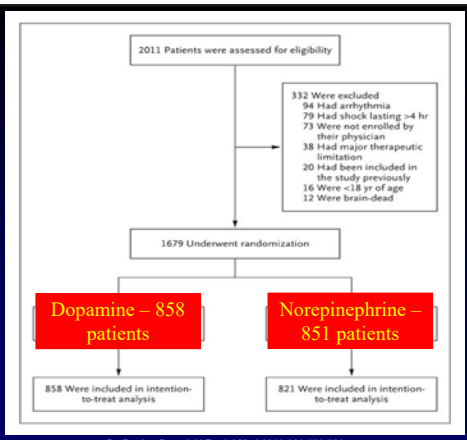
Comparison of Dopamine and Norepinephrine in the Treatment of Shock

Daniel De Backer, M.D., Ph.D., Patrick Biston, M.D., Jacques Devriendt, M.D., Christian Madl, M.D., Didier Chochrad, M.D., Cesar Aldecoa, M.D., Alexandre Brasseur, M.D., Pierre Defrance, M.D., Philippe Gottignies, M.D., and Jean-Louis Vincent, M.D., Ph.D., for the SOAP II Investigators*



De Backer D et al. N Engl J Med 2010;362:779-789

Norepinephrine vs. Dopamine -Mortality

Table 2. Mortality Rates.*

Time Period	Dopamine	Norepinephrine	Odds Ratio (95% CI)†	P Value
	percent mortality			
During stay in intensive care unit	50.2	45.9	1.19 (0.98–1.44)	0.07
During hospital stay	59.4	56.6	1.12 (0.92–1.37)	0.24
At 28 Days:	**52.5%**	**48.5%**		**0.1**

* Data were available for 1656 patients in the intensive care unit, in the hospital, and at 28 days; for 1443 patients at 6 months; and for 1036 patients at 12 months.
† Odds ratios for death are for the comparison of the dopamine group with the norepinephrine group.

De Backer D et al. N Engl J Med 2010;362:779-789

Table 3. Secondary Outcomes and Adverse Events.*

Variable	Dopamine (N=858)	Norepinephrine (N=821)	P Value
Support-free days through day 28			
Vasopressors not needed			
Trial drug	11.0±12.1	12.5±12.1	0.01
Open-label vasopressors	12.6±12.5	14.2±12.3	0.007
Mechanical ventilation not needed	8.5±11.2	9.5±11.4	0.13
Renal support not needed	12.8±12.4	14.0±12.3	0.07
Intensive care not needed	8.1±10.3	8.5±10.3	0.43
Length of stay — no. of days			
Intensive care unit			0.12
Median	5	5	
Interquartile range	1–11	2–12	
Hospital			0.22
Median	11	12	
Interquartile range	2–28	3–28	
Cause of death in hospital — no./total no. (%)			0.31
Refractory shock			
Withdrawal of life support because of brain death			
Adverse events			
Arrhythmias			
Atrial fibrillation			
Ventricular fibrillation	10 (1.2)	4 (0.5)	
Myocardial infarction — no. (%)	19 (2.2)	25 (3.0)	0.29
New infectious episode			
No. of episodes			0.69
Median	1	1	
Interquartile range	0–1	0–1	
Patients with at least one episode — no. (%)	674 (78.6)	619 (75.4)	0.35
Skin ischemia — no. (%)	56 (6.5)	34 (4.1)	0.09
Mild†	46 (5.4)	28 (3.4)	
Severe‡	10 (1.2)	6 (0.7)	
Arterial occlusion — no. (%)§	23 (2.7)	20 (2.4)	0.12
Arms or fingers	5 (0.6)	1 (0.1)	
Legs	7 (0.8)	13 (1.6)	

Arrhythmias: Dopamine 24% Norepine 12% <0.001

Possible Scenarios

BP 80/30 HR 106

BP 80/30 HR 148

BP 80/30 HR 50

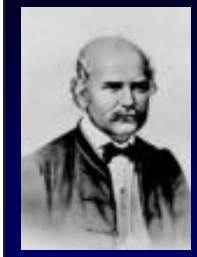
Possible Scenarios – Consider the Heart Rate (HR)

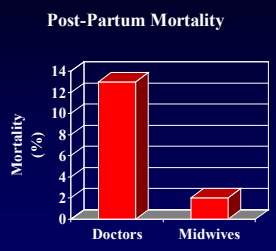
BP 80/30	HR 106	Norepinephrine
BP 80/30	HR 148	Phenylephrine
BP 80/30	HR 50	Epinephrine/Dopamine

Steroids in Septic Shock

- Long history of mixed trial results
- Concept of shock reversal versus immune system modulation
- Current guidelines and my approach focus on use for refractory shock – what defines "refractory shock" differs between clinicians. In the ED, can always start them upfront and they can be discontinued in the ICU if improving

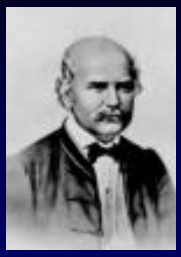
Sepsis: The Beginning

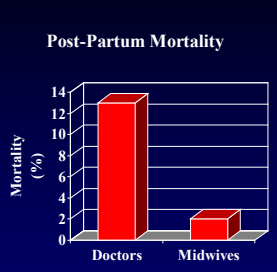




Ignaz Semmelweis

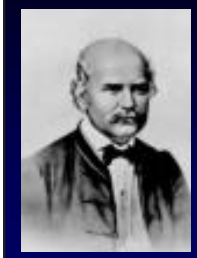
Sepsis: The Beginning

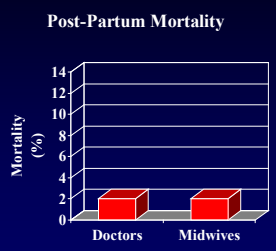




Ignaz Semmelweis

Sepsis: The Beginning





Ignaz Semmelweis

Conclusions

1) First, identify sepsis and shock
2) Provide early antibiotics and address source control
3) Restore tissue hypoperfusion with attention to:
   a) Preload (Fluids)
   b) Maintain Afterload (Vasopressors)
   c) Evaluate for rare myocardial suppression (Inotropes)
4) Careful not to rely on just one endpoint → consider multiple endpoints and the entire patient
5) Remember ancillary tips like increasing pacemaker rate, steroids for refractory shock, and prevention secondary nosocomial infections

SELF EVALUATION

Diagnosis and Management of Sepsis and Septic Shock

1. T/F - A normal white blood cell (WBC) count can rule out sepsis in patients with infection.

2. Which of the following best defines sepsis according to the 2016 JAMA criteria?

a. Infection with fever and tachycardia
b. Life-threatening organ dysfunction caused by a dysregulated host response to infection
c. Hypotension requiring vasopressors
d. Bacteremia confirmed by blood cultures

3. T/F - Blood pressure alone is a reliable indicator of tissue perfusion in early septic shock.

4. Which vasopressor is generally preferred as the first-line agent in septic shock?

a. Dopamine
b. Norepinephrine
c. Phenylephrine
d. Epinephrine

5. T/F - Lactate elevation is always due to hypoperfusion in septic shock.

6. Which of the following is the correct sequence emphasized in sepsis resuscitation?

a. Vasopressors → Fluids → Inotropes
b. Fluids → Vasopressors → Consider Inotropes
c. Inotropes → Steroids → Fluids
d. Antibiotics → Vasopressors → Fluids

Answer Key: 1. F, 2. B, 3. F, 4. B, 5. F, 6. B

Critical Concepts in Pediatric Trauma: Imaging, Intervention, and Risk Reduction

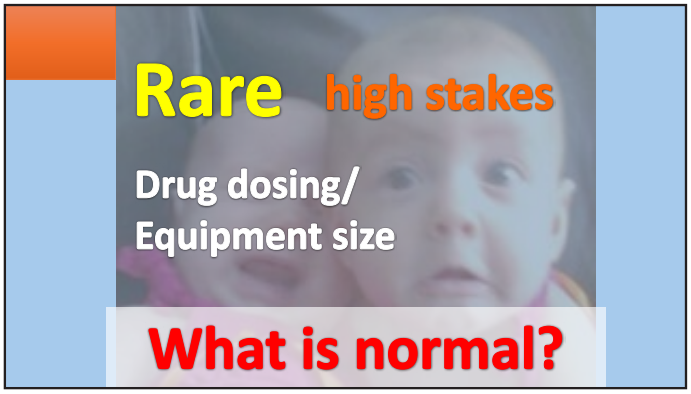
Emily Rose, MD, FAAP, FAAEM, FACEP

Disclosures

- No financial relationships to disclose
- No off-label medication or therapeutics will be discussed
  - (TXA dosing and indications remain unclear)

Objectives

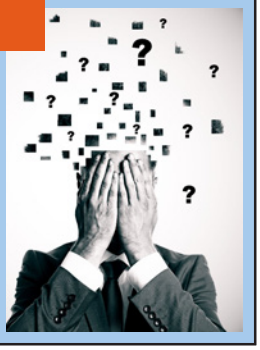
- Compare and contrast care of the injured child with management of the adult
- Discuss how to clinically clear a young child's cervical spine
- Formulate an evidence based diagnostic plan for utilization of CT in pediatric trauma victims
- Implement radiation sparing techniques in the diagnostic work-up of trauma patients



Peds weight?

Infant: (months of age+9)/2
Age 1-4 : 2x (age+5)
Age 5-14 years: 4 x age

Weight=2 (age+4)
2x age + 10



Years	KG
1	10
3	15
5	20
7	25

Years	KG
1	10
3	15
5	20
7	25

Birth 3 kg
4 mo 6 kg

AGE	HR	RR
0-1 YR	140	40
1-4	120	30
4-12	100	20
>12	80	15

BP (5th %)	
NEONATE	60 (mmHg)
INFANT	70
1-10 yr	(age x 2) + 70
>10 yr	90

Peds Airway

ETT Simplified

4.0 at 1 year

5.0 at 5

6.0 at 8-10 years

BLADE	AGE
Miller 0	Premature/Newborn
Miller 1	1mo-2 years
Wis-Hipple 1.5	2 years
Miller 2	3-6 years
Macintosh 2	3 years

BLADE	AGE
Miller 0	Premature/Newborn
Miller 1	Mo-2 years
Wis-Hipple 1.5	2 years
Miller 2	3-6 years
Macintosh	3 years

2 at 2

Sedation

Etomidate
Ketamine
Fentanyl
Midazolam
Propofol

Paralysis

Succinylcholine
Atropine 0.02 mg/kg
Min 0.1 mg/Max 0.5 mg
Rocuronium

RSI Facilitates Intubation Reduces Adverse Events

Head Trauma

Huge Heads

Bad news: High risk for trauma; thinner cranium is less protective

Good news: open sutures can tolerate expanding intracranial hematoma

MOST head injuries are **MINOR** and requires

NO intervention

Clinical Decision Rules for Minor Head Trauma

PECARN (US)—most sensitive and most widely used 10,000 <2; 30,000 >2

CATCH (Canada) 3,000

CHALICE (Europe) 22,000

Predictor variables	PECARN <2 (N=10,718)	PECARN ≥2 (N=31,695)	CATCH (N=3,866)	CHALICE (N=22,772)
Mechanism of injury	Severe mechanism of injury (MVC with patient ejection, death of another passenger, or rollover; pedestrian or bicyclist without helmet struck by motorized vehicle; falls >0·9 m; or head struck by high-impact object)	Severe mechanism of injury (MVC with patient ejection, death of another passenger, or rollover; pedestrian/bicyclist without helmet struck by motorized vehicle; falls >1·5m; or head struck by high-impact object)	Dangerous mechanism of injury (e.g. MVC; fall from elevation ≥3 ft (≥91 cm) or ≥5 stairs; or fall from bicycle with no helmet)	High-speed RTA as pedestrian, cyclist, or occupant (defined as accident with speed >40 miles per h or 64 km/h); fall >3 m in height; or high-speed injury from projectile or object
History	LOC for ≥5 s Not acting normally per parent report	Any LOC History of vomiting Severe headache	History of worsening headache	Witnessed loss of consciousness for >5 min ≥3 discrete episodes of vomiting after head injury Amnesia (antegrade or retrograde; >5 min) Suspicion of non-accidental injury (any suspicion by the examining doctor) Seizure in patient with no history of epilepsy
Examination	GCS score <15 Other signs of altered mental status (agitation, somnolence, repetitive questioning, slow response to verbal communication) Palpable or unclear skull fracture Occipital, parietal, or temporal scalp hematoma	GCS score <15 Other signs of altered mental status (agitation, somnolence, repetitive questioning, slow response to verbal communication) Clinical signs of basilar skull fracture (e.g. hemotympanum, "raccoon" eyes, otorrhea o rhinorrhea of CSF, Battle's sign	GCS score <15 at 2 h after injury§ Irritability on examination§ Any sign of basal skull fracture (e.g., hemotympanum, "raccoon" eyes, otorrhea or rhinorrhea of CSF, Battle's sign) Suspected open or depressed skull fracture§ Large, boggy scalp hematoma	GCS score <14, or <15 if aged <1 year Abnormal drowsiness (in excess of that expected by examining doctor) Positive focal neurology (motor, sensory, coordination, or reflex abnormality) Signs of basal skull fracture (hemotympanum, "raccoon" eyes, otorrhea or rhinorrhea of CSF, Battle's sign, facial crepitus, or severe facial injury)

Statistics*	PECARN <2 (N=10,718)	PECARN ≥2 (N=31,695)	CATCH (N=3,866)	CHALICE (N=22,772)
Sensitivity (95% CI)	100.0% (90.7-100.0	99.0% (94.4-100.0)	88.7% (82.2-93.4)	92.3% (89.2-94.7
Specificity (95%CI	53.8% (52.3-55.4)	45.8% (44.9-46.8)	56.4% (55.0-57.8)	78.1% (77.5-78.7)
PPV (95% CI)	2.0% (1.4-2.8)	1.6% (1.3-1.9)	5.6% (4.7-6.7)	7.9% (7.2-8.7)
NPV (95% CI)	100.0% (99.8-100.0)	100.0% (99.9-100.0)	99.4% (99.1-99.7)	99.8% (99.7-99.9)

PECARN (<2 yrs)

- Normal mental status
- No scalp hematoma (except frontal)
- LOC <5 seconds
- Non-severe mechanism
- No palpable skull fracture
- Acting normally per parents

Negative Predictive Value

99.99%

Severe Mechanism

- MVC
  - Ejection
  - Passenger death
  - Rollover
- Auto vs Peds/Bicycle (no helmet)
- Fall
- Head struck by high impact object

< 2 Years Old - Rates of ciTBI

	Intermediate Risk Variables: Isolated – Extensive definition	Isolated – PECARN risk based definition	Severe mechanism of injury	Nonfrontal scalp hematoma	Loss of consciousness +5 sec	Not acting normally per parent	High Risk Variables: Palpable or unclear fracture	GCS <15 or Altered Mental Status
Severe mechanism of injury	0.1% (1/756) (0-0.7 CI)	0.3% (4/1327) (0.1-0.8 CI)		3.5% (9/257) (2-7 CI)	3.5% (2/57) (0.4-12 CI)	0.6% (1/156) (0-4 CI)	0% (0/21) (0-16 CI)	3% (3/103) (0.6-8 CI)
Nonfrontal scalp hematoma	0.4% (12/2998) (0.2-0.7 CI)	0.5% (4/820) (0.1-1.2 CI)	3.6% (7/231) (1.2-6.1 CI)		0% (0/16) (0-20.6 CI)	1.7% (1/59) (0-9.1 CI)	1.4% (1/72) (0-7.5 CI)	2.2% (1/45) (0.1-11.8 CI)
Loss of consciousness +5sec	0.2%* (1/576) (0-1.0 CI)	0.6% (1/857) (0-3.2 CI)	3.5% (2/57) (0.6-12 CI)	0% (0/16) (0-20.6 CI)		0% (0/20) (0-16.8 CI)	Data Not Available	0% (0/16) (0-20.6 CI)
Not acting normally per parent	0% (0/188) (0-1.9 CI)	0.2% (1/411) (0-1.3 CI)	0.7% (1/141) (0-3.8 CI)	1.7% (1/59) (0-9.1 CI)	0% (0/20) (0-16.8 CI)		0% (0/10) (0-30.8 CI)	0.8% (4/494) (0.2-3.0 CI)
Vomiting	0.2%* (2/815) (0-0.9 CI)	0% (0/567) (0-0.6 CI)	0.6% (1/181) (0-3.0 CI)	0% (0/76) (0-4.7 CI)	5.6% (1/18) (0.1-27.3 CI)	0.6% (1/158) (0-3.5 CI)	0% (0/5) (0-52.2 CI)	0% (0/35) (0-10 CI)

*Aggregate data for all patients <18 years

PECARN (>2 yrs)

- Normal mental status
- No LOC
- No vomiting
- Non-severe mechanism
- No basilar skull fracture
- No severe headache

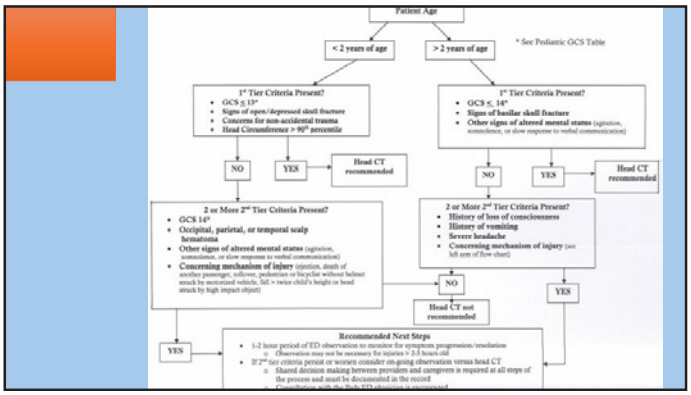
Negative Predictive Value

99.95%

2 – 18 Years Old - Rates of ciTBI

	Intermediate Risk Variables: Isolated – Extensive definition	Isolated – PECARN risk based definition	Severe mechanism of injury	Loss of consciousness	Vomiting	Severe headache	High Risk Variables: Signs of basilar skull fracture	GCS <15 or Altered Mental Status
Severe mechanism of injury	0.3% (2/730) (0-1.0 CI)	0.6% (5/1975) (0.3-1 CI)		2.3% (14/601) (1-4 CI)	4.5% (5/110) (1-10 CI)	0% (0/27) (0-12.8 CI)	0% (0/12) (0-26 CI)	3.8% (10/261) (2-7 CI)
Loss of consciousness	0.2%* (1/576) (0-1.0 CI)	0.5% (12/2623) (0.2-0.8 CI)	2.3% (14/601) (1-4 CI)		0.9% (3/321) (0.2-2.7 CI)	0% (0/121) (0-3.0 CI)	10% (2/20) (1.2-31.7 CI)	1.9% (13/695) (1.0-3.2 CI)
Vomiting	0.2%* (2/815) (0-0.9 CI)	0.7% (10/1501) (0.3-1.2 CI)	4.5% (5/110) (1-10 CI)	0.9% (3/321) (0.2-2.7 CI)		1.4% (1/69) (0-7.8 CI)	18.8% (3/16) (4-45.6 CI)	1.8% (9/487) (0.8-3.5 CI)
Severe headache	0% (0/2462) (0-0.1 CI)	1.4% (3/209) (0.3-4.1 CI)	0% (0/27) (0-12.8 CI)	0% (0/121) (0-3.0 CI)	1.4% (1/69) (0-7.8 CI)		0% (0/3) (0-70.8 CI)	2.7% (2/74) (0.3-9.4 CI)

*Aggregate data for all patients <18 years

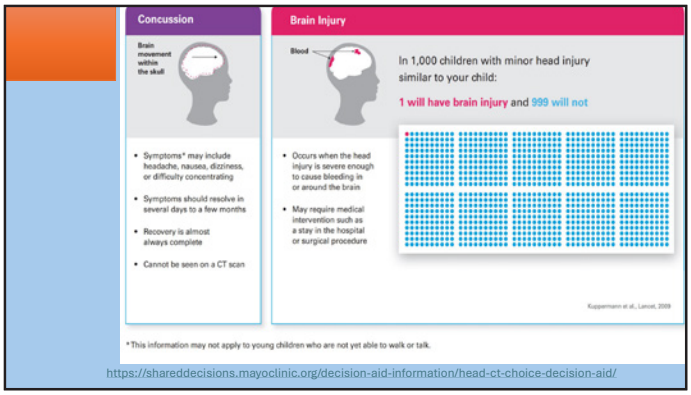


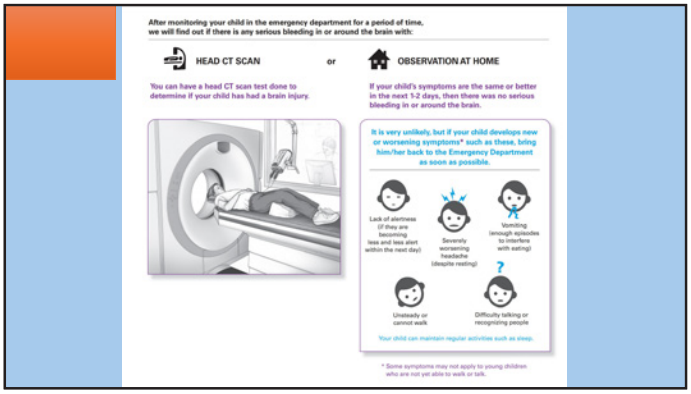
< 2 years old

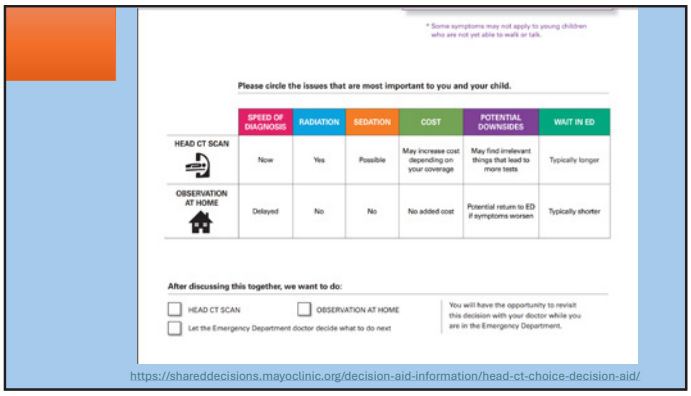
First Risk Factor		None	GCS < 15 or AMS	Scalp Haematoma	Loss of Consciousness +5sec	Severe Mechanism of Injury	Palpable or Unclear Fracture	Not acting normally per parent
GCS < 15 or AMS	risk							
	DA							
Scalp Haematoma	risk	0.5 (0.1,1.2)			0.5 (0.1,1.2)	3.0 (1.2,6.1)		1.7 (0.9,9.1)
	DA	1:100			1:100	3:100		2:100
Loss of Consciousness +5sec	risk	0.6 (0.0,3.5)		0.6 (0.0,3.5)		3.9 (0.5,13.5)		0.6 (0.0,3.5)
	DA	1:100		1:100		4:100		1:100
Severe Mechanism of Injury	risk	0.2 (0.1,0.7)		3.0 (1.2,6.1)	3.9 (0.5,13.5)			0.7 (0.0,3.8)
	DA	2:1000		3:100	4:100			1:100
Palpable or Unclear fracture	risk							
	DA							
Not acting normally per parent	risk	0.2 (0.0,1.3)		1.7 (0.0,9.1)	0.6 (0.0,3.5)	0.7 (0.0,3.8)		
	DA	2:1000		2:100	1:100	1:100		

2 + years old

First Risk Factor		None	GCS < 15 or AMS	Loss of consciousness	Vomiting	Signs of basilar skull fracture	Severe mechanism of injury	Severe headache
GCS < 15 or AMS	risk							
	DA							
Loss of consciousness	risk	0.5 (0.2,0.8)			0.9 (0.2,2.7)		2.4 (1.3,4.1)	1.4 (0.3,4.1)
	DA	1:100			1:100		2:100	1:100
Vomiting	risk	0.7 (0.3,1.2)		0.9 (0.2,2.7)			2.4 (0.3,8.3)	1.4 (0.7,8)
	DA	1:100		1:100			2:100	1:100
Signs of basilar skull fracture	risk							
	DA							
Severe mechanism of injury	risk	0.3 (0.1,0.6)		2.4 (1.3,4.1)	2.4 (0.3,8.3)			1.4 (0.3,4.1)
	DA	3:1000		2:100	2:100			1:100
Severe headache	risk	1.4 (0.3,4.1)		1.4 (0.3,4.1)	1.4 (0, 7.8)		1.4 (0.3,4.1)	
	DA	1:100		1:100	1:100		1:100	







PECARN High Risk Features

AMS/depressed GCS

Signs of skull fracture

ciTBI ≈4%

PECARN High Risk Features

AMS/depressed GCS

Signs of skull fracture

ciTBI ≈4%

Imaging probably appropriate

PECARN Intermediate Risk

Other features

ciTBI=0.9%

PECARN Intermediate Risk

Isolated severe mechanism

ciTBI=0.1-0.3%

PECARN Intermediate Risk

Extensive vomiting

ciTBI=0.2%

PECARN Intermediate Risk

Isolated non-frontal scalp hematoma

ciTBI=0.4%

PECARN Intermediate Risk

Isolated LOC

ciTBI=0.2%

PECARN Intermediate Risk

"Not acting normally" and severe headache

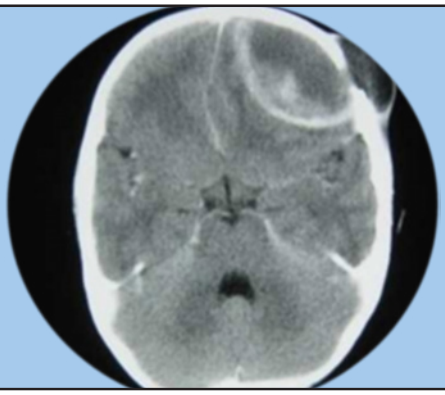
ciTBI=0%

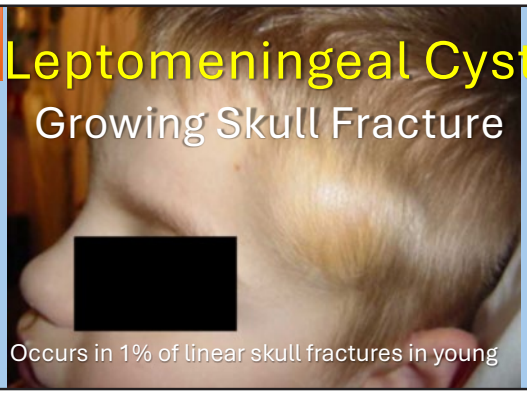
Observation decreases scans

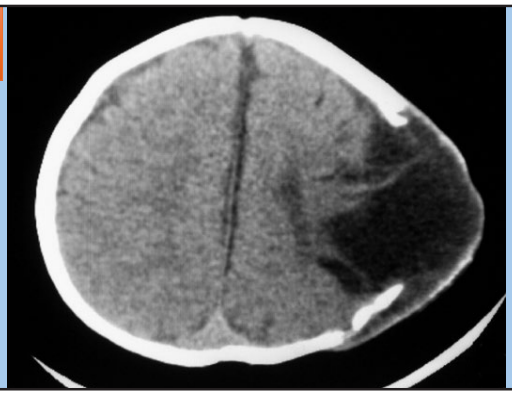
Most symptoms improve significantly

Unlikely to develop findings after 6 hours

Abnormal CT?







Pitfall to decision rules:

NAT

Pediatric Glasgow Coma Scale

Eye opening: open to sound (command)
Verbal response: age-appropriate (oriented)
cries/irritable (confused)
cries to pain (inappropriate)
moans to pain (incomprehensible)
Motor response: spontaneous movements
(obey commands)
withdraws to touch (or localizes)

Injury Prevention

Primary: prevent the incident

Secondary: decrease serious injury

Tertiary: decrease complications of injuries

Severe TBI Management

- Elevate HOB
- Pain management
- Avoid hypoxia
- Normoglycemia
- Euvolemia
- Normothermia
- Treat coagulopathy
- Seizure prevention

Cervical Spine Injury

Location of CSI

Clearing the C-Spine

- NEXUS
- Mechanism of injury
- Neurologic symptoms
- Physical evidence of significant trauma to head/neck
- Altered mental status/inconsolable

NEXUS

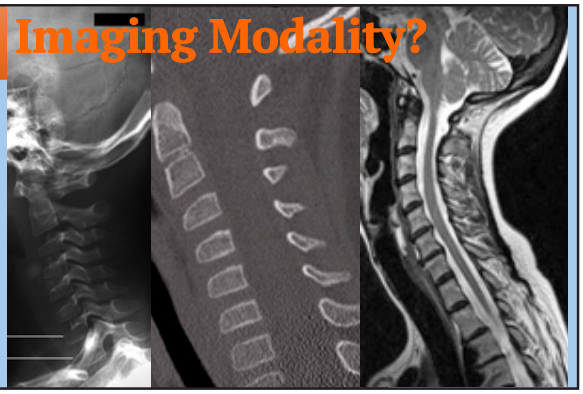
- No midline cervical tenderness
- No focal neurologic deficit
- Normal alertness
- No intoxication
- No painful, distracting injury

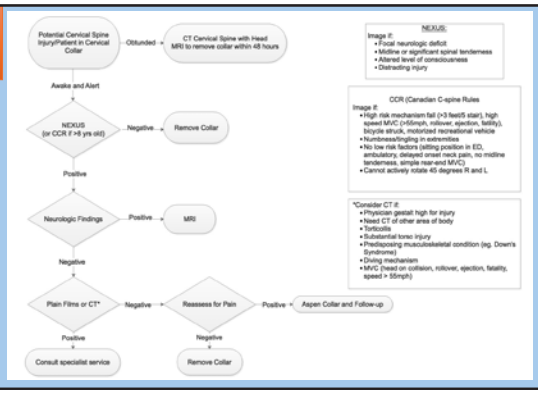
Clearing the C-Spine

- NEXUS
- Mechanism of injury
- Neurologic symptoms
- Physical evidence of significant trauma to head/neck
- Altered mental status/inconsolable

	NEXUS	CCR	PECARN
Rule	• No posterior midline tenderness • No evidence of intoxication • Normal level of alertness • No focal neurologic deficit • No painful distracting injuries	1. No high-risk factors* 2. Simple rear-end MVC+ or sitting in the ED or ambulatory or delayed onset of pain or absence of cervical spine 3. Able to rotate 45 left and right	• Altered mental status • Focal neurologic findings • Neck pain • Torticollis • Substantial torso injury± • Conditions predisposing to cervical spine injury° • Diving • High-risk mechanismΨ
# Children	3701	None	540 patients with injury and 2774 controls
Sensitivity	0.57 (0.18-0.9) 0.98 (0.91-1.0) 1.00 (0.88-1.00)	0.86 (0.42-1.00)	0.98 (96-99)
Specificity	0.35 (0.25-0.45) 0.54 (0.45-0.62) 0.20 (0.18-0.21)	0.15 (0.08-0.23)	0.26 (0.23-0.29)

Imaging Modality?





Neck Trauma

Neck Trauma Red Flags

- Neurologic Deficit
- Abnormal Pulse
- Bruit
- Stridor/Signs of airway compromise

Violation of the Platysma

Seat Belt Sign

- Neurologic Deficit
- Abnormal Pulse
- Bruit
- Significant facial/chest trauma
- Stridor/Signs of airway compromise

Arterial injury unlikely without additional red flags

Torso Trauma

Chest

- Compliant chest walls
- Pulmonary injury
- LESS rib fractures
- LESS aortic injuries
- MORE tracheal-bronchial injuries
- MORE likely tension pneumothorax

Chest

Traumatic asphyxia

Conjunctival hemorrhages

Facial edema

Petechial hemorrhage of upper chest and face

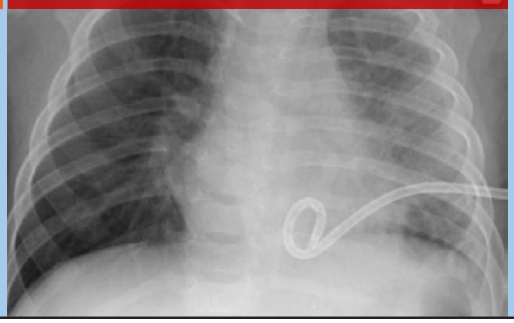
Severe cases→ hemoptysis, exophthalmos, vision loss

PECARN Chest Rule

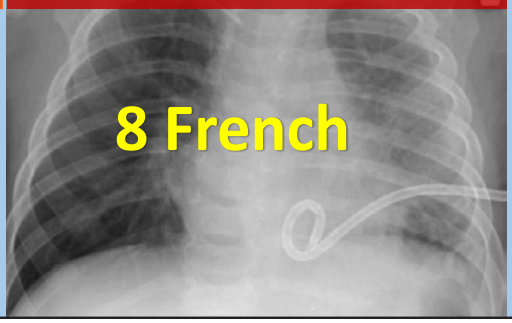
1. Low systolic BP
2. Elevated age-adjusted respiratory rate
3. Abnormal thoracic exam
4. Abnormal auscultation of lung fields
5. Femur fracture
6. GCS < 15

Negative predictive value of 99%

Chest Tube Size?



Chest Tube Size?



Abdominal Trauma

- Abnormal vitals
- Abnormal abdominal exam
- Abnormal labs
- Hematuria

PECARN Abdominal Trauma

1. PE: (-) abdominal wall trauma, seat belt sign
2. GCS>13
3. No abdominal wall tenderness
4. No chest wall/thoracic tenderness
5. No complaints of abdominal pain
6. No decreased bowel sounds
7. No Vomiting

Negative predictive value of 99.9%

Abdomen

Seat belt sign

Increased risk of Injury

Hollow Viscous Injury

(may have negative CT)

Photo courtesy Nathan Kuppermann

Abdomen

(Mostly)

Non-operative management

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http://dx.doi.org/10.1016/j.jemermed.2016.01.003

Ultrasound in Emergency Medicine

CrossMark

PELVIC FREE FLUID IN ASYMPTOMATIC PEDIATRIC BLUNT ABDOMINAL TRAUMA PATIENTS: A CASE SERIES AND REVIEW OF THE LITERATURE

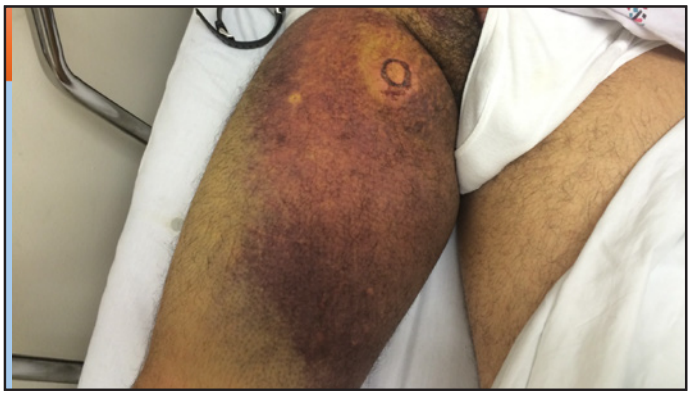
Kristin Berona, MD, Tarina Kang, MD, FACEP, and Emily Rose, MD, FAAP, FAAEM, FACEP

Keck School of Medicine of the University of Southern California, Department of Emergency Medicine, Los Angeles County + USC Medical Center, Los Angeles, California

Reprint Address: Kristin Berona, MD, Keck School of Medicine of the University of Southern California, Department of Emergency Medicine, Los Angeles County + USC Medical Center, 1200 North State Street, Room 1011, Los Angeles, 90033, CA

TXA?

Current suggested dosing is **a loading dose of 15mg/kg tranexamic acid (max 1g) over 10 minutes followed by 2mg/kg per hour over 8 hours.** There is expert opinion that the trauma resuscitation be guided by Thromboelastogram (TEG) to identify candidates with thrombolysis (Elevated LY30) that may yield maximum benefit from TXA infusion.



Motor Scooter Handlebar

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http://dx.doi.org/10.1016/j.jemermed.2015.12.005

Visual Diagnosis in Emergency Medicine

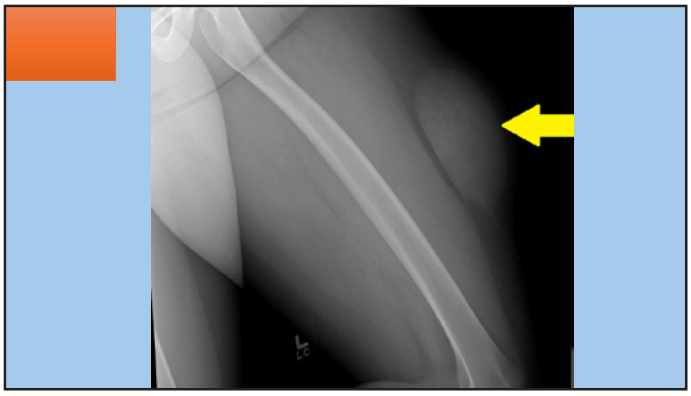
CrossMark

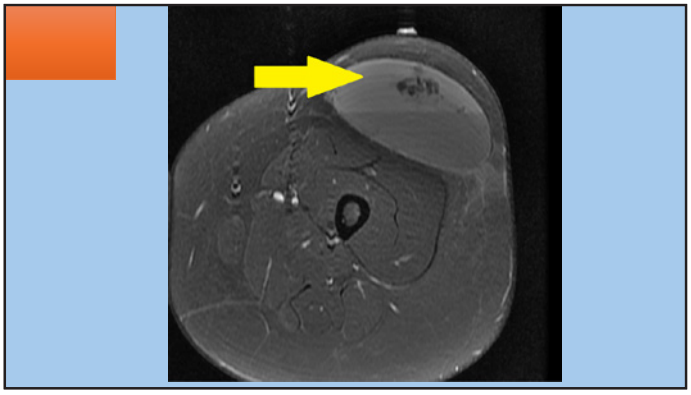
THE MOTOR-SCOOTER HANDLEBAR SYNDROME: RIGHT COMMON FEMORAL ARTERY OCCLUSION SECONDARY TO BLUNT TRAUMA

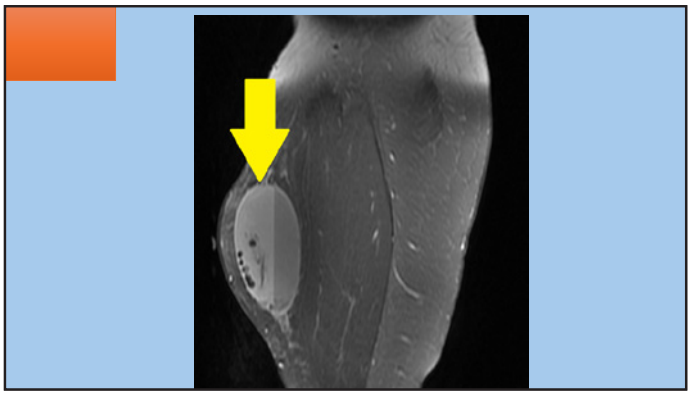
Emily Rose, MD, FACEP, FAAP and Madhu Hardasmalani, MD

Department of Clinical Emergency Medicine, Keck School of Medicine of the University of Southern California, Los Angeles County + USC Medical Center, Los Angeles, California

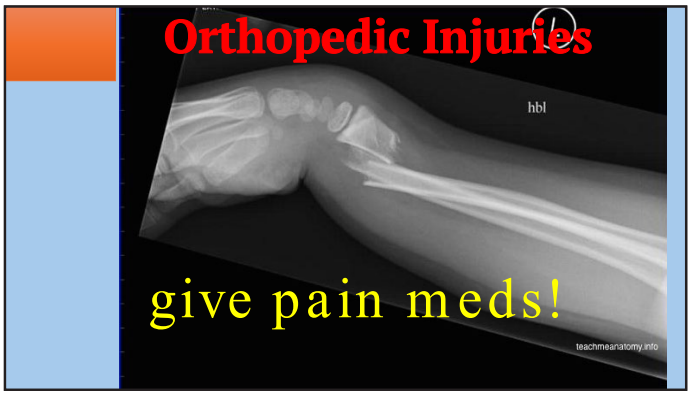
Reprint Address: Emily Rose, MD, FACEP, FAAP, Clinical Emergency Medicine, Keck School of Medicine of the University of Southern California, Los Angeles County + USC Medical Center, 1200 North State Street, Los Angeles, CA 90033

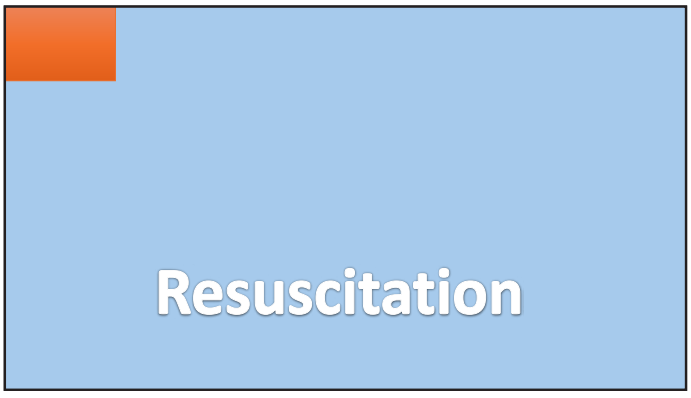


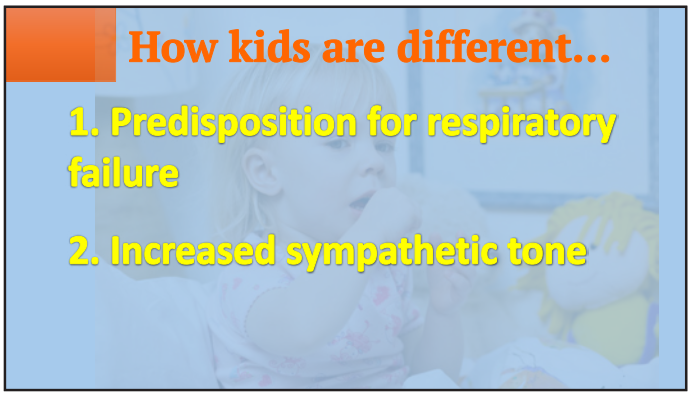


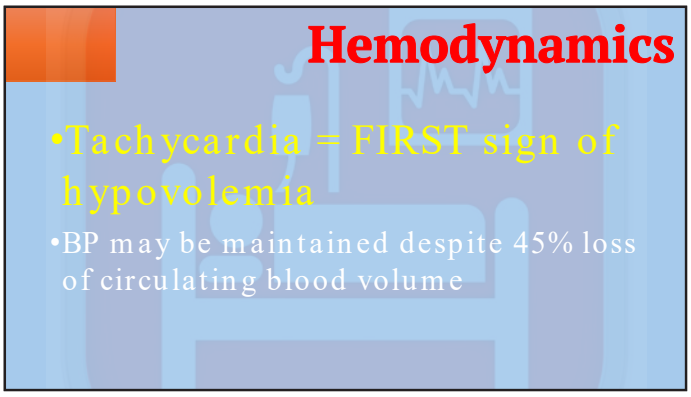



Moral Lavalee Lesion
(closed degloving injury)


Orthopedic Injuries
give pain meds!
teachmeanatomy.info


Resuscitation

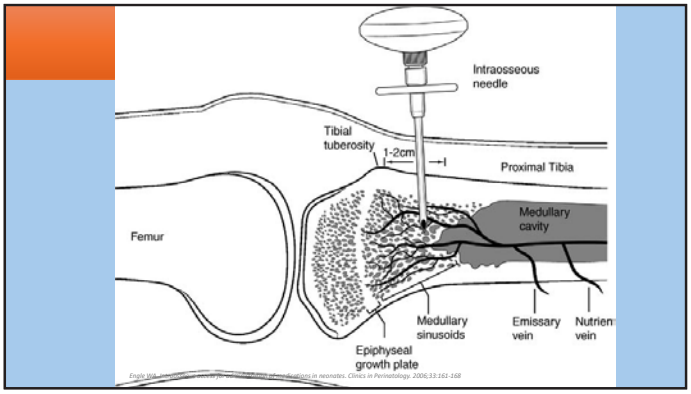

How kids are different…
1. Predisposition for respiratory failure
2. Increased sympathetic tone


Hemodynamics
•Tachycardia = FIRST sign of hypovolemia
•BP may be maintained despite 45% loss of circulating blood volume

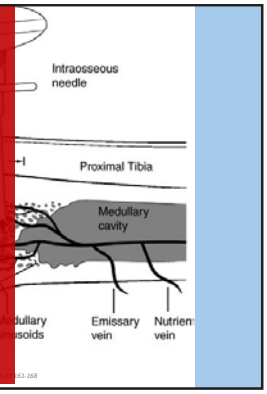
Resuscitation

20 mL/kg crystalloid

10-20 mL/kg PRBCs



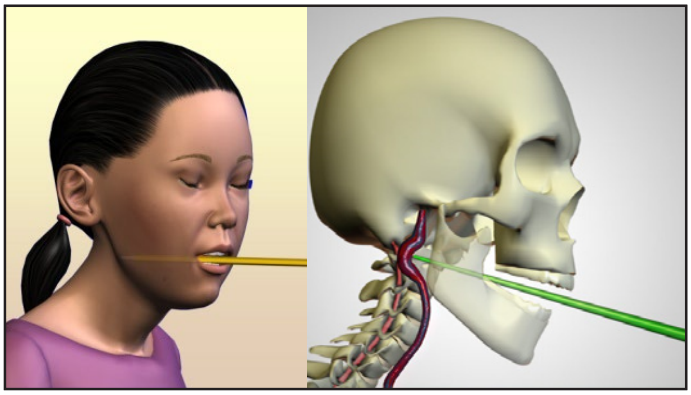
- Proximal tibia
- Distal tibia
- Distal femur
- Proximal humerus
- Sternum (age >12 yr)
- Anterior iliac spine
- Distal radius
- Clavicle
- Os calcis



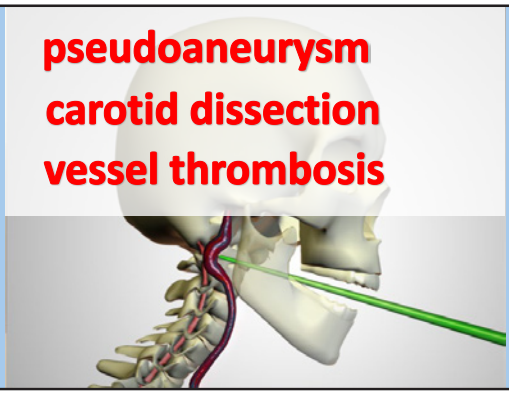
Pediatric Trauma

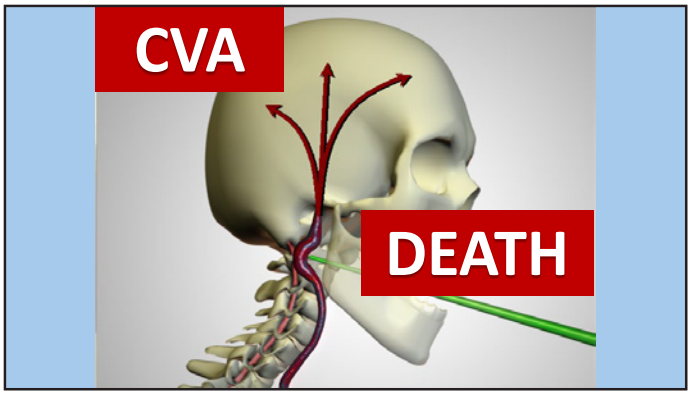
- Your odds of saving a life are best with kids
- Maintain the ALARA concept
- Don't rely on BP to clue you into hemorrhage

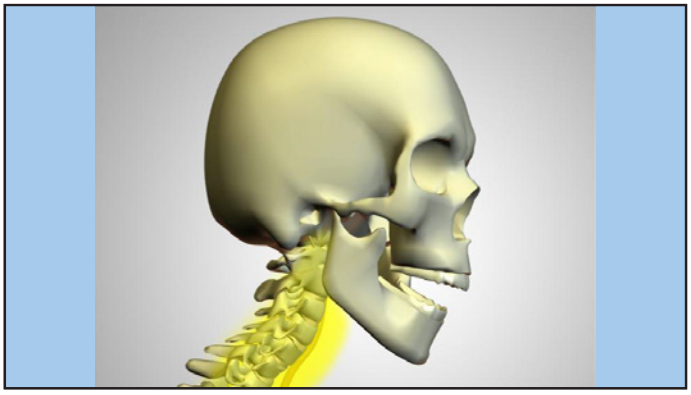
Posterior Palatal Trauma

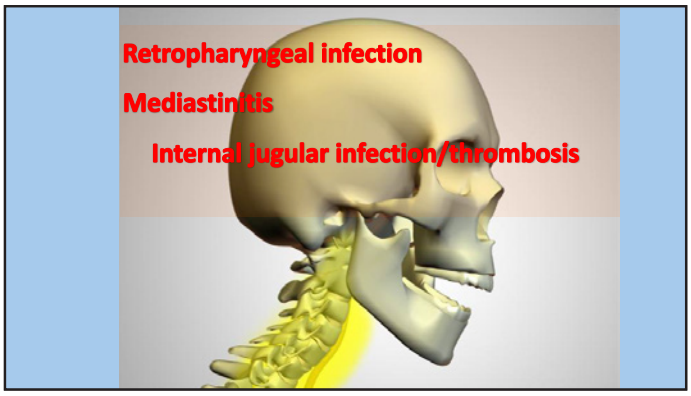


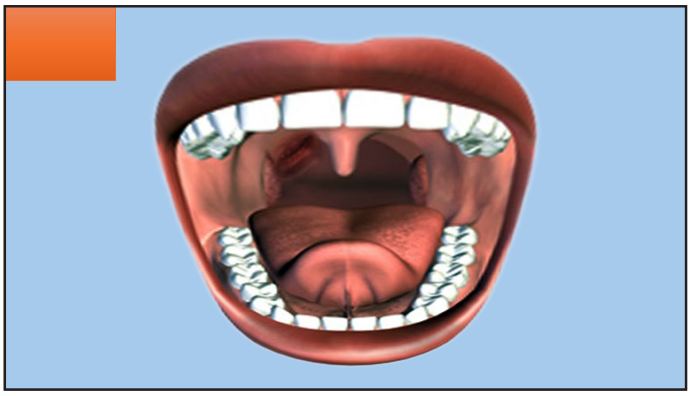
- pseudoaneurysm
- carotid dissection
- vessel thrombosis

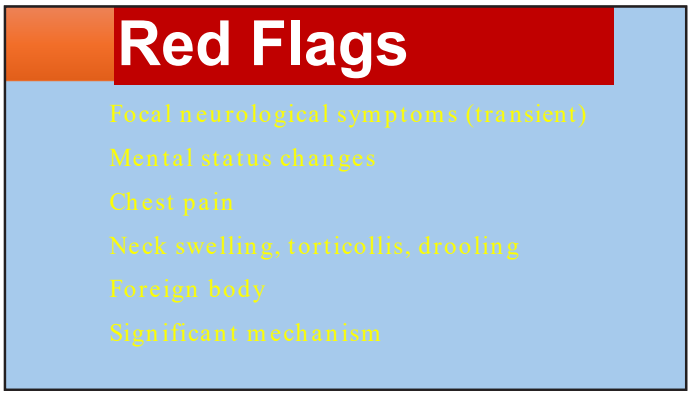


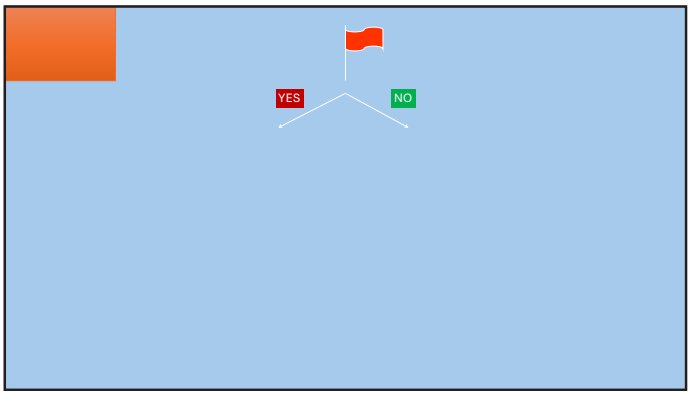

CVA
DEATH

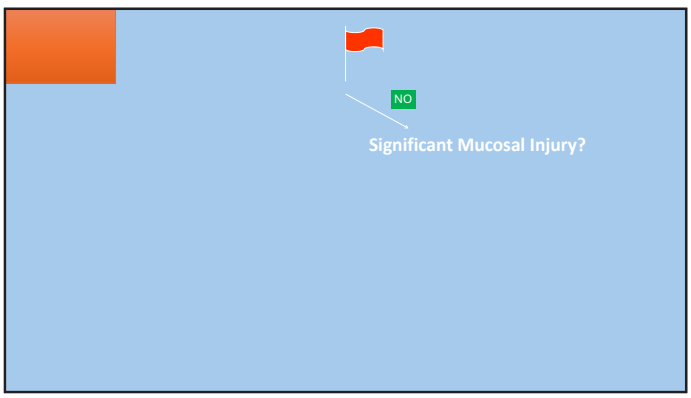


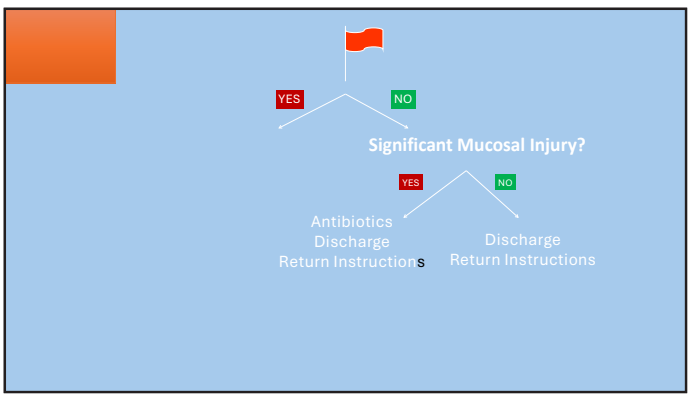

Retropharyngeal infection
Mediastinitis
Internal jugular infection/thrombosis

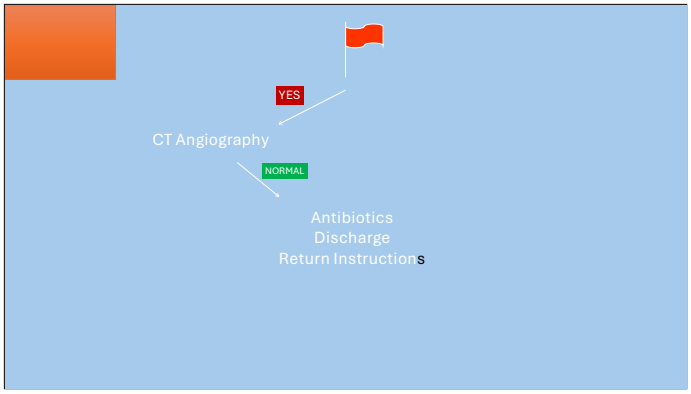


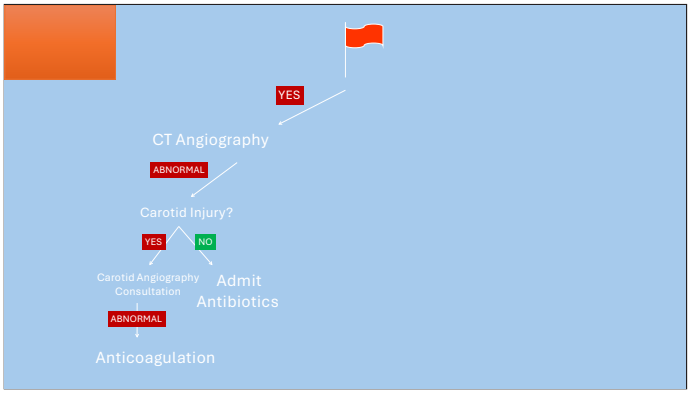

Red Flags
Focal neurological symptoms (transient)
Mental status changes
Chest pain
Neck swelling, torticollis, drooling
Foreign body
Significant mechanism

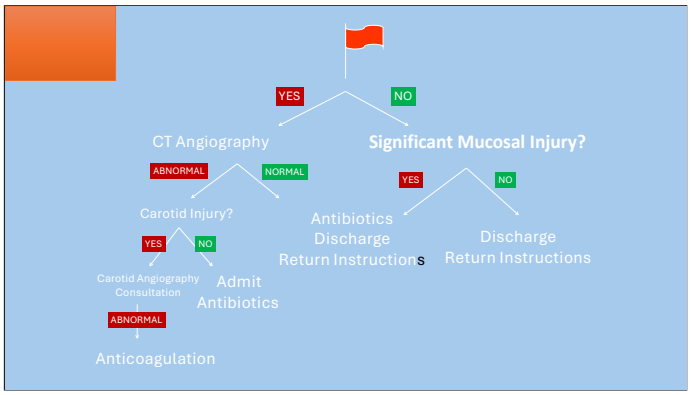

YES
NO

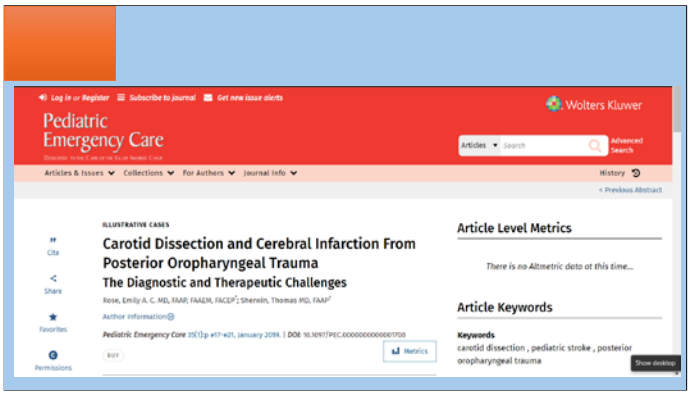

NO
Significant Mucosal Injury?


YES
NO
Significant Mucosal Injury?
YES
NO
Antibiotics
Discharge
Return Instructions
Discharge
Return Instructions









Thank you!

emilyros@usc.edu
@EmilyRoseMD1

SELF EVALUATION

Critical Concepts in Pediatric Trauma: Imaging, Intervention, and Risk Reduction

1. Which of the following is the most common cause of traumatic death in children?

a. Abdominal trauma
b. Head injury
c. Thoracic trauma
d. Extremity fractures

2. Which imaging modality is most appropriate as the first-line study in a hemodynamically stable child with blunt abdominal trauma, abdominal tenderness and a seat belt sign?

a. Diagnostic peritoneal lavage (DPL)
b. FAST (Focused Assessment with Sonography for Trauma)
c. Abdominal X-ray
d. CT scan of the abdomen with IV contrast

3. What is the primary goal of the PECARN head injury decision rule?

a. Identify all skull fractures
b. Determine which patients require hospitalization
c. Reduce unnecessary CT imaging in pediatric head trauma
d. Guide management of spinal cord injuries

4. Which of the following is considered a high-risk mechanism of injury according to PECARN?

a. Fall from bicycle while wearing a helmet
b. Fall from ≥5 feet in children ≥2 years
c. Tripping and hitting the head on carpeted floor
d. Running into a wall at home

5. A 3-year-old fell off a couch (2 feet) and briefly cried but did not lose consciousness, vomit, or show signs of altered mental status. On exam: GCS 15, no scalp hematoma, normal behavior. According to PECARN, what is the appropriate next step?

a. Obtain immediate head CT
b. Observe for 6 hours in ED
c. Discharge without imaging
d. Admit for overnight observation

6. In infants <2 years, which of the following is considered a significant predictor of ciTBI per PECARN?

a. Scalp hematoma located on the frontal region
b. Acting normally according to parents
c. Palpable skull fracture
d. Isolated vomiting without other findings

7. Which imaging modality is most sensitive for detecting cervical spine injury in children?

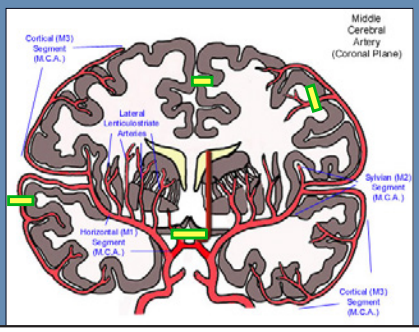
a. Plain radiography
b. CT scan
c. MRI
d. Ultrasound

Answer Key: 1. B, 2. D, 3. C, 4. B, 5. C, 6. C, 7. C

Atypical Presentations of Acute Stroke: Recognizing Chameleons and Mimics

Jonathan A. Edlow, MD

Atypical Stroke Presentations



	Typical presentation	Atypical presentation
True stroke	STROKE	STROKE CHAMELEON
Not a stroke	STROKE MIMIC	NON-STROKE

	Typical presentation	Atypical presentation
True stroke	STROKE	STROKE CHAMELEON
Not a stroke	STROKE MIMIC	NON-STROKE

How Common Are Stroke Mimics?

- Most studies report 25-40%
- In one study of patients hospitalized with a stroke ED diagnosis, 719/1861 (~40%) had discharge diagnoses of stroke mimics

What Happens if I Thrombolyse a Patient Who Has Stroke Mimic?

CT positive CT negative

CT Positive Stroke Mimics

- ICH
  - SDH & EDH
  - IPH
  - SAH
- Tumor
- Abscess

CT Negative Stroke Mimics

- **Migraine**
- **Todd's paralysis**
- **Conversion syndrome**
- Todd's (post-seizure) paralysis
- Metabolic disorders (sodium, glucose, drug toxicity)
- Bell's palsy, vestibular disorders
- Encephalopathy (hypertensive, Wernicke's)

Safety of Intravenous Thrombolysis in Stroke Mimics
Prospective 5-Year Study and Comprehensive Meta-Analysis

Georgios Tsivgoulis, MD; Ramin Zand, MD; Aristeidis H. Katsanos, MD; Nitin Goyal, MD; Ken Uchino, MD; Jason Chang, MD; Efthimios Dardiotis, MD; Jukka Putaala, MD; Anne W. Alexandrov, PhD; Marc D. Malkoff, MD; Andrei V. Alexandrov, MD

2015

- Of 516 consecutive patients at a tertiary center given IV t-PA, 75 (14.5%) were stroke mimics:
  - 1 patient (1.3%) had a sICH
  - 0 had oro-lingual edema or non-CNS bleed
- Of the pooled analysis of 8942 t-PA treated patients, patients, 392 (4.4%) were mimics:
  - sICH rate was 0.5%
  - Orolingual edema was 0.3%

Review article

Ischemic stroke mimics: A comprehensive review

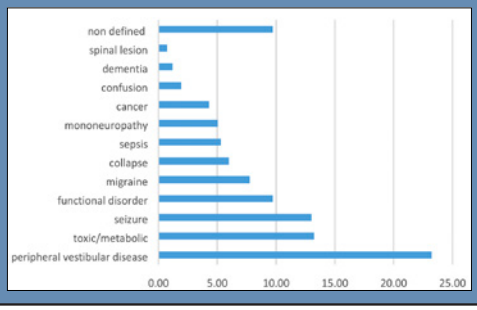
Marietta Pohl[a], David Hesszenberger[b], Krisztian Kapus[a], Janos Meszaros[a], Andrea Feher[c], Imre Varadi[d], Gabriella Pusch[d], Eva Fejes[e], Antal Tibold[a], Gergely Feher[a,f,*]

[a] Centre for Occupational Medicine, Medical School, University of Pécs, Pécs, Hungary
[b] Department of Laboratory Medicine, Medical School, University of Pécs
[c] Szent Rafael Hospital, Zalaegerszeg, Hungary
[d] Department of Neurology, Medical School, University of Pécs
[e] Hospital of Komlo, Komlo, Hungary
[f] Neurology Outpatient Clinic, ÚJ-MED KFT, Komlò, Hungary

2021

- Review of 61 studies (n = 62,664 patients)
- 25% were stroke mimics
- Approximately 4000 of the stroke mimic patients were treated with IV thrombolysis

Etiology of Stroke Mimics



What Happened to Those 4000?

- 26% of the mimics were treated with IV thrombolysis ≈ 4000 patients so it happens
- ICH rate was 0.7%

Stroke Mimic Predictors

TABLE 3. Logistic Regression Model for Predicting the Diagnosis of Brain Attack

Variable	OR	95% CIs
Known cognitive impairment	0.33	(0.14–0.76)
An exact onset could be determined	2.59	(1.30–5.15)
Definite history of focal neurological symptoms	7.21	(2.48–20.93)
Any abnormal vascular findings*	2.54	(1.28–5.07)
Abnormal findings in any other system†	0.44	(0.23–0.85)
NIHSS=0‡		
NIHSS 1–4	1.92	(0.70–5.23)
NIHSS 5–10	3.14	(1.03–9.65)
NIHSS >10	7.23	(2.18–24.05)
The signs could be lateralized to the left or right side of the brain	2.03	(0.92–4.46)
OCSP classification was possible	5.09	(2.42–10.70)

Stroke Mimics in tPA-Treated Patients

Safety of Intravenous Thrombolysis in Stroke Mimics
Prospective 5-Year Study and Comprehensive Meta-Analysis

Georgios Tsivgoulis, MD; Ramin Zand, MD; Aristeidis H. Katsanos, MD; Nitin Goyal, MD; Ken Uchino, MD; Jason Chang, MD; Efthimios Dardiotis, MD; Jukka Putaala, MD; Anne W. Alexandrov, PhD; Marc D. Malkoff, MD; Andrei V. Alexandrov, MD

2015 - Stroke

Meta-analysis of 9 studies of IV tPA treated patients (N=8941)

Of these, 392 patients ended up having a final diagnosis of a stroke mimic (4.4%)
Most common causes: conversion reaction, seizure, complex migraine

Of these 392 stroke mimic patients:

- **0.5% incidence of sICH**
- **0.3% incidence of orolingual edema**

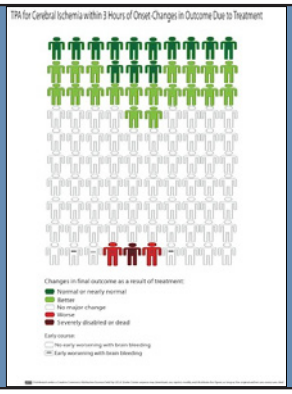
sICH After Thombolysing a Stroke Mimic Patient

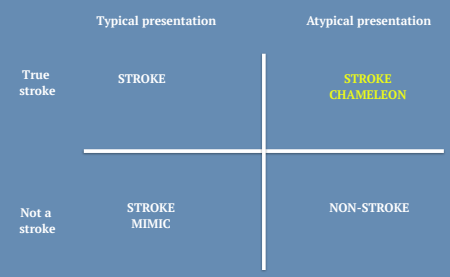
- Older data from the early thrombolysis studies of MI
  - GUSTO trial of 41,000 patients - ICH rate 0.49-0.93% (all patients received heparin + SK or tPA)

The risk of an ICH in a stroke mimic patient treated with IV thrombolysis is very low

Medico-Legal Implications

- The ***overwhelming majority*** of lawsuits related to IV thrombolysis and stroke relate to failure to administer the lytic drug
  - Given that the very low ICH risk treating a CT negative mimic, and that nearly all suits are due to not giving the drug, it's usually both medico-legally and medically safer to treat in uncertain situations.
  - Shared decision-making





Atypical Stroke Presentations

- Non-localizing symptoms
- Abnormal movement or seizures
- Symptoms suggesting a mono-neuropathy
- Atypical central symptoms
- Isolated headache
- Neurological symptoms with normal brain imaging

Non-Localizing Symptoms

- Neuropsychiatric symptoms
- Acute confusional state
- Altered level of consciousness

Neuropsychiatric Symptoms

- Pseudobulbar affect (laughter, crying, anger) due to disruption of pathways from frontal lobe to lower centers
- Thalamic stroke
  - excessive crying
  - agitation
  - aggression
  - apathy

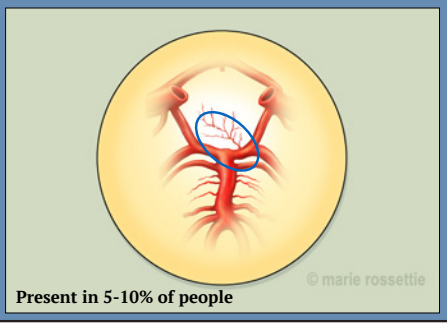
Acute Confusional State

- Transient global amnesia (stroke mimic)
- Acute confusion from stroke
  - Thalamic strokes
  - Inferior division of MCA

Altered Level of Consciousness

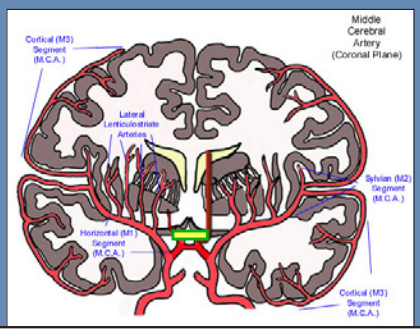
- Thalamic stroke - artery of Percheron
- Inferior division of the MCA (especially left side)
- Brainstem stroke, especially from basilar artery clot, that affects the ARAS

Artery of Percheron

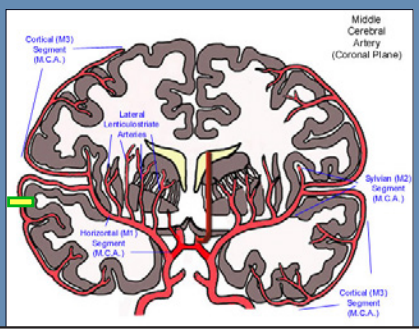


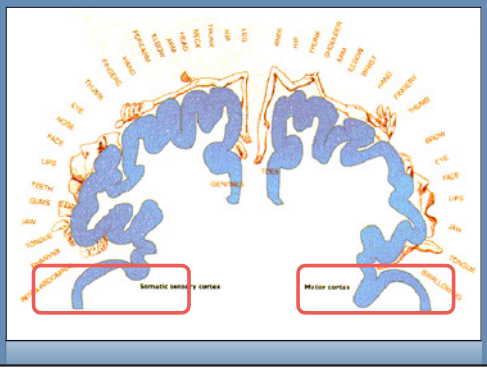
Present in 5-10% of people

Bilateral Thalamic Stroke



Inferior Division of MCA

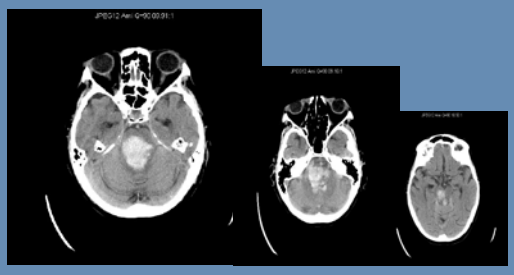




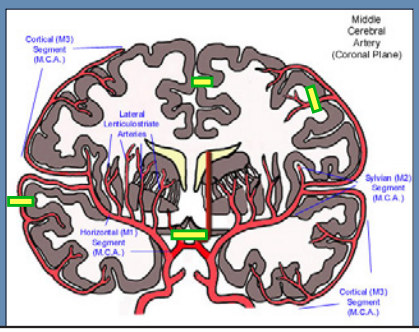
Inferior Division of MCA

- Left side
  - Right homonymous hemianopsia
  - Wernicke's aphasia (fluent, non-sensical speech with poor comprehension)
- Right side
  - Behavioral symptoms
  - Visual-spacial neglect

Large Brainstem Hemorrhage Affecting the ARAS



Atypical Stroke Presentations



KEY CONCEPT:

Distinguish Negative From Positive Symptoms

Negative or Positive?

	Negative symptom	Positive symptom
Motor	weakness	involuntary movements (shaking or seizure)
Sensory	numbness or anesthesia	tingling, dysesthesias
Visual	blindness	flashes of light, zig-zag patterns, hallucinations
Speech	decreased speech output	speaking non-sensical words

Positive Speech Symptoms

KEY CONCEPT:

Negative = Ischemic/Infarction
Positive = Migraine/Seizure

Abnormal Movement or Seizures

- Abnormal movements
- Limb-shaking TIA
- Seizures
- Alien hand syndrome
- Localized asterixis
- Isolated hemifacial spasms
- Disappearance of prior essential tremor

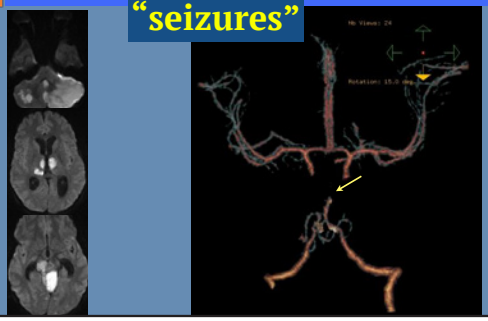
Seizure At Onset

- Initially used as a contraindication to tPA to eliminate treating patients with Todd's paralysis
- If one is fairly sure that the seizure is a result of an acute ischemic stroke (not a Todd's phenomenon), then seizure at onset is NOT a contra-indication
- Would have neurology involved in this decision

Abnormal Movements

- Occasionally will see unilateral hemiballismus from a thalamic small vessel stroke
- This is to be distinguished from the "alien hand" syndrome, also characterized by involuntary movements, but the patient perceives them as being controlled by some external force, usually from lesions in the corpus callosum, frontal or parietal lobes

Altered Mental Status, Vomiting, "seizures"



Symptoms Suggesting a Mono-Neuropathy

- Cranial neuropathy
- Peripheral neuropathy
- Mono-paresis

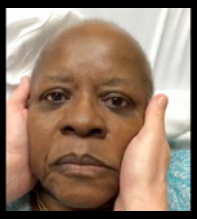
Symptoms Suggesting a 8th Cranial Neuropathy

- Bell's palsy
- Acute vestibular syndrome & acute audio-vestibular syndrome
- Third nerve palsy

Acute Vestibular (And Audio-Vestibular) Syndrome

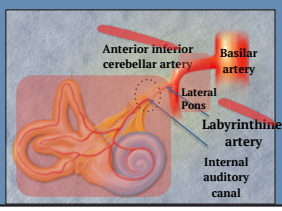
- Major differential diagnosis is stroke versus 8th nerve inflammation (vestibular neuritis)
- The HINTS (and HINTS +) battery of tests (if performed correctly) will reliably distinguish stroke from neuritis
- Simultaneous balance and hearing deficit ≠ labyrinthitis.

Positive head impulse test



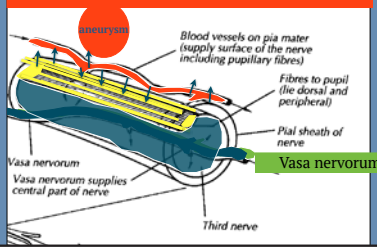
- 25% of patients with acute "audio-vestibular" loss have strokes
- 55% are unaware of the hearing loss

Test the hearing in every dizzy patient!!



Symptoms Suggesting a 3rd Cranial Neuropathy

Posterior communicating artery

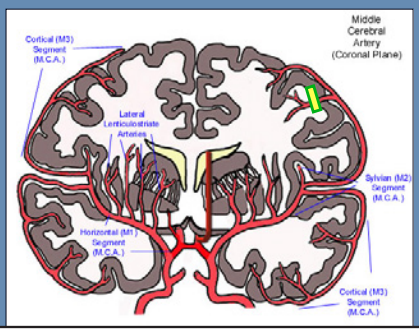


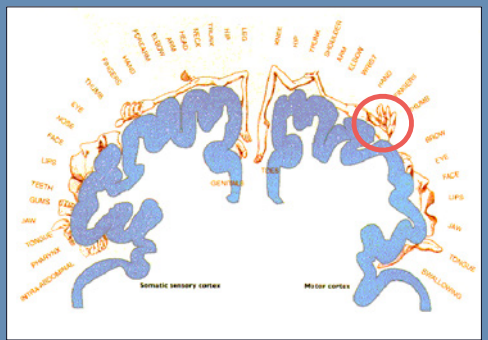
Vasa nervorum

Mono-Paresis and Isolated Sensory Symptoms

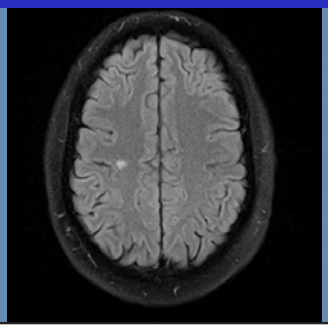
- Cortical hand syndrome
- Cortical leg syndrome
- Isolated sensory loss

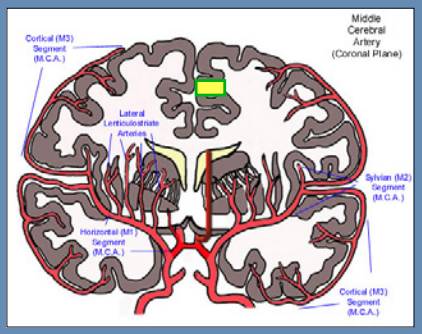
Cortical Hand Syndrome

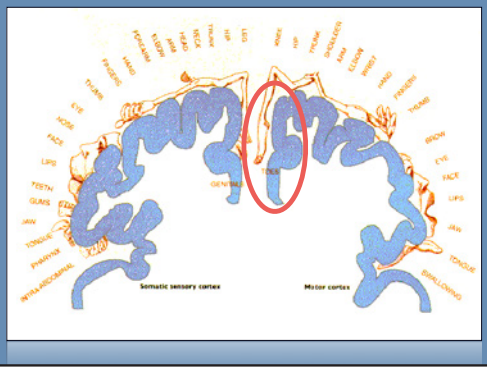




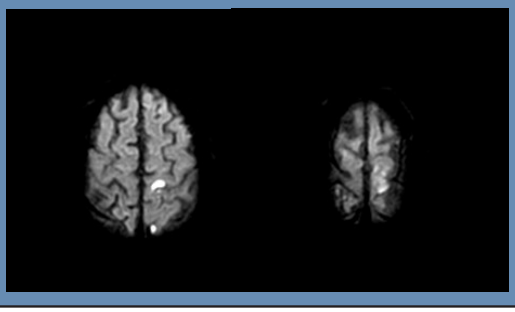
Cortical Hand Syndrome



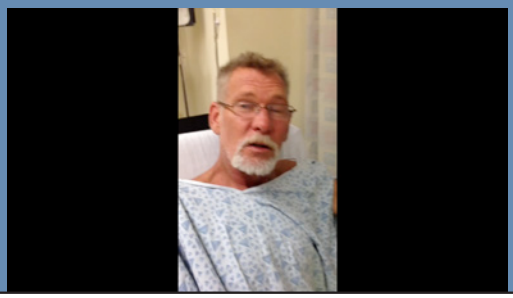




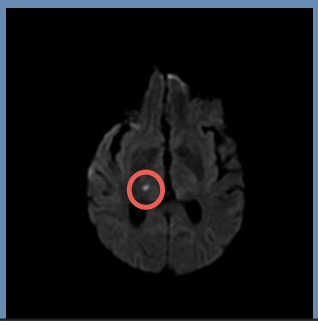
Cortical Leg (ACA) Syndrome



Isolated Sensory Symptoms



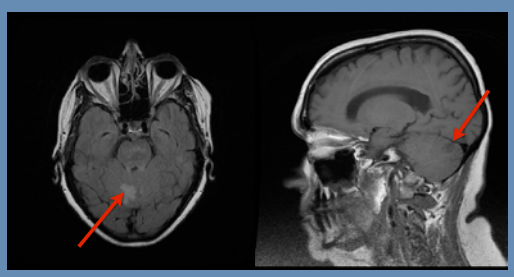
Thalamic Stroke



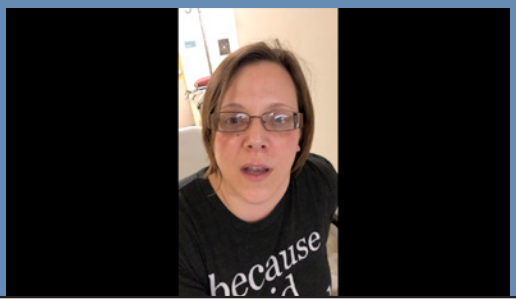
Atypical Central Symptoms

- Isolated dysarthria
- Isolated visual symptoms
- Foreign accent syndrome
- Isolated dysphagia
- Limb ataxia

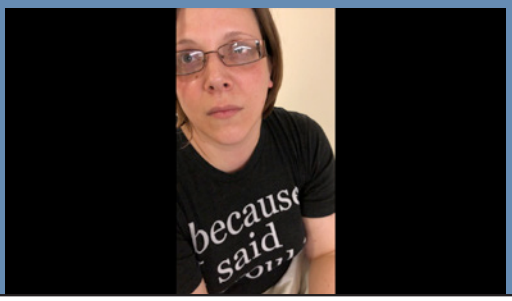
Dysarthria (SCA ischemic stroke)



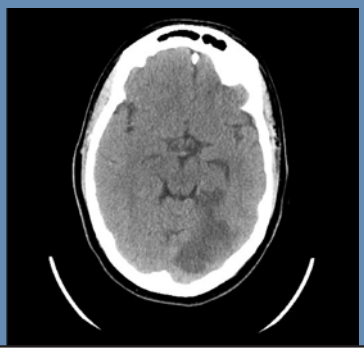
Visual symptoms



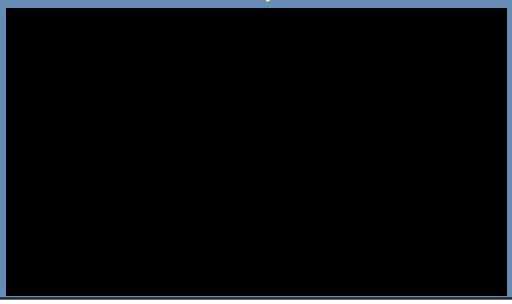
Visual symptoms

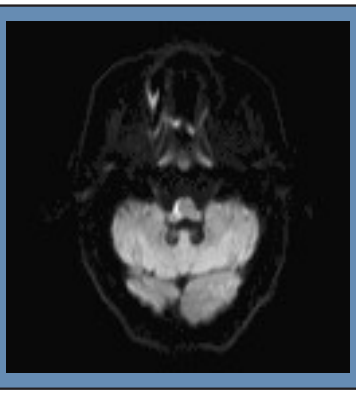


Left Occipital Stroke



Dysphagia & Dysarthria In Lateral Medullary Stroke

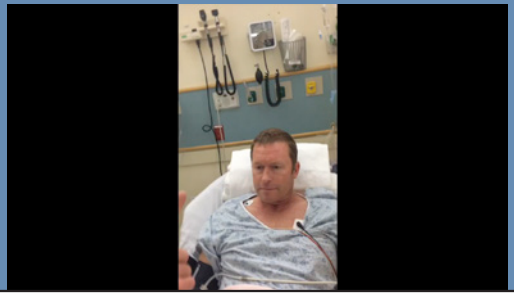




Dysphagia - Lateral Medullary Infarct

Symptom	**Frequency** (approximate)	Anatomic structure
Ataxia, dizziness, (nausea/vomiting)	90%	Cerebellar peduncle, Vestibular nucleus
Dysphagia, hoarseness, occ. dysarthria	60-75%	Nucleus ambiguous (center for CN 9, 10, 11)
Horner's syndrome	75%	Descending sympathetic pathway
Contralateral hemi-hypesthesia (pain/temp)	90%	Lateral spinothalamic tract
Ipsilateral facial hypesthesia (pain/temp)	40-50%	Spinal trigeminal nucleus

Limb Ataxia

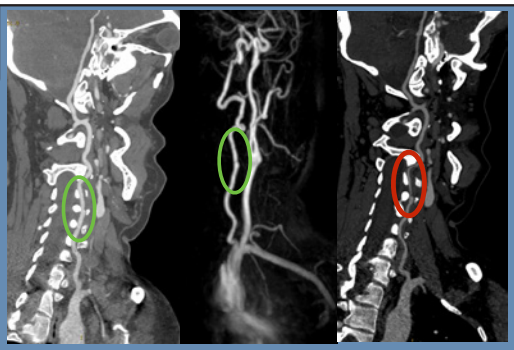


Isolated Headache

- Cranio-cervical artery dissections
- Cerebral venous sinus thrombosis (CVST)
- Subarachnoid hemorrhage (SAH)

Neurological Symptoms With Normal Brain Imaging

- No test is perfect
- Know the limitations of brain CT, MRI and vascular imaging



CTA 5-17 MRA 5-18 CTA 5-21

Tips To Avoid Misdiagnosis

- Were the neurological ***symptoms abrupt in onset***?
- Know that "***atypical" stroke presentations*** are not that uncommon
- Do a ***systematic neurological examination*** - including
  - Language evaluation
  - Visual field testing
  - Gait assessment
- Remember that ***young people have strokes*** too!

SELF EVALUATION

Atypical Presentations of Acute Stroke: Recognizing Chameleons and Mimics

True/False

1. An important clue of a stroke is the gradual onset of symptoms.

2. Stroke mimics are rare.

3. Patients with stroke mimics treated with an intravenous thrombolytic drug have a high incidence of the complication of intracranial bleeding.

4. Two cerebral arteries that are involved in acute ischemic stroke that present with acute onset of confusion are the artery of Percheron and the inferior branch of the middle cerebral artery.

5. "Negative" neurological symptoms (the absence of a function, such as a visual field loss) far more commonly indicate a stroke compared to a "positive" neurological symptom (the accentuation of a function, such as flashed of light).

6. The cortical hand syndrome is usually seen in a stroke that only involves weakness of the hand or even some individual fingers.

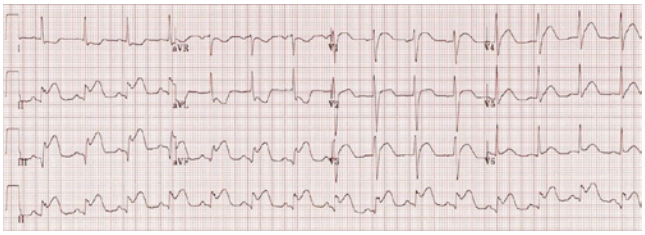
Answer Key: 1. F, 2. F, 3. F, 4. T, 5. T, 6. T

Beyond ACS: Recognizing Other Life-Threatening Causes of Chest Pain

Robert Tubbs, MD

- Discuss the presentation of potentially lethal causes of chest pain
- Discuss the approach to diagnosing these disorders including imaging modalities

Prehospital EKG

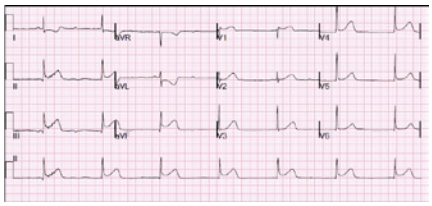


- Cardiology waiting in the ED
- Patient receives aspirin and clopidogrel
- Heparin started
- Portable CXR performed
- Patient rushed to cath lab

Total time in ED =
15 minutes

Differential DDx

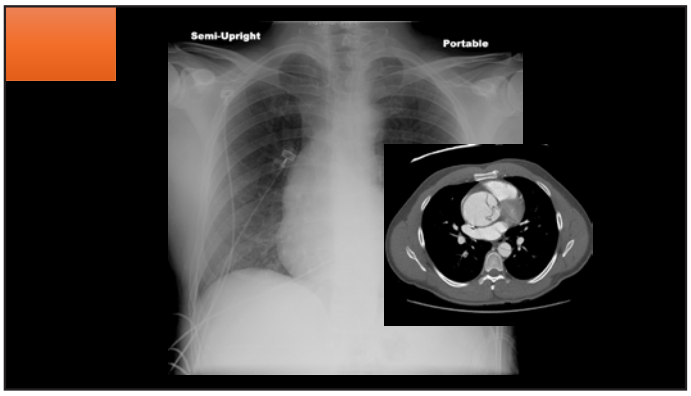
1. STEMI
2. STEMI
3. STEMI



Or is it???

Call comes from the cath lab:

"Did this patient get a CXR in the ED?"



Aortic Dissection

Highest mortality of the life-threatening causes of chest pain

<u>1-2%</u> per **HOUR** for first 48 hours!

This is an easy diagnosis, right?

Aortic Dissection

Diagnostic delays
> 24 hours
occur in up to 39% of admitted patients

Actor John Ritter

So how *DO* we diagnose it?

JAMA. 2000 Feb 16;283(7):897-903.

The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease.

Hagan PG[1], Nienaber CA, Isselbacher EM, Bruckman D, Karavite DJ, Russman PL, Evangelista A, Fattori R, Suzuki T, Oh JK, Moore AG, Malouf JF, Pape LA, Gaus C, Sechtem U, Lanfranchi S, Deutsch HJ, Diedrichs H, Marcos y Robles J, Llovet A, Gilon D, Das SK, Armstrong WF, Deeb GM, Eagle KA.

- Largest database of aortic dissection cases
- 30 centers in 11 countries
- Established in 1996
- 464 patients in original publication in 2000
- Currently >3800 patients in the database

IRAD

Aortic Dissection – IRADS Data

- **85%** reported *abrupt* onset
- **73%** chest pain
- **53%** back pain
- Tearing or ripping pain 50%
- 49% hypertensive
- 15% had pulse deficit

Michael DeBakey MD

Don't rule out with D dimer

A number of studies looking at this, but numbers are small, they are all observational, and methodologies vary significantly

Sensitivities and specificities vary widely

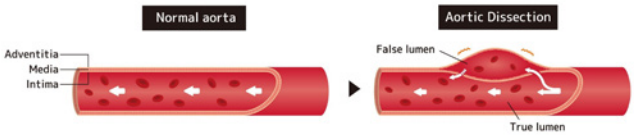
Aortic Dissection: Plain Film Findings

Mediastinal widening:

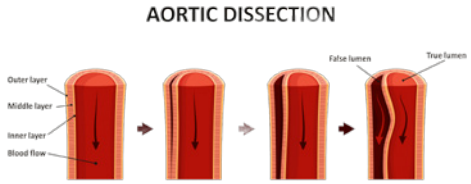
Type A = 63%

Type B = 56%

What *DOESN'T* Happen



What *REALLY* Happens

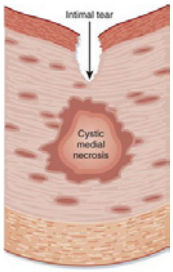


Acute Aortic Dissection is an INTRA-Luminal Event

Cystic Medial Necrosis

With age, vascular risk factors such as hypertension, connective tissue disorders, the media starts to degenerate and the aorta widens out

The intima becomes prone to tearing from the media



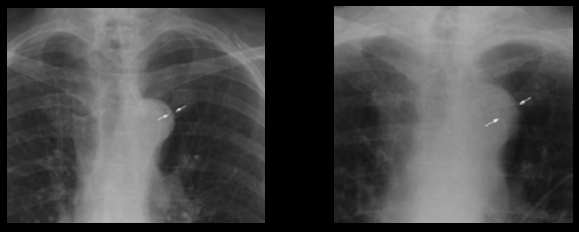
Aortic Dissection: Other X-ray Findings

Pleural effusion = **19%**

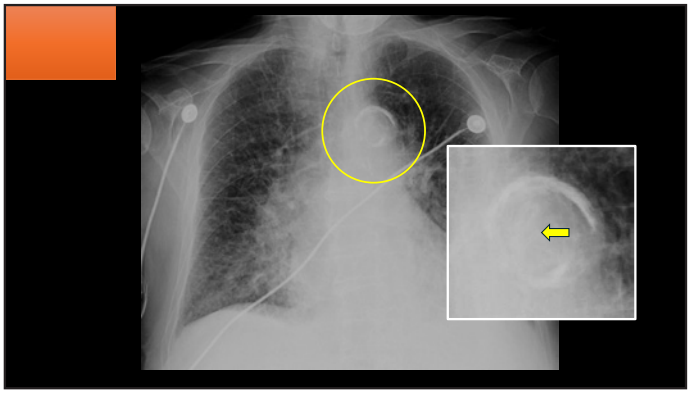
Other findings:

- Widening of the aortic contour
- Displaced calcification – "Calcium sign"
- Loss of AP window

The Calcium Sign



Normal Displaced



11-15%
have normal CXR's!!

Transesophageal Echocardiogram

Transesophageal Echocardiogram

Reported sensitivities:
97-99%

However:
Limited availability
Operator dependent
Invasive

CTA:
The Gold Standard

What do you get when you order a dissection study?

You actually get 2 scans!

#1: A *non-con* chest and limited abdomen

Why would we do a non-contrast CT when we're looking for a vascular issue?

Intramural Hematoma

Blood dissecting in the media *without* a dissection flap

Often shows as a hyperdensity in the wall

Contrast can obscure it

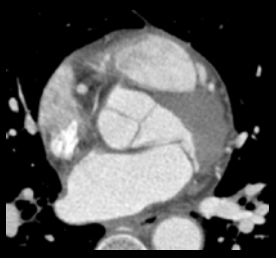
#2: A full CTA of chest and abdomen

Allows evaluation of the full extent of the dissection and organ involvement

Watch for fake-outs!

Motion at the aortic root is common, and can be mistaken for a dissection flap

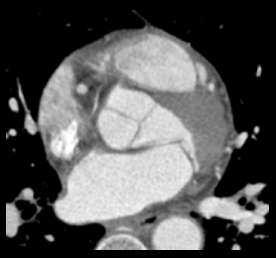
ECG-Gated CTA



Patient is attached to the CT scanner with ECG leads

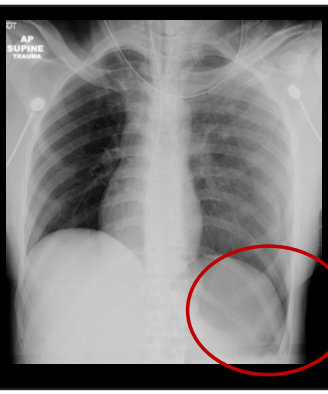
Scanner acquires images at the same point in the cardiac cycle, then stacks them together

ECG-Gated CTA



Essentially eliminates motion artifact

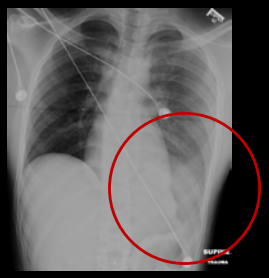
Can't be used in patients with arrhythmias

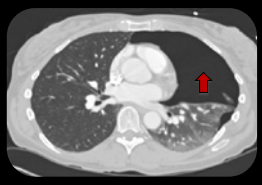


Know how the X-ray was taken!!

Understanding how the patient is positioned is key!

The "Deep Sulcus" Sign





Air rises ***anteriorly*** in a supine patient

CXR for PTX:

Just how good are the options?

Sensitivity of CXR for PTX

Lateral Decubitus = **88%**

Upright CXR = **59%**

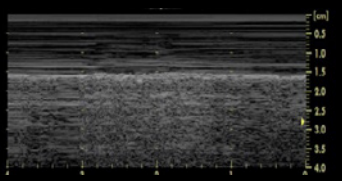
Supine AP = **37%**

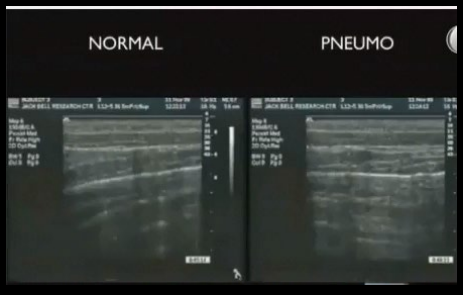
What about Ultrasound?

Lung Ultrasound

Sensitivities of 79-100%*

*With experienced providers





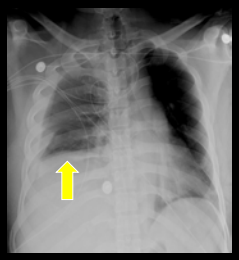
When in doubt…

CT:
The Gold Standard

Patient positioning is key to interpretation!

Blood layers posteriorly when supine!

Appearances of hemothorax

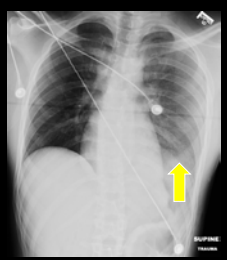


Upright

In an *upright* patient, the effusion will create a horizontal fluid level

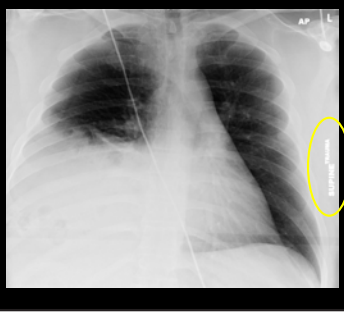
Appearances of hemothorax

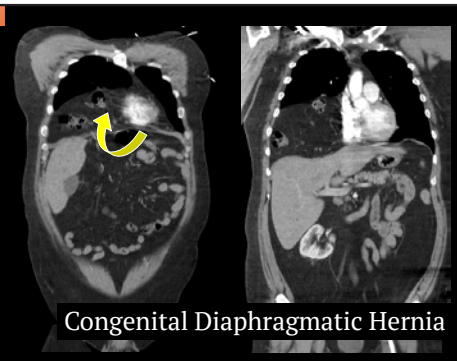
In a *supine* patient when the blood layers posteriorly, aerated lung lies on top and creates a hazy appearance to the lung compared to the other side



Supine

Back to our guy…





Congenital Diaphragmatic Hernia

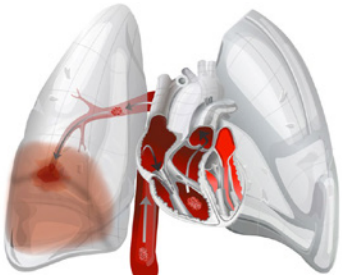
Case #3:
72 yo female with chest pain

72 y.o. with history of uterine cancer now with pleuritic chest pain, SOB, and near syncope

ECG

"Sinus tachycardia, incomplete RBBB"

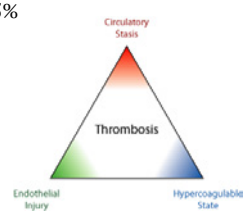
Pulmonary Embolism



How do we diagnose it?

Risk Factors for PE

- Active Malignancy →25-30%
- Previous DVT or PE especially within 3 months → 12-20%
- Immobility (Bed Rest > 5 days) → 15-55%
- Recent Surgery → 15% -50%
- Family history of PE →5%
- Estrogen Therapy (OCP, other) → 2-3%
- Known Hypercoagulable State → 3-4%
- Sickle Cell Disease → 1%



Risk Factors for PE

20-50% of patients do not have a known risk factor at time of diagnosis

Signs and Symptoms… Helpful?

- Tachypnea:
  - present in **54-73%** of PE cases
  - present in 43-68% of non PE cases
- Tachycardia
  - present in **24-30%** of PE cases
  - present in 14-24% of non PE cases
- Combination Features
  - Dyspnea or Tachypnea or Pleuritic CP was present in 97% of cases with PE (**97% sensitive**)
  - Present in 93% of cases **without** PE

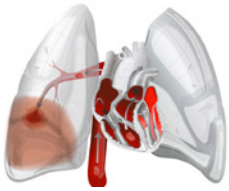
So, risk factors may not be helpful.

Signs and symptoms also not always helpful

What are we supposed to do?

Pulmonary Embolism Rule-Out Criteria (PERC)

- Age ≥ 50
- HR > 100
- O_2 sat on RA <95%
- Prior history of DVT/PE
- Recent trauma or surgery
- Hemoptysis
- Exogenous estrogen
- Unilateral leg swelling



Kline *Thrombosis and Hemostasis 2008*

PERC Rule

- If **NO** criteria are positive and the *pre-test probability is low*, the PERC rule is satisfied
- No further work-up needed as < 2% chance of the patient having a PE

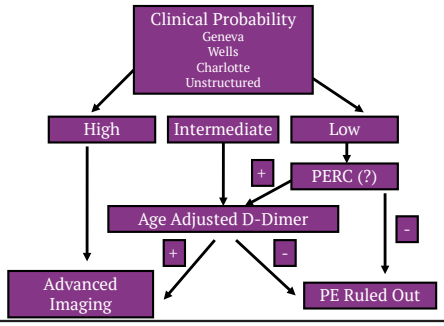
Conditions in which the PERC rule cannot be used
Concurrent β-blocker use (may blunt tachycardia)
Transient tachycardia
Thrombophilia
Strong family history of thrombosis
Patients with amputations
Massively obese patients (may be difficult to clinical assess leg swelling)
Patients with baseline hypoxemia

Original Investigation

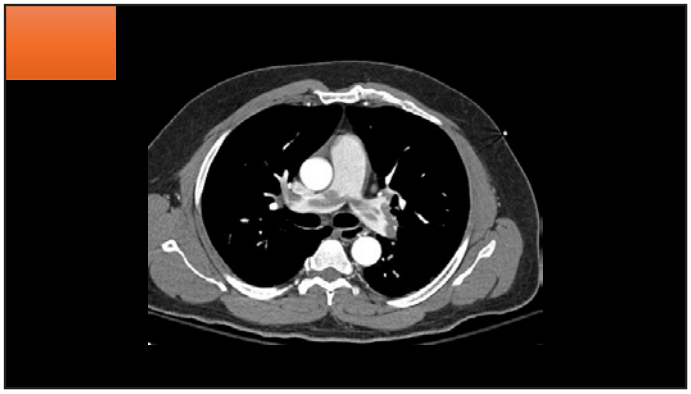
Age-Adjusted D-Dimer Cutoff Levels to Rule Out Pulmonary Embolism
The ADJUST-PE Study

- D-Dimer Rises with Age making 500 (or 250) cut point often useless
- Study of 2900 patients > 50 years old
- Normal D-Dimer was 10 x Age (in FEU) or 5 x Age in DDU
- Could reduce CT use from 72% to 60% (w/o any misses)
- Even true in the > age 75 subgroup!!!

PE Diagnostic Strategy

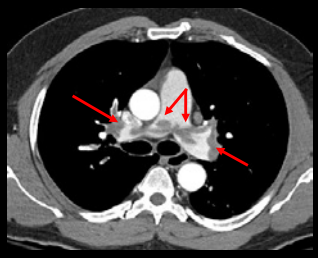


Back to our patient…



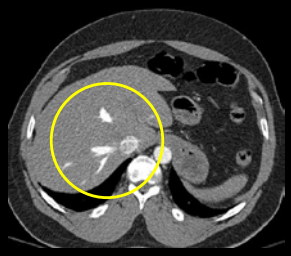
Yikes!!!

Saddle Embolus



Large filling defects crossing over and extending into bilateral main pulmonary arteries

Look for signs of right heart strain



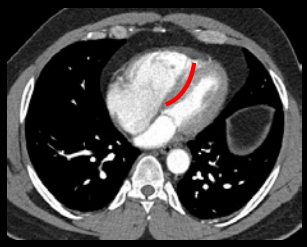
Reflux of contrast into the IVC and liver

Not specific for PE

Look for signs of right heart strain

Septal bowing

CT may overestimate this



Look for signs of right heart strain

The Right Ventricular Diameter (RVD) : Left Ventricular Diameter (LVD) Ratio

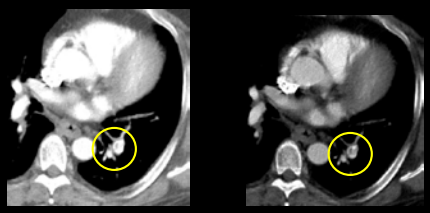
RVD:LVD ratio > 1 predictive of right heart strain

Time is critical!!

Majority of deaths in PE occur in the first **1-2 hours** of treatment

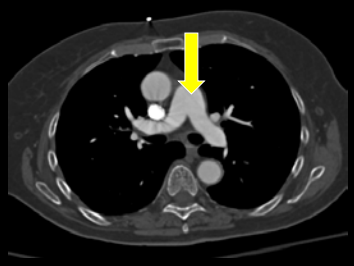
How do I read these things?

First thing is to drop the brightness and contrast



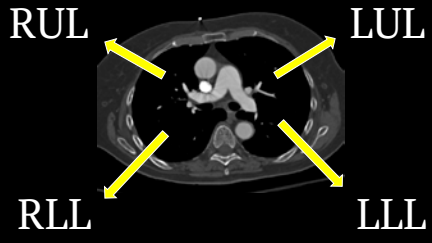
Brighter is not always better

Next step is to start here

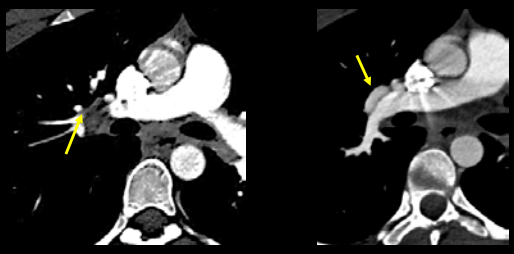


It takes 4 passes

Trace out each pulmonary artery



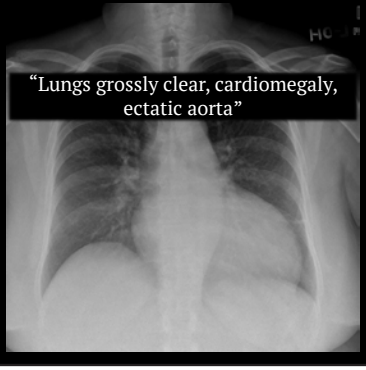
Then trace back to make sure the defect you're seeing is not in a pulmonary vein



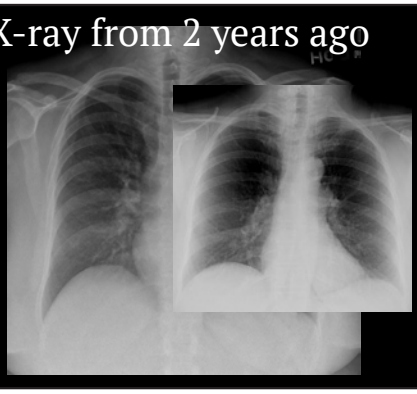
Physical Exam

HR: **120**
RR: **22**
BP: **95/53**
SaO_2: 97% RA

Lungs: CTA
Heart: **Distant heart sounds**



X-ray from 2 years ago



Pericardial effusions

Be suspicious…

With new (or possibly new) cardiomegaly

Patients with SOB and clear lungs

Etiologies of Pericardial Effusions

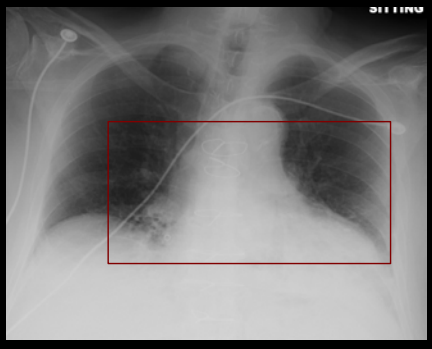
- Acute pericarditis
- Post MI
- Trauma
- Aortic Dissection
- Malignancy
- Uremia
- Hypothyroidism
- Drugs
- Autoimmune disease

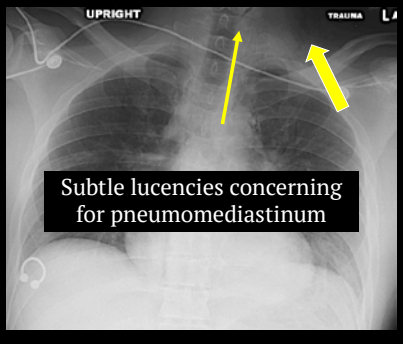
Echocardiography

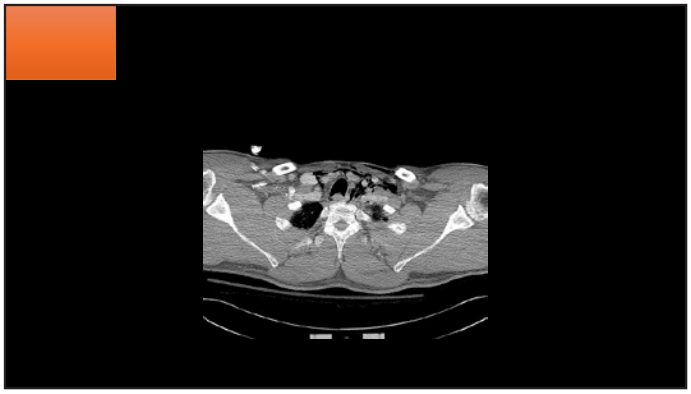
- Study of choice for evaluating pericardial disease
- Gives both anatomic and physiologic / hemodynamic data

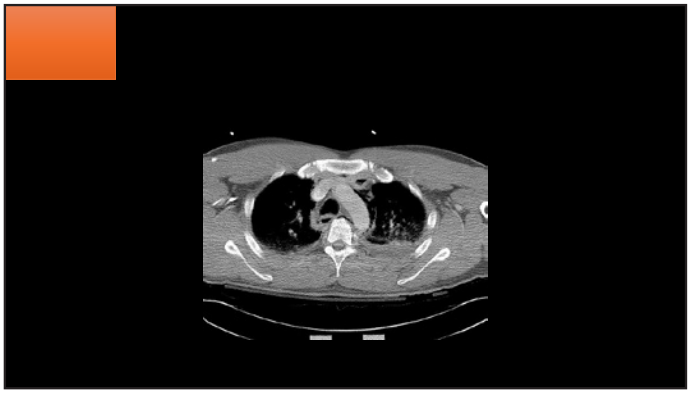
Case #5:

45 yo male with chest pain after vomiting

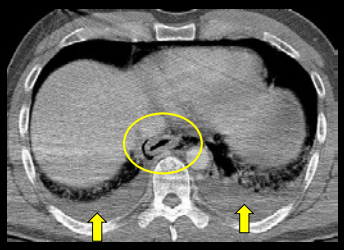








Esophageal Perforation



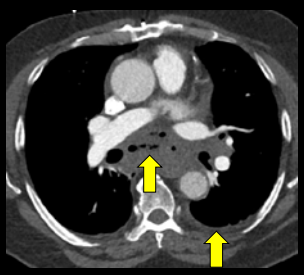
Extra-luminal air

Often see pleural effusions (left is more common)

Esophageal Perforation

Pneumomediastinum

Small pleural effusions

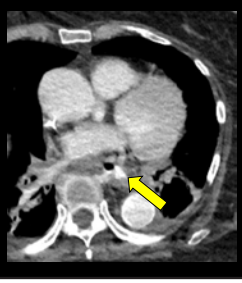


CT Esophageal Perforation Protocol

Has replaced fluoroscopic swallow studies in many institutions

Patient drinks oral contrast on the table

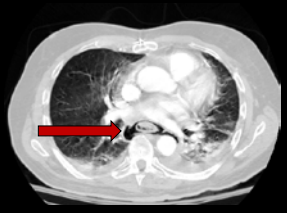
Looking for extravasation of contrast



Emesis + Pneumomediastinum

= Boerhaave's Syndrome!!

Boerhaave's Syndrome



Watch for pneumomediastinum with a pleural effusion!!

Boerhaave's Syndrome

Up to **40%** of cases related to ETOH intoxication!

FINAL THOUGHTS

- Remember all the life-threatening causes of chest pain
- If you don't consider the diagnosis, you will miss it!
- Use your clinical decision rules when appropriate
- Use labs and imaging judiciously, and know their limitations

SELF EVALUATION

Beyond ACS: Recognizing Other Life-Threatening Causes of Chest Pain

1. Widened mediastinum is present in which percentage of chest radiographs in cases of Ty pe A aortic dissection?

a. 20%
b. 45%
c. 63%
d. 85%

2. Each of the following is a component of the Pulmonary Embolism Rule-out Criteria (PERC) Rule EXCEPT:

a. Age >50
b. Heart rate >100
c. Unilateral leg swelling
d. History of cancer
e. Exogenous hormone use

3. CT findings of right heart strain include each of the following EXCEPT:

a. Reflux of contrast into the IVC
b. Dilated left ventricle
c. Bowing of the interventricular septum
d. RV:LV ratio of >1

4. T/F - Cases of effort-related esophageal rupture, aka Boerhaave's Syndrome, are most often associated with Left sided pleural effusions.

5. Alcohol intoxication is present in up to which percentage of cases of effort-related esophageal rupture (aka Boerhaave's Syndrome)?

a. 10%
b. 20%
c. 40%
d. 75%
e. 90%

Answer Key: 1. C, 2. D, 3. B, 4. T, 5. C

Brian Fuehrlein, MD, PhD

Brian Fuehrlein, MD, PhD, of New Haven, Connecticut, graduated from the M.D. Ph.D. program at the University of Florida in 2008, adult psychiatry residency program at the University of Texas Southwestern Medical Center in 2012 and addiction psychiatry fellowship at Yale University in 2013. He is currently an Associate Professor and the director of the psychiatric emergency room at the VA Connecticut. Dr. Fuehrlein has a strong interest in medical student and resident education, particularly surrounding addiction psychiatry and serves on multiple local and national committees in this role. In 2017 he was awarded the Irma Bland award for excellency in psychiatry resident education through the APA. In 2018 he was awarded the Clerkship Faculty Teaching Award for Outstanding Medical Student Educator and Role Model. He is also passionate about emergency psychiatry and substance use disorders and has presented and published his work surrounding opioid use disorder in the emergency room setting. In 2019 he was inducted into the American College of Psychiatrists, an organization that recognizes excellence in clinical practice, research, academic leadership, or teaching.

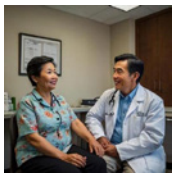
You may contact Dr. Fuehrlein with your comments or questions at brian.fuehrlein@yale.edu.

Recognizing and Managing Alcohol Withdrawal

Brian Fuehrlein, MD, PhD

Clinical Presentation

- Sun presents to the ED chest pain
- She is cleared medically
- She reports drinking "1-2 drinks" before bed, a few nights each week
- Her husband reports that she drinks nearly a fifth of alcohol every night



Standard Drinks

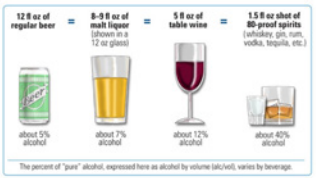
- **12 oz regular beer (5% alcohol)**
  - Light beer contains slightly less alcohol (4.2%)
  - Malt beverages contain approximately 7% alcohol
- **5 oz of table wine (12% alcohol)**
- **1.5 oz of 80 proof spirits (40% alcohol)**
- **14 grams of alcohol**

Remember 60 as an easy way to figure out drink size and percent

Common Terms

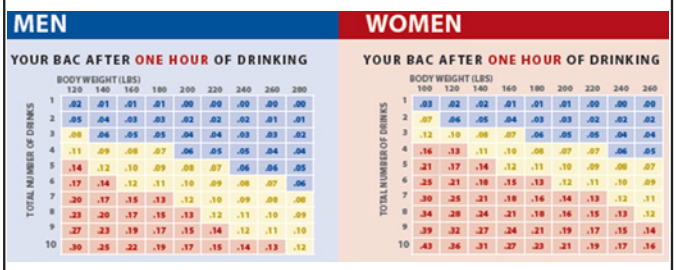
- Nip = common airplane bottle = 50 ml = 1.7 oz = slightly more than 1 standard drink
- Pint = less than a real pint = 375 ml = 12.7 oz = 8.5 standard drinks
- Fifth = fifth of a gallon = 750 ml = 25.4 oz = 17 standard drinks
- Handle = approximately half gallon = 1.75 L = 59 oz = 39 standard drinks

Standard drink of alcohol



National Institute on Alcohol Abuse and Alcoholism (NIAAA). What's a standard drink measurement? Rethinking Drinking | NIAAA. Rethinking Drinking.
Accessed June 4, 2024https://rethinkingdrinking.niaaa.nih.gov/how -much-too-much/whats-standard-drink

Impact on BAL

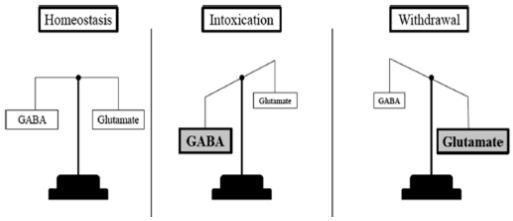


BAL 0.10 = 0.10 grams of alcohol per 100ml blood

Alcohol Metabolism

- On average, one standard drink is metabolized per hour (0.02)
- Hence a BAL of 0.30 will take approximately 15 hours to metabolize to zero
- People with tolerance start to develop withdrawal approximately 6 hours from last drink
- Withdrawal will develop long before the BAL reaches 0

Simplified neurobiology of alcohol intoxication and withdrawal

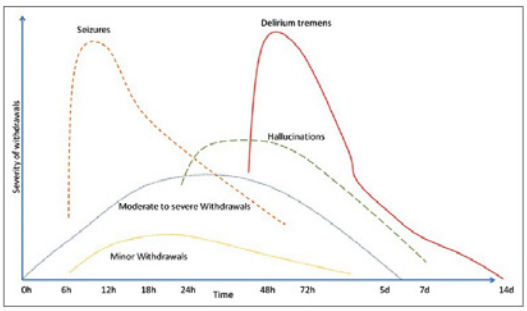


Cohen S, Alexander R, Holt S. The spectrum of alcohol use: epidemiology, diagnosis, and treatment. 2022 *Med Clin North Am* 2022; 106(1):43-60. doi:10.1016/j.mcna.2021.08.003

Syndrome	Time after last drink	Symptoms/Characteristics
Stage 1: Initial withdrawal symptoms	6-8 hours	-Tachycardia -Anxiety -Hypertension -Tremulousness -Hyperthermia -Nausea/Vomiting -Diaphoresis -Headache -Insomnia
Stage 2: Alcoholic hallucinosis	12-24 hours	-7 to 8% of patients with AWS -Most commonly visual -Normal sensorium differentiates from delirium tremens
Stage 3: Withdrawal seizures	12-48 hours	-Generalized tonic-clonic -Shorter duration with little post-ictal period -1/3 of patients with withdrawal seizures progress to delirium tremens
Stage 4: Delirium tremens (DT)	3 to 5 days after initial withdrawal symptoms; lasting up to 8 days	-Rapid onset -Fluctuating disturbance of attention/cognition + alcohol withdrawal symptoms and autonomic instability -3-5% of patients hospitalized with AWS with a 1-4% mortality

Yaroy, J. Alcohol Withdrawal Syndrome: Identification and Management. *EM.Docs*, online publication, accessed 7/31/24 at emDOCs.net – Emergency Medicine EducationAlcohol Withdrawal Syndrome: Identification and Management - emDOCs.net - Emergency Medicine Education

Time course for alcohol withdrawal



Haber NL, et al.*Guidelines for the Treatment of Alcohol Problems*.1989.

Who is at risk for alcohol withdrawal?

- Alcohol consumption of 4+ drinks daily for > 4 weeks
- Pattern of binge drinking (4+ drinks in 1 sitting) > 3 times a week
- Recent symptoms of alcohol withdrawal

Clinical Institute Withdrawal Assessment for Alcohol – revised (CIWA-Ar) scale

Clinical Institute Withdrawal Assessment for Alcohol revised	
Symptoms	**Range of scores**
Nausea or vomiting	0 (no nausea, no vomiting) –7 (constant nausea and/or vomiting)
Tremor	0 (no tremor) – 7 (severe tremors, even with arms not extended)
Paroxysmal sweats	0 (no sweat visible) – 7 (drenching sweats)
Anxiety	0 (no anxiety, at ease) – 7 (acute panic states)
Agitation	0 (normal activity) – 7 (constantly trashes about)
Tactile disturbances	0 (none) – 7 (continuous hallucinations)
Auditory disturbances	0 (not present) – 7 (continuous hallucinations)
Visual disturbances	0 (not present) – 7 (continuous hallucinations)
Headache	0 (not present) – 7 (extremely severe)
Orientation/clouding of sensorium	0 (orientated, can do serial additions) – 4 (Disorientated for place and/or person)

Complicated alcohol withdrawal

- 5% of people with alcohol withdrawal
- The Prediction of Alcohol Withdrawal Severity Scale (PAWSS)
- History: severe withdrawal (DTs), withdrawal seizures, drinking pattern, symptoms while alcohol level elevated
- Labs: BAL >200 on admission, thrombocytopenia, hypokalemia, BUN > 26
- Older age
- Concurrent acute illness

1. Wood E, Albarouni L, Tkachuk S, et al. Will this hospitalized patient develop severe alcohol withdrawal syndrome? The clinical examination systematic review. *JAMA* 2018;320(8):825-833. doi:10.1001/jama.2018.10574.
2. Maldonado JR, Sher Y, Ashouri J, et al.The "prediction of alcohol withdrawal severity scale" (PAWSS): systematic literature review and pilot study of a new scale for the prediction of complicated alcohol withdrawal syndrome. *Alcohol* 2014;48(4):375-390. doi: 10.1016/j.alcohol.2014.01.004.

Assessing Withdrawal Risk

Risk Points on Presentation

1) History of DTs **
2) History of Seizures**
3) History of multiple withdrawals**
4) Quantity of drinking ≥ 20 servings/day**
   OR
   Quantity of drinking ~ 10 servings/day
5) Age > 55
6) Breathalyzer > 200 or CIWA > 8
7) Significant medical Co-morbidity
8) History of withdrawal Hallucinosis

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Prediction of Alcohol Withdrawal Severity Scale (PAWSS)

Maldonado et al, 2015

Part A: Threshold Criteria: ("Y" or "N", no point)

Have you consumed any amount of alcohol (i.e., been drinking) within the last 30 days? OR did the patient have a "+" BAL on admission? _____

IF the answer to either is YES, proceed with test:

Part B: Based on patient interview: (1 point each)

1. Have you been recently intoxicated/drunk, within the last 30 days? _____
2. Have you ever undergone alcohol use disorder rehabilitation treatment or treatment for alcoholism? (i.e., in-patient or out-patient treatment programs or AA attendance) _____
3. Have you ever experienced any previous episodes of alcohol withdrawal, regardless of severity? _____
4. Have you ever experienced blackouts? _____
5. Have you ever experienced alcohol withdrawal seizures? _____
6. Have you ever experienced delirium tremens or DT's? _____
7. Have you combined alcohol with other "downers" like benzodiazepines or barbiturates, during the last 90 days? _____
8. Have you combined alcohol with any other substance of abuse, during the last 90 days? _____

Part C: Based on clinical evidence: (1 point each)

9. Was the patient's blood alcohol level (BAL) on presentation ≥ 200? _____
10. Is there evidence of increased autonomic activity? (e.g., HR > 120 bpm, tremor, sweating, agitation, nausea) _____

Total Score: _____

Notes: Maximum score = 10. This instrument is intended as a SCREENING TOOL. The greater the number of positive findings, the higher the risk for the development of AWS.
A score of ≥ 4 suggests HIGH RISK for moderate to severe (complicated) AWS; prophylaxis and/or treatment may be indicated.

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PAWSS

- Screens for complicated alcohol withdrawal syndrome (AWS)
  - Alcohol withdrawal seizures
  - Alcoholic hallucinosis
  - Delirium Tremens (DTs)
- Standardized method to assess for and document clinical factors associated with development of AWS
- Complement to CIWA - assesses risk independent of current signs and symptoms

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PAWSS Interpretation

- Likelihood ratio based on systematic review of 71296 patients and 1355 cases of severe alcohol withdrawal syndrome
- 93% sensitivity; 99.5% specificity

PAWSS Score	Risk of complicated AWS	Likelihood Ratio
<4	Average	0.07
≥4	High	174

References:
Wood E, Albarqouni L, Tkachuk S, et al. Will This Hospitalized Patient Develop Severe Alcohol Withdrawal Syndrome? The Rational Clinical Examination Systematic Review. JAMA. 2018;320(8):825–833. doi:10.1001/jama.2018.10574
Maldonado, José R et al. "The "Prediction of Alcohol Withdrawal Severity Scale" (PAWSS): systematic literature review and pilot study of a new scale for the prediction of complicated alcohol withdrawal syndrome." Alcohol (Fayetteville, N.Y.) vol. 48,4 (2014): 375-90. doi:10.1016/j.alcohol.2014.01.004

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CIWA + PAWSS

Utilizing both screens is consistent with American Society for Addiction Medicine:

- Incorporate universal screening for unhealthy alcohol use into medical settings
- For patients known to be using alcohol recently, regularly, and heavily, assess their risk of developing alcohol withdrawal *even in the absence of signs and symptoms*
- For patients who have signs and symptoms suggestive of alcohol withdrawal, assess the quantity, frequency, and time of day when alcohol was last consumed to determine whether the patient is experiencing or is at risk for developing alcohol withdrawal

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ASAM Recommendations

- To diagnose alcohol withdrawal, alcohol withdrawal delirium, and alcohol use disorder, use diagnostic criteria such as those provided by the Diagnostic and Statistical Manual 5 (DSM-5)
- Alcohol withdrawal severity assessment scales (including the Clinical Instrument Withdrawal Assessment for Alcohol, Revised [CIWA-Ar]) should not be used as a diagnostic tool because scores can be influenced by conditions other than alcohol withdrawal
- Do not rule in or rule out the presence of alcohol withdrawal for patients who have a positive blood alcohol concentration
- Do not rule out the risk of developing alcohol withdrawal if the result of an alcohol test is negative

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VACT Internal Medicine Alcohol Withdrawal Guideline 5/24/2022

Goals of inpatient "detox"
1.Minimize Symptoms
2.Avoid delirium
3.Avoid pneumonia
4.Avoid seizures
5.If patient agreeable, d/c to structured SA program

Pt admitted for alcohol withdrawal/intoxication†

Note: ASAM recommends STT for most, with front load if at risk for complicated withdrawal, preferably with long-acting benzo (diazepam)

Supportive care:
1.IVF for most
2.Electrolyte and Mag repletion
3.PO PPI if abdominal or chest pain
4.Early social work consult for possible post-discharge programs
5.Consider medications to reduce craving at time of discharge

Low risk patients:

Symptom Triggered Therapy (STT) alone:

1. STT with diazepam**
2. STT with lorazepam
3. Fixed dose Gabapentin

Prior complicated withdrawal or presenting with Sz, high CIWAs

STT with 24hr front-load:

4. STT with one time Valium*
5. STT with 24-hr diazepam front load**

****Preferred regimens**

If current Bz failure only:

For Benzo Failures with agitated delirium:

7.Fixed dose Phenobarbital†

PCSS Providers Clinical Support System 20

Ambulatory Detox from VISN 1

Inclusion criteria

1. No history of withdrawal complications [seizures or DTs]
2. No active psychiatric symptoms affecting safety [e.g., psychosis, mania, dementia, delirium, suicidal or homicidal ideation, intent or plan]
3. No acute or unstable medical problems [e.g., uncontrolled hypertension]
4. Not pregnant
5. No current abuse of or dependence on other substances such as opioids, sedative-hypnotics, stimulants.
6. Agrees and can pursue active follow-up during the detoxification period, which would consist of daily follow up via telephone or VVC
7. Able to tolerate oral medication
   a. Agrees to take detoxification medications as prescribed.
   b. Is not taking medications likely to affect alcohol withdrawal symptoms [e.g., chronic anticonvulsant therapy, beta blocker, clonidine, gabapentin, benzodiazepines]
8. Has a reliable method to access emergency care if needed
   a. Has an identified significant other or individual to serve as resource when needed
   b. Has a home blood pressure cuff or has received one from the VA
9. Understands and accepts potential risks [e.g., seizure]

PCSS Providers Clinical Support System 21

3. The prescribing clinician may elect to use one of three recommended medication protocols for detoxification:
   a. Gabapentin
      i. 400 mg tid po, for three days
      ii. 400 mg bid po, for two days
      iii. 400 mg qd po, for one day
   b. Depakote
      i. 500 mg tid po, for four days
      ii. 250 mg tid po, for one day
      iii. 250 mg bid po, for one day
      iv. 250 mg qd po, for one day
   c. Carbamazepine
      i. 200 mg qid po, for one day
      ii. 200 mg tid po, for one day
      iii. 200 mg bid po, for one day
      iv. 200 mg qd po, for one day
4. Adjunctive medications are prescribed as appropriate
   a. Thiamine, 100 mg po daily
   b. Multivitamins daily
   c. Clonidine, 0.1 mg po q 6 h
5. A follow up schedule will be created with the patient. Daily follow up is the standard of care for ambulatory detoxification from alcohol.
   a. Medication assisted treatment for Alcohol Use Disorders (MAT4AUD) will be offered
6. Veteran will be referred to Alcohol/Substance Use Disorder Treatment concomitant with the ambulatory detoxification procedure. Treatment may include psychosocial and Alcohol Use Disorder pharmacotherapies.

PCSS Clinical Support System 22

Most Importantly

- Detox is not treatment!
- Alcohol withdrawal is an acute complication of a chronic medical condition
- Treating the acute complication does not treat the underlying condition

PCSS Providers Clinical Support System 23

SELF EVALUATION

Recognizing and Managing Alcohol Withdrawal

1. What will be the approximate blood alcohol level for a 180 pound man after consuming 2 standard drinks?

a. 0.01
b. 0.03
c. 0.08
d. 0.30

2. T/F - The symptoms of alcohol withdrawal typically begin approximately 48 hours after the last drink.

3. Which of the following is a risk factor for the development of complicated alcohol withdrawal?

a. A prior history of complicated alcohol withdrawal
b. Younger age
c. At least 2 weeks from the last drink of alcohol
d. Legal stressors

4. T/F - PAWSS is a clinical tool for screening for complicated alcohol withdrawal syndrome.

5. T/F - When alcohol withdrawal is managed appropriately there is no need to also manage the alcohol use disorder.

Answer Key: 1. B, 2. F, 3. A, 4. T, 5. F

Pediatric Fever in the Emergency Department

Emily Rose, MD, FAAP, FAAEM, FACEP

Disclosures

No financial relationships with a commercial interest to disclose

Outline

- Resuscitation of the Critically Ill Pearls
- Key Take-Home Points
- Age based approach to fever in kids with updated literature
- Parental Anticipatory Guideline Pearls

Peds Fever Resuscitation Pearls

- 95% of Peds Cardiac Arrest has a Respiratory Etiology
- Hypotension is not required in children with sepsis
- Pediatric shock is more commonly cold shock and best treated with epinephrine
- Consider early ECMO for refractory shock

Fever Key Points

- Neonates and young infants are evaluated and treated differently than older, vaccinated, and clinically evaluable children
- Most fevers in children are self-limited of viral etiology
- Bacterial infection evaluation should occur in:
  - Febrile neonates
  - Risk stratification in young infants
- All ill-appearing and immunocompromised patients should be evaluated for bacterial infection regardless of age

Fever Truths

- Fever inhibits bacterial and viral replication and activates the immune response
- Temperature will not exceed 42°C (107.6°F) in normal patients (rarely >106/41.1)
- A normal immune response fever does not cause brain damage

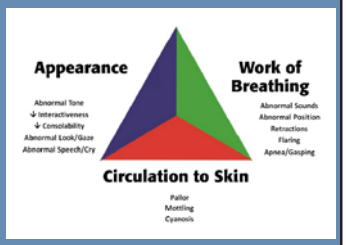
Predicting an invasive bacterial infection?

Age

Appearance

NOT talking about ill appearing…

- Unresponsive, AMS
- Limp, decreased tone
- Weak cry
- Mottled, petechiae
- Poor cap refill
- Tachycardic, bradycardic, hypotensive
- Tachypneic, bradypneic, apneic



Serious Bacterial Illness

- Meningitis
- Bacteremia
- Sepsis
- Bone, joint, significant soft tissue infections
- UTI
- Pneumonia
- Bacterial Enteritis

Serious Bacterial Illness

- Meningitis
- Bacteremia
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- Bone, joint, significant soft tissue infections
- UTI
- Pneumonia
- Bacterial Enteritis

Maybe a term of the past?

Invasive Bacterial Illness (IBI)

4 wk | 4-8 wk | >8 wk

Decreasing risk of SBI

↓ Immunity
↑ SBI
Unreliable exam

↑ Immunity
↓ SBI
Interactive

2025 ?

Viral Infections are the most common

75% of fever without a source

5-6% of neonates determined to be "low-risk" have an SBI

up to 20% overall risk

4 wk | 4-8 wk | >8 wk

Decreasing risk of SBI

↓ Immunity
↑ SBI
Unreliable exam

↑ Immunity
↓ SBI
Interactive

4 wk | 4-8 wk | >8 wk

20% (week 1) | 12% (week 4)

15% | ~5%

Risk of SBI

4 wk | 4-8 wk | >8 wk

5% IBI | 4% IBI | <1% IBI

15% | ~5%

Risk of SBI

IBI=Invasive Bacterial Infection (bacteremia/sepsis, meningitis)

Missed infection= death or permanent neurologic sequelae/disability

Risk Factors for Significant Infection in Neonates

- AGE
- Ill-Appearance
- Potential HSV infection (1st 3 weeks of life)
- Prematurity
- Prior hospitalization/NICU
- Co-morbidities
- Tech dependent
- Recent antibiotics (up to 7 days)
- Focal soft tissue infection (with abnormal labs)

Fever >38.5 deg C is a predictor for increased risk of IBI in young infants

Neonatal Herpes

<50% have rash or (known) risk factors

Herpes until proven otherwise…

Seizures

Sepsis-like illness

Abnormal mental status/lethargy

Conjunctival injection

Mucocutaneous vesicles

CSF pleocytosis

Thrombocytopenia

Elevated transaminases

PEDIATRICS

OFFICIAL JOURNAL OF THE AMERICAN ACADEMY OF PEDIATRICS

From The American Academy Of Pediatrics

Evaluation and Management of Well-Appearing Febrile Infants 8 to 60 Days Old

Robert H. Pantell, Kenneth B. Roberts, William G. Adams, Benard P. Dreyer, Nathan Kuppermann, Sean T. O'Leary, Kymika Okechukwu and Charles R. Woods; Subcommittee On Febrile Infants

Pediatrics July 2021, e2021052228; DOI: https://doi.org/10.1542/peds.2021-052228

Pantell RH, Roberts KB, Adams WG, Dreyer BP, Kuppermann N, O'Leary ST, Okechukwu K, Woods CR Jr; Subcommittee On Febrile Infants. Evaluation and Management of Well-Appearing Febrile Infants 8 to 60 Days Old. Pediatrics. 2021 Jul 19:e2021052228. doi: 10.1542/peds.2021-052228. Epub ahead of print. PMID: 34281996.

Inflammatory Markers

Labs

- CRP < 20 mg/L*
- PCT < 0.5 ng/mL
- ANC > 4000-5200

Clinical

- T > 38.5C

August 2021, Pediatrics Clinical Practice Guideline: Evaluation and Management of Well-Appearing Febrile Infants 8 to 60 Days Old Pantell et al.

Excluded babies

Sick

- Suspected HSV (vesicles)
- Focal bacterial infection
  - Omphalitis
  - Osteomyelitis
- Bronchiolitis (less IBI risk)

Complicated

- Prematurity (<37 weeks)
- Mom had fevers or abx*
- Chromosomal problem/cong anomalies
- s/p surgery or prior infection
- Immune compromised
- Tech dependent
- s/p vaccine w/in 48 hrs

August 2021, Pediatrics Clinical Practice Guideline: Evaluation and Management of Well-Appearing Febrile Infants 8 to 60 Days Old Pantell et al.

8-21 days: Assess HSV Risk

Maternal factors

- maternal history genital HSV lesions
- Maternal fevers from 48 hours before to 48 hours after delivery

Infant Factors

- Infants with vesicles,
- Seizures
- Hypothermia,
- Mucous membrane ulcers
- CSF pleocytosis w/absence of a (+) Gram stain
- Leukopenia
- Thrombocytopenia
- Elevated ALT levels

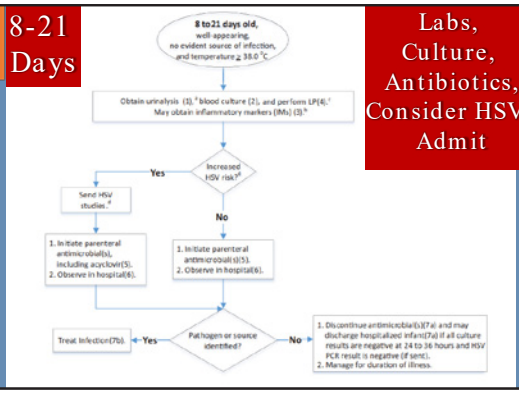
August 2021, Pediatrics Clinical Practice Guideline: Evaluation and Management of Well-Appearing Febrile Infants 8 to 60 Days Old Pantell et al.

Neonates are nearly a different species!

Viruses can be life-threatening

Decreased T cell immunity

8-21 Days



Labs, Culture, Antibiotics, Consider HSV, Admit

AAP Clinical Guidelines 2021

*may be a role for admission for OBS +/- antibiotics

*Usually doing everything

22-28 days

22-28 days: Assess IBI risk risk

Quiet IMs

- "May" NOT do LP!
- If No LP done admit +/- abx (1-2% bacteremia risk)

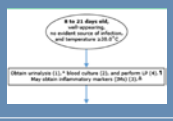
If LP done:

- If (+) admit, abx
- **If (-), dc home with CTX***,
  - 1-2% risk bacteremia
  - f/u PMD in AM
- Interpret WBC in CSF at face value for RBC counts <10,000

August 2021, Pediatrics Clinical Practice Guideline: Evaluation and Management of Well-Appearing Febrile Infants 8 to 60 Days Old Pantell et al.

0-7 days: different animal. Wide ddx

8-21 days:



Antibiotics, Admit

22-28 days:*

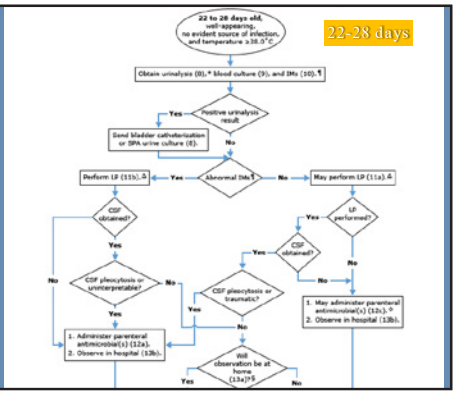
August 2021, Pediatrics Clinical Practice Guideline: Evaluation and Management of Well-Appearing Febrile Infants 8 to 60 Days Old Pantell et al.

AAP Clinical Guidelines 2021

*may be a role for admission for OBS +/- antibiotics

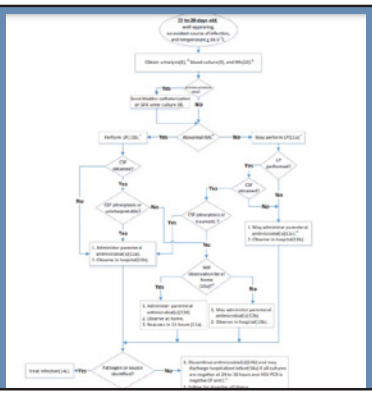
*Usually doing everything

22-28 days



ADMIT Febrile Neonates

almost always



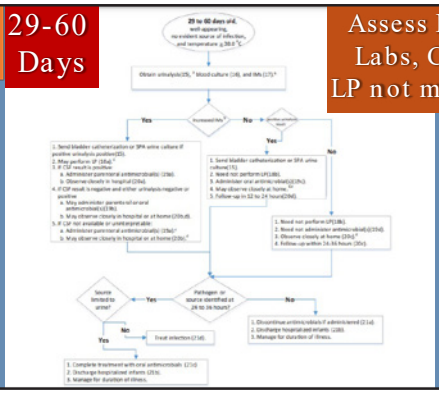
Discharge 22-28 day old if:
LP negative
Give ceftriaxone
Follow up in 12-24 hours

1-2% bacteremia risk

4-8 Weeks

work up may be amended
use a clinical decision tool with laboratory risk stratification

29-60 Days



Assess IBI Risk
Labs, Cultures
LP not mandatory

29-60 days: IBI risk

Quiet IMs

No LP needed!
No empiric antibiotics!
Check blood, urine
If UA (+), PO abx!
If UA (-), no abx
DC home!

Elevated IMs

"May" do LP
If no LP done (or uninterpretable):
Empiric CTX→dc home or admit (comfort level)

August 2021, Pediatrics Clinical Practice Guideline: Evaluation and Management of Well-Appearing Febrile Infants 8 to 60 Days Old Pantell et al.

4-8 Weeks

RSV?

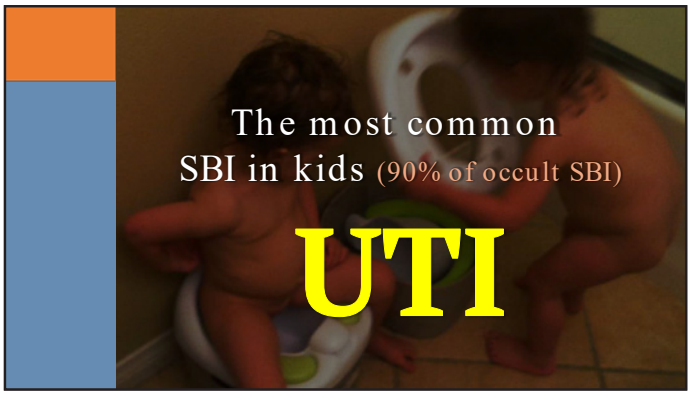
influenza?

DO the LP IF YOU GIVE ANTIBIOTICS

With unknown source of infection
UNLESS 21-28 day old and low risk

NEONATE:
Ampicillin and Ceftazidime

>1 month
Ceftriaxone



5-10% of young infants with UTIs have associated bacteremia

Associated meningitis is rare and is almost always described with additional symptoms in the literature

CSF pleocytosis correlates with peripheral WBC

Risk Factors

Prior UTI (LR 2.3-2.9)
Temp >39 C
Fever w/o Source
Fever >24 hr
Ill appearance
Suprapubic TTP (LR 4.4)

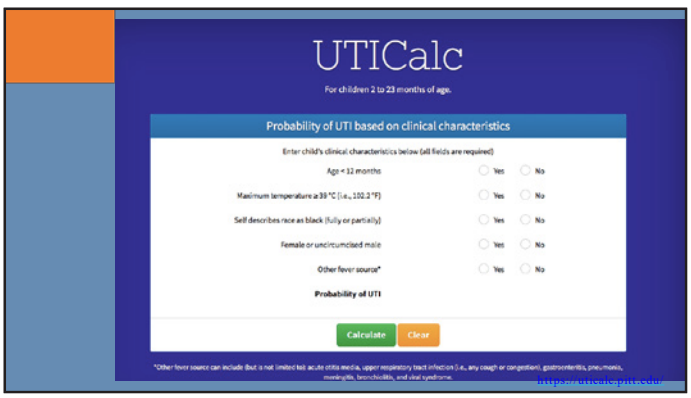
(2-24 Mo)

Underlying Neuro/anatomy abnormality

UTI symptoms

Infant: previous UTI, temp >40C, suprapubic tenderness

Verbal child: abdominal/back pain, dysuria/frequency, new-onset incontinence (LR 4.6)



Labrosse, M., A. Levy, J. Autmizguine, S. Gravel (2016) Evaluation of a New Strategy for Clean-Catch Urine in Infants. Pediatrics, 138 https://www.ncbi.nlm.nih.gov/pubmed/27542848

Bagged Urine?

85% false positive culture results

Don't send this for culture

AAP def of UTI: presence of pyuria and/or bacteriuria on UA and > 50,000 CFU/mL of a uropathogen

Or 10,000 if fever and pyuria

Culture

UTI treatment (local resistance patterns vary)

Outpatient: TMP/SMX, amox/clav, cephalosporins (cephalexin, cefixime, cefpodoxime) x 7-10 days (F, RF)

Inpatient: ceftriaxone, cefotaxime, ceftazidime, gentamicin

80% E. coli

(50% resistant to amoxicillin/ampicillin)

Unvaccinated febrile child?

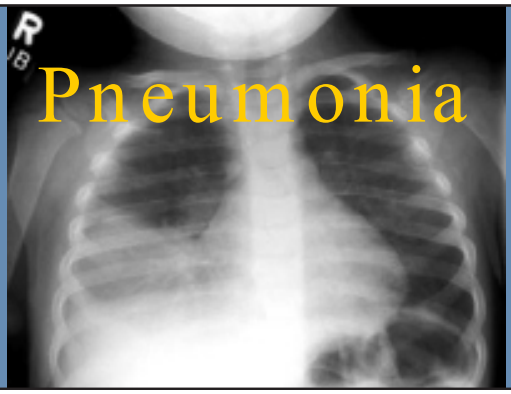
Short answer→nobody really knows

Prior (hib/s.pneumo) 3-11% occult bacteremia

5% invasive disease

Now herd immunity decreases risk, but not as low as fully immunized kids

(<1% bacteremia rate in vaccinated non-young infants)



Pathogens?

- Neonates: Grp B Strep, Gram negative enteric bacteria, Listeria, Chlamydia
- Infants/Toddlers: Viral (RSV, influenza, parainfluenza, metapneumovirus, adenovirus); Baterial (Strep pneumo, Haemophalus, S. aureus, Pertussis, atypicals?)
- >4-5 Years: Mycoplasma

Antibiotics?

- Neonate→Sepsis
- Afebrile staccato cough → Azithromycin
- > 3 Months → Amoxicillin
- > 4-5 Years → Macrolides

Occult bacteremia <1%

Otitis Media tx

Bilateral (<24 mo)
>48 Hours
Severe sx
<6 mo old

Otitis Media tx

Amoxicillin
80-90 mg/kg (max 2 g)
Pain control

Otitis Media tx

Amoxicillin
80-90 mg/kg
10 day course <24 months
7 days 2-5 years
5 days if >5 years and mild symptoms

Otitis Media

Amoxicillin-Clavulanate (Augmentin)

Bilateral
Conjunctivitis
Recent OM Infection

Otitis Media tx

Watch and wait therapy
OM is often self-resolving

Antibiotics DO NOT reduce complication rates

PMID: 26099233, 26451229

Summary

- Most kids with fever have self-limited viral infection
- Vaccinations have changed how we manage Peds Fever
- Neonates have decreased immune function and limited clinical exam so do full sepsis work-up
- All infants <2 months of age should have risk stratification labs
- UTI is the most common 'occult SBI'
- Most OM resolves on its own

Thank You!

emilyros@usc.edu
@EmilyRoseMD1

SELF EVALUATION

Pediatric Fever in the Emergency Department

1. A 5-week-old baby comes into the ED with a fever. What is the best work up for management of this infant?

a. Automatic LP and admission
b. Clinical evaluation only
c. Laboratory risk stratification and defer LP if all inflammatory markers are quiet
d. Prophylactic management for UTI only

2. What is the most common source of bacterial infection in children?

a. Bacteremia
b. Meningitis
c. Osteomyelitis
d. Urinary tract infection

3. At what age should lumbar puncture and admission occur for all febrile children?

a. ≤7 days only
b. ≤21 days only
c. ≤30 days only
d. ≤60 days only

4. A 2-year-old child presents with a fever of 39.2°C (102.6°F) for the past 24 hours. The child is active, well-hydrated, and otherwise healthy. Which of the following is the most appropriate next step in management?

a. Administer empirical antibiotics immediately
b. Recommend acetaminophen for comfort and monitor at home
c. Order blood cultures and a chest X-ray
d. Admit the child for observation and IV fluids

5. Which of the following best describes the role of antipyretics in the management of pediatric fever?

a. They are necessary to prevent febrile seizures
b. They should be used routinely to normalize temperature
c. They are used to improve comfort, not to prevent complications
d. They eliminate the need for further diagnostic evaluation

Answer Key: 1. C, 2. D, 3. B, 4. B, 5. C

Unusual Causes of Acute Generalized Weakness: Recognizing Neurologic Emergencies

Jonathan A. Edlow, MD

- Review uncommon but important conditions presenting as acute generalized weakness
- Understand physiology of the nicotinic acetylcholine receptor, the basis of neuromuscular transmission and our RSI medications
- Discuss issues relating to intubating patients with myasthenic crisis

General medical causes of diffuse weakness

- Hypotension, hypoxia, fever
- Sepsis, occult infections & acute febrile states
- GI bleeds & severe anemia
- CHF
- Thyroid disease
- Electrolyte, acid-base disorders & dehydration . . . and

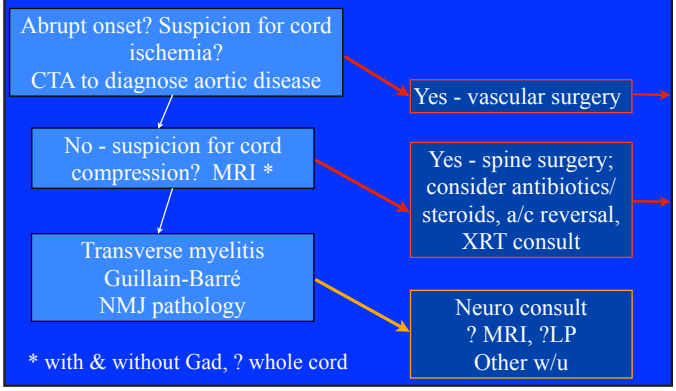
. . . pretty much anything else that affects homeostasis - mechanism unclear and likely multi-factorial

Grading weakness

- 0 - no moment
- 1 - flicker or trace muscle contraction
- 2 - limb or joint movement possible only with gravity eliminated
- 3 - limb or joint movement against gravity
- 4 - strength decreased but limb or joint movement possible against resistance
- 5 - normal strength against resistance

Medical Research Council

Lower Extremity Weakness



Uncommon causes of generalized weakness

- Guillain-Barré syndrome
- Myasthenia gravis & Lambert-Eaton syndrome
- Transverse myelitis
- Hypokalemic paralysis
- Tick paralysis, botulism, organophosphate poisoning

Epidemiological Context

- Recent URI, vaccination, gastroenteritis
- Long-haired child in the summer
- Asian male after large carbohydrate meal
- Common source case clustering
- Farmer with exposure to pesticides

This course book is available in full color and expandable form at www.AEIseminars.com/Syllabus

Jean Baptiste Octave Landry de Thézillat

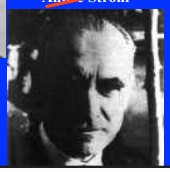
1859 - Landry describes 10 cases of ascending paralysis

George Guillain

1916 - Guillain, Barré and Strohl write the classic paper, but Strohl's name gets dropped and Guillain argues that Landry's cases were a hodegpodge of various causes.

Alexandre Barré

André Strohl



What are the clinical features of GBS?

- Ascending paralysis starting in the legs, typically symmetric
- Loss of deep tendon reflexes
- Sensory symptoms (numbness, paresthesias) common at onset
- Rapidity of evolution is variable
- Morbidity/mortality due to respiratory muscle involvement

Guillain-Barré syndrome

• What part of the nervous system is affected?	PNS - polyradiculopathy
• What are some known triggers?	Infections (2/3 of patients), immunizations
• In what percent of cases is the CSF protein elevated?	Depends on timing often normal in 1st week usually high by end of 2nd week

What should every GBS patient have done in the ED?

- **Forced vital capacity** (FVC) & negative inspiratory force (NIF) & consider ABG - respiratory failure
- **Telemetry** - autonomic instability
- **Check for an embedded, engorged tick**
- Lumbar puncture is **NOT required**

Intubate for hypoxia, suspected aspiration, or FVC < 15cc/kg

Admit to neurology ICU (or closely monitored) setting
IV-Ig (or plasma exchange)

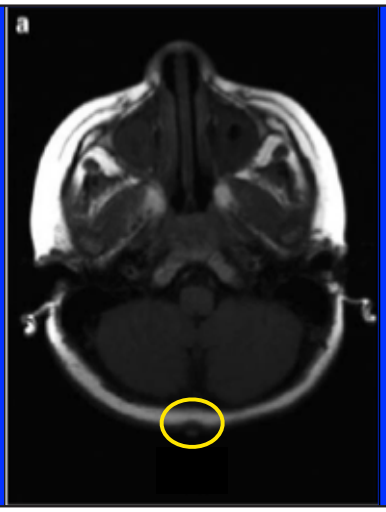
Miller Fisher variant GBS

- Triad of ophthalmoplegia, ataxia & diminished deep tendon reflexes
- Due to a specific antibody; treated like GBS

Miller Fisher variant GBS

Bickerstaff encephalitis

- A 6 year-old girl is brought to the ED for inability to walk and slurred speech. She was normal when she went to sleep the night before after an outdoors July 4th celebration.
- General exam is normal except for a P-110. She has mild bilateral facial weakness, and diffuse motor weakness, as well as moderate ataxia.
- CT scan and CSF analysis are normal.



Tick paralysis

- Toxin blocks acetylcholine release at neuromuscular junction
- Symptoms evolve over 12-24 hours
- Do not diagnose GBS without a complete check for ticks
- Examine the skin (especially the scalp) carefully
- Symptoms resolve over the ensuing hours after tick removal

- 48-year old woman with 4 weeks of intermittent diplopia and ptosis
- Diplopia has waxed and waned over last 4 days. Ptosis is bilateral and also varies in intensity
- No weakness in extremities; denies headache, neck or eye pain; % mild tremor
- No significant past medical history or medication use

This course book is available in full color and expandable form at www.AEIseminars.com/Syllabus

What is the diagnosis? What are clues to the diagnosis?

Myasthenia gravis

- Non-specific background symptoms in setting of recent psychological stressor
- Symptoms are intermittent over weeks
- Localize poorly (diplopia, bilateral ptosis)
- Ptosis is present on exam

Myasthenia Pearls

- The pupils are normal in myasthenia
- **Painless** ptosis or diplopia
- Variable, shifting findings over time, worse with increased use and end of day
- Ptosis
  - Contralateral lid is usually retracted upwards (higher than normal)

Why get a CXR in a patient with myasthenia?

Tensilon test
(now of historic interest only)

- **Define a "testable" muscle** (usually lid or EOM)
- Cardiac monitoring in place and atropine readily available (side effects - bradycardia, salivation)
- Tensilon (edrophonium) has half-life of 10 minutes
- Draw up 10cc (10mg) as well as 10cc saline in 2nd syringe
- Give 2cc of syringe A or B, repeat with 3mg (may go up to full 10mg if needed), followed by the 2nd syringe; blinded observer

Tensilon test
(now of historic interest only)

Before tensilon

After tensilon

Tensilon test: + LR 15, - LR 0.11

Before ice

After ice

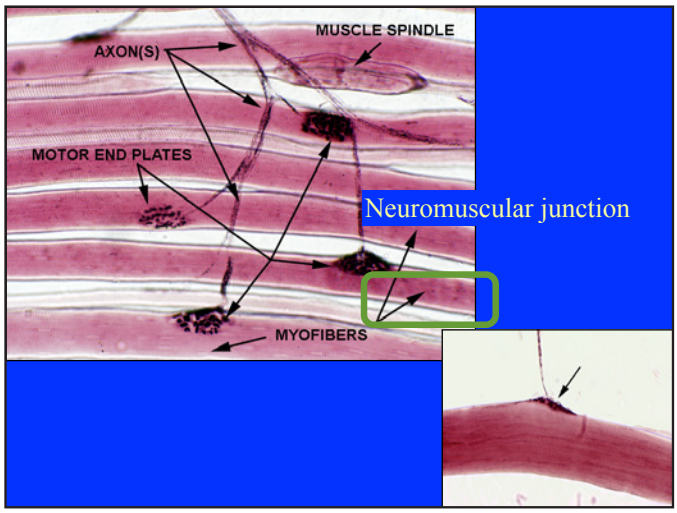
Ice test

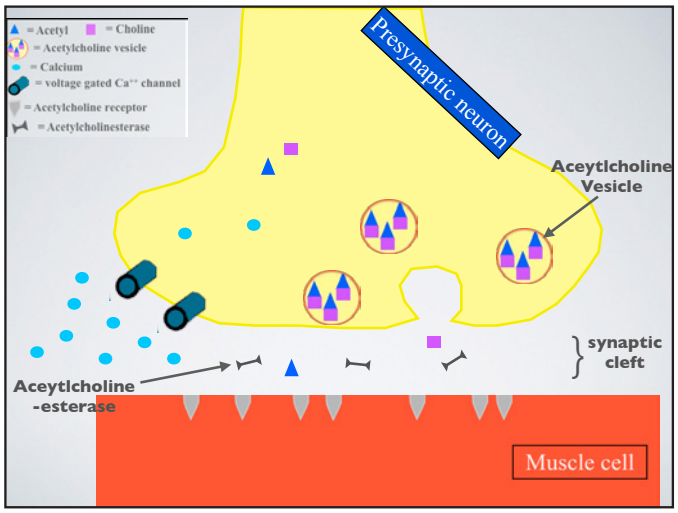
Tensilon test: + LR 15, - LR 0.11
Ice test: + LR 26, - LR 0.16

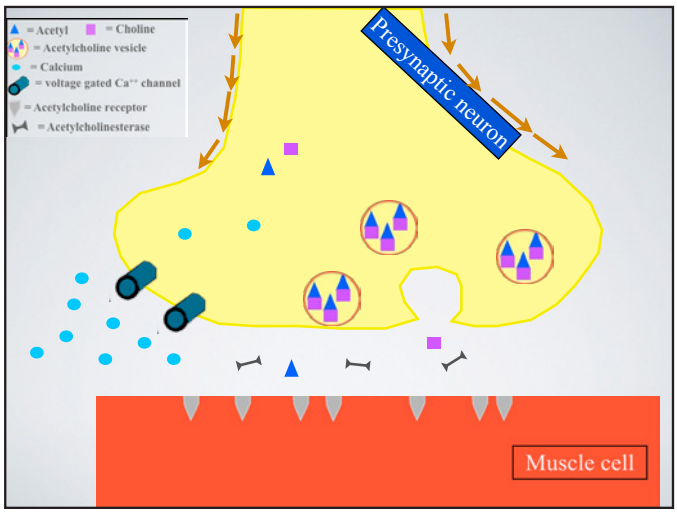
The neuromuscular junction

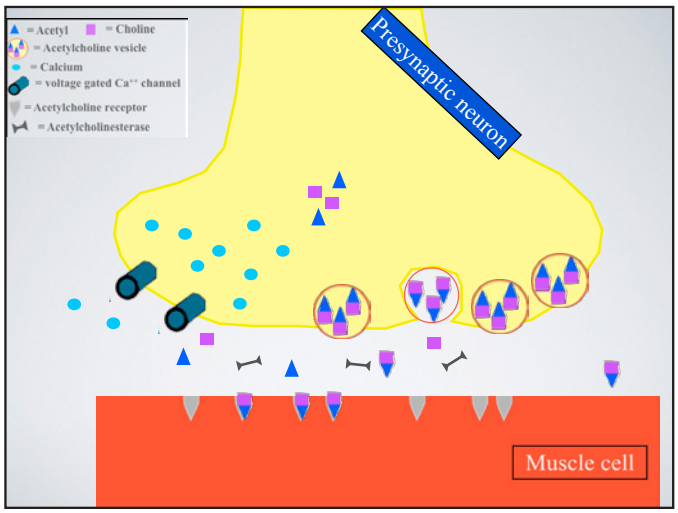
- Myasthenia gravis
- Lambert Eaton syndrome
- Organophosphate poisoning

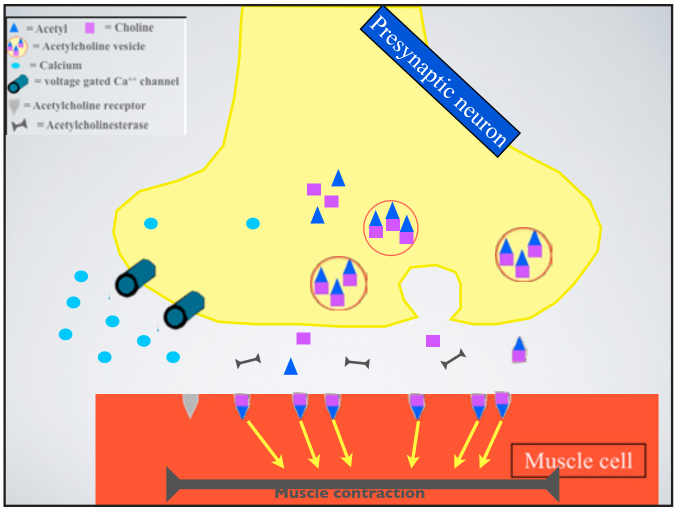
- Medications to facilitate intubation
  - Depolarizing agents (succinylcholine)
  - Non-depolarizing agents (rocuronium)







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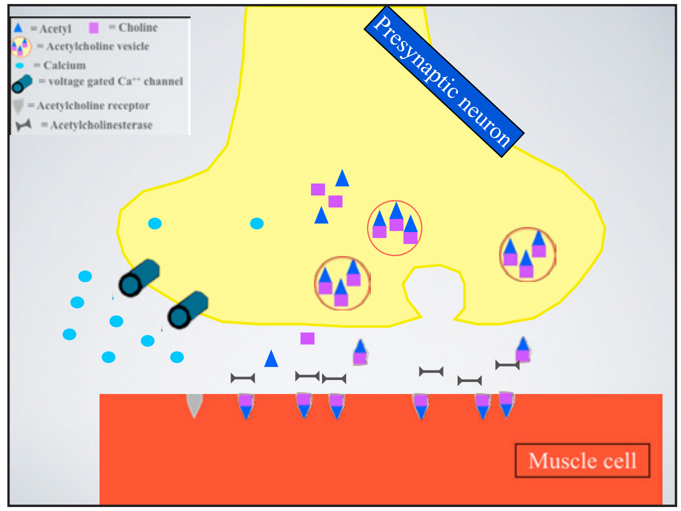
= voltage gated Ca^{++} channel
= Acetylcholine receptor
= Acetylcholinesterase
Presynaptic neuron
Muscle cell
Muscle contraction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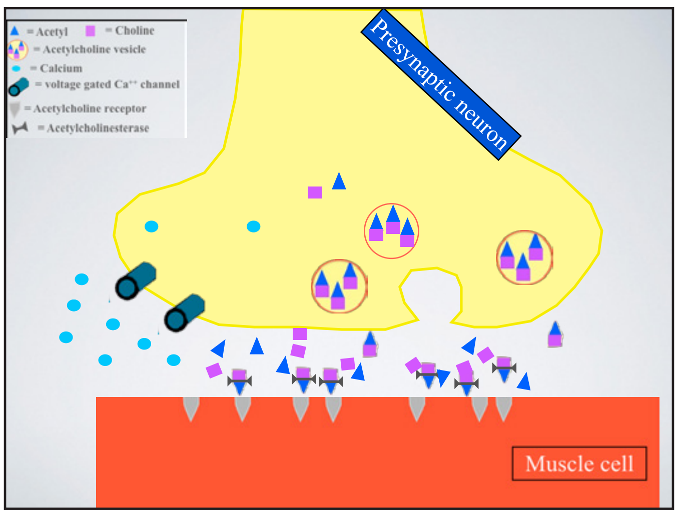
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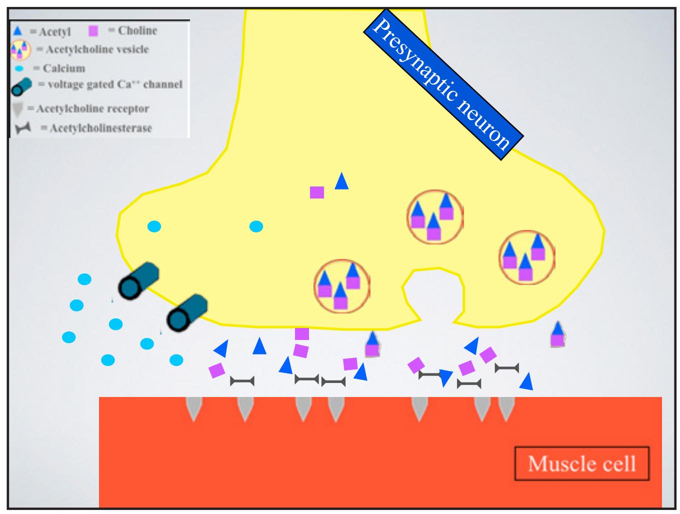
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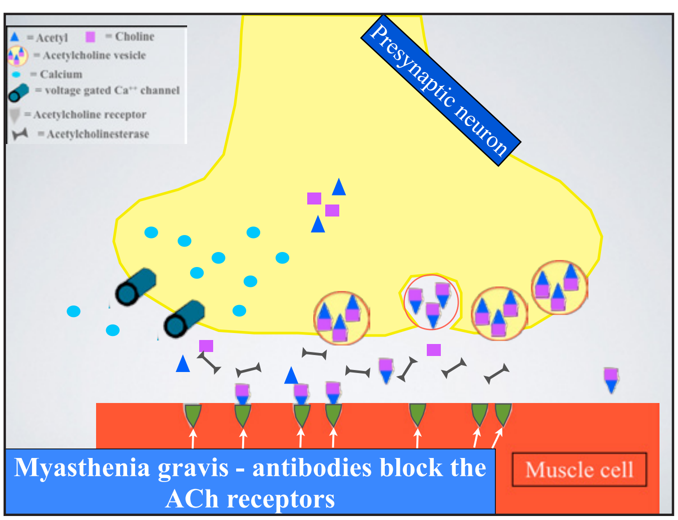
Muscle cell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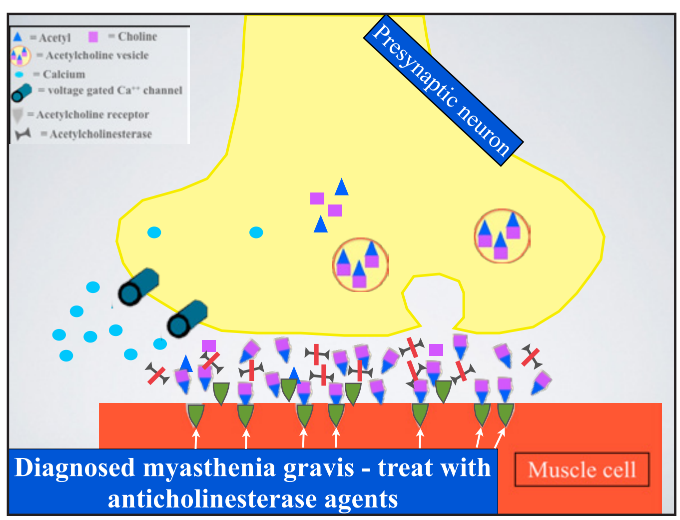
Presynaptic neuron
Myasthenia gravis - antibodies block the ACh receptors
Muscle cell


= Acetyl
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= Acetylcholine vesicle
= Calcium
= voltage gated Ca^{++} channel
= Acetylcholine receptor
= Acetylcholinesterase
Presynaptic neuron
Diagnosed myasthenia gravis - treat with anticholinesterase agents
Muscle cell

What is a myasthenic crisis?

- Myasthenia gravis that is acutely associated with respiratory failure

- Must distinguish myasthenic crisis (too little meds) from cholinergic crisis (too much meds)
- Cholinergic crisis (similar to organophosphate toxicity)
  - Muscarinic (SLUDGE)
  - Nicotinic (on skeletal muscle - flaccid paralysis)
- Look for triggers (infection, physiologic stress, medications and medication non-compliance)
- Rare MG patients will present in myasthenic crisis

What are some medications that trigger myasthenic crises?

- Antibiotics
  - gentamicin, quinolones
- Antihypertensive & anti-arrythmics
  - β and Ca channel blockers, quinidine
- Magnesium
- Neuromuscular blockers
  - Succinylcholine

Myasthenic crisis - treatment

- Assess respiratory mechanics, ICU admit
- IV pyrodostigmine (Mestinon)
  - **Caution**: is it a cholinergic crisis?
- Neurology consult
- Intubation as needed

Intubating the patient in myasthenic crisis

Can I use succinylcholine?

- Will it lead to exaggerated hyperkalemia?
- Will it result in prolonged paralysis?

Will it lead to exagerated hyperkalemia?

- With Sux, usual ↑ in K is 0.5-1 mEQ/L
- With many muscle diseases (dystrophies, burns, crush injuries, denervation), ACh receptors ↑-regulate after several days
- In these patients, the hyperkalemic response to Sux is exaggerated
- In myasthenia, the ACh receptors are functionally ↓ so hyperkalemia does not occur

Will neuromuscular blockers cause prolonged paralysis?

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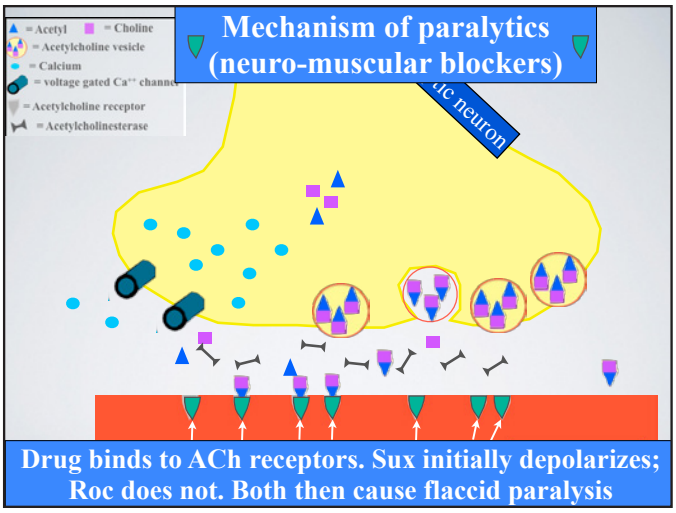


Will neuromuscular blockers cause prolonged paralysis?

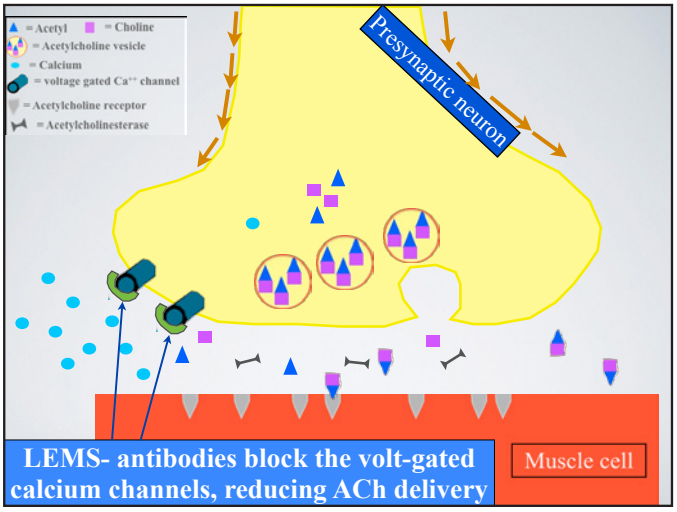
- Depolarizing agents (Sux)
  - less predictable dose response
  - need to ↑ dose (1.5-2 mg/kg)
  - often results in prolonged paralysis
- Non-depolarizing agents (vec- and rocuronium)
  - may require a ↓ dose, may take longer for effect
  - less risk of prolonged paralysis
- In some profoundly weak and fatigued patients, can intubate with a sedating agent, no paralytic

What is the average duration of intubation in patients with myasthenic crisis?

12 days

Lambert-Eaton myasthenic syndrome (LEMS)

- Opposite of MG - IMPROVES with repetitive stimulation
- More proximal muscle involvement compared to myasthenia gravis



Myasthenia vs Lambert-Eaton

	Myasthenia Gravis	LEMS
Pathophysiology	Antibodies to ACh **post-synaptic** receptor	Autoimmune impairment of VGCC → decreased **pre-synaptic** release of ACh
Clinical presentation	Oculo-bulbar presentation common Cranio-caudad evolution	Proximal leg muscles, rare to present with oculo-bulbar Caudal-cranial evolution
Association with cancer	No, but look for thymoma	Yes in half, most often SCLC (usually precedes cancer diagnosis)
Reflexes	Normal	Usually decreased or absent
Autonomic involvement	Not present	Very common, dry mouth > erectile dysfunction/constipation > orthostasis
Respiratory symptoms	More common	Much less common

- A 25 year-old man has 2 days of mid-thoracic back pain. Today, he develops gradually increasing numbness "like I've got saran wrap on my legs" and leg weakness. Yesterday, at an OSH, his exam was "normal". He denies head or back trauma. No chest pain
- PMH: none
- No meds or allergies; he drinks 2 beers 5x/week
- Exam - mental status & cranial nerves are normal; he is paraplegic with a T-8 sensory level & decreased sphincter tone

MRI - no tumor, abscess, hematoma or large disc protrusion

CSF - 20 lymphocytes and protein of 72
DX: transverse myelitis
Lyme serology is negative
Treated with steroids with minimal response

Returns 2 weeks later with large DVT

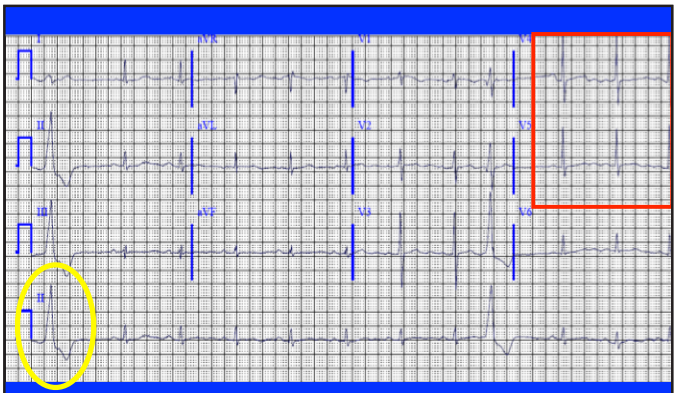
Transverse Myelitis

- If complete, fairly typical with a sensory level, bladder dysfunction and paralysis below the level of inflammation
- If incomplete (or evolving) the findings can be quite variable
- Often begins with vague back pain, many times "odd" sensory symptoms

Transverse Myelitis

- Vascular
  - aortic dissection with cord ischemia
- Inflammatory
  - post-infectious or post-vaccine (ADEM), lupus, multiple sclerosis
- Infections
  - Lyme, schistosomiasis, others

- A 26 year-old Asian male arrives at the ED profoundly and diffusely weak, which has developed over the last few hours. He is a grad student in the middle of exams.
- Vital signs & general exam are normal except for a slightly irregular pulse
- Neurological exam shows profound weakness, legs greater than arms



Treatment

- **Remember that total body potassium is normal**
- **KCl, 10mEq/hour, < 50 total (less likely to have rebound hyperkalemia**
- **Propranolol (especially in thyrotoxic cases)**

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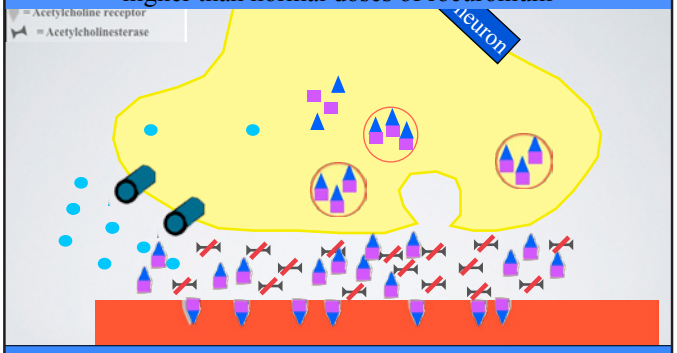
Organophosphate poisoning

- Binds to acetylcholinesterase leading to over supply of ACh at the neuromuscular junction
- Symptoms begin within hours (oral/respiratory) or up to 12 hours after (dermal) exposure
- Symptoms last up to 2 weeks!
- Epidemiological context important

Organophosphate poisoning

- Muscarinic symptoms early
  - SLUDGE with bradycardia, bronchospasm and bronchorrhea
- Nicotinic symptoms follow
  - Fasciculations followed by neuromuscular paralysis
  - Similar to what one sees with succinylcholine, to be AVOIDED when intubating)

When intubating patients with organophosphate poisoning, avoid succinylcholine (prolonged effect); but may require higher than normal doses of rocuronium



Organophosphate binds irreversibly to anticholinesterase, ↑ the ACh, acting like succinylcholine

Organophosphate poisoning

- Atropine (for muscarinic effects) - LARGE doses
  - Initially, 1mg, then double the dose (1/2/4/8/16/. . .) every 5-10 minutes
  - End-point = no respiratory secretions or bradycardia
  - Will not affect paralysis
- 2-PAM, pralidoxime (for nicotinic effects)
- Decontamination of skin and clothing

Acute flaccid paralysis

- More common in children with frequent preceding or concurrent GI or respiratory illness
- Anterior horn cell injury caused by an enterovirus (D68 & other sero-types) is the likely cause; late summer/ early fall
- Clinical manifestations (pure motor - no sensory level)
  - Flaccid quadraparesis
  - Cranial nerve findings (30%), altered mental status (~30%)

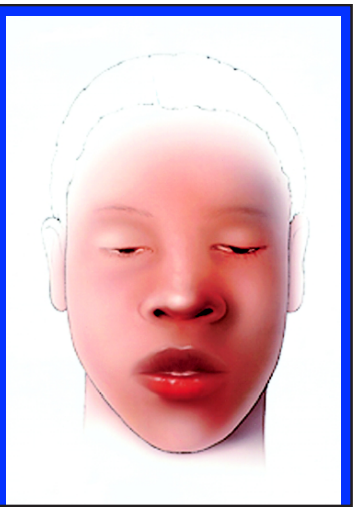
Polio

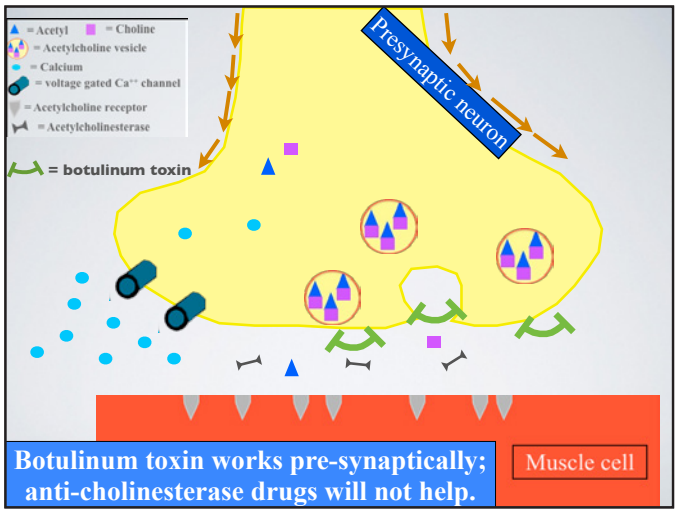
- Another enterovirus, most common in children
- Recent NYC outbreak in ***unvaccinated*** people
- Most infections are either asymptomatic or cause fever, fatigue, HA, sore throat, vomiting, neck stiffness and limb pain
- 1/25 (4%) develop a viral meningitis picture and only 1/200 (0.5%) of infected patients develop a flacid paralysis
- Treatment is supportive

- A 36 year-old woman arrives at the ED c/o diplopia and generalized weakness, both gradually increasing for 2 days. She has difficulty combing her hair. Today, while trying to pull out of the driveway, she could not do it because "everything looked blurry and double." An acquaintance is currently in the ER being evaluated for GBS.
- Exam shows normal vital signs and general exam
- Neurological exam shows severe weakness of proximal arms and mild weakness of hip flexors. She has mild diplopia.

Botulism

1. **Clinical – always with cranial neuropathy**
   - **Ptosis & diplopia (3, 4, 6), bi-facial palsy (7), diplopia, dysphagia (10)**
   - **Flaccid descending paralysis**
   - **Alert, normal mental status**
2. **Normal CSF**
4. **Wound botulism –injected drugs, often "black tar" heroin**
5. **Infant botulism – floppy baby, ↓ suck & swallow**





Botulinum toxin works pre-synaptically; anti-cholinesterase drugs will not help.

Mis-diagnoses in a botulism outbreak

Table 3. Working Diagnosis of 31 Patients with Botulism at the Time of Outbreak Recognition*

Working Diagnosis	Number of Patients
Myasthenia gravis	7
Psychiatric illness†	4
Viral syndrome	4
Botulism‡	3
Stroke	3
Guillain-Barré syndrome	3
Inflammatory myopathy	2
Diabetic complications	1
Hyperemesis gravidarum	1
Hypothyroidism	1
Laryngeal trauma	1
Overexertion	1

* Excludes 5 patients first evaluated after the outbreak was recognized.
† Includes hysteria, agitated depression, separation reaction, factitious weakness.
‡ Includes the 3 family members whose diagnosis represented the initial recognition of the outbreak.

Anti-toxin access

- Call your state DPH first
- If difficulty connecting at the state level, can call the CDC 24-hour telephone number: **770-488-7100**

Closing thoughts

- Acute morbidity & mortality is ***respiratory*** - CHECK respiratory mechanics early
- ***Consider myasthenia*** in the absence of anatomically localizing symptoms or signs in patients with oculomotor presentations & fluctuating symptoms
- Use the exam thoughtfully
  - Check for a sensory level
  - Do a bladder scan, even if not a PVR
  - Check the Babinski sign
- Check potassium

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SELF EVALUATION

Unusual Causes of Acute Generalized Weakness: Recognizing Neurologic Emergencies

True/False

1. Guillain-Barre syndrome is a disease of the spinal cord.

2. A typical case of Guillain-Barre syndrome is mild sensory symptoms that leads to an ascending paralysis and loss of deep tendon reflexes.

3. In a 5-year-old girl with long hair who presents with severe weakness and ataxia in the summer time, a thorough examination of the scalp may lead to a specific diagnosis.

4. The pupils are always normal in myasthenia gravis.

5. In myasthenia gravis, weakness in a given muscle gets better with repetitive use whereas in Lambert-Easton syndrome, the opposite occurs.

6. One epidemiological clue for hypokalemic periodic paralysis is Asian descent.

Answer Key: 1. F, 2. T, 3. T, 4. T, 5. F, 6. T

Challenging Cases in Emergency Critical Care

Michael W. Donnino, MD

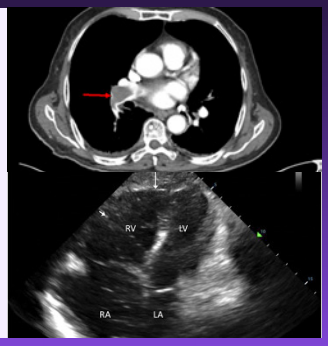
DISCLOSURES

1. RESEARCH GRANTS: NIH, DAY ZERO DIAGNOSTICS, FOUNDATIONS, PHILANTHROPY

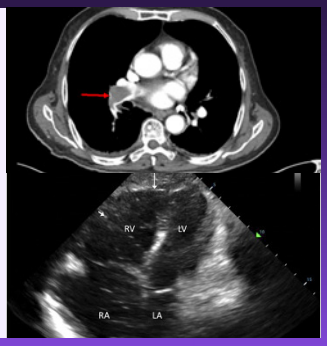
OBJECTIVES

1. TO EXAMINE CHALLENGING CASES IN CRITICAL CARE
2. TO DEVELOP STRATEGIES TO RAPIDLY EVALUATE AND TREAT SUDDEN DETERIORATION IN CRITICALLY ILL PATIENTS

- 65 YEAR OLD WITH SOB AND FOUND TO HAVE CT AND ECHO AS DISPLAYED
- VITALS: HR 102, BP 130/73, RR 18, SPO2 = 95% ON 2 LITERS
- STARTED ON HEPARIN, ADMITTED TO THE ICU

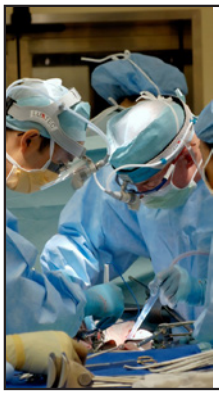


- 12 HRS AFTER ADMISSION, PATIENT BECOMES HYPOTENSIVE
- HR 110, BP 70/30, RR 22, SPO2 = 95% ON 2L NC
- DESPITE A 1 LITER FLUID BOLUS THE BP REMAINS LOW (74/40)



YOUR NEXT MOVE?

1. GIVE ADDITIONAL HEPARIN BOLUS
2. SYSTEMIC THROMBOLYSIS
3. GIVE ANOTHER 1 LITER OF LR AND REASSESS
4. CALL CARDIOLOGY TO CONSIDER EKOS
5. SOMETHING ELSE



- **REMEMBER THE CLOT-TREATMENT PARADOX**
  When treating an underlying thrombotic condition with anti-coagulation, remember that a crashing patient can be exhibiting thrombosis complications OR bleeding!
- **ALWAYS RUN THE SHOCK DIFFERENTIAL**
  Regardless of your initial instinctive move, step back and run the "shock differential" on crashing hypotensive patients to ensure you are not missing something

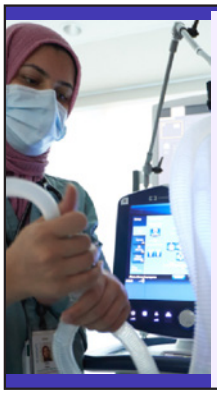
"NOISE" WHILE ON THE VENTILATOR

- 65 YEAR OLD WITH COVID PNEUMONIA ON THE VENTILATOR
- NURSE REPORTS HEARING A NOISE EACH TIME BREATH IS DELIVERED
- THE VENT MECHANICS ARE UNCHANGED & SPO2 >95%

WHAT IS YOUR NEXT STEP?

1. REMOVE A FEW CCS FROM CUFF
2. PUT A FEW CCS OF AIR IN CUFF AND/OR CHECK CUFF PRESSURE
3. INCREASE THE TIDAL VOLUME
4. GO BACK TO BED - NOT A BIG DEAL
5. SOMETHING ELSE

YOU PUT AIR IN THE CUFF BUT NOTHING CHANGES- WHAT NEXT?



DIFFERENTIAL OF "NOISE" WITH VENTILATOR BREATHS

1) CUFF NEEDS SOME AIR - COMMON
Typically resolves with a few ccs of air, cuff pressures should be adequate.

2) CUFF LEAK/RUPTURE- TUBE EXCHANGE
Cuff pressures also likely low and illustrate leak

3) PARTIAL EXTUBATION - URGENCY!
Noise won't improve with air cuff pressures normal or even high. Requires urgent attention to reposition tube

YOU ARE CALLED TO THE ED TO HELP A 65 YEAR OLD FEMALE WHO PRESENTS WITH ALTERED MENTAL STATUS, ATRIAL FIBRILLATION WITH RVR (170) AND HYPOTENSION (70/30) - WHAT IS YOUR IMMEDIATE MOVE?

1. CARDIOVERT AT 200J
2. AMIODARONE
3. CARDIOVERT AT 360J
4. ESMOLOL WITHOUT A BOLUS
5. 1 LITER OF LR +/- VASOPRESSOR
6. SOMETHING ELSE

YOU ARE CALLED TO THE ED TO HELP A 65 YEAR OLD FEMALE WHO PRESENTS WITH ALTERED MENTAL STATUS, ATRIAL FIBRILLATION WITH RVR (170) AND HYPOTENSION (70/30) - WHAT IS YOUR IMMEDIATE MOVE?

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*** TEMPERATURE 103.5 ***

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2. AMIODARONE
3. CARDIOVERT AT 360J
4. ESMOLOL WITHOUT A BOLUS
5. 1 LITER OF LR +/- VASOPRESSOR
6. SOMETHING ELSE

✔ **SEEK AND TREAT UNDERLYING CAUSE**

Often the rapid heart rate is just compensatory to hypotension much like sinus tachycardia.

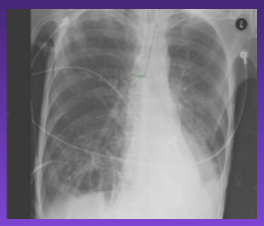
✔ **CONSIDER THE CONTEXT & TIMING**

Patients in ED likely with hypotension/afib with RVR are most likely septic/hypovolemic. Post-cardiac surgery patients are more likely to have issue originating from the heart.

✖ **AVOID LUMPING AS A "CARDIAC" PROBLEM**

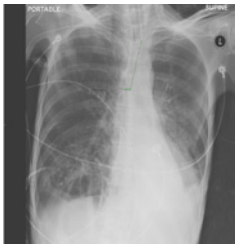
Avoid tunnel vision of considering a primary cardiac issue.

74 Y/O STABLE VENTED PT. ON HEPARIN FOR AFIB WITH SUDDEN HYPOXIA AND HYPOTENSION- WHAT NEXT?



1. THROMBOLYSIS
2. NEEDLE DECOMPRESSION OR CHEST TUBE
3. PERICARDIAL TAP
4. WAIT UNTIL PATIENT ARRESTS - I KNOW WHAT TO DO THEN
5. SOMETHING ELSE

74 Y/O VENTED PT. WITH SUDDEN HYPOXIA AND HYPOTENSION



A CASE OF SEVERE ACIDOSIS

- 42 YEAR OLD MALE WITH NO KNOWN PAST MEDICAL HISTORY PRESENTED WITH CONFUSION AND SEVERE ABDOMINAL PAIN
- TEMP: 99.3, HR: 108 BP: 98/50 RR: 30 SAT: 98%
- EXAM: DRY MUCOUS MEMBRANES AND DIFFUSE ABDOMINAL TENDERNESS
- KEY LABS: PH = 6.8 PCO2=5 BICARB=3 LACTATE = 27 MMOL/DL

A CASE OF ACIDOSIS -> TO THE OR!

- PRE-OP DIAGNOSIS: PRESUMED ISCHEMIC BOWEL
- POST-OP DIAGNOSIS: NORMAL BOWEL
- WHOOPS- LACTATE WAS 27 MMOL/DL!
- NOW WHAT?

POST-OPERATIVE COURSE

- LACTATE RAPIDLY NORMALIZED
- PATIENT EXTUBATED WITHIN HOURS
- ASKING FOR A FOOD TRY AND WHEN HE CAN GO HOME!
- REVIEW OF CASE REVEALS HE ONLY RECEIVED 500 CC OF FLUID AND ONE INTRAVENOUS MEDICATION IN THE ED BUT NO OTHER THERAPIES???

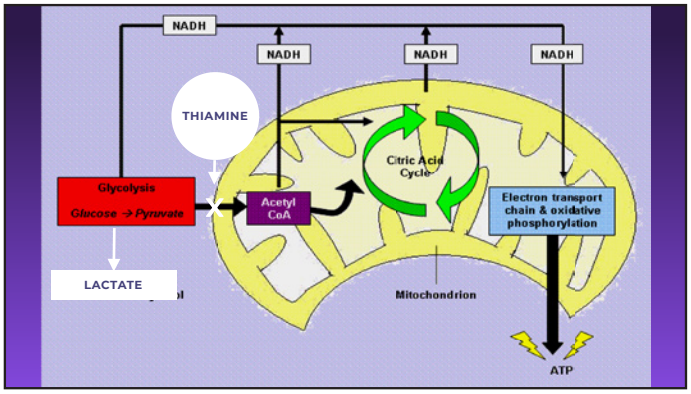
WHAT JUST HAPPENED?

1. DIET-RELATED PROBLEM
2. SEIZURE WITH SOMATIC MANIFESTATIONS
3. TOXIC INGESTION WITH RAPID IMPROVEMENT
4. INCARCERATED HERNIA THAT SELF-RESOLVED DURING EX-LAP
5. GEEZ, I HAVE NO IDEA
6. SOMETHING ELSE

GASTROINTESTINAL BERIBERI: A PREVIOUSLY UNRECOGNIZED SYNDROME

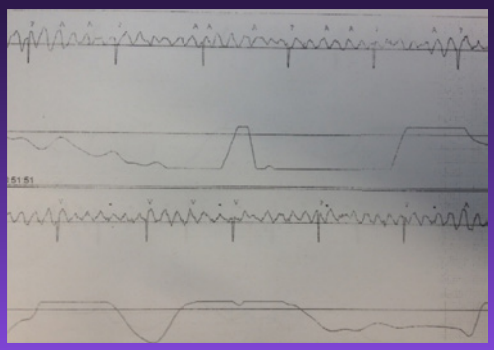
Patient	Date	pH	Bicarbonate	Lactate
LU	03/2002	6.82	4	27.2
LU	08/2002	6.83	4	30.0
LU	03/2003	6.73	5	23.8
MS	03/2004	7.06	5	18.6

DONNINO M. GASTROINTESTINAL BERIBERI: A PREVIOUSLY UNRECOGNIZED SYNDROME. ANNALS OF INTERNAL MEDICINE. (2004) 141 (11): 898-99

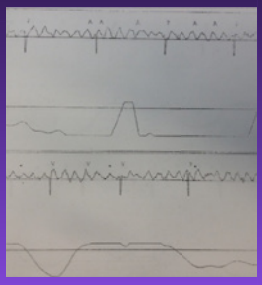


71 YEAR OLD POST-OP DAY #5 FROM CABG IS NOW IN CARDIAC ARREST

WHAT DO YOU WANT TO KNOW NEXT???



APART FROM CPR, WHAT NEXT??



1. DEFIBRILLATE
2. GIVE EPINEPHRINE 1 MG IV
3. GIVE AMIODARONE 300 MG IV
4. CALL SURGEON FOR THORACOTOMY
5. SOMETHING ELSE

THE ULTIMATE CHALLENGE

- 72 YEAR OLD MALE NO PMH WITH CHEST PAIN SENT TO THE CATH LAB WITH QUESTION OF STEMI (SUBTLE ECG CHANGES)
- T: 99, HR 102, BP 98/40, RR 22, SPO2 98% RA
- CATH = NORMAL CORONARIES
- AFTER CATH, DROPS BP 80/34, HR 120, ALTERED MENTAL STATUS & IS INTUBATED
- ECHO: > 55% EF AND NO EFFUSION
- PULM ANGIO: NO PE
- MIXED OXYGEN SAT = 35%, CI = 1.7
- CVP/PCWP = "NORMAL"

THE ULTIMATE CHALLENGE

1. PROVIDE SUPPORTIVE CARE FOR PRESUMED CARDIOGENIC SHOCK
2. ADD ANTIBIOTIC COVERAGE FOR POSSIBLE SEPSIS AND SEEK POSSIBLE SOURCES
3. CHECK FOR BLEEDING - YOU FOOLED ME ON THE FIRST CASE AND I WON'T BE FOOLED AGAIN!
4. CALL A SURGEON - MAYBE THERE IS AN OPERATION OF SOME SORT THAT CAN HELP?
5. I HAVE NO CLUE BUT WILL PROVIDE GOOD SUPPORTIVE CARE
6. SOMETHING ELSE

DIFFERENTIAL DIAGNOSIS FOR NORMAL/HIGH EJECTION FRACTURE BUT LOW CARDIAC OUTPUT (CARDIOGENIC SHOCK)

1. PROFOUND HYPOVOLEMIA
2. VENTRICULAR SEPTAL DEFECT
3. MITRAL REGURGITATION
4. AORTIC INSUFFICIENCY
5. LEFT VENTRICULAR OUTFLOW TRACT OBSTRUCTION (LVOT0)

SO, WHAT DISEASE PROCESS PRESENTS WITH CHEST PAIN AND ONE OF THE ACUTE FINDINGS BELOW?

1. PROFOUND HYPOVOLEMIA
2. VENTRICULAR SEPTAL DEFECT
3. MITRAL REGURGITATION
4. AORTIC INSUFFICIENCY
5. LEFT VENTRICULAR OUTFLOW TRACT OBSTRUCTION (LVOTO)

SO, WHAT DISEASE PROCESS PRESENTS WITH CHEST PAIN AND ONE OF THE ACUTE FINDINGS BELOW?

HINT: Blood pressure was 80/34

1. PROFOUND HYPOVOLEMIA
2. VENTRICULAR SEPTAL DEFECT
3. MITRAL REGURGITATION
4. AORTIC INSUFFICIENCY
5. LEFT VENTRICULAR OUTFLOW TRACT OBSTRUCTION (LVOTO)

SELF EVALUATION

Challenging Cases in Emergency Critical Care

1. A patient on heparin for PE becomes hypotensive despite fluids. What should always be done before giving more anticoagulation?

a. Give thrombolysis immediately
b. Add more heparin
c. Run the “shock differential”
d. Call cardiology

2. T/F - A ventilated patient with a persistent “noise” on breaths that does not improve after inflating the cuff may be partially extubated.

3. In the ED, a hypotensive patient with atrial fibrillation and RVR should be managed by:

a. Immediate cardioversion for presumed arrhythmia cause
b. Assuming the tachycardia is compensatory and treating underlying shock
c. Giving amiodarone to slow the heart rate
d. Ignoring context and focusing only on rhythm

4. T/F - Sudden hypoxia and hypotension in a ventilated ICU patient may indicate tension pneumothorax, which requires needle decompression or chest tube placement.

5. A patient with severe lactic acidosis (lactate 27) and normal bowel at surgery was later diagnosed with:

a. Ischemic bowel missed on exam
b. Gastrointestinal beriberi (thiamine deficiency)
c. Seizure with lactic acidosis
d. Incarcerated hernia

6. T/F - Left ventricular outflow tract obstruction (LVOTO) can present with chest pain and low blood pressure despite preserved ejection fraction.

Answer Key: 1. C, 2. T, 3. B, 4. T, 5. B, 6. T

Sticks and Stones May Break My Bones: Avoiding Orthopedic Pitfalls

Robert Tubbs, MD

- Briefly review a systematic way of interpreting MSK radiographs
- Examine the radiographic findings of common ED orthopedic injuries
- Review the common pitfalls in high-risk injuries

So how do we read these things?

You need to develop a system!!!

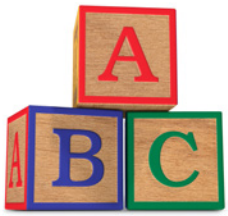
The ABC'S of MSK Radiology

A = Adequacy and Alignment

B = Bones

C = Cartilage (Joints)

S = Soft tissues and Spacing

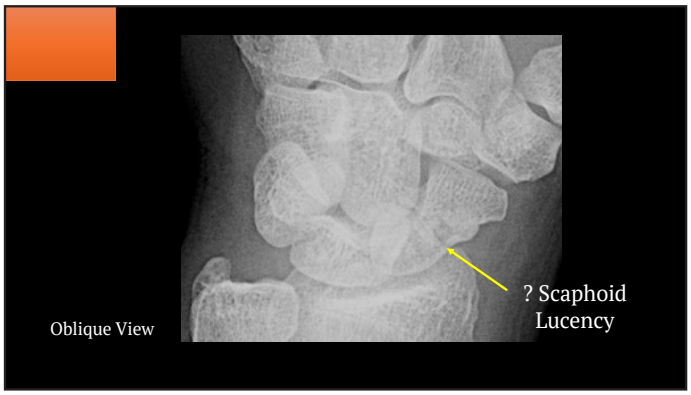


Let's look at some cases

23 y.o. female s/p fall on outstretched hand

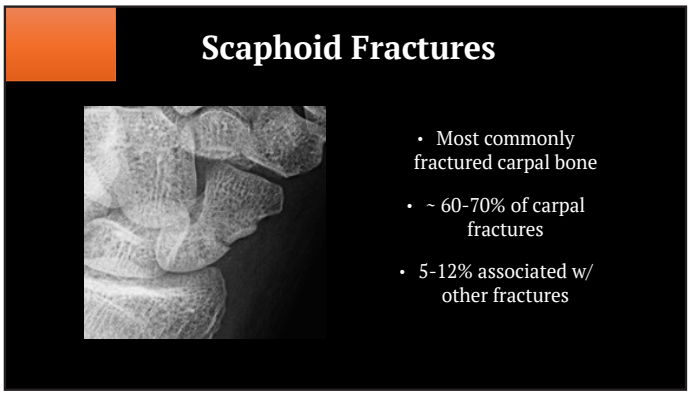

PA View

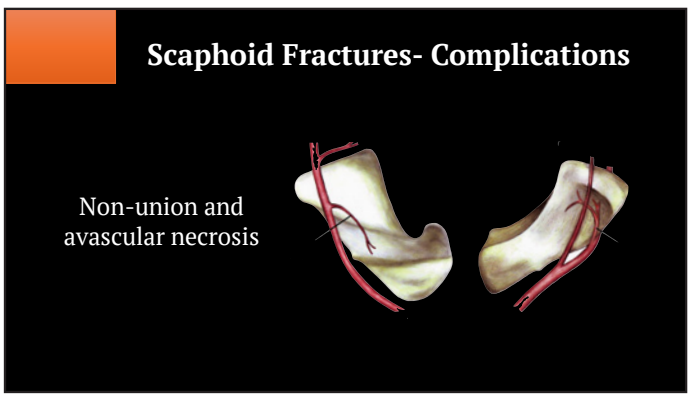

Oblique View


Oblique View
? Scaphoid Lucency


Lateral View
Loss of Pronator Quadratus Fat Stripe


"Scaphoid view"


Scaphoid Fractures
• Most commonly fractured carpal bone
• ~ 60-70% of carpal fractures
• 5-12% associated w/ other fractures


Scaphoid Fractures- Complications
Non-union and avascular necrosis


Scaphoid Avascular Necrosis

Initial negative radiographs **DO NOT** rule out a scaphoid fracture!
May miss up to **30%!**

MRI of the wrist is the gold standard for diagnosing occult scaphoid fractures with **100% Sensitivity**

Obviously, availability is often limited

IF clinical concern for scaphoid fracture:
- FOOSH mechanism
- Tenderness in the anatomic snuffbox

Place in a good thumb spica & arrange repeat radiographs in 10-12 days

36 y.o. male FOOSH

“Terry Thomas Sign”

AKA Scapholunate Dissociation

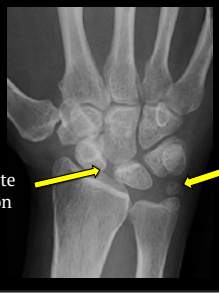
Scapholunate Dissociation

Most common and most significant ligament injury of the wrist

Distance between scaphoid and lunate should be **<3 mm** in adults

Elderly female s/p fall

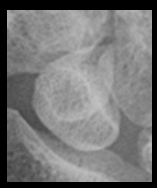
A lot going on here…



Scapholunate dissociation

Old ulnar styloid fracture

But what about that scaphoid?



"Signet Ring Sign"

Normal

Rotary Subluxation of the Scaphoid

- Scaphoid is rotated volarly
- Complete disruption of the scapholunate and radioscaphoid ligaments

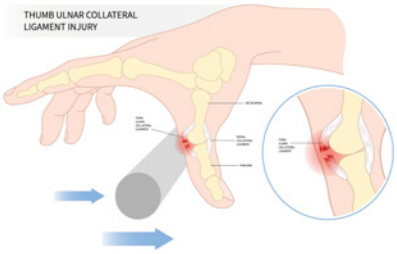
17 y.o. MVA

Remember to look at EVERYTHING!!!

Gamekeeper's AKA Skier's Thumb

- Classically an overuse injury to the **ulnar collateral ligament (UCL)**
- Now describes an acute hyperabduction injury to the UCL:
  - Skiing / Falls
  - MVA
  - Ball sports

Gamekeeper's AKA Skier's Thumb



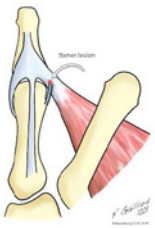
X-rays may be negative!

If significant pain at the insertion site of the UCL, or laxity with radial deviation of the thumb:

Splint the patient in position of comfort and get prompt follow-up

Stener Lesion

With a complete tear of the UCL:
- Aponeurosis of the adductor pollicis can become interposed between the UCL and insertion site
- Prevents healing
- All require surgical repair



23 y.o. male with a finger injury

Jersey Finger

Hyperextension injury at the DIP with Flexor Digitorum Profundus (FDP) in flexion

Results in FDP rupture and inability to flex at the DIP

75% involve ring finger

Jersey Finger

May have a pure tendon injury without bony injury

Physical exam is critical!!

Prognosis worsens with delayed treatment

32 y.o. with finger injury during basketball

Mallet Finger

- **Forced flexion** injury of the extended DIP joint
- Leads to rupture of the extensor tendon
- Requires splinting in full extension for good result

Mallet Finger

Again, may have a pure tendon injury without bony injury

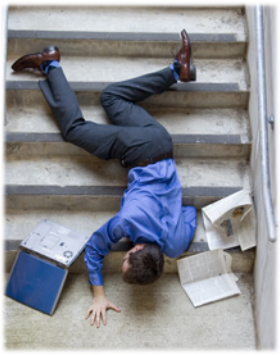
X-rays may appear normal

Physical exam is critical!!

Mallet Finger

Finger resting in ~ 45 degrees flexion

30 y.o. male falls down the steps



Galeazzi Fracture/Dislocation

Riccardo Galeazzi

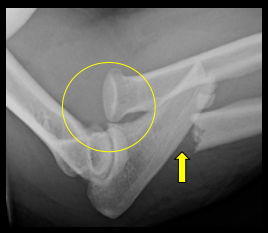
Galeazzi Fracture / Dislocation

Fracture of the mid to distal
Radius
with
Dislocation of the ***Ulna*** *(DRUJ)*

20 y.o. male falls off skateboard

Monteggia
Fracture/Dislocation

Monteggia Fracture/Dislocation



Fracture of the proximal ***Ulna***

with

Dislocation of the ***Radial Head***

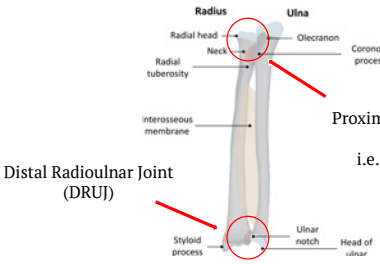
Monteggia vs Galeazzi

MUGR

Why do we care about these eponyms anyway?

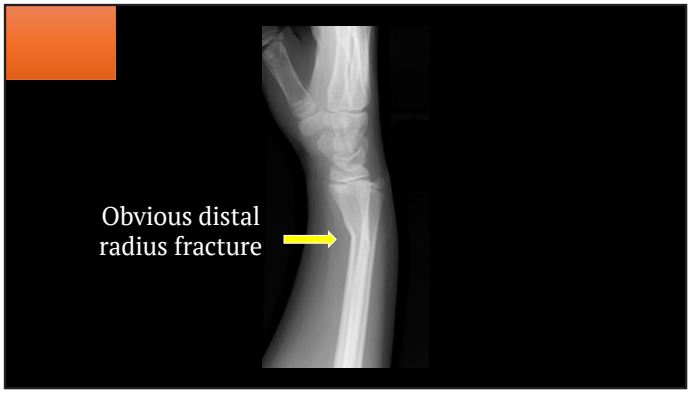
The forearm is a ring,
and it's hard to break a ring in just one spot

The forearm is a ring

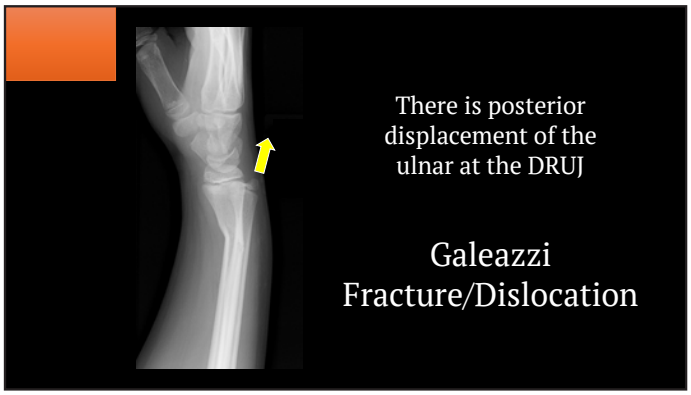


Proximal Radioulnar Joint
(PRUJ)
i.e. the Radial Head

Distal Radioulnar Joint
(DRUJ)

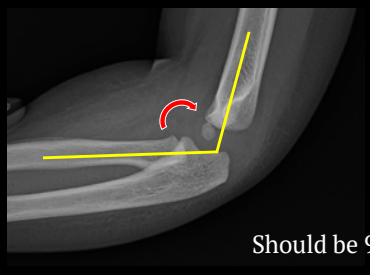


Look carefully at this one…

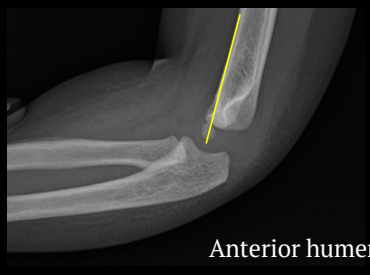


2 y.o. old with elbow pain

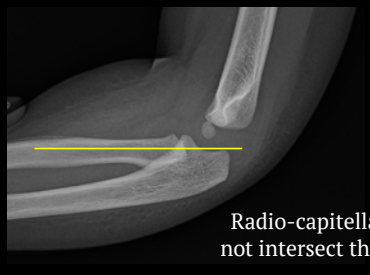
Adequacy and Alignment



Adequacy and Alignment



Adequacy and Alignment



Radial head dislocation AKA Nursemaid's Elbow

Rarely see these on plain film

When you have…

Adult patient
Fat pads without obvious fracture

= Radial Head Fracture

Radial Head Fractures

Most common elbow fracture in adults
(one third of elbow fractures)

Mechanism is usually FOOSH

Often occult, so look for those fat pads!

Management?

Radial Head Fractures

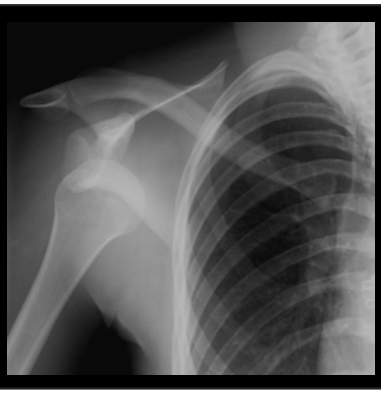
Non-displaced or
Non-depressed

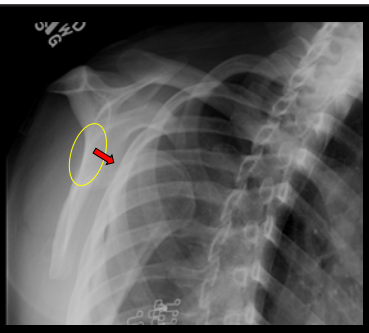
= Sling & early mobilization

Radial Head Fractures

Depressed/Displaced
>2 mm

= Call Ortho





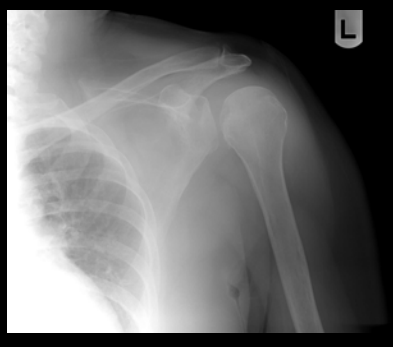
Scapular Y view

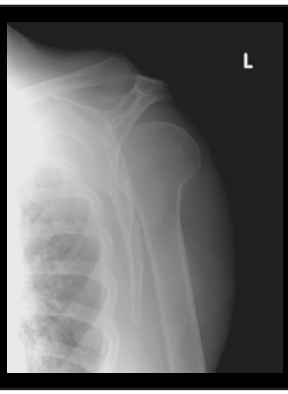
Shoulder Dislocations

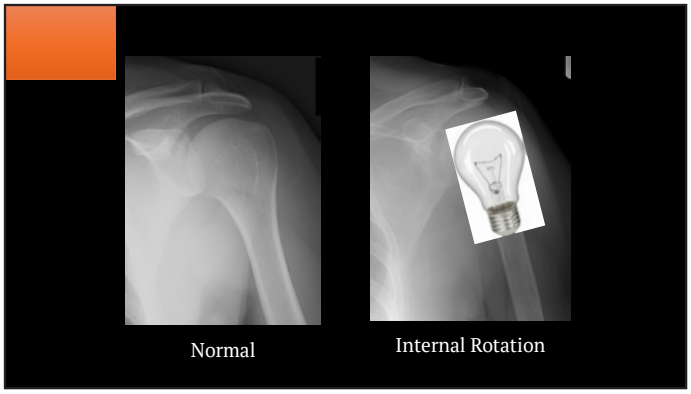
One of the most common dislocations in the ED
- 50% of all dislocations

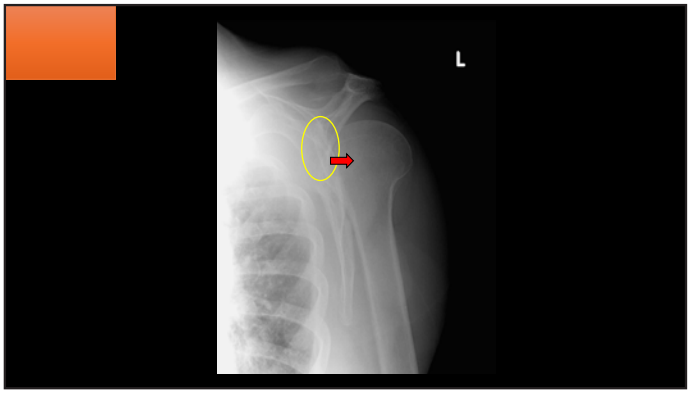
Divided into:
Anterior = **90-95%**
Posterior = **5-10%**
Inferior = **0.5%**

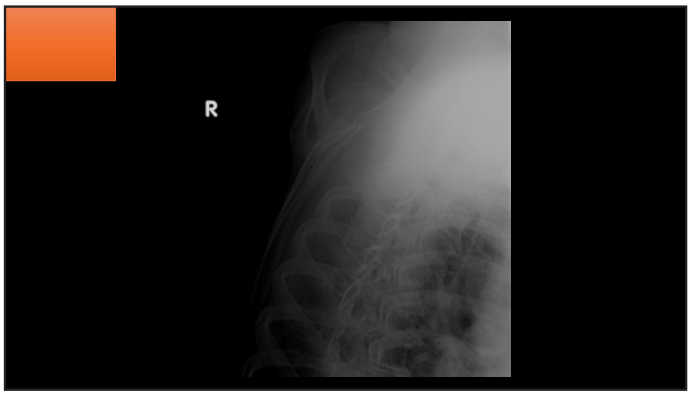
25 y.o. shoulder pain after electrocution











Luxatio Erecta

This is a pure *INFERIOR* shoulder dislocation

Least common type of shoulder dislocation

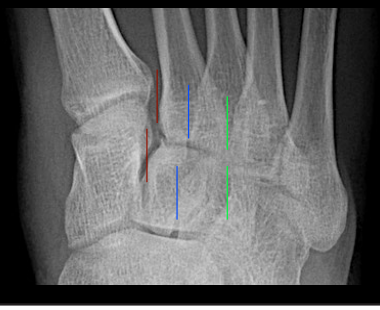
0.5%

Luxatio Erecta

Arm held in characteristic fixed upward position that is essentially *pathognomonic* for this injury

Let's look at some cases…

A = Alignment



B = Bones

C = Cartilage (Joints)

Look carefully at the articulation of the 1st and 2nd metatarsals with the cuneiforms

C = Cartilage (Joints)

Space between 1st metatarsal and medial cuneiform should be <3 mm, and similar to the other articulations

C = Cartilage (Joints)

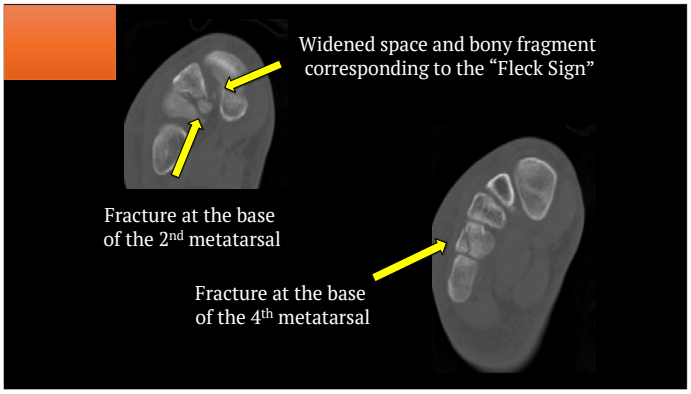
There are bony fragments in the joint space

"Fleck sign"

Lisfranc Fracture-Dislocation

Fracture and/or dislocation of the tarsal-metatarsal junction

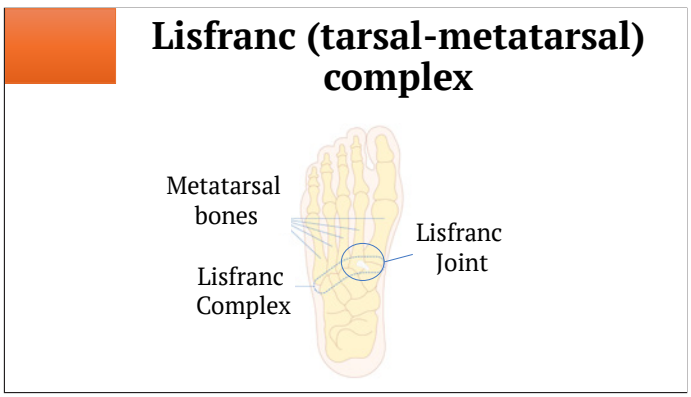
Jacques Lisfranc de St. Martin

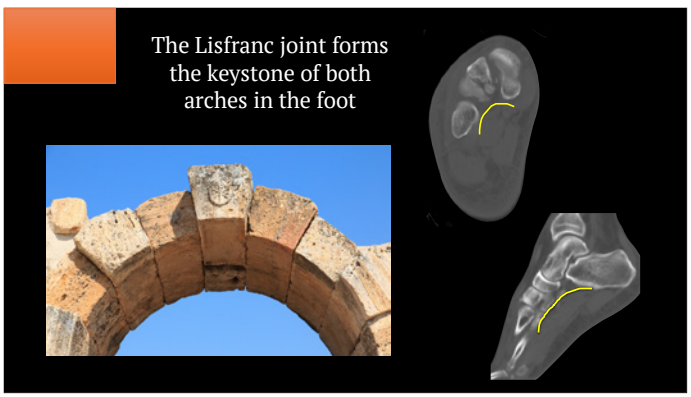

Widened space and bony fragment corresponding to the "Fleck Sign"
Fracture at the base of the 2nd metatarsal
Fracture at the base of the 4th metatarsal


A word about mechanisms…


Up to 30% from trivial mechanisms!!!


Why do we care so much?


Lisfranc (tarsal-metatarsal) complex
Metatarsal bones
Lisfranc Joint
Lisfranc Complex


The Lisfranc joint forms the keystone of both arches in the foot

Stuff that scares your pants off!

20% initial rate of misdiagnosis!!!

One of the most common reasons EM docs (and radiologists) get sued!!

Complications

Compartment syndrome

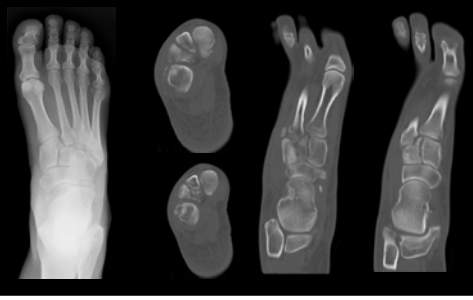
Chronic pain

Gait instability

There is a branch of the dorsalis pedis that dives between the 1st and 2nd metatarsals

With severe disruptions to the Lisfranc joint, this may be compromised

Have a very low threshold for obtaining a CT in patients with significant midfoot pain or tenderness



24 y.o. female trip and fall

Remember the *oblique* view gives a better view of the lateral structures

So, which one is it?

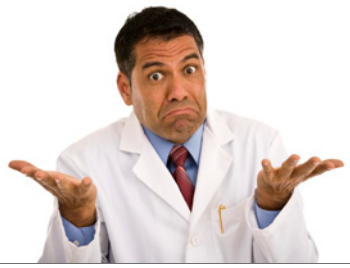
Pseudo-jones

Jones

Helpful Radiology Read:

"Transverse fracture at the base of the fifth metatarsal"

Why do we care so much?



Jones vs Pseudo-Jones

There are MAJOR differences in management and prognosis

Radiology will (almost) never make the distinction in their reports

You have to look at your own films!!

Jones vs Pseudo-Jones

Strict non-weightbearing	Hard-soled shoe and weight bearing as tolerated
High risk for delayed or non-union	Excellent prognosis
Usually requires operative fixation	Conservative management

Our Case?

Pseudo-Jones
AKA
Dancer's Fracture

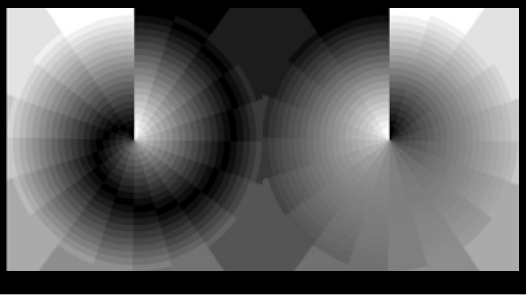
Forced inversion with foot in plantar flexion

Avulsion by the peroneus brevis tendon and plantar aponeurosis

90% of fx at base of 5th metatarsal

Occurs *proximal* to articulation between 4th and 5th MT i.e. the intermetarsal facet

In Contrast…



Jones Fracture

Fracture at junction of diaphysis and metaphysis

Extends into intermetatarsal facet

When in doubt, treat as a Jones

Posterior splint

Crutches with strict non-weightbearing

Prompt orthopedic Follow-up

So what is this?

It's normal!

This is a normal apophysis, or ossification center

- Longitudinally oriented
- Not horizontal, which would be concerning for a fracture

Unlike this, where there is a horizontal fracture across the base and through the apophysis

45 y.o. male fell off house

So, what's the diagnosis?

Calcaneal Fracture

These can be *very* subtle!!!

Look for:
Subtle heterogeneity in the bone

Disruption of the normal trabecular bone

Lucencies within the bone

Often don't see cortical disruptions

When in doubt…

Obtain a CT scan

There are dedicated calcaneal x-ray view's but their sensitivity is poor

Calcaneus Fractures

Most commonly fractured tarsal bone (60%)

75% of calcaneal fractures are intra-articular

Calcaneus Fractures

30% have associated other injuries to lower extremities

7% are bilateral

Calcaneus Fractures

10-15% have associated thoraco-lumbar compression fractures

Pretty normal

Large suprapatellar effusion

But there's more…

This is the position the pt was in when the x-ray was taken

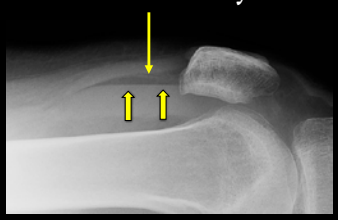
Notice the perfectly straight line in the effusion

Lipohemarthrosis

Formed by marrow fat leaking into the joint space and layering out atop the blood

By definition indicates an intra-articular fracture

Fat is dark and ***hypo***dense on x-ray



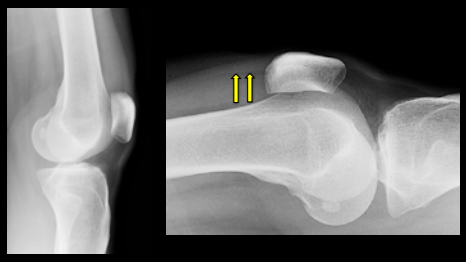
Blood is bright and ***hyper***dense

Lipohemarthrosis

These can be very subtle on an upright x-ray

I recommend flipping your lateral knee x-rays sideways

We evolved from hunters, and our brains are hard-wired to see horizons (backs of prey)



Flipping your lateral x-rays makes subtle lipohemarthroses more obvious

Segond Fracture

Small avulsion off the lateral tibial plateau

75% are associated with ACL injuries

This guy comes in after a fall

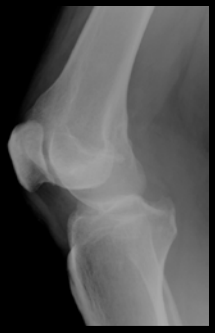
Alignment is off

No obvious fracture

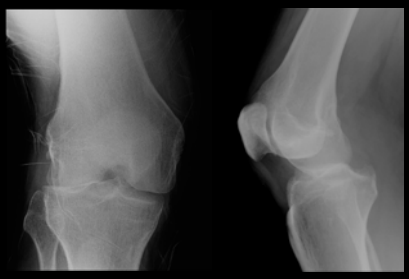
Asymmetric joint space

Femur appears rotated

Unequal spacing between the femoral condyles and tibial plateau



Knee Dislocation



Knee Dislocation

Posterior Dislocation

Grossly unstable injuries

Almost all will have ACL/PCL rupture

+ MCL in 50%

+ LCL in 28%

Knee Dislocations

Many patients will spontaneously reduce prior to radiographs

A careful physical exam for stability is critical!

Associated Injuries

1. Popliteal Artery

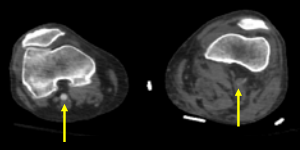
2. Nerve injuries:
- Common Peroneal
- Tibial
1.

Anterior Dislocation

Popliteal Artery Injuries

Rates reported from **3.3% – 40%**

Delay in repair > 8 hours leads to amputation in up to 86% of patients

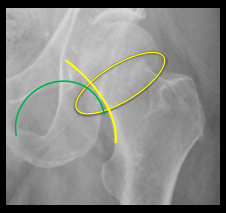


"I've fallen and I can't get up!"

Femoral Neck Fracture

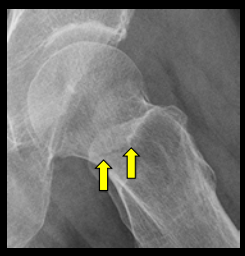
Abnormal Shenton line

Heterogeneity in the trabecular bone

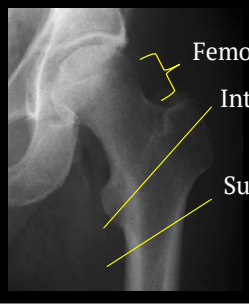


Femoral Neck Fracture

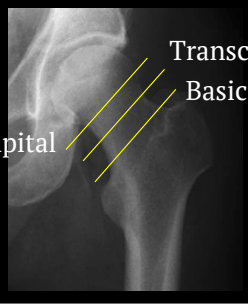
Lucency extending across femoral neck



Hip Fractures



Femoral Neck Fractures



This one is pretty obvious, but…

Hip fractures can be incredibly subtle, or even occult

Plain film sensitivities are reported as 90-98%

In clinical practice these numbers seem generous

Beware Occult Hip Fractures!!

Watch out for especially in:

- Elderly
- Female (4:1)
- Osteoporosis
- Poorly positioned patients

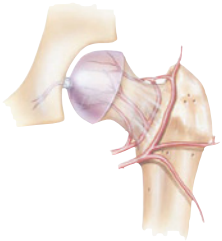
Some people with hip fractures can still walk on them!

CANNOT use trial of ambulation to rule out a hip fracture!

The Worrisome Ones

The Intracapsular Fractures:

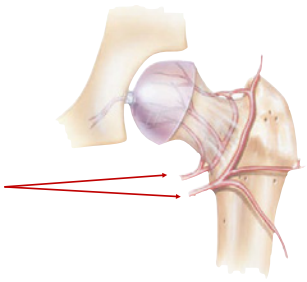
Subcapital
Transcervical
Basicervical



Why it matters...

The vascular supply to the femoral head is tenuous

The femoral circumflex and retinacular arteries extend under the capsule and feed the femoral head



Why it matters...

Displaced fractures are at high risk of AVN and require a hemiarthroplasty

MUCH bigger surgery and MUCH higher morbidity and mortality

Why it matters...

Non-displaced fractures are often treated with screw fixation

Much less invasive, allows for much faster rehab, and less morbidity and mortality

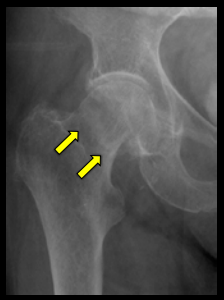
Femoral Neck Fractures

The femoral neck is made of trabecular bone

Often hard to see due to the way trabecular bone tends to break, especially with compressive forces

It often causes interdigitation of the trabeculae, rather than a frank cortical break

Femoral Neck Fracture



Abnormal bone density from interdigitated trabeculae

Compare to the other side

Femoral Neck Fracture Signs

Cortical discontinuity or sudden angulation

Disruption of normal trabeculae

Altered bone density

Foreshortened femoral neck

Abnormal angle between femoral head and neck

Radiographic Signs

Shortened femoral neck

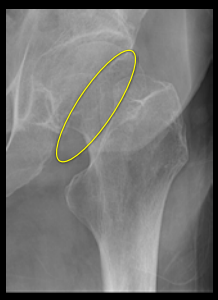
Abnormal angle

Abnormal bone density

Radiographic Signs

Abnormal bone density

Altered trabecular pattern



1 week later…

Another fall…

Abnormal angle of femoral head

Abnormal bone density

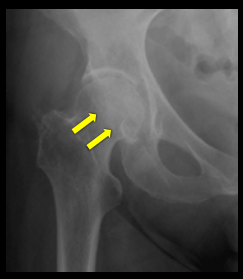
2 days later…

Watch for fake-outs

Overlying skin folds can look like lucencies in the bone

Watch for fake-outs

Overlapping bone (acetabulum and femoral head) can look like altered bone density



What should we order?

Should always get a pelvis x-ray

Sometimes dedicated hip films don't adequately visualize other structures:

Pubic rami

Acetabulum

What should we order?

Also gives you a built-in contralateral view to compare which can be very helpful

The Tubbs method for suspected hip fractures

1. Hip films – PA and Lateral
2. Pelvis
3. Femur +/- knee
4. Pre-op chest x-ray - make sure to order as a *single view supine*

If you're still suspicious…

MRI is the test of choice for occult hip fx:

Looking for bone marrow edema and hemorrhage on T1 images

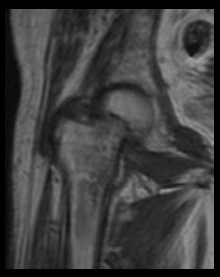
Hip MRI

100% sensitivity and specificity

Also identifies other injuries that could be causing symptoms:

Pelvic fractures

Gluteal tears or other soft tissue injuries



Take home points

- Be systematic – Remember the ABCS
- Thorough physical exam is critical
- Remember, you can have significant tendon or ligament injuries minimal x-ray findings
- Have a low threshold for getting advanced imaging with potential high-risk injuries like Lisfranc fracture-dislocations or hip fractures

SELF EVALUATION

Sticks and Stones May Break My Bones: Avoiding Orthopedic Pitfalls

1. The most commonly fractured carpal bone is:

a. Lunate
b. Triquetrum
c. Hamate
d. Scaphoid
e. Pisiform

2. A Gamekeeper's or Skier's thumb refers to an injury to which structure?

a. Flexor Digitorum Profundus
b. Ulnar Collateral Ligament
c. Extensor Pollicis Brevis
d. Extensor Pollicis Longus
e. Palmaris Longus

3. A Galeazzi Fracture-Dislocation refers to which injury pattern?

a. Fracture of the mid to distal radius with a DRUJ dislocation
b. Fracture of the mid to distal ulna with a DRUJ dislocation
c. Fracture of the radial head with a DRUJ dislocation
d. Fracture of the proximal ulnar with a radial head dislocation

4. T/F - A Jones fracture refers to a transverse fracture at the base of the fifth metatarsal that occurs proximal to the articulation between the fourth and fifth metatarsals

5. A Segond Fracture is associated with which of the following injuries:

a. Knee dislocation
b. Medial meniscus tear
c. Lateral collateral ligament tear
d. Anterior cruciate ligament tear
e. Patellar dislocation

Answer Key: 1. D, 2. B, 3. A, 4. F, 5. D

Pediatric Cardiac Emergencies: Recognizing the Subtle and Saving Lives

Emily Rose, MD, FAAP, FAAEM, FACEP

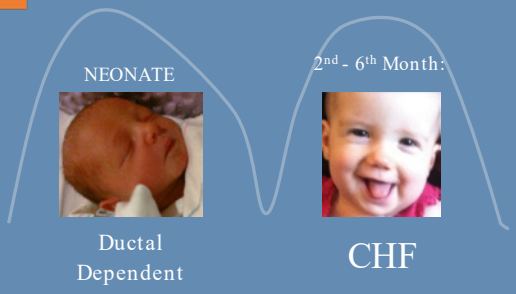
Disclosures

None

Objectives

1. Detail the presentation of congenital heart disease in children
2. Implement an algorithm for the management of ductal dependent cardiac lesions
3. Formulate a differential diagnosis for chest pain in children

Congenital Heart Disease



Ductal Dependent Lesions

Pulmonary Blood Flow ↓ CYANOSIS

Systemic Blood Flow ↓ SHOCK

Prostaglandin E1

Hypoxic/Cyanotic

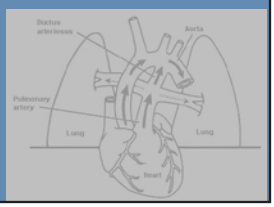
Shocky/Acidotic

Ductal Dependent Baby

PGE1 gtt
- Monitor for side effects

Consider differential diagnoses and treat concomitantly
- IV Fluids
- Antibiotics
- Glucose
- Supportive Care

TRANSFER!



Congenital Heart Disease

NEONATE

Ductal Dependent

2nd - 6th Month:

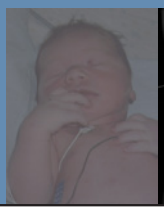
CHF

Congestive Heart Failure in Infants

Respiratory Symptoms
- Wheezing
- Retractions
- Tachypnea

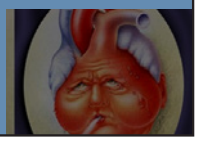
Difficulty Feeding
- Infant stress-test

Hepatomegaly



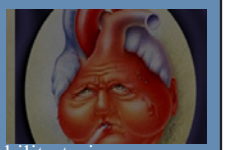
CHF in Children

- Excessive Pressure Loads
  - Left sided obstruction
- Excessive Volume Loads
  - Left-to-right shunt, regurg, anemia
- Primary Inotropic Depression
  - Myocarditis, coronary perfusion irregularities
- Rhythm Abnormalities
  - SVT
  - Heart Block



CHF Treatment

Optimize
- Preload
- Contractility
- Afterload
- Heart rate



Infants and children have limited ability to increase stroke volume, therefore cardiac output is increased by HR only

Management includes
- Diurectics
- Inotropes

12 y/o with chest pain

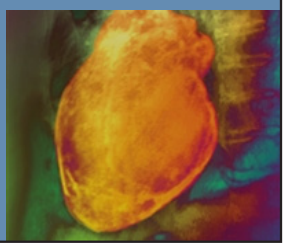
Pediatric Chest Pain

Most is NOT Cardiac

History, Physical Exam

EKG

CXR



Chest Pain in Kids is Rarely MI

Common ED chest pain diagnoses
- Musculoskeletal
- GI
- Asthma
- Psychiatric/panic (20-47%)

~0.6% of kids in ED with chest pain have cardiac diagnoses

If no risk factors 0% chance cardiac dx

Chest Pain in Kids is Rarely MI

<10% referred to cards have cardiac abnormality

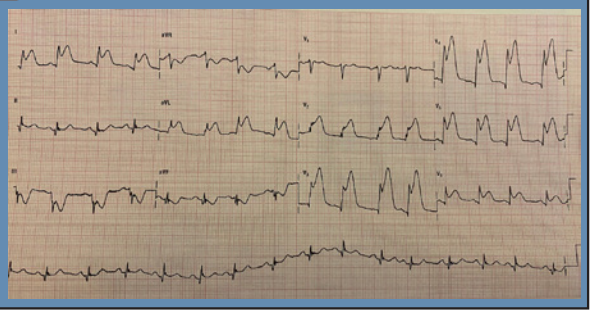
~0.6% of kids in ED with chest pain have cardiac diagnoses

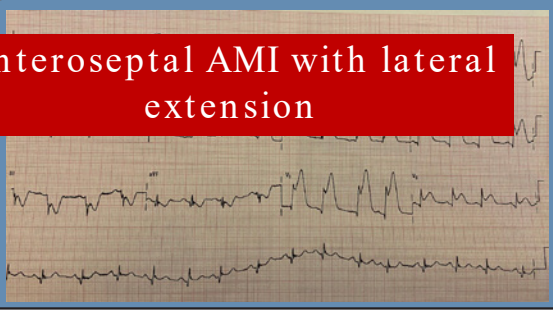
If no risk factors 0% chance cardiac dx

Chest Pain Morbidity

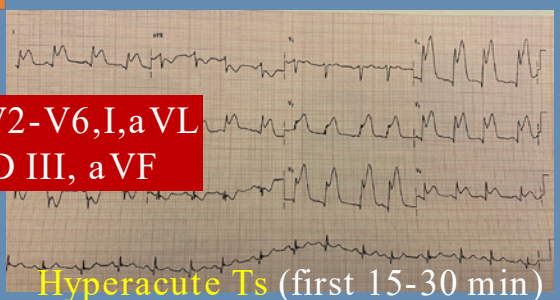
1/3 awake from sleep
1/3 miss school
Typically >1 ED visit

12 y/o with chest pain

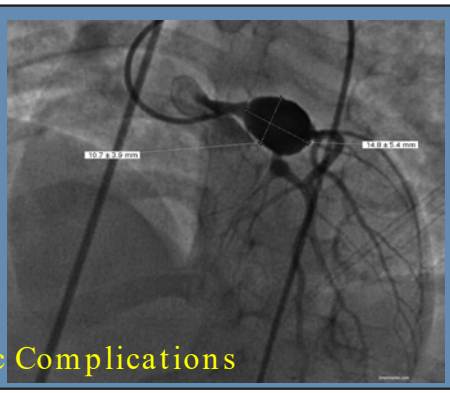




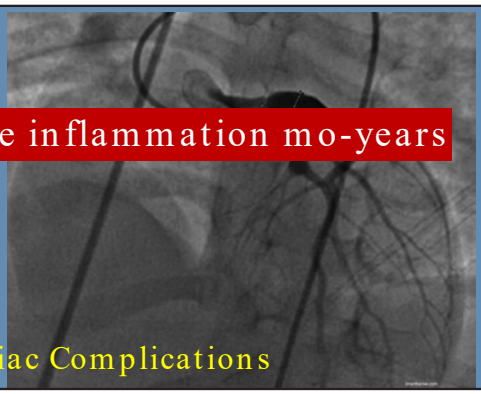
Anteroseptal AMI with lateral extension



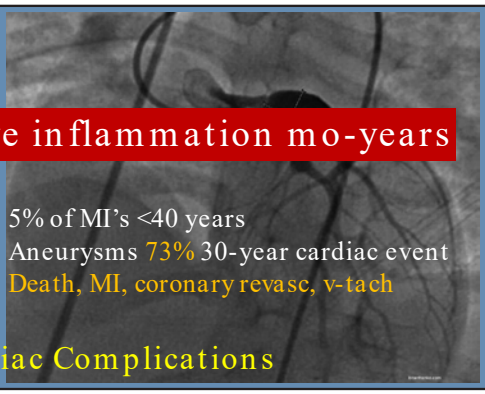
Hyperacute Ts (first 15-30 min)



KD—Cardiac Complications



KD—Cardiac Complications



5% of MI's <40 years
Aneurysms 73% 30-year cardiac event
Death, MI, coronary revasc, v-tach

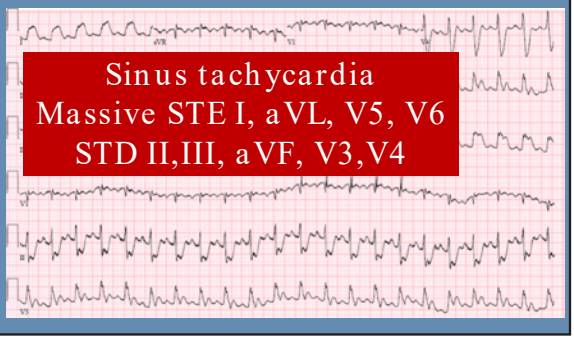
KD—Cardiac Complications

Pediatric EKG

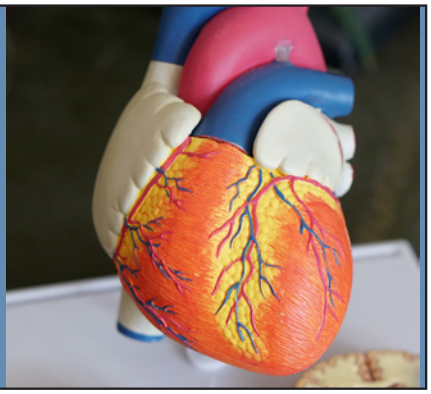
- Right Heart Dominant at Birth
- Heart Rate is Faster
- Intervals are Shorter (except QTc in infants)
- T wave inversions V1-3
- Q waves in inferior/left precordial leads

EKG Interpretation

- Be systematic—always
- Sinus?
- Intervals, rate, rhythm, axis
- Signs of ischemia—reciprocal change
- REPEAT with concern



Myocardial Infarction



Chest Pain in Kids is Rarely MI

Risk factors

- Positive family history
- Abnormal EKG
- Concerning PMH
- Abnormal exam
- Exertional CP with no other reason at high level of exertion

Why MI?

Congenital
Acquired

- Drugs
- Myocarditis
- Kawasaki Disease
- Post-transplant rejection atherosclerosis
- Congenital coronary artery anomalies

Congenital heart disease
Innate disease (eg, HOCM, aortic stenosis)
Surgery (eg, atrial switch)

Congenital Coronary Artery Anomalies

Left anterior descending artery bridging

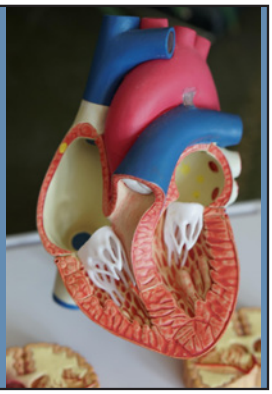
Artery tunnels under muscle leading to compression in systole

Can cause ischemia or progress to dissection

Coronary ostial stenosis/Single ostium

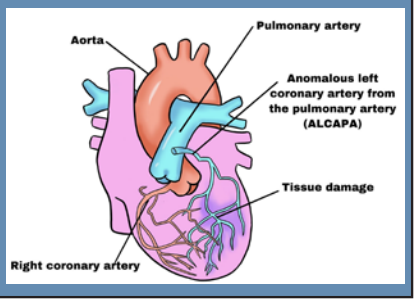
Coronary artery dissection

(pregnancy related)



Congenital Coronary Artery Anomalies

ALCAPA



ALCAPA- Anomalous left coronary artery from the pulmonary artery

Around 2 mo of life, pulmonary pressures fall → Anginal spells cause crying/colic

Consider EKG if A LOT of crying or BRUEs

Can also present in teens: Syncope/sudden death with activity

EKG: massive MI
CXR: cardiomegaly

Treatment: bypass or re-implantation

Presentation

Infant
Crying
Feeding problems/ vomiting
Cold extremities/ pallor

Older children
Similar to adult

Exam
Normal
Tachycardia
Signs of Failure

Diagnosis

EKG

May be across >1 region of heart

Even a single deep and/or wide q wave can be of concern
- >3 small squares in duration are likely ischemic
- >3-4 mm deep concerning (or 25% depth of qrs)
- Q wave notching can represent ischemia

Troponin
- Can also be elevated by HF, cardiac contusion, myocarditis, PE, sepsis, strenuous exercise, drugs, tachyarrhythmias, Covid-19 (MIS-C)

Treatment

No great studies

Adult principles applied
PCI
ASA 5 mg/kg/d
Clopidogrel 0.2 mg/kg/day

Heparin for all PCI patients
70-100 U/kg □10-15 U/h

If thrombolysis with TNK
30 mg for < 60 kg
35 mg for 60-70
40 mg for >70 kg

12 y/o after new seizure

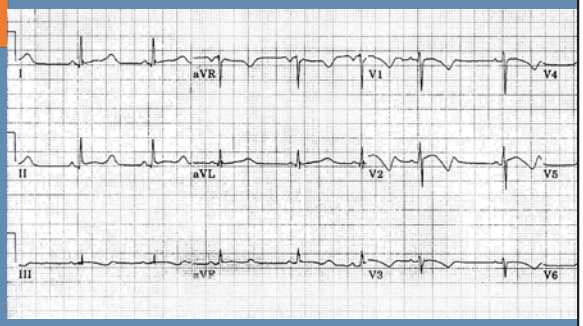
Concerning HISTORY in syncope

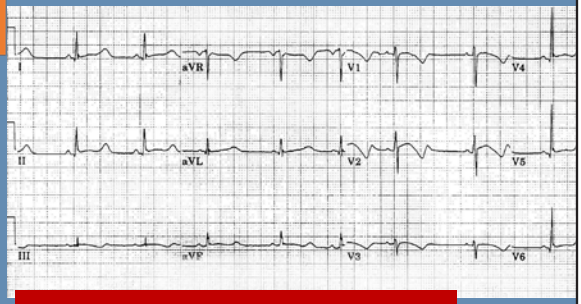
Exertion
No prodrome
Preceding chest pain/palpitations
Triggered by fright/loud auditory stimulus
Posturing or "seizure-like" event

Reading the ECG in Syncope

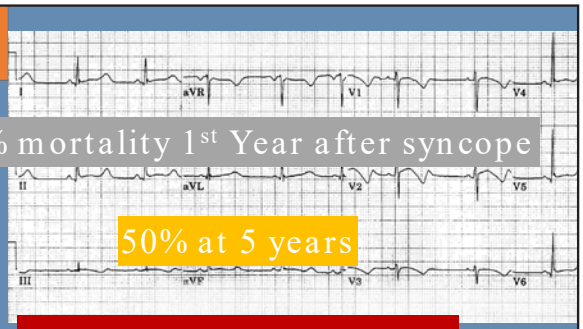
Rate, rhythm, intervals, ischemia

WPW
Prolonged/short QT
Brugada
Arrhythmogenic right ventricular cardiomyopathy
Hypertrophic cardiomyopathy





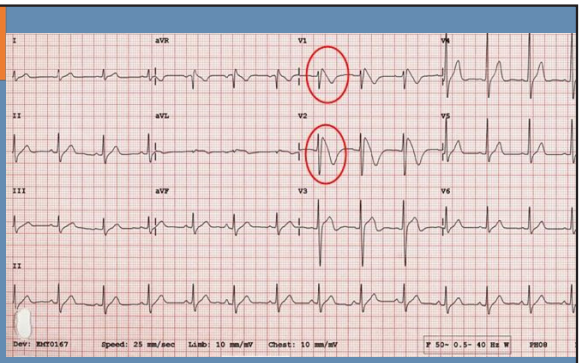
Long QT Syndrome

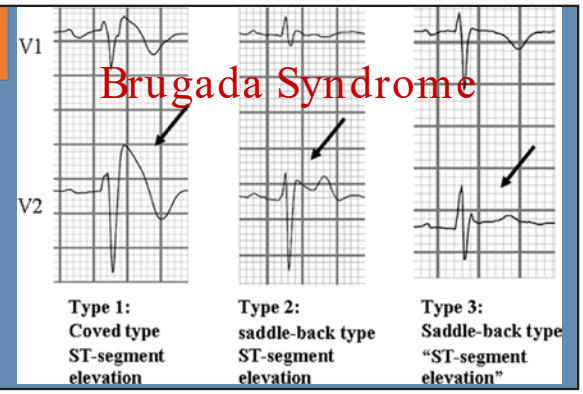


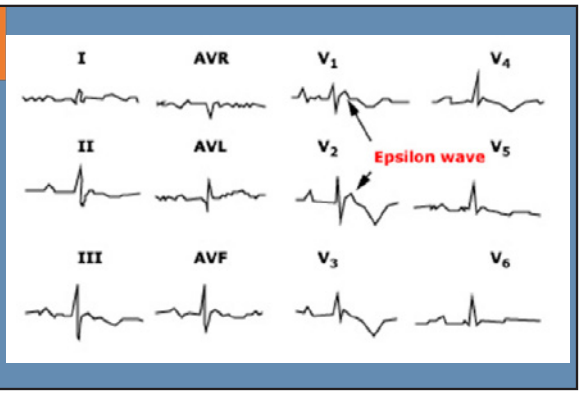
20% mortality 1st Year after syncope

50% at 5 years

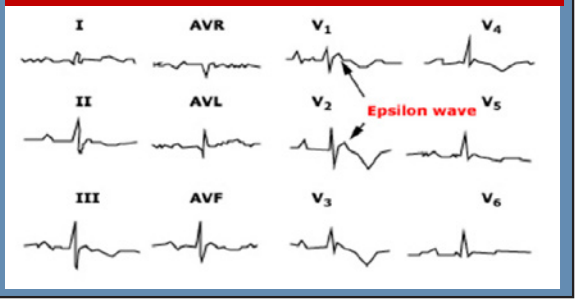
Long QT Syndrome





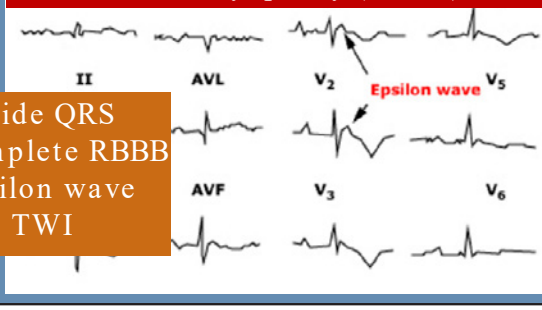


Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)



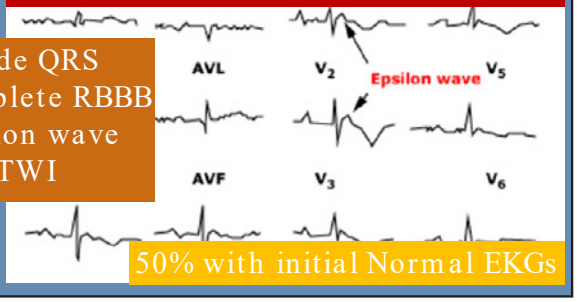
Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)

Wide QRS
Incomplete RBBB
Epsilon wave
TWI



Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)

Wide QRS
Incomplete RBBB
Epsilon wave
TWI



50% with initial Normal EKGs

Reading the ECG in Syncope

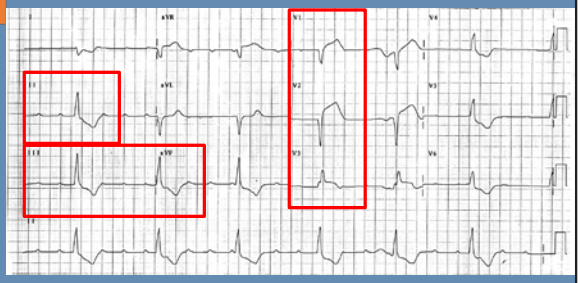
Rate, rhythm, intervals, ischemia

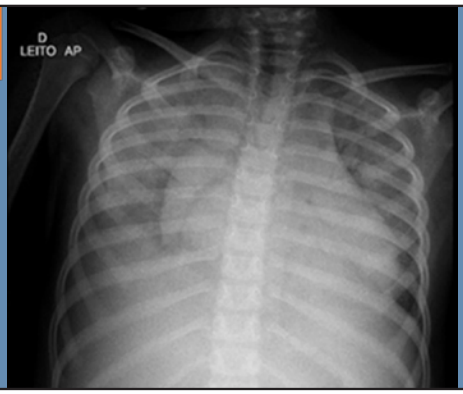
WPW
Prolonged/short QT
Brugada
Arrhythmogenic right ventricular cardiomyopathy
Hypertrophic cardiomyopathy

New Onset Seizure?

EKG was it V-tach? Pathologic syncope?

12 y/o with chest pain





Labs

138 | 103 | 11 | 110
4.5 | 27 | 0.6

12.6
15.8 | 321
36.8

Ca: 9.2
Mg: 1.9
Ph: 4.2

LFT: wnl
AST: 148
GGT: 21

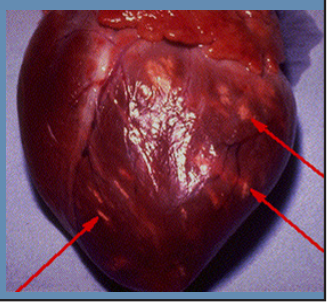
BNP: 26
ESR: 66
CRP < 0.5

CPK: 832
Trop: 32.6

Myocarditis

Myocardial Inflammation/ Necrosis

YOUNG — FULMINANT DISEASE

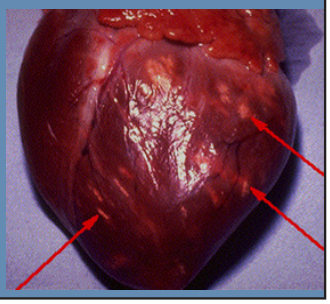


Myocarditis Sx

<12 years: Resp, GI

Adolescent: Cardiac

Pain only with pericardial involvement



Congestive Heart Failure in Infants

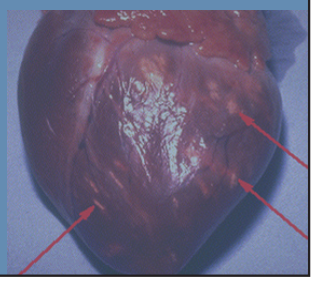
Respiratory Symptoms
Wheezing
Retractions
Tachypnea

Difficulty Feeding
Infant stress-test

Hepatomegaly

Myocarditis Sx

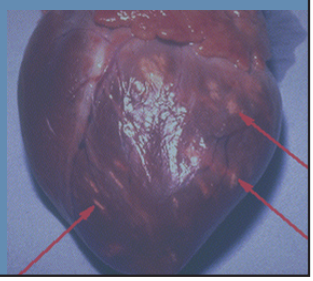
Dyspnea, Chest Pain, CHF, Palpitations, Sudden Death



Myocarditis Sx

<12 years: Resp, GI

Irritability, lethargy, diaphoresis

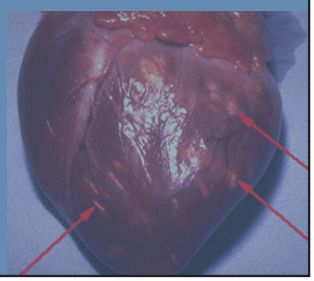


Myocarditis Sx

<12 years: Resp, GI

Irritability, lethargy, diaphoresis

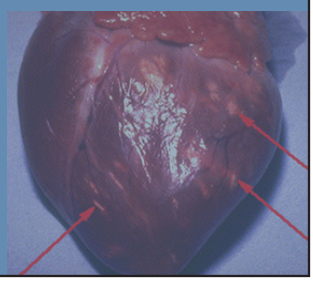
Cough, tachypnea, abd pain, n/v



Myocarditis Sx

Chest pain, exercise intolerance, palpitations

Adolescent: Cardiac

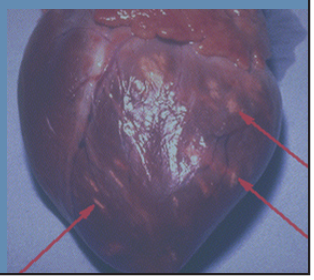


Myocarditis Sx

<12 years: Resp, GI

Adolescent: Cardiac

Pain only with pericardial involvement



Myocarditis Sx

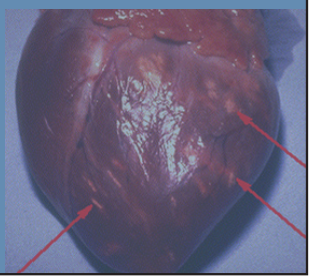
Most common signs:

hypoperfusion

lethargy, syncope, seizure

ALL had abnormal EKGs

Persistent tachycardia



Normal Vital Sign Ranges by Age

	Age	Weight (kg)	Respiratory Rate (bpm)	Heart Rate (bpm)	Systolic BP (mm Hg)
>60	Premie	<3	30-60	120-160	52 ± 10
	Newborn/ Neonate	3-4	30-60	90-160	60 ± 10
	1-6 mo	4-6	24-30		
	6-12 mo	6-10	24-30		
	1-2 yr	10-13	20-24		
	2-4 yr	13-17	20-24		
>30	4-6 yr	17-20	20-24		
	6-8 yr	20-25	12-20	60-120	105 ±13
	8-10 yr	25-30	12-20	60-120	110 ± 15
	10-12 yr	30-40	12-20	60-120	112 ± 15
	12-14 yr	40-50	12-20	60-120	115 ± 20
>15	14-adult	>50	10-16	60-120	120 ± 20

Tachypnea
Tachycardia

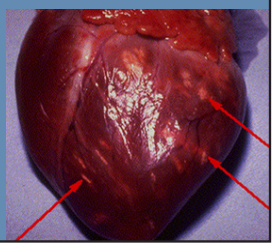
[2 x age] + 90

Myocarditis Sx

<12 years: Resp, GI

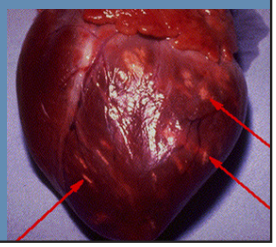
Adolescent: Cardiac

Tachycardia (50%)
AST most sensitive test (85% in one study)
Troponin (71%)



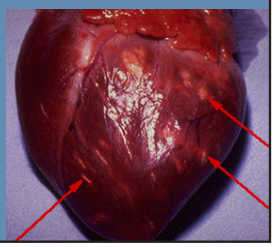
Myocarditis

25% Mortality Rate



Myocarditis Treatment

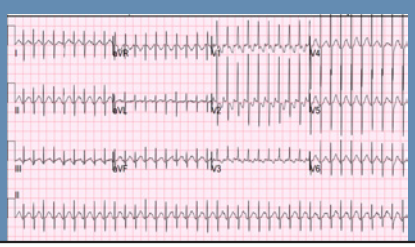
Caution with fluid/diuretics
Inotropic support
ECMO



ARRHYTHMIAS

SVT

>220 Infant >180 Child



SVT

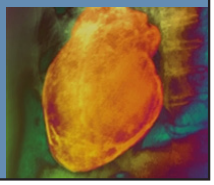
90% accessory pathway
~50% have normal hearts
~50% occur in infancy
~1/4 have WPW
~1/4 have CHD

SVT Management

Vagal maneuvers
Adenosine
Cardioversion
Amiodarone
Procainamide
Verapamil (NOT in young)
Beta-blockers
Rapid atrial pacing

Pediatric Chest Pain

Most is benign
History (including FH) and PE very important
EKG and CXR highest yield (but require correct interpretation)



Thank you!

emilyros@usc.edu
@EmilyRoseMD1

SELF EVALUATION

Pediatric Cardiac Emergencies: Recognizing the Subtle and Saving Lives

1. Which of the following is the most common cause of chest pain in children and adolescents?

a. Myocardial ischemia
b. Costochondritis
c. Pneumothorax
d. Pericarditis

2. A 14-year-old boy collapses during basketball practice. He regains consciousness within 1 minute. Which diagnosis is most concerning?

a. Vasovagal syncope
b. Hyperventilation syndrome
c. Long QT syndrome
d. Dehydration

3. Which of the following historical features is most suggestive of a cardiac cause of syncope in a child?

a. Syncope after prolonged standing in a warm environment
b. Syncope with prodromal lightheadedness and nausea
c. Syncope occurring suddenly without warning during exercise
d. Syncope triggered by emotional distress

4. Which of the following is the most common cause of myocardial infarction in children?

a. Atherosclerotic coronary artery disease
b. Kawasaki disease
c. Hypertrophic cardiomyopathy
d. Myocarditis

5. T/F - Prostaglandin E1 infusion is indicated in suspected ductal dependent cardiac lesions and should be initiated in a hypoxic baby not responsive to oxygen or a hypotensive/shocky infant not responsive to a fluid bolus.

Answer Key: 1. B, 2. C, 3. C, 4. B, 5. T

FACULTY

Kevin Klauer, DO, EJD

Kevin Klauer, DO, EJD, of Winter Park, Florida, is currently the Chief Executive Officer of the American Health Information Management Association and was formerly the System Chief Medical Officer HCA Florida and Ocala hospitals. He formerly served as CEO of the American Osteopathic Association and the Chief Medical Officer and Chief Risk Officer for TeamHealth. He holds the following faculty appointments: Clinical Asst. Professor, Michigan State University College of Osteopathic Medicine; Clinical Asst. Professor, Ohio University Heritage College of Osteopathic Medicine. He is the co-author of five risk management books: *Bouncebacks: Critical Care*, *Bouncebacks: Pediatrics, Emergency Medicine*; *Bouncebacks: Medical and Legal* (1st and 2nd Editions) and *Risk Management and the Emergency Department: Executive Leadership for Protecting Patients and Hospitals*. Dr. Klauer also served as the American College of Emergency Physicians Council Speaker and subsequently as a member of their Board of Directors. Dr. Klauer earned his Executive JD, with honors, from Concord Law School in 2011.

You may contact Dr. Klauer with your comments or questions at kevinklauer1@gmail.com.

Medical Documentation and Liability: Avoiding Common Pitfalls

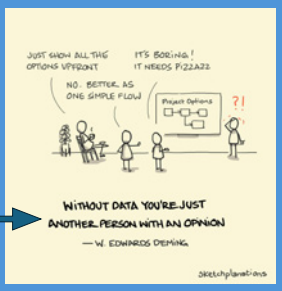
Kevin Klauer, DO, EJD

Documentation
Your Voice Matters!

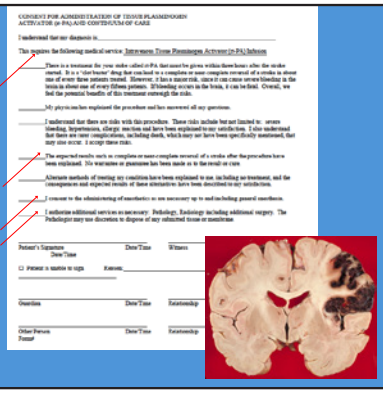
Kevin Klauer, DO, EJD, FACEP, FACOEP
Assistant Clinical Professor
Michigan State University College of Osteopathic Medicine
Ohio University Heritage College of Osteopathic Medicine
Orlando College of Osteopathic Medicine

So many ways to be "wrong" even when you're right!

10+ Examples!



Informed Consent, When?

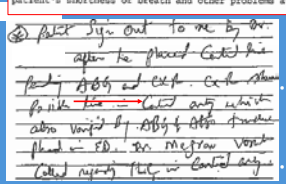


Medical Legal Case

- October 18, 2018, at 1510
- 72 y/o male presented to the ED
- CC: Cough and shortness of breath
- HPI: He reported a non-productive cough with subjective fever for 5 days.
- BP: 176/96, HR: 104, RR: 18, T: 99.4, SaO_2: 92%
- PMHx: COPD, NIDDM
- P .E.: Ill-appearing, but non-toxic with diminished lung sounds and mild increased work of breathing.

REASON FOR CONSULTATION:
Central line placement in right common carotid artery.

HISTORY OF PRESENT ILLNESS:
I was called to the emergency room to see the patient in whom a central line had been placed in his common carotid artery. At this point, our plan will be to take him to the operating room, remove the line and place a central line in the correct position. Unless there is significant bleeding from the insertion site, we will place a Perclose and hold pressure and hope that there is no bleeding given the patient's shortness of breath and other problems at this time.



gm IVPB, Azithromycin 500mg IVPB, IV fluids, aerosols and prednisone.

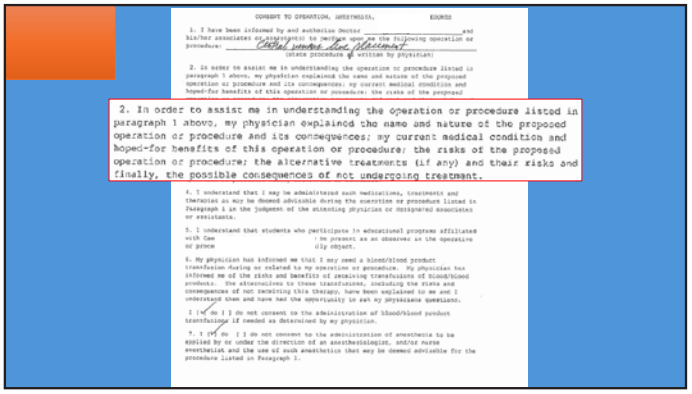
- Central venous line insertion (Rt. internal jugular)
  - No ultrasound guidance
- Shift change: "ABG and CXR for line placement pending"

Verdict(S)

- Court directed defense verdict on the neg
- Jury only allowed to deliberate on the que
- $100,000 for the lack of informed consent and the balance for additional care.

7/20/19
Demand: $500,000

Plaintiff verdict
$121,145



2. In order to assist me in understanding the operation or procedure listed in paragraph 1 above, my physician explained the name and nature of the proposed operation or procedure and its consequences; my current medical condition and hoped-for benefits of this operation or procedure; the risks of the proposed operation or procedure; the alternative treatments (if any) and their risks and finally, the possible consequences of not undergoing treatment.

Juror Feedback

"While understanding this is a complication, because it involved an artery, they believed that this was a real "accident" and the agreement was there was insurance to pay for an accident, and it would not hurt the doctor."

Risk Reduction Recs

1. The Doctrine of Informed Consent requires:
   a. Capacity to consent or refuse
   b. Risks and benefits of treatment options, including non-treatment
   c. Disclosure of conflicts of interest.
2. Patient autonomy
3. Reasonable patient v. Reasonable physician standard
4. Written consent if helpful, but not worth paper it's written on without the informed discussion

Administrative Info

ADDENDUM: Billing coders have asked me to do a procedure note.

Again, the original dictation was read. The second page of the dictation under ED course outlines the procedure. Again, I will re-read what has already been dictated for their benefit.

"DX STATES ACUTE RECTAL PACING WITH NO MENTION OF IT IN THE DICTATION. PLEASE CLARIFY."

Signing Does Not = Editing

(_____); however, she did become aggressive and violent in the emergency department again necessitating medication with Haldol and Ativan SINCE SHE IS ALLERGIC TO GEODON, chloroform was the only thing that really works for her awaited for its arrival. She was given another 2 mg of Ativan. The patient's care was discussed with Dr

Anger Management

ADDENDUM: Please see the previous dictation. A complaint was made by the patient and his wife who said that I was polite, but also disrespectful. They said that while I was going over the x-rays that I used profanity in front of their 8-year-old daughter, which I do not remember at all. It is possible I used a "hell or damn" but I do not remember specifically saying that. The wife also states that I misdiagnosed the patient with muscle spasm.

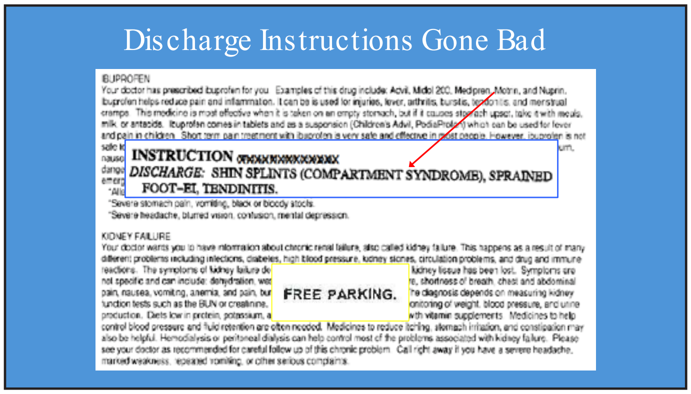
DIAGNOSIS:
1. Acute cervical strain.
2. Upper respiratory infection.
3. Tobacco abuse.

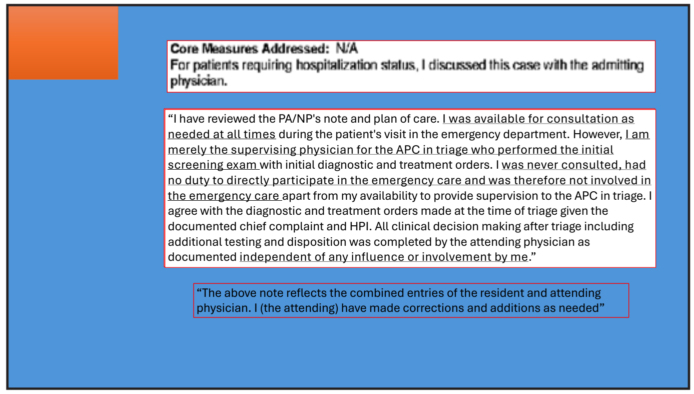
ADDENDUM: The patient is a 44 year-old female, who is a patient I admitted to the Hospitalist Service with an upper GI bleed. I attempted a central line in the right femoral vein and for the first time in 22 years after many experiences putting in a central line, I actually punctured the bladder and aspirated urine from the needle that accepts the guidewire.

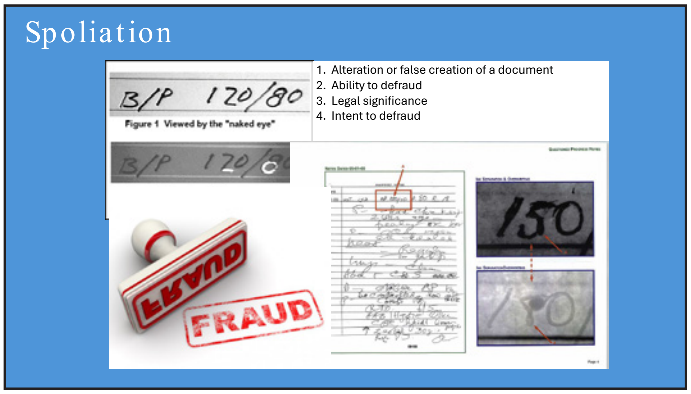
I am adding to my diagnosis of iatrogenic bladder puncture, unintentional, under sterile conditions.

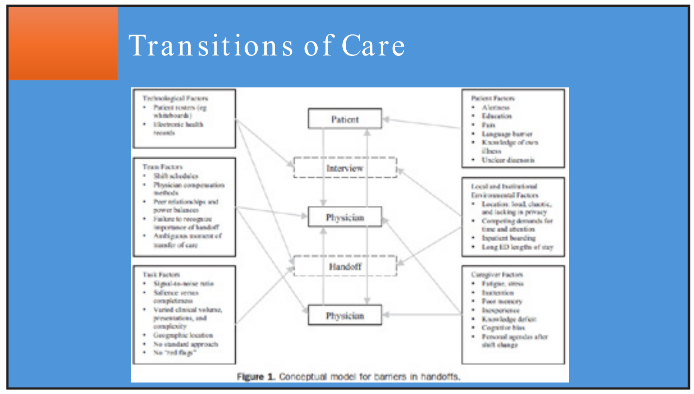
Documenting Errors

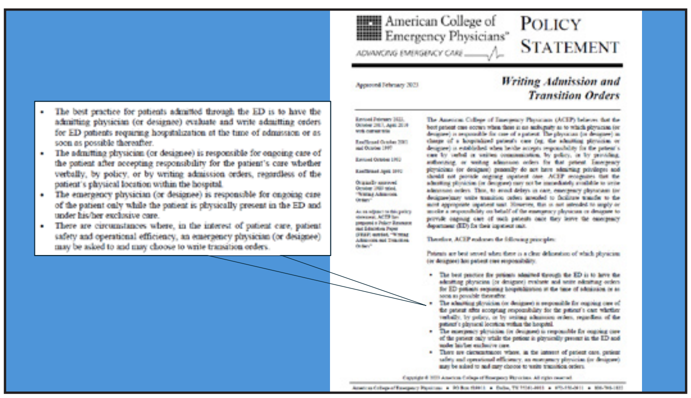
LABS/X-RAY: She had an emergent ultrasound, transvaginal which showed a normal uterus, normal right ovary and a small 1.4 cm cyst in the left. There is a moderate amount of fluid in the cul-de-sac surrounding the left adnexa as well.

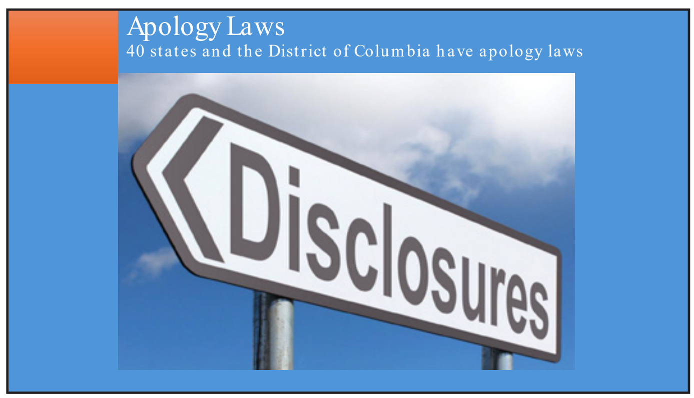

Discharge Instructions Gone Bad
IBUPROFEN
INSTRUCTION
DISCHARGE: SHIN SPLINTS (COMPARTMENT SYNDROME), SPRAINED FOOT-EI, TENDINITIS.
KIDNEY FAILURE
FREE PARKING.

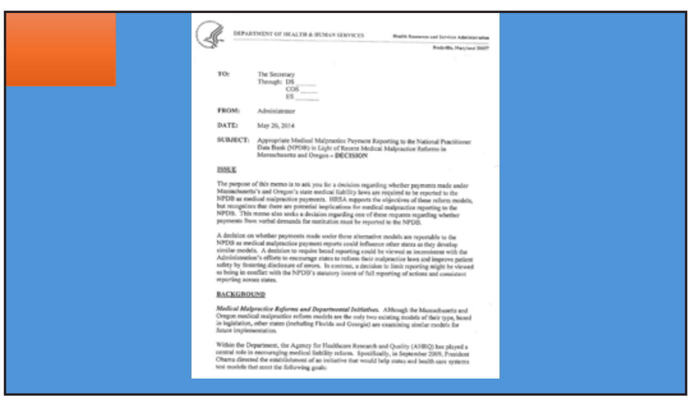

Core Measures Addressed: N/A
For patients requiring hospitalization status, I discussed this case with the admitting physician.
"I have reviewed the PA/NP's note and plan of care. I was available for consultation as needed at all times during the patient's visit in the emergency department. However, I am merely the supervising physician for the APC in triage who performed the initial screening exam with initial diagnostic and treatment orders. I was never consulted, had no duty to directly participate in the emergency care and was therefore not involved in the emergency care apart from my availability to provide supervision to the APC in triage. I agree with the diagnostic and treatment orders made at the time of triage given the documented chief complaint and HPI. All clinical decision making after triage including additional testing and disposition was completed by the attending physician as documented independent of any influence or involvement by me."
"The above note reflects the combined entries of the resident and attending physician. I (the attending) have made corrections and additions as needed"

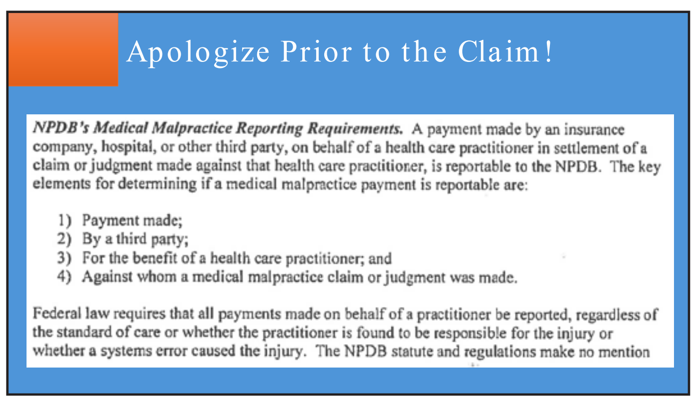

Spoliation
1. Alteration or false creation of a document
2. Ability to defraud
3. Legal significance
4. Intent to defraud
B/P 120/80
Figure 1 Viewed by the "naked eye"
FRAUD
150


Transitions of Care
Patient
Interview
Physician
Handoff
Physician
Figure 1. Conceptual model for barriers in handoffs.


American College of Emergency Physicians
POLICY STATEMENT
Writing Admission and Transition Orders
The best practice for patients admitted through the ED is to have the admitting physician (or designee) evaluate and write admitting orders for ED patients requiring hospitalization at the time of admission or as soon as possible thereafter.
The admitting physician (or designee) is responsible for ongoing care of the patient after accepting responsibility for the patient's care whether verbally, by policy, or by writing admission orders, regardless of the patient's physical location within the hospital.
The emergency physician (or designee) is responsible for ongoing care of the patient only while the patient is physically present in the ED and under his/her exclusive care.
There are circumstances where, in the interest of patient care, patient safety and operational efficiency, an emergency physician (or designee) may be asked to and may choose to write transition orders.


Apology Laws
40 states and the District of Columbia have apology laws
Disclosures


DEPARTMENT OF HEALTH & HUMAN SERVICES
TO: The Secretary
FROM: Administrator
DATE: May 20, 2014
ISSUE
BACKGROUND


Apologize Prior to the Claim!
NPDB's Medical Malpractice Reporting Requirements. A payment made by an insurance company, hospital, or other third party, on behalf of a health care practitioner in settlement of a claim or judgment made against that health care practitioner, is reportable to the NPDB. The key elements for determining if a medical malpractice payment is reportable are:
1) Payment made;
2) By a third party;
3) For the benefit of a health care practitioner; and
4) Against whom a medical malpractice claim or judgment was made.
Federal law requires that all payments made on behalf of a practitioner be reported, regardless of the standard of care or whether the practitioner is found to be responsible for the injury or whether a systems error caused the injury. The NPDB statute and regulations make no mention

Laboratory Diagnostic Testing

- **Own what you order**
  - Delayed results
  - Patient elopements/Lost to follow up
- **Don't order what you don't need**
- **Address abnormals: MDM**
- **Common errors: Physiologic connection to Dx**
  - Creatinine
  - Potassium, Sodium, etc.
  - WBC and differential

Medical Legal Case

- March 28, 2021: Initial ED Visit
- CC: Fever and sweating for 2 week
  - No other symptoms
  - He had been recently Tx with Azithromycin and was not improving
- PMHx: None
- P.E.: Thorough, but no abnormalities noted

Management

- CBC: NL (excluding WBC left shift)
- Two sets of blood Cxs were ordered
- Ceftriaxone 2gm IVPB administered
- Discharged: Rx for Doxycycline
- Follow up with PCP in 48 hours
- "You will be notified of positive results."

Outcome

- First blood culture returned 2 days later
  - "Gram positive cocci"
  - Reported to the ED
  - EP determined it was a contaminant
  - Patient not notified
- Final: Staphylococcus aureus
  - Reported to the ED
  - Patient not notified

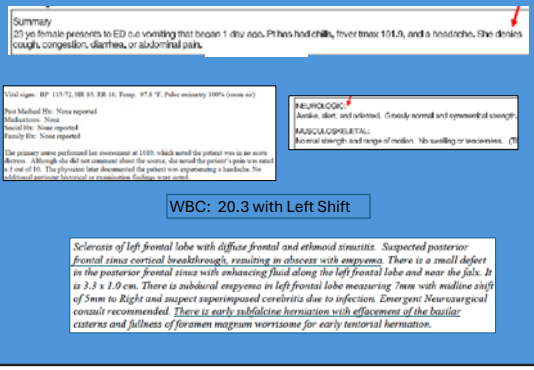
Outcome

- April 10, 2021: ED visit #2
- Patient presented to different ED
- CC: Fever, Chills, Rash
- Dx: Subacute Bacterial Endocarditis
  - Mitral Valve Replacement

Demand letter: $1,800,000

Settlement
Dr. 1: $700,000
Dr. 2: $400,000
Hospital: $400,000

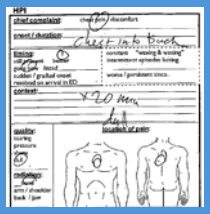
- Outpatient blood cultures may create more risk than they are worth.
- 50% false positive rate
- Gram + cocci: Staph aureus, Viridans Strep
  - Two of the most common causes of SBE
- Staphylococcus epidermidis is also a Gram + cocci
- When looking for bad disease …
- Most don't need it and those that do need admission



The DDX

- Avoid all-inclusive drop downs for billing
  - New documentation guidelines
- Included in the DDX ⟶ Must address in MDM
  - The choice not to pursue a Dx …?

Nursing/Physician Documentation Discrepancies



Chaperones

AMA *Code of Medical Ethics*

1.2.4 Use of Chaperones

Efforts to provide a comfortable and considerate atmosphere for the patient and the physician are part of respecting patients' dignity. These efforts may include providing appropriate gowns, private facilities for undressing, sensitive use of draping, and clearly explaining various components of the physical examination. They also include having chaperones available. Having chaperones present can also help prevent misunderstandings between patient and physician.

Physicians should:

(a) Adopt a policy that patients are free to request a chaperone and ensure that the policy is communicated to patients.

(b) Always honor a patient's request to have a chaperone.

(c) Have an authorized member of the health care team serve as a chaperone. Physicians should establish clear expectations that chaperones will uphold professional standards of privacy and confidentiality.

(d) In general, use a chaperone even when a patient's trusted companion is present.

(e) Provide opportunity for private conversation with the patient without the chaperone present. Physicians should customize inquiries or history taking of a sensitive nature during a chaperoned examination.

AMA Principles of Medical Ethics: I,IV

Shared Decision Making

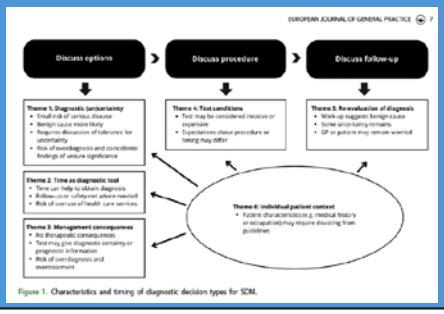


Figure 1. Characteristics and timing of diagnostic decision types for SDM.

Offering v. Recommending

Scribe Abuse?

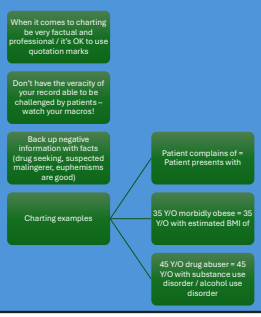
FAQs, regarding scribes, published by The Joint Commission on July 12, 2012.

- "Scribed for Dr. X by name of the scribe and title" with the date and time of the entry.
- "Scribed" entries-- the physician or practitioner must actually sign or authenticate through the clinical information system.
- The authentication must take place before the physician or practitioner and scribe leave the patient care area
- Scribes cannot perform CPOE

21st Century Cures Act

- One provision allows patients access to their EHR
- They can read and potentially challenge what's written (incorrect? / disparaging? / test necessity?)
- Physicians can block access to the chart, but only for a very defensible reason (e.g., spousal abuse)
- Big fines are allowed (up to $1m per chart!!!)

21st Century Cures Act



- 9/1: ED Visit.
- 9/3: Gradual paralysis. Pt researched symptoms on internet: transverse myelitis.
- 9/4: Admitted to other hospital.
  - Dx: Transverse Myelitis.
  - Reviewed 9/1 ED record.
- "I saw the Munchausen comment …my family exploded."

I was not trying to find something wrong with me; I was ill. I greatly resent the mention of Munchausen Syndrome. That is a dx that is considered only after time, repeat MD, ER visits, and can affect the attitude of any following professionals. To put that in a summary is irresponsible.

If I ever decide to 'fake' an illness, I will choose something more glamorous than bladder/bowel retention, numb buttocks, rectum, and urethra.

I demand that the 'Munchausen' comment be redacted. I have very real, serious health issues that are unusual and I do not need that following me. If this ER report cannot be corrected to reflect the errors I want it purged.

Do not bill me for anything.

Psychiatric Diagnoses without basis exposes you to serious liability!

Remember! Your Patients are Reading Their Records

Thank you!

SELF EVALUATION

Medical Documentation and Liability: Avoiding Common Pitfalls

1. T/F - Informed consent requires not just a signed form but also a meaningful discussion of risks, benefits, and alternatives.

2. In the case where blood cultures returned positive for gram-positive cocci, what key error led to a malpractice claim?

a. Ordering unnecessary cultures
b. Failing to notify the patient of positive results
c. Misinterpreting the chest x-ray
d. Not prescribing antibiotics

3. T/F - Documentation that conflicts between nurses and physicians can expose providers to legal risk.

4. Which of the following is a risk of using drop-down menus for differential diagnoses?

a. Encourages more detailed charting
b. Ensures billing compliance
c. May include irrelevant diagnoses that must be defended
d. Automatically generates discharge instructions

5. T/F - Patients now have the legal right to access their electronic health records and can challenge or demand removal of disparaging comments.

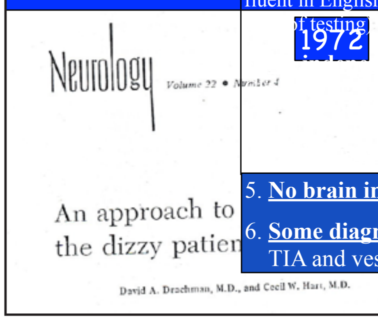
6. What is the main risk of assigning unsupported psychiatric labels (e.g., “Munchausen”) in the medical record?

a. Better continuity of care
b. Enhanced billing documentation
c. Exposure to significant liability and patient backlash
d. Avoiding repeat testing

Answer Key: 1. T, 2. B, 3. T, 4. C, 5. T, 6. C

Diagnosing Acute Dizziness in the Emergency Department
Jonathan A. Edlow, MD

Episode I
The Phantom Paradigm



cts
rolled over a 2-year period (21 inadequate data) ➛ 104 (9 rejected gnosis) ➛ **95 patients**.

ected population
natic dizzy patients (who had to be fluent in English, and available to return for 4 half of testing, referred to a dizziness clinic.

1972

dent verification of diagnosis
is assigned by lead investigator, in e word used by the patient

m follow-up

5. **No brain imaging** (except skull x-ray)
6. **Some diagnoses not recognized in 1970** TIA and vestibular migraine

For the traditional approach to work,

- Patients must consistently choose 1 'dizzy type'
- Each 'dizzy type' must be tightly linked to a given DDx

Patients DO NOT reliably select a single dizziness subtype:

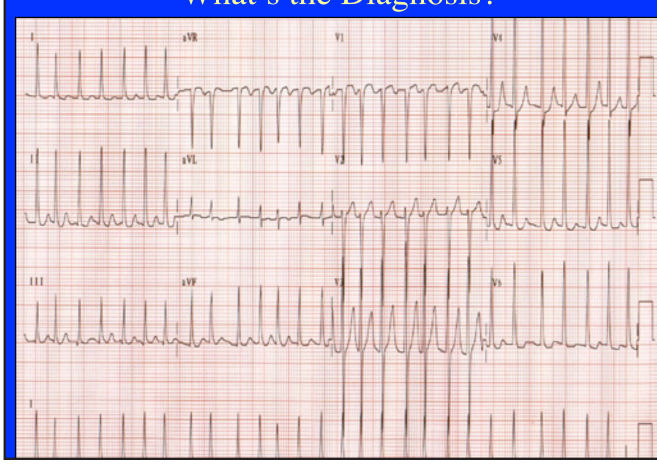
- Sensory symptoms are hard to describe
- Half of patients change dizzy category < 10 minutes
- Most endorse > 1 dizzy types

There is NO TIGHT LINK between type & differential diagnosis:

- "Vertigo" (in dizzy patients) does not predict stroke diagnosis
- 37% with cardiac causes (of dizziness) endorse "vertigo"
- In BPPV, mean number of dizzy symptoms = 3; only 25% endorsed "vertigo" as the main symptom

A dizzy patient in the Emergency Department

What's the Diagnosis?



Another dizzy patient in the Emergency Department

Around 2 or 3 this morning, I felt "lightheaded", "dizzy", "dizziness"

I was in bed when it happened. When I tried to walk to the bathroom, I felt "unsteady on my feet".

Because I had "vertigo"

Definitely not, nothing close to that . . . The vertigo, the whole place was spinning like I was on a ferris wheel.

What's the Diagnosis?

BPPV

(positive Dix-Hallpike
And resolution of all symptoms
with Epley maneuver)

NEW APPROACH: "ATTEST"

Associated symptoms

Timing

Triggers

Exam **S**igns

Testing to confirm diagnosis

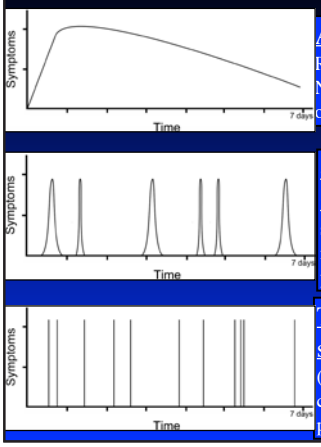
Associated symptoms — Identify obvious, worrisome associated symptoms, signs and basic ancillary tests that suggest a particular diagnosis or group of diagnoses **(this will account for 50% of ED dizzy cases!)**

Timing / **Triggers** — Define the timing of the dizziness. Ask about the onset and duration of attacks? Is the dizziness episodic or persistent? Is the dizziness triggered by specific head movements, or change in body position? Was there preceding trauma? A new medication?
Each Timing & Triggers category is tightly linked with a specific differential diagnosis!

Exam Signs — Within each timing & trigger category, distinguish benign from dangerous causes by carefully looking for exam signs using a targeted bedside physical examination

Testing — If clinically important diagnostic ambiguity remains, exclude dangerous causes by appropriate testing; non-contrast CT is rarely the best test!

TT (timing & triggers) Patterns

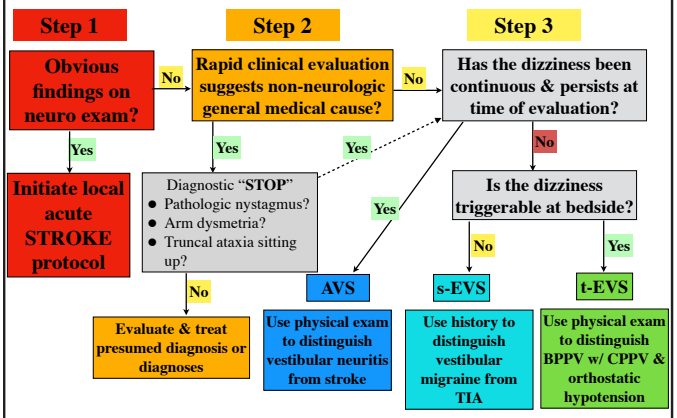


Acute vestibular syndrome (AVS)
Rapid onset, persistent, continuous dizziness, N/V, head motion intolerance that improves over days

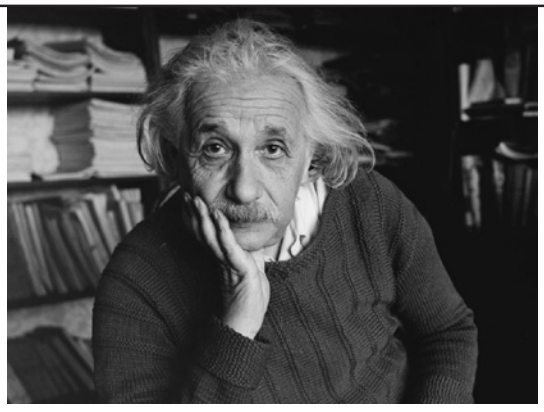
Spontaneous episodic vestibular syndrome (s-EVS)
spontaneous episodes (minutes to hours) not provoked by changes in head or body position

Triggered episodic vestibular syndrome (t-EVS)
(<1 minute) episodes with N/V that are clearly triggered by a change in body posture or head position

ED Diagnostic Approach to the Acutely Dizzy Patient



AVS: acute vestibular syndrome, s-EVS: spontaneous episodic vestibular syndrome, t-EVS triggered episodic vestibular syndrome. Only most common diagnoses are listed for each vestibular syndrome.



"Everything should be made as simple as possible, but not simpler"

ED Diagnostic Approach to the Acutely Dizzy Patient

Step 1

Obvious findings on neuro exam?

Yes → **Initiate local acute STROKE protocol**

No → **Step 2**

Rapid clinical evaluation suggests non-neurologic general medical cause?

Yes → Diagnostic "**STOP**"
- Pathologic nystagmus?
- Arm dysmetria?
- Truncal ataxia sitting up?

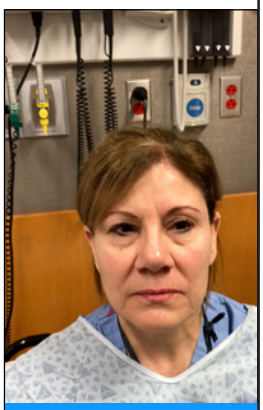
AVS: acute vestibular syndrome, s-EVS: spontaneous episodic vestibular syndrome, t-EVS triggered episodic vestibular syndrome. Only most common diagnoses are listed for each vestibular syndrome.

ED Diagnostic Approach to the Acutely Dizzy Patient

Step 2

No → **Rapid clinical evaluation suggests non-neurologic general medical cause?**

Yes → Diagnostic "**STOP**"
- Pathologic nystagmus?
- Arm dysmetria?
- Truncal ataxia sitting up?



26 seconds

AVS: acute vestibular syndrome, s-EVS: spontaneous episodic
episodic vestibular syndrome. Only most common diagnoses ar

ED Diagnostic Approach to the Acutely Dizzy Patient

Step 2

No → **Rapid clinical evaluation suggests non-neurologic general medical cause?**

Yes → Diagnostic "**STOP**"
- Pathologic nystagmus?
- Arm dysmetria?
- Truncal ataxia sitting up?

No (reassuring STOP) → **Evaluate & treat presumed diagnosis or diagnoses**

AVS: acute vestibular syndrome, s-EVS: spontaneous episodic vestibular syndrome, t-EVS triggered episodic vestibular syndrome. Only most common diagnoses are listed for each vestibular syndrome.

ED Diagnostic Approach to the Acutely Dizzy Patient

Step 3

Has the dizziness been continuous & persists at time of evaluation?

No → **Is the dizziness triggerable at bedside?**

Yes (worrisome STOP) → **AVS**

Use physical exam to distinguish vestibular neuritis from stroke

No → **s-EVS**

Use history to distinguish vestibular migraine from TIA

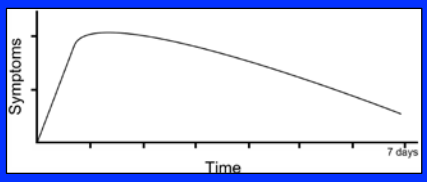
Yes → **t-EVS**

Use physical exam to distinguish BPPV w/ CPPV & orthostatic hypotension

AVS: acute vestibular syndrome, s-EVS: spontaneous episodic vestibular syndrome, t-EVS triggered episodic vestibular syndrome. Only most common diagnoses are listed for each vestibular syndrome.

Diagnose AVS

Vestibular neuritis or posterior circulation stroke?



Common causes (~97% of cases):

Vestibular neuritis

Posterior circulation stroke

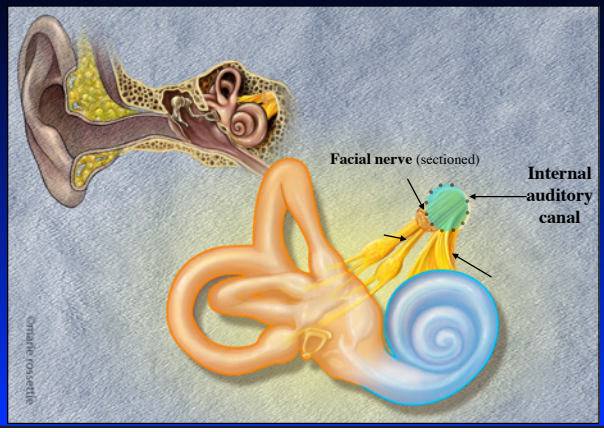
Labyrinthitis

Multiple sclerosis

Uncommon causes:

labyrinthine trauma/concussion
gentamicin toxicity
acute alcohol intoxication
acute medication side effects
bacterial otomastoiditis
Wernicke's
syphilitic labyrinthitis
autoimmune labyrinthitis

Vestibular Neuritis or Labyrinthitis ?

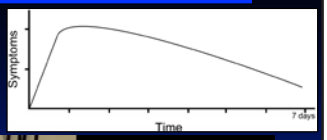


Acute Vestibular Syndrome

Nystagmus present	Nystagmus absent
• Official definition of AVS - acute onset of persistent dizziness lasting > 24 hours with head-motion intolerance, N/V **and nystagmus** • HIT testing is ONLY validated on AVS patients WITH nystagmus • Nearly all neuritis patients seen in first few days have nystagmus	• Some AVS patients have all criteria but **no nystagmus** • Nearly all neuritis patients have nystagmus; only half of cerebellar stroke so the absence of nystagmus is more worrisome* in AVS patients • HIT not validated in this group • Gait testing is far more important&

* Machner; 2020; J Neurol
& Carmona; 2023; Eur J Neurol

ACUTE VESTIBULAR SYNDROME



"I was feeling really dizzy & nauseous . . . and couldn't even keep my head up or stand up."

"The dizziness came on very abruptly."

Vestibular neuritis ? . . .

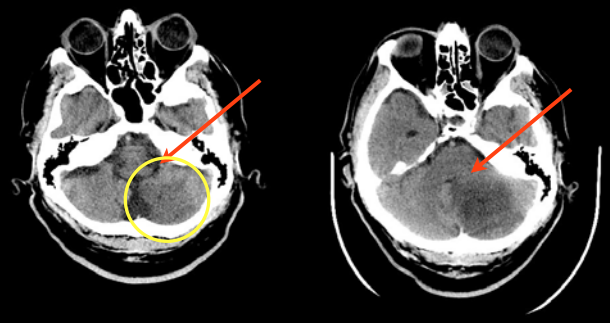
or

1. How often do small strokes mimic a peripheral vestibular syndrome? Posterior circulation stroke ??
   . . . ~ 10-25% of cases
2. Are we missing them clinically?
   Yes, stroke risk within 7d is ~ 1/500 patients (0.14-0.5%)
3. If we are missing them, does it matter?
   Yes, brainstem compression due to cerebellar edema & a recurrent stroke
4. Can we distinguish between central and peripheral causes at the bedside?
   Yes . . .

Cerebellar infarct

70 year old man with abrupt onset of vomiting & dizziness

8 hours after symptom onset | 48 hours after symptom onset



Acute Vestibular Syndrome

Ask and answer 5 questions in the following sequence:

- Is there a central pattern of nystagmus ?
- Is skew deviation present ?
- Is the head impulse test negative? (only applies to patients with nystagmus)
- Are there any CNS signs on focused neurological exam ?
- Is the patient unable to sit or walk unassisted ?

"Yes" answer to ***any*** question: Posterior circulation stroke till proven otherwise

"No" answer to ***all*** questions: Treat as vestibular neuritis

Can we clinically distinguish central & peripheral? Exam Signs

	Peripheral	Central	
Is there a central pattern of nystagmus ?	Horizontal Unidirectional	Horizontal, direction-changing, torsional, vertical	~ 50%
Is skew deviation present ?	Absent	May be present (vertical correction)	~ 25%
Is the head impulse test negative? (only in patients with nystagmus*)	Corrective saccade present	Corrective saccade absent	~ 85%
Are there CNS signs on focused neurological exam?	Absent	May be present	~ 65%
Is the patient unable to sit or walk unassisted ?	Usually can	Often cannot	~ 65%

Uni-directional (direction-fixed) nystagmus

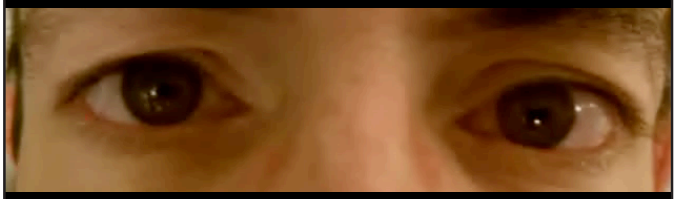
Reassuring

Direction-changing nystagmus
+ HIT (AICA stroke)

Worrisome

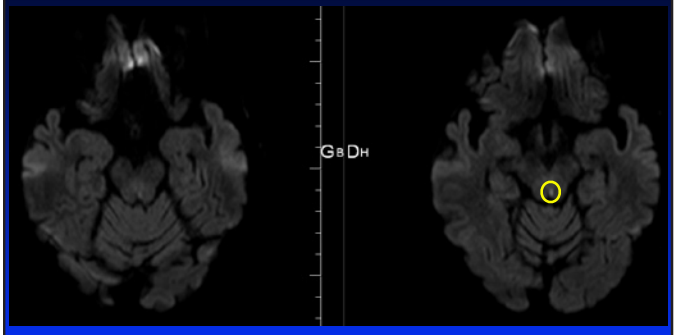
Gaze-evoked direction changing nystagmus is present in ~ 50% of patients with posterior circulation strokes presenting as acute dizziness

Direction-changing nystagmus . . . in a patient on phenytoin



A 92-year old with dizziness, diplopia & torsional nystagmus

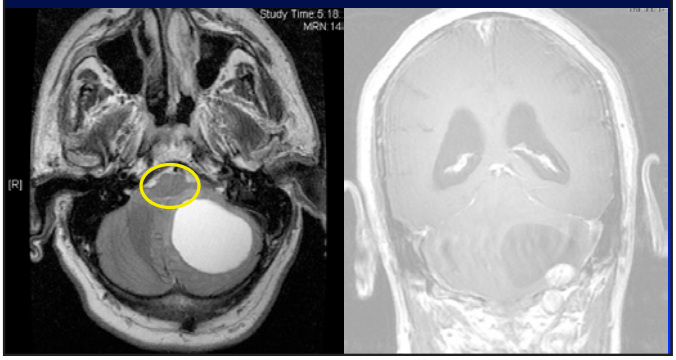
Worrisome



Vertical Nystagmus
Also had direction-changing nystagmus

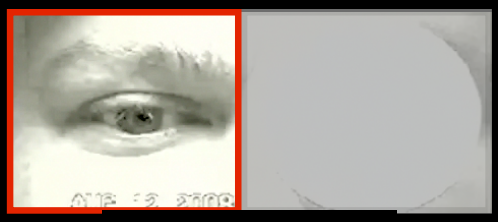
Worrisome

This patient had a benign hemangioblastoma that was resected; he recovered and was neurologically normal.



Skew Deviation

perform alternate cover test; look for a small corrective vertical movement



Worrisome

Skew deviation (presence of a small ***vertical*** correction) is is not very sensitive (~ 35%) but is very specific for brainstem lesions

Head Impulse Test

Positive: presence of corrective saccade = neuritis
Negative: no corrective saccade = stroke

Positive HIT

(Corrective saccade when patient moves to left)

Rapid movement (~ 200⁰/sec)

Unpredictable (to patient) side to which head is turned

Low amplitude (10-20⁰)

? Eccentric to midline

Reassuring

Positive HIT

(Corrective saccade when patient moves to right)

Reassuring

Positive HIT (to left)

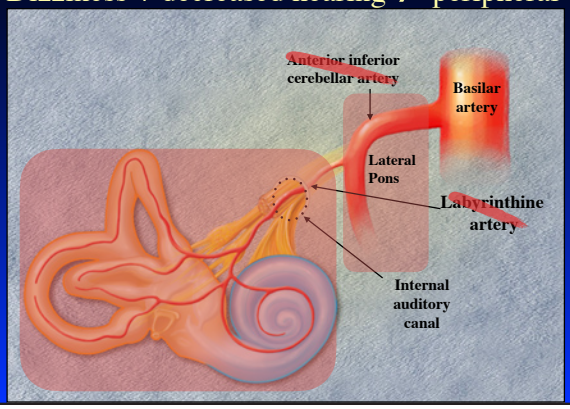
False "positive" HIT
(falsely reassuring HIT)

	Imaging negative (no stroke, N=8)	Imaging positive (stroke, N=34)
Negative test (no corrective saccade)	0% (N=0)	91% (N=31)
Positive test (corrective saccade +)	100% (N=8)	**9% (N=3)**

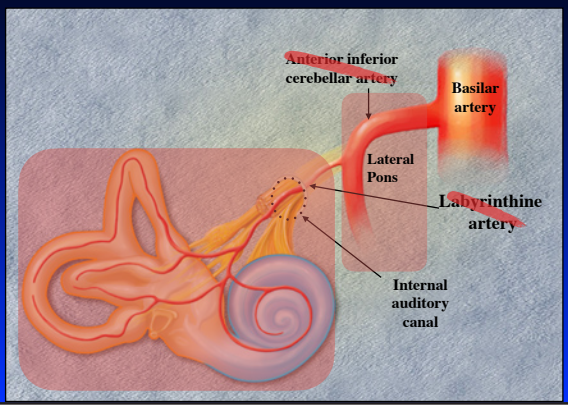
False positive HIT

Dizziness + decreased hearing = peripheral - right?

Dizziness + decreased hearing ≠ peripheral

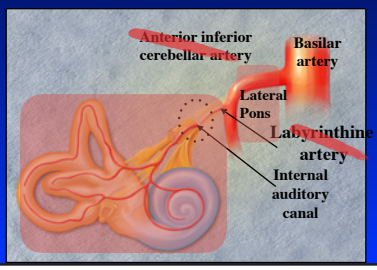


Hearing test is the '+' in HINTS +



- 25% of patients with acute "audio-vestibular" loss have strokes
- 55% are unaware of the hearing loss

Test the hearing by finger rub!!



Imagine a physical exam test for appendicitis that is more accurate than an abdominal CT scan!

HINTS is more sensitive (~ 98%) than early MRI (~ 80%) in detecting stroke in AVS patients!

HOW good are emergency physicians at doing HINTS in routine practice?

. . . pretty bad

- Ohle 2020 Acad EM - Systematic review & meta-analysis of literature
  - 5 studies, N= 617
  - Sub-specialists: Sens/spec: 96.7% & 94.8%
  - EPs/***gen neurol***: Sens/spec: 83% & 44%

Can Emergency Physicians Accurately Rule Out a Central Cause of Vertigo Using the HINTS Examination? A Systematic Review and Meta-analysis

To fix this W I D E knowledge gap, we need

. . . pretty bad

- Dmitriew 2021 Acad EM - Canadian retrospective chart review, tertiary medical center 2309 patients
  - HINTS documented in ~ 20% of dizzy patients
  - 97% did not meet criteria (AVS with nystagmus)
  - Many patients had both HINTS & Dix-Hallpike
- Much better clinician education, and/or
- A change in the paradigm to a goggles- and/or tele-neurology-based approach, and/or
- More rational use of imaging

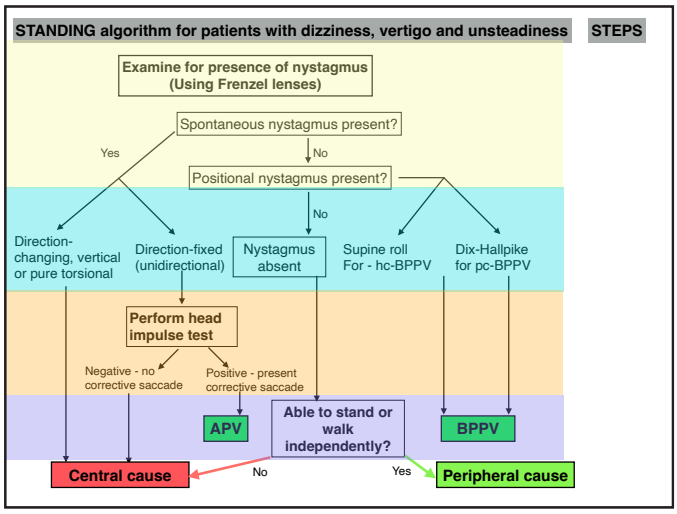
Episode VI

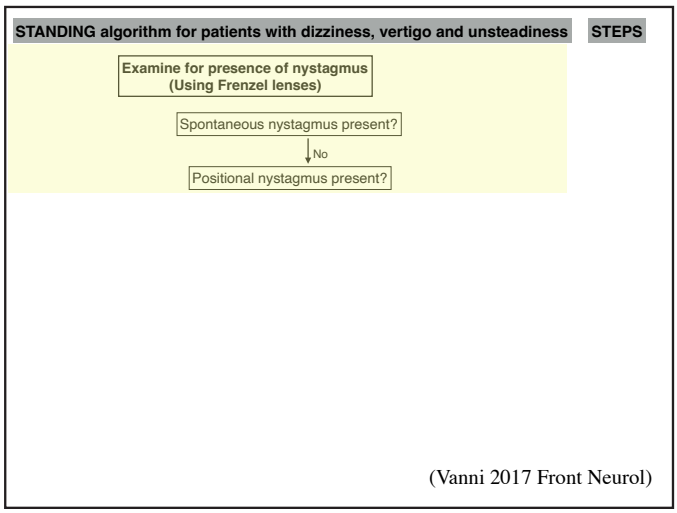
The Return of the Jedi

. . . Europeans come to the rescue!

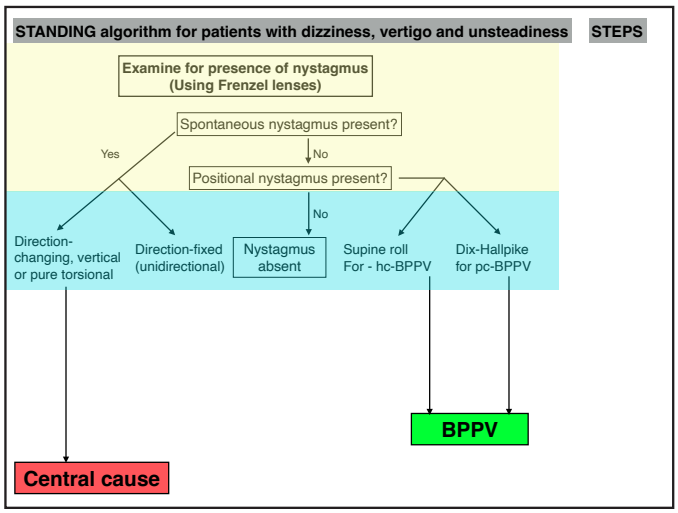
Florence
Simone Vanni

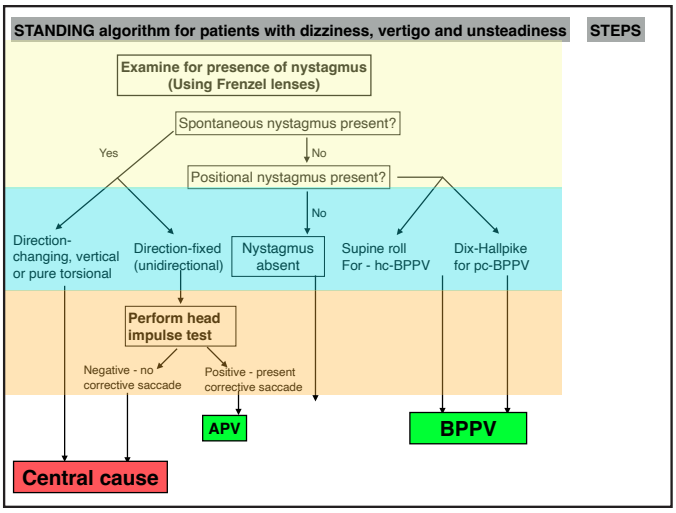
Paris
Camille Gerlier

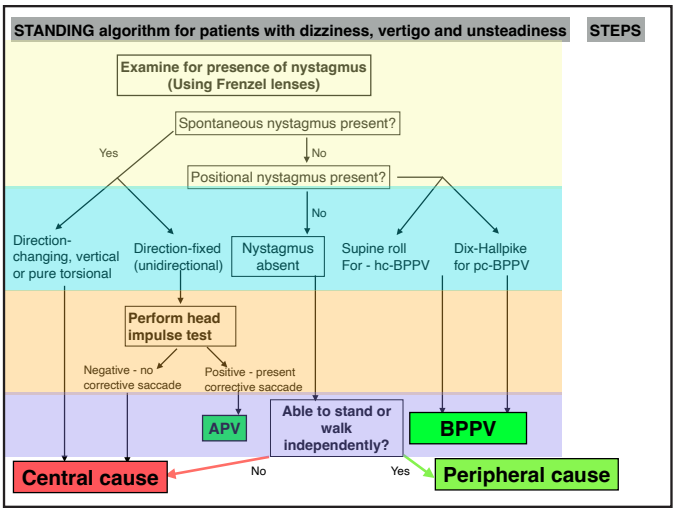




(Vanni 2017 Front Neurol)







STANDING protocol

Single center prospective diagnostic accuracy study of 6 trained emergency physicians (using Frenzel lenses)

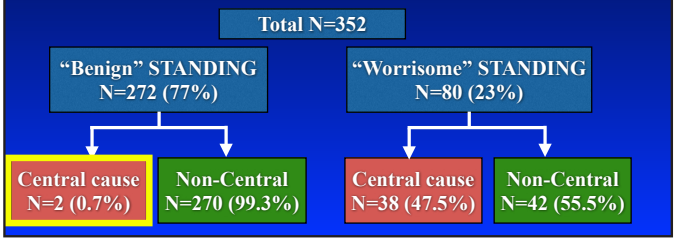
- 4h lecture & 2h demonstration on normal subjects
- 10 patient examinations proctored by a specialist)

- Compared usual care (blinded) to STANDING results; outcome = central cause
- Final diagnosis assigned by consensus, based on all clinical data at 3 month follow-up. MRI ***not*** applied to all patients.

Larochelle - ongoing study (Vanni 2017 Front Neurol)

STANDING protocol

- 391 screened, 4 LTFU, 352 (90%) included
  - 40 (11.4%) had central causes (mostly ischemic stroke)
- Overall OR for central causes was 122 (95% CI 15-943)



HINTS v STANDING v ABCD2

French mono-center prospective diagnostic cohort study of 9 trained emergency physicians (using Frenzel lenses) and 300 patients

- 4h lecture & 2h video demonstrations, repeated in 6 m
- No proctored exams by a specialist

- Compared usual care to STANDING, HINTS and ABCD2 using MRI as criterion standard
- All patients had MRI (not all in ED)

Gerlier; 2021; Acad EM
Gerlier; 2023; Acad EM

How much time does it take to do HINTS or STANDING testing?

	Number of patients	HINTS (seconds)	STANDING (seconds)
Emergency physicians (7)	48	64	157
Otoneurologists (2)	9	32	65

HINTS has no gait or positional testing for BPPV
STANDING has no test of skew deviation

Vanni; 2023; Acad EM

These studies show that, ***with training***, emergency physicians can learn to correctly perform & interpret these bedside examination techniques

Given this gap, err towards patient safety
Do a focused posterior circulation exam

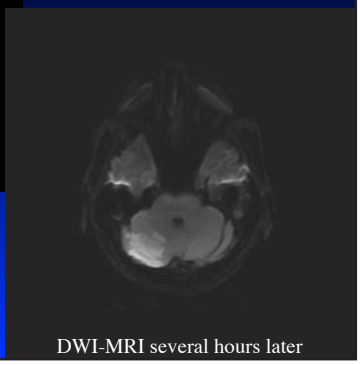
decreased hearing (the "+" in HINTS +)
dysmetria (FTN and HTS)
anisocoria (easier to see in the dark)
dysarthria, dysphagia & hoarseness
facial hypesthesia (↓ pain/temperature, not light touch)
visual field cut

and test the **GAIT!!**

Can the patient walk?
TEST THE GAIT !!

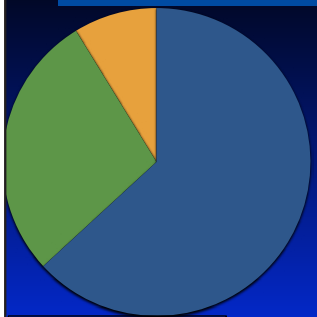
70 year-old acutely dizzy,
normal mentation & strength
no dysmetria or nystagmus

CT at presentation to ED



DWI-MRI several hours later

Gait abnormality in AVS



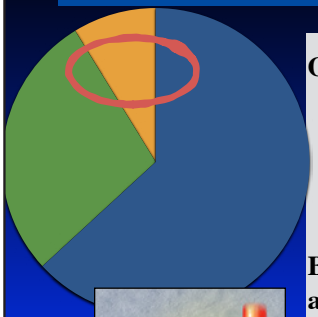
Ataxia Grade	Neuritis N (%)	Stroke N (%)
0	5 (7%)	0
1	39 (54%	3 (7%)
2	28 (39%)	11 (26%)
3	0	28 (67%)

114 patients with AVS
72 (63%) neuritis
42 (37%) stroke

0 = normal
1 = mild/moderate imbalance walking independently
2 = severe imbalance standing, can't walk without support
3 = falls with upright posture

Carmona 2016 Front Neurol

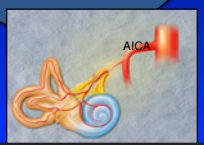
Gait abnormality in AVS



Of the 10 AICA strokes,

- Only 4 had decreased hearing
- 7 had + HIT (falsely reassuring)

But all 10 had grade 2 or 3 ataxia!



Carmona 2016 Front Neurol

Acute Vestibular Syndrome
Ask and answer 5 questions in the following sequence:

- **Is there a central pattern of nystagmus ?**
- **Is skew deviation present ?**
- **Is the head impulse test negative?** (only applies to patients with nystagmus)
- **Are there any CNS signs on focused neurological exam ?**
- **Is the patient unable to sit or walk unassisted ?**

"Yes" answer to ***any*** question: Posterior circulation stroke till proven otherwise

"No" answer to ***all*** questions: Treat as vestibular neuritis

---

How useful is imaging?

How does it compare to physical examination?

How BAD is CT?

CT is a <u>REALLY BAD</u> test for early ischemic stroke in general !!

- It's worse for posterior circulation stroke (< 20% sensitive)
- The vast majority of CT scans done for dizziness do not supply useful information
- ICH presenting as **<u>isolated</u>** dizziness without other worrisome findings (headache, altered mental status, hard neurologic findings) is very uncommon

---

Delusions of (CT) grandeur

- Retrospective cohort study of patients discharged from Canadian EDs with a peripheral dizziness diagnosis
- Primary outcome: stroke outcome at 30 days
- Patients who HAD a CT scan were 2.3x more likely to have a stroke versus those who had no CT

Grewal 2015 Stroke

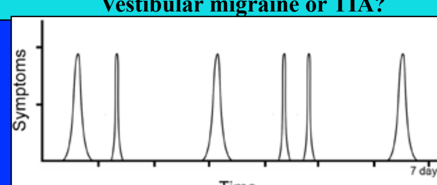
What about MRI ?

"We now have ... technological
renders your old-fashioned clinical; the ability to
paradigm useless, ... blood vessels is
insignificant next to the power of a careful
history & targeted physical exam"

MRI <u>misses ~20%</u> of posterior circulation strokes presenting as an AVS in the first 48 hours

---

Diagnose spontaneous-EVS
Vestibular migraine or TIA?



Common causes:
Vestibular migraine
Posterior circulation TIA
Arrhythmia, PE
Meniére's disease

Uncommon causes:
Peri-lymphatic fistula
Seizure

~ 8% of posterior circulation strokes are preceded by an episode of isolated vertigo (Paul; 2013; Lancet Neurol)

Clinical Factor	Vestibular Migraine	Posterior circulation TIA
Epidemiological context • Age • Sex • Vascular risk factors • Past history of migraine • Family history of migraine • Recent head or neck trauma	• Younger (mean age of onset ~ 40 years) • More often female • Fewer vascular risks • Past history nearly always present • Family history in ~ 50-70% • Recent trauma less likely	• Older (usually older than 60 years) • More often male • More vascular risks • Past history of migraine much less common • Family history of migraine less common • If recent trauma is present, consider vertebral artery dissection
Symptom timing • Onset • Duration of attacks • Number of attacks over time	• Gradual onset in ~ 40%; different symptoms may evolve over time • Variable but more often > 1 hour • Multiple prior attacks common*, occurring over months-years	• Usually sudden; all symptoms present at onset • Variable but more often < 1 hour • Fewer number of attacks usually occurring over days-weeks
Symptom quality • Migraine-related symptoms • Positive vs Negative symptoms • Concurrent headache	• Symptoms often present • Positive symptoms • Present ~ 50% of time (not always simultaneous with the dizziness)	• Symptoms far less common • Negative symptoms • Less common (if present, consider vertebral artery dissection)

* Formal diagnostic criteria for definite vestibular migraine include ≥ attacks

^ Migraine-related symptoms include photophobia, phonophobia, visual aura, nausea or vomiting

Important note - There is overlap between vestibular migraine and pc-TIA for each row of the table. Combinations of factors will be more helpful than any one factor in isolation.

SAEM GRACE-3 Clinical Guideline

Search: SAEM GRACE

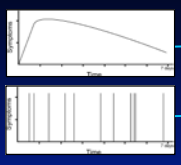
VertiGuide App

https://t.co/JOWNYF9bf0

Timing & Triggers informs . . .

Differential diagnosis → **Head impulse**

Appropriate testing to help make a specific diagnosis → **Dix-Hallpike & Supine Roll**



Interpretation of those tests

Nystagmus pattern means different things depending on the timing & triggers category

Treatment of the specific diagnosis BPPV (vertigo) ≠ **meclizine**

"Use the history and physical exam, Luke"

"Forget the CT"

Luke tears up the CT requisition

"Why did you cancel the CT?"

Luke nails the diagnosis using clinical skills . . .

. . . avoiding a potentially disastrous misdiagnosis ALWAYS

THE END

Acknowledgments:

My patients

Dr. Richard Baloh

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Drs. Louis Caplan & Sean Savitz

Dr. Anita Bhandari

Dr. Terry Fife, Barrows Institute

AAN BPPV practice parameter video clips

George Lucas

THE END

SELF EVALUATION

Diagnosing Acute Dizziness in the Emergency Department

True/False

1. Patients' use of a particular word to describe their dizziness (e.g., vertigo vs lightheadedness vs unsteadiness) is extremely helpful in creating a differential diagnosis for the cause of their symptoms?

2. Patients who present with simultaneous onset of both dizziness and hearing loss nearly always have a peripheral cause, labyrinthitis.

3. The most common cause of the acute vestibular syndrome is vestibular neuritis.

4. In patients with the acute vestibular syndrome, nystagmus that changes direction depending on the direction the patient is looking (e.g., beats to the right on rightward gaze and towards the left on leftward gaze) means that the cause is central.

5. In patients with the acute vestibular syndrome who have nystagmus, the HINTS battery of bedside testing, when done by trained individuals, is more sensitive for central causes than MRI done in the first 48 hours.

6. The STANDING protocol does not include testing for BPPV.

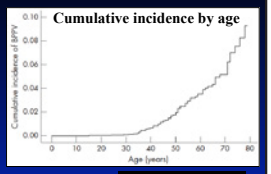
Answer Key: 1. F, 2. F, 3. T, 4. T, 5. T, 6. F

Benign Paroxysmal Positional Vertigo (BPPV): Diagnosis and Bedside Management

Jonathan A. Edlow, MD

Epidemiology of BPPV

- **Very common**
  - Cumulative lifetime incidence of ~ 10%
  - Median age ~ 55 years; ♀:♂ ~2:1
  - Incidence ~ 110/100,000 (PE is ~ 65)



Von Brevern 2007

- **Not always so "benign"**
  - Persistent symptoms, time out of work
  - Falls (especially in the elderly) with injuries
  - Side effects from non-indicated meclizine/others

How common is it?

(A conservative back-of-the envelope calculation)

- You work #14 8-hour shifts/month x 11 months = 1232 hours/year
- You see 2.5 patients/hour = 3080 patients/year
- 2% of ED patients are dizzy 0.02 x 3080 = 62 dizzy patients/year
- Half of the 62 have general medical causes (minus) 31 patients/year
- Of the remaining 31, ~ 50% have BPPV **~ 15 BPPV patients/year**

Diagnosis of BPPV

- **Brief episodes** of dizziness usually lasting ~ 30 seconds that resolves without further motion (***often at night in bed***)
- **DO a PROVOCATIVE test!!**
  - Dix-Hallpike for posterior canal BPPV (pc-BPPV) - transient upbeating torsional nystagmus
  - Supine roll for horizontal canal BPPV (hc-BPPV) - transient horizontal nystagmus
- (Latency after maneuver before dizziness/nystagmus begin)

KEY POINT

***CAUTION*:**
dizziness that worsens with head movement IS NOT diagnostic of a peripheral cause !!!

Misdiagnosis & resource over-utilization

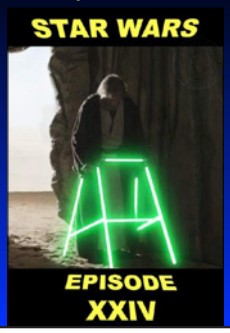
- The Dix-Hallpike test is underused
- When used, it may be performed or interpreted incorrectly
- Nystagmus is poorly documented and when it is, the details are often inconsistent with the ED diagnosis
- Imaging tests are commonly performed but almost never useful

Potential sources of implicit bias

- Not considering BPPV in elderly patients
- Not considering BPPV in patients who present with new falls (and not other reason for the fall)

BPPV in the Elderly

- Atypical presentations of BPPV are ***common*** in the elderly
- Elderly BPPV patients are > 6x more likely to fall than those without BPPV; ***falls*** lead to ***fractures*** & other injuries
- ***Falls are reduced by treatment*** with a canalith repositioning maneuver
- ***Meclizine is a BAD*** drug in the elderly
  - AGS recommends ***against*** its use
  - Meclizine → ***hip fractures***



Another dizzy patient in the Emergency Department

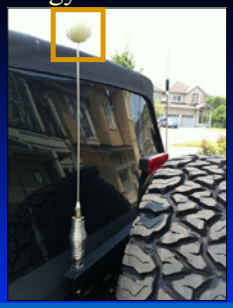
Around 2 or 3 this morning, I felt "lightheaded", "dizzy", "dizziness"

I was in bed when it happened. When I tried to walk to the bathroom, I felt "unsteady on my feet".

Because I had "vertigo" last year . . .

Definitely not, nothing close to that . . . The vertigo, the whole place was spinning like I was on a ferris wheel.

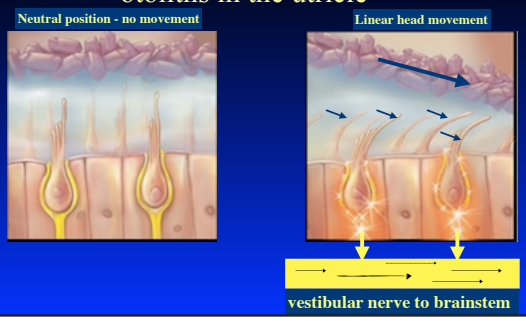
Normal physiology

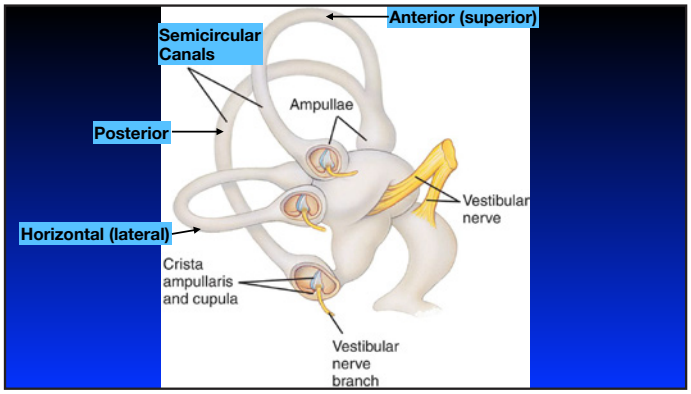


Thought experiment:

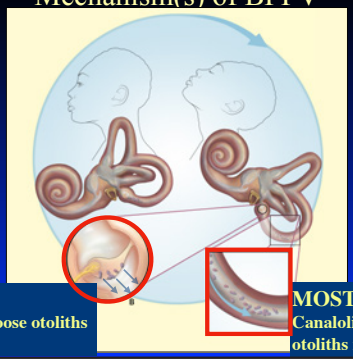
What happens to the tennis ball as the car accelerates?

Vestibular anatomy & physiology otoliths in the utricle



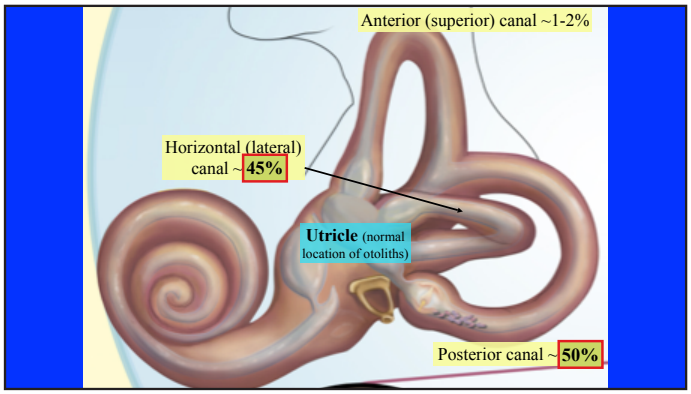


Mechanism(s) of BPPV



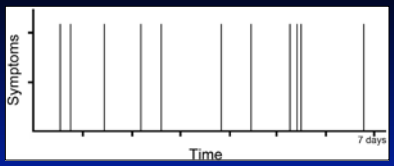
LESS common
Cupulolithiasis - loose otoliths stick to the cupula

MOST common
Canalolithiasis - free-flaoting otoliths in a semi-circular canal



TRIGGERED-EVS

BPPV or orthostatic hypotension?

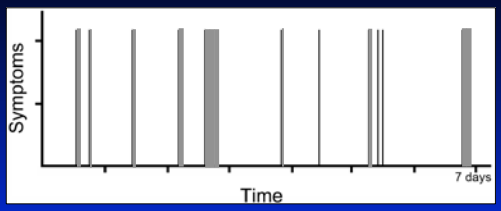


BPPV

Orthostatic hypotension

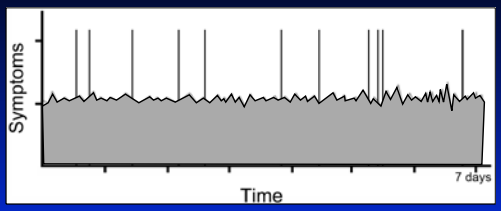
Structural CNS lesion- CPPV

TRIGGERED-EVS



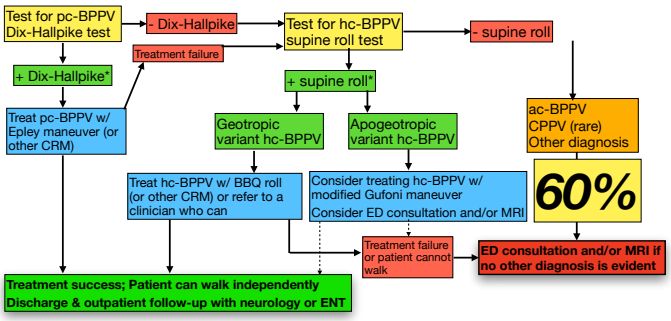
Patients may overestimate duration length because there is concomitant nausea and vomiting (which outlasts the dizziness), or because it's middle of the night, it's dark (and patient loses accurate sense of time).

TRIGGERED-EVS



Other times, patients will report that their dizziness is "continuous", lasting for days (due to very frequent episodes or due to low grade head motion or anticipation of dizziness with motion or persistent nausea)

t-EVS - Diagnostic algorithm for patients with suspected BPPV



* A **positive Dix-Hallpike test** is the reproduction of symptoms with transient upbeat torsional nystagmus.
* A **positive supine roll test** is the reproduction of symptoms with transient horizontal direction changing nystagmus

t-EVS - Diagnostic algorithm for patients with suspected BPPV

Test for pc-BPPV Dix-Hallpike test

When should I do the Dix-Hallpike test?

- Patients who have a clear t-EVS
- No obvious general medical cause
- No obvious central neurological findings & can walk unassisted
- Patents with dizziness whose history is ambiguous* (?t-EVS) but
  - Do **NOT** have spontaneous nystagmus at rest
  - Do **NOT** have gaze-evoked (direction-changing) nystagmus

* - Leibnitz's "best of all possible worlds"

CAUTION: is this an AVS . . .

. . . or a triggered-EVS ?

"For about 28 hours or so, I've been having dizziness. It comes in waves . . ."

"It's not a dizzy type of thing where the room is spinning, it's just that I walk towards one side."

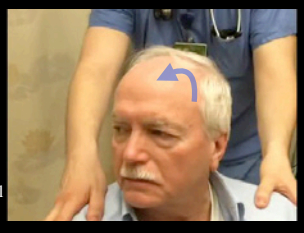
Dix-Hallpike test: what's happening? What should you see?

Courtesy of Dr. Anita Bhandari

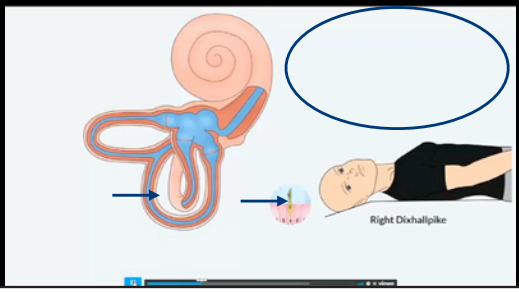
No obvious general medical cause nor obvious CNS findings so . . .

5-7 seconds of latency

15-20 seconds of symptoms & upbeating torsional nystagmus



Dix-Hallpike test: what's happening (with the hair cells & endolymph) that you don't see?



t-EVS - Diagnostic algorithm for patients with suspected BPPV

Test for pc-BPPV
Dix-Hallpike test

When should I ***NOT*** do the Dix-Hallpike test?

- Presence of new CNS findings
- Inability to walk or sit up unassisted
- Nystagmus at rest
  - Spontaneous nystagmus
  - Gaze-evoked (direction-changing) nystagmus

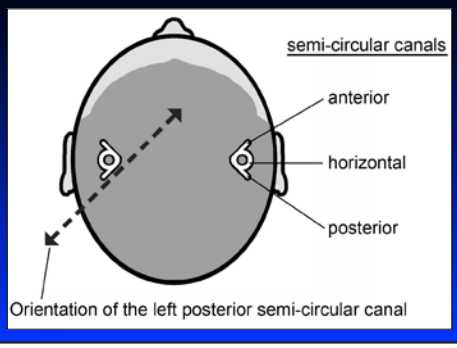
t-EVS - Diagnostic algorithm for patients with suspected BPPV

Test for pc-BPPV
Dix-Hallpike test

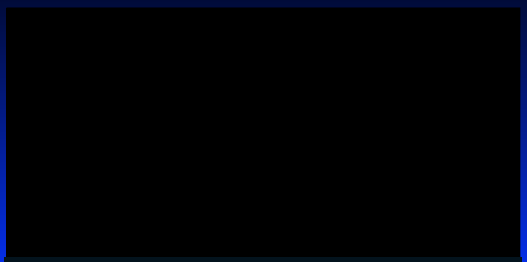
How should I do the Dix-Hallpike test?

- Explain to the patient what you are going to do and why.
- Position patient in bed so that the shoulders line up with edge of bed. With patient sitting up, turn head 45º to the side tested
  - Move patient down moderately fast
  - Be sure that the head is hanging 20-30 degrees off the edge of bed
  - Coach patient not to sit up if symptomatic
- Wait at least 30 seconds before coming back up

Straightening the Hoola-hoop



Dix-Hallpike maneuver



Eyes open & looking into the distance (not fixating on a near point)

What's happening when you do a Dix-Hallpike test in a patient with pc-BPPV?

Courtesy of Dr. Anita Bhandari

t-EVS - Diagnostic algorithm for patients with suspected BPPV

Test for pc-BPPV
Dix-Hallpike test

↓

+ Dix-Hallpike*

What is a positive Dix-Hallpike test?

- Symptoms of dizziness on one side ***PLUS***
- Nystagmus which is
  - Triggered by the maneuver ***and***
  - Transient, lasting < 1 minute
  - Upbeating & torsional

* A **positive Dix-Hallpike test** is the reproduction of symptoms with transient upbeat torsional nystagmus.
* A **positive supine head roll test** is the reproduction of symptoms with transient horizontal direction changing nystagmus

t-EVS patient with definite BPPV

Possible findings on Dix-Hallpike testing

What the patient says?	What's the nystagmus quality?	Clinical Significance – What does it mean?	Notes
Nothing– ***no symptoms***	No nystagmus	Extremely unlikely to be BPPV	BPPV has spontaneously resolved or DHT was not done properly (done too slow or without enough head tilt).
Patient feels dizzy on ***both*** sides	No nystagmus	Very unlikely to be BPPV	Many people without BPPV report mild dizziness with a DHT, or the BPPV has spontaneously resolved, or the DHT was not performed properly, or patient has bilateral subjective BPPV (very unlikely)
Patient only feels dizzy on ***one*** side	No nystagmus	Possible "subjective" BPPV if there are too few particles in the canal to create enough drag.	Many subjective BPPV patients respond to an Epley (or other CRM). If the maneuver helps, BPPV is likely. If not, pc-BPPV is unlikely but consider hc-BPPV.
Patient only feels dizzy on ***one*** side	Triggered & transient upbeating torsional	Classic pc-BPPV; treat with an Epley maneuver (or other CRM)	
Patient only feels dizzy on ***one*** side	Triggered & transient horizontal	Likely hc-BPPV; consider re-test with supine head roll test. Treat with Lempert BBQ roll (or other CRM).	Alternate CRMs: Gufoni Some hc-BPPV patients may respond to the Epley maneuver. (Distinguish geotropic from apogeotropic)
Patient only feels dizzy on ***one*** side	Triggered & transient vertical downbeating	Patient either has rare ac- BPPV or a central cause.	Caution: unless clear-cut ac-BPPV that resolves 100% with an Epley maneuver, work-up for a central cause with MRI in the ED and/or neurology consult.

Right side = definite symptoms and nystagmus

Left side = no symptoms and no nystagmus

What if the Dix-Hallpike test shows unilateral symptoms but no nystagmus?

- Possibility 1: the nystagmus amplitude is so small, it's not visible with naked eye; fixation needs to be removed
- Possibility 2: subjective BPPV - not uncommon (BUT the symptoms should still be clear-cut & unilateral). The diagnosis is less definite than with nystagmus

Several studies show that treatment works equally well in patients with "subjective" BPPV with or without nystagmus

t-EVS - Diagnostic algorithm for patients with suspected BPPV

Test for pc-BPPV Dix-Hallpike test

↓

+ Dix-Hallpike*

↓

Treat pc-BPPV w/ Epley maneuver (or other CRM)

↓

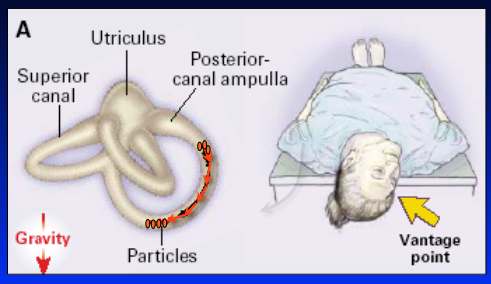
Treatment success; Patient can walk independently Discharge & outpatient follow-up with neurology or ENT

60%

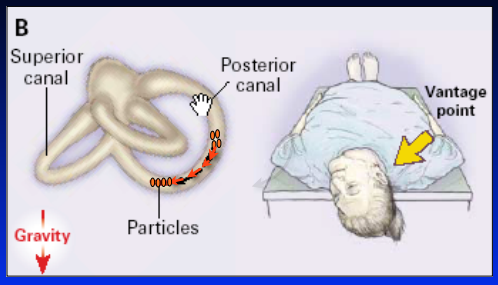
* A **positive Dix-Hallpike test** is the reproduction of symptoms with transient upbeat torsional nystagmus.
* A **positive supine roll test** is the reproduction of symptoms with transient horizontal direction changing nystagmus

Epley Maneuver

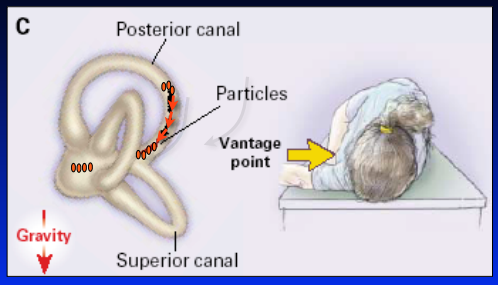
Should be head hanging ~ 30 degrees



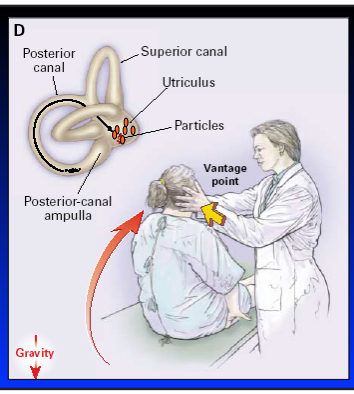
After dizziness & nystagmus resolve (~ 30-45 seconds) turn head 90°



After dizziness & nystagmus resolve (~ 30-45 seconds), turn head 90°



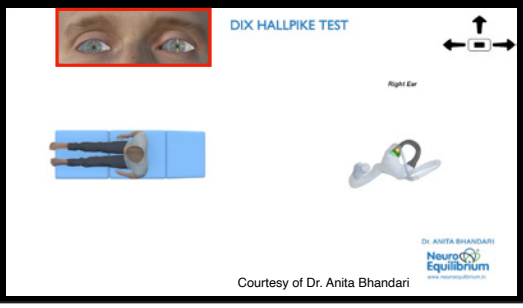
After dizziness & nystagmus resolve (~ 30-45 seconds) help patient up



History sounds like BPPV . . . but Dix-Hallpike is negative, or Epley doesn't work - WHY?

1. **Wrong diagnosis**
   Distinguish dizziness that is **provoked or triggered by** versus ***exacerbated by*** head or body position changes. Is patient normal at rest and becomes dizzy on motion OR has fixed pathology (tumor, MS, labyrinthitis) & are dizzy at baseline but are more dizzy with movement.
2. **Wrong technique**
3. **Wrong canal**
   ~ 40% of BPPV is the horizontal (lateral) canal
4. **Wrong "PPV"** - C(central)PPV instead of B(benign) PPV

Simulation of Dix-Hallpike test done in hc-BPPV



Courtesy of Dr. Anita Bhandari

t-EVS - Diagnostic algorithm for patients with suspected BPPV

Test for pc-BPPV Dix-Hallpike test
- Dix-Hallpike
Treatment failure
+ Dix-Hallpike*
Treat pc-BPPV w/ Epley maneuver (or other CRM)

CLUES on the Dix-Hallpike test that suggest hc-BPPV:
- Definite reproduction of transient unilateral symptoms but no nystagmus
- Definite symptoms + ***horizontal*** nystagmus

Treatment success; Patient can walk independently
Discharge & outpatient follow-up with neurology or ENT

* A **positive Dix-Hallpike test** is the reproduction of symptoms with transient upbeat torsional nystagmus.
* A **positive supine roll test** is the reproduction of symptoms with transient horizontal direction changing nystagmus

Horizontal canal-BPPV

- Classic teaching is that hc-BPPV accounts for ~ 15% of BPPV, but newer data suggest that hc-BPPV is likely more common in an ED population & early presenters
- Diagnostic test - supine roll test
- Therapeutic maneuver - Lempert BBQ roll
- Two types
  - **GEO**tropic (canalolithiasis) fast-component beats towards the ground
  - **APOGEO**tropic (cupulolithiasis) fast-component beats towards the sky

t-EVS - Diagnostic algorithm for patients with suspected BPPV

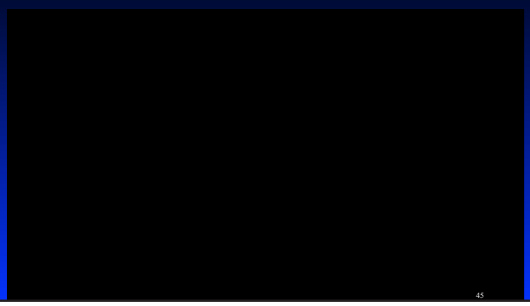
Test for pc-BPPV Dix-Hallpike test
- Dix-Hallpike
Treatment failure
Test for hc-BPPV supine roll test
+ supine head roll*
+ Dix-Hallpike*
Treat pc-BPPV w/ Epley maneuver (or other CRM)

85%

Treatment success; Patient can walk independently
Discharge & outpatient follow-up with neurology or ENT

* A **positive Dix-Hallpike test** is the reproduction of symptoms with transient upbeat torsional nystagmus.
* A **positive supine roll test** is the reproduction of symptoms with transient horizontal direction changing nystagmus

hc-BPPV diagnostic test Supine Roll



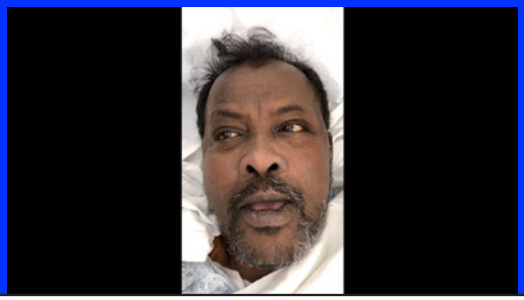
t-EVS - Diagnostic algorithm for patients with suspected BPPV

Test for pc-BPPV Dix-Hallpike test
- Dix-Hallpike
Treatment failure
Test for hc-BPPV supine roll test
+ supine roll*
+ Dix-Hallpike*
Treat pc-BPPV w/ Epley maneuver (or other CRM)
Geotropic variant hc-BPPV
Apogeotropic variant hc-BPPV
Treat hc-BPPV w/ BBQ roll (or other CRM) or refer to a clinician who can

Treatment success; Patient can walk independently
Discharge & outpatient follow-up with neurology or ENT

* A **positive Dix-Hallpike test** is the reproduction of symptoms with transient upbeat torsional nystagmus.
* A **positive supine roll test** is the reproduction of symptoms with transient horizontal direction changing nystagmus

Geotropic hc-BPPV



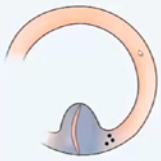
Geotropic hc-BPPV
Supine roll

More intense nystagmus

Less intense nystagmus

www.neuroequilibrium.in

What's happening when you do a supine roll test in a patient with hc-BPPV?



Courtesy of Dr. Anita Bhandari

hc-BPPV therapeutic maneuver:
Lempert BBQ roll

Turn head AWAY from the affected side
(away from the side with the most intense nystagmus)

t-EVS - Diagnostic algorithm for patients with suspected BPPV

Test for pc-BPPV Dix-Hallpike test

\- Dix-Hallpike

Test for hc-BPPV supine roll test

Treatment failure

\+ Dix-Hallpike*

\+ supine roll*

Treat pc-BPPV w/ Epley maneuver (or other CRM)

Geotropic variant hc-BPPV

Apogeotropic variant hc-BPPV

Treat hc-BPPV w/ BBQ roll (or other CRM) or refer to a clinician who can

Consider treating hc-BPPV w/ modified Gufoni maneuver
Consider ED consultation and/or MRI

98%

Treatment success; Patient can walk independently
Discharge & outpatient follow-up with neurology or ENT

* A **positive Dix-Hallpike test** is the reproduction of symptoms with transient upbeat torsional nystagmus.
* A **positive supine roll test** is the reproduction of symptoms with transient horizontal direction changing nystagmus

Apogeotropic hc-BPPV
Supine roll to left

horizontal nystagmus - less intense side

Apogeotropic hc-BPPV
Supine roll to right

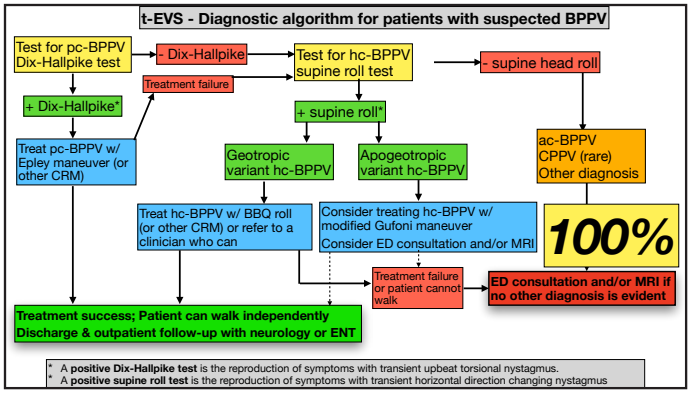
horizontal nystagmus - more intense side

Anterior canal BPPV
(don't worry about it)

- Rare (~ 2% of BPPV cases)
- Tends to quickly resolve spontaneously due to the physical position of the canal
- The nystagmus is downbeat vertical, which more commonly indicates a central cause

Sounds like BPPV . . .
. . . but the Dix-Hallpike is negative (or positive) bilaterally, or the Epley doesn't work - WHY?

4 **Wrong "PPV"** - C(central)PPV instead of B(benign) PPV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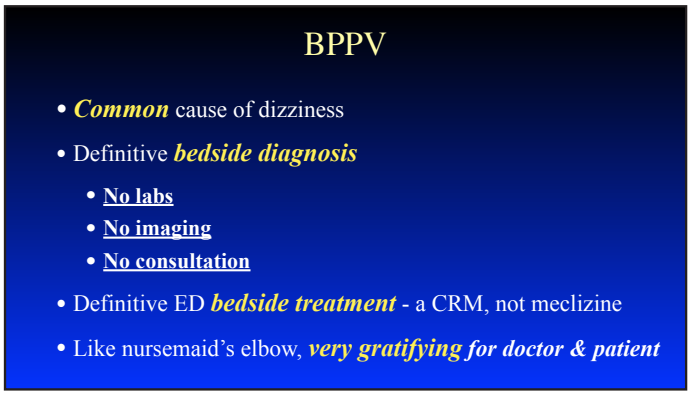
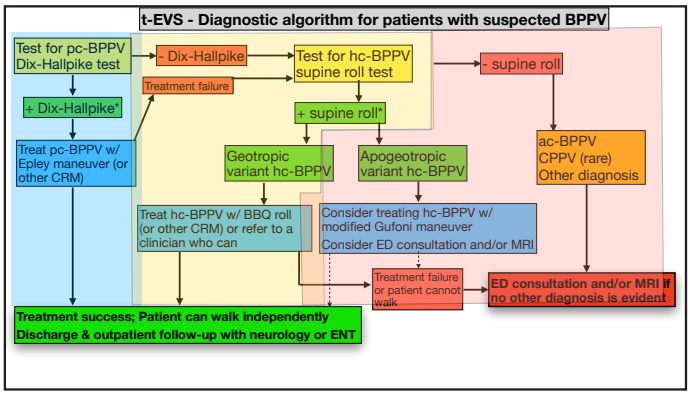
Geotropic variant hc-BPPV
Apogeotropic variant hc-BPPV
ac-BPPV CPPV (rare) Other diagnosis
Treat hc-BPPV w/ BBQ roll (or other CRM) or refer to a clinician who can
Consider treating hc-BPPV w/ modified Gufoni maneuver
Consider ED consultation and/or MRI
100%
Treatment failure or patient cannot walk
ED consultation and/or MRI if no other diagnosis is evident
Treatment success; Patient can walk independently
Discharge & outpatient follow-up with neurology or ENT
* A positive Dix-Hallpike test is the reproduction of symptoms with transient upbeat torsional nystagmus.
* A positive supine roll test is the reproduction of symptoms with transient horizontal direction changing nystagmus


Treat BPPV with a canalith repositioning maneuver NOT meclizine
• Epley for pc-BPPV
• Lempert BBQ roll for hc-BPPV
Reflex use of meclizine to treat BPPV or "vertigo"


BPPV
• Common cause of dizziness
• Definitive bedside diagnosis
• No labs
• No imaging
• No consultation
• Definitive ED bedside treatment - a CRM, not meclizine
• Like nursemaid's elbow, very gratifying for doctor & patient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Treatment failure or patient cannot walk
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Treatment success; Patient can walk independently
Discharge & outpatient follow-up with neurology or ENT

SELF EVALUATION

Benign Paroxysmal Positional Vertigo (BPPV): Diagnosis and Bedside Management

True/False

1. The lifetime incidence of BPPV is approximately 10%.

2. The pathophysiology of BPPV is post-viral inflammation of the vestibular nerve.

3. The most common canal for BPPV to occur in is the posterior semicircular canal.

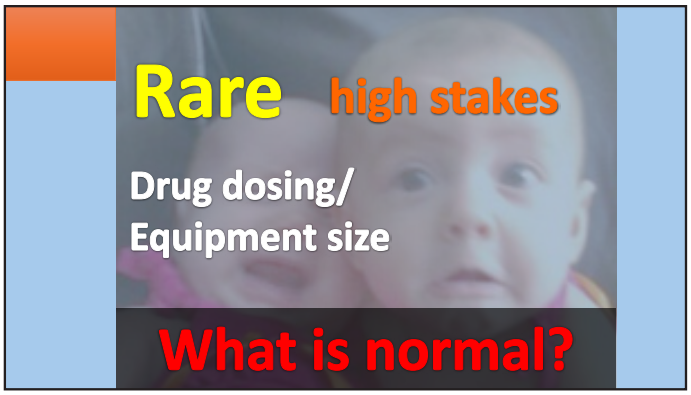
4. The most common diagnostic test for posterior canal BPPV is the Dix-Hallpike test.

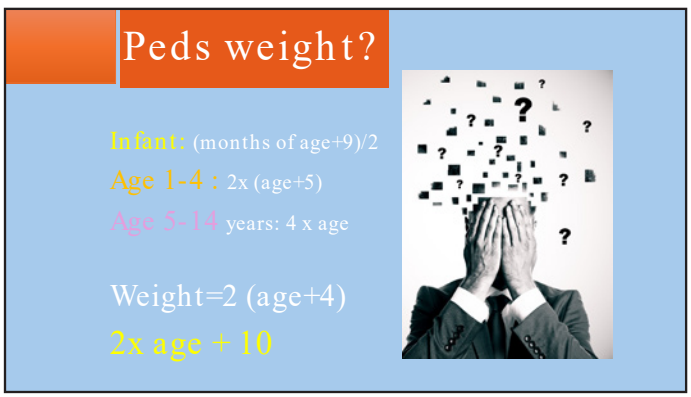
5. In a patient with right sided posterior canal BPPV, the Dix-Hallpike will be positive when the patient's head is turned towards the right, but negative when the head is turned towards the left.

6. The best treatment for BPPV is meclizine, or some other vestibular sedative.

Answer Key: 1. T, 2. F, 3. T, 4. T, 5. T, 6. F

Pediatric Resuscitation Essentials
Emily Rose, MD, FAAP, FAAEM, FACEP





Years	KG
1	10
3	15
5	20
7	25

Birth 3 kg
4 mo 6 kg

AGE	HR	RR
0-1 YR	140	40
1-4	120	30
4-12	100	20
>12	80	15

BP (5th %)	
NEONATE	60 (mmHg)
INFANT	70
1-10 yr	(age x 2) + 70
>10 yr	90

How kids are different...

1. Predisposition for respiratory failure
2. Increased sympathetic tone

Pediatric Trauma

MOST head injuries are **MINOR** and requires

NO intervention

PECARN (<2 yrs)

- Normal mental status
- No scalp hematoma (except frontal)
- LOC <5 seconds
- Non-severe mechanism
- No palpable skull fracture
- Acting normally per parents

Negative Predictive Value

99.99%

Severe Mechanism

- MVC
  - Ejection
  - Passenger death
  - Rollover
- Auto vs Peds/Bicycle (no helmet)
- Fall
- Head struck by high impact object

PECARN (>2 yrs)

- Normal mental status
- No LOC
- No vomiting
- Non-severe mechanism
- No basilar skull fracture
- No severe headache

Negative Predictive Value

99.95%

Hemodynamics

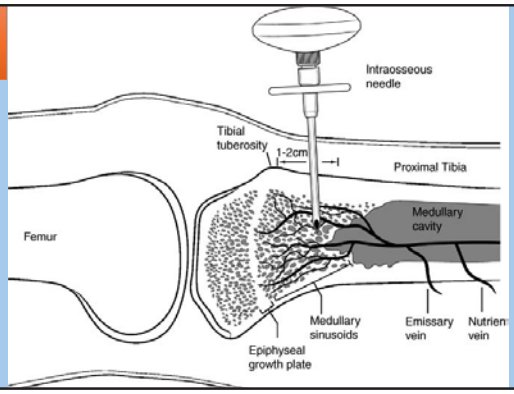
Tachycardia = FIRST sign of hypovolemia

BP may be maintained despite 45% loss of circulating blood volume

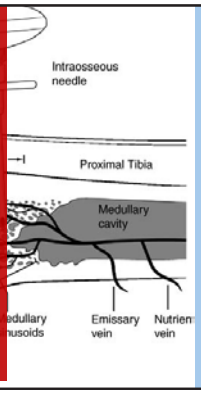
Resuscitation

20 mL/kg crystalloid

10-20 mL/kg PRBCs



Proximal tibia
Distal tibia
Distal femur
Proximal humerus
Sternum (age >12 yr)
Anterior iliac spine
Distal radius
Clavicle
Os calcis



Pediatric Trauma

- Your odds of saving a life are best with kids
- Maintain the ALARA concept
- Don't rely on BP to clue you into hemorrhage

Pediatric Sepsis

Sepsis Hemodynamics

Hypotension = LATE finding in children

COLD shock is more common than WARM shock

Sepsis Treatment Goals

- Distal pulses = central
- Capillary refill <2 sec
- Normal mental status
- Normal urinary output
- BP ≥ 5th %ile for age
- Lactate <4 mmol/L
- Hgb >10 g/dL
- ScvO2 ≥70% (if available)

Fluid Resuscitation

20 mL/kg crystalloid (60 mL/kg 60')

Goal: Perfusion or Overload

Antibiotics (<1 Hr)

Glucose

Calcium

Fluid Resuscitation

20 mL/kg crystalloid (60 mL/kg 60')

Goal: Perfusion or Overload

Antibiotics (<1 Hr)

Glucose

Calcium

Caution: Resource-Limited Setting

Vasopressors in Children

Dopamine if poor perfusion and normal BP

Cold Shock (hypotensive and vasoconstricted): give Epinephrine

Warm Shock (hypotensive and vasodilated): give Norepinephrine

Vasopressors in Children

Milrinone

Dopamine if poor perfusion and normal BP

Cold Shock (hypotensive and vasoconstricted): give Epinephrine

Warm Shock (hypotensive and vasodilated): give Norepinephrine

Hydrocortisone if catecholamine-resistant

Peds Sepsis Management:

Intravenous Fluids (5 min)

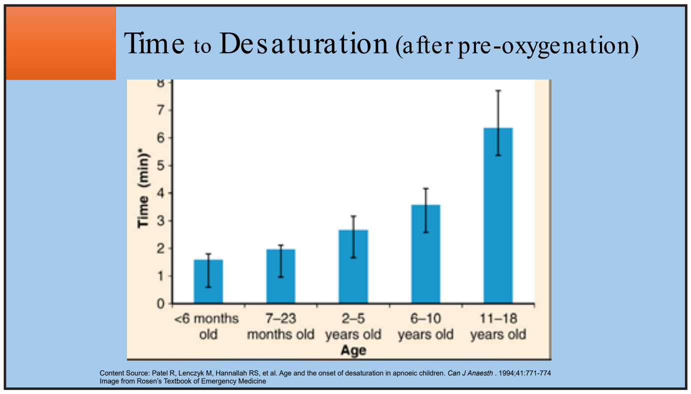
Antibiotics (<1 hour)

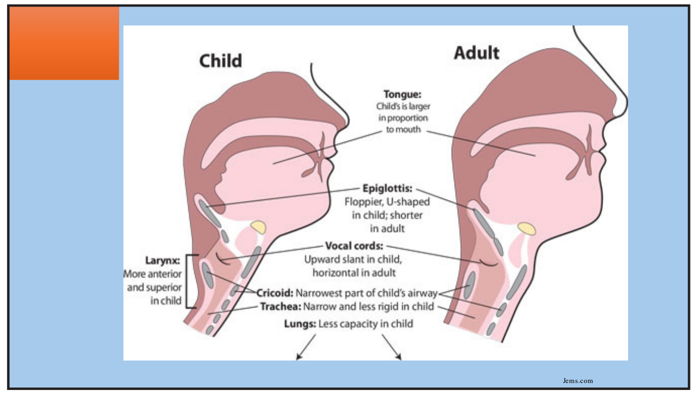
Pressors

Hydrocortisone if refractory to catecholamines

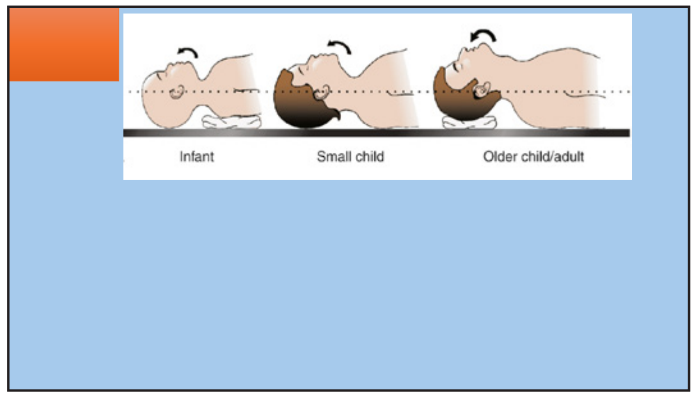
Pediatric Airway

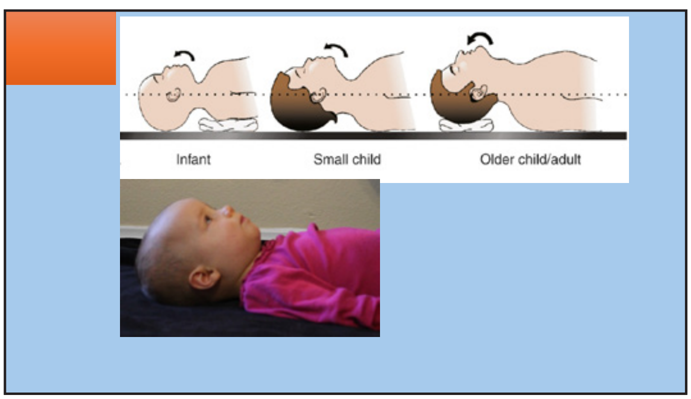
95% of all cardiac arrest in children is of a respiratory etiology


Time to Desaturation (after pre-oxygenation)
Time (min)*
0
1
2
3
4
5
6
7
<6 months old
7–23 months old
2–5 years old
6–10 years old
11–18 years old
Age
Content Source: Patel R, Lenczyk M, Hannallah RS, et al. Age and the onset of desaturation in apnoeic children. Can J Anaesth. 1994;41:771-774
Image from Rosen's Textbook of Emergency Medicine


Child
Adult
Tongue: Child's is larger in proportion to mouth
Epiglottis: Floppier, U-shaped in child; shorter in adult
Vocal cords: Upward slant in child, horizontal in adult
Larynx: More anterior and superior in child
Cricoid: Narrowest part of child's airway
Trachea: Narrow and less rigid in child
Lungs: Less capacity in child
Jems.com


PEDIATRIC


Infant
Small child
Older child/adult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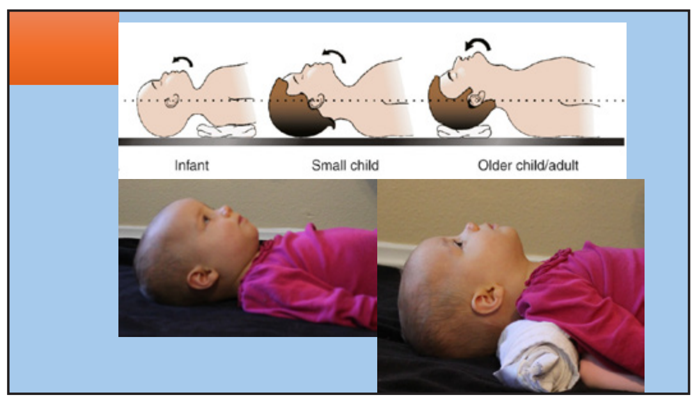
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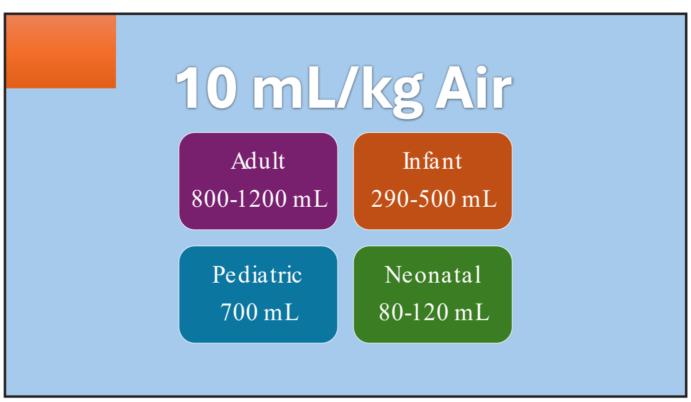
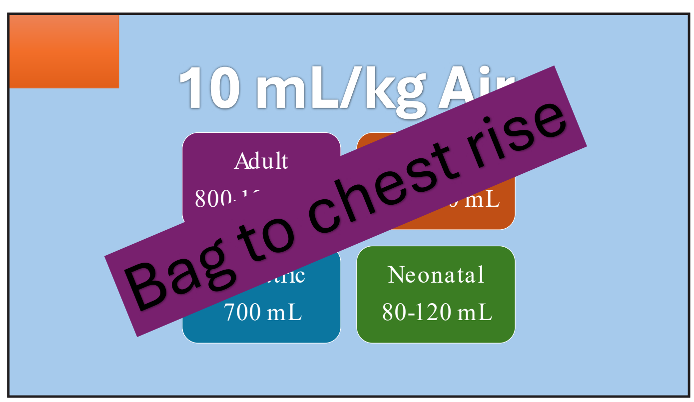
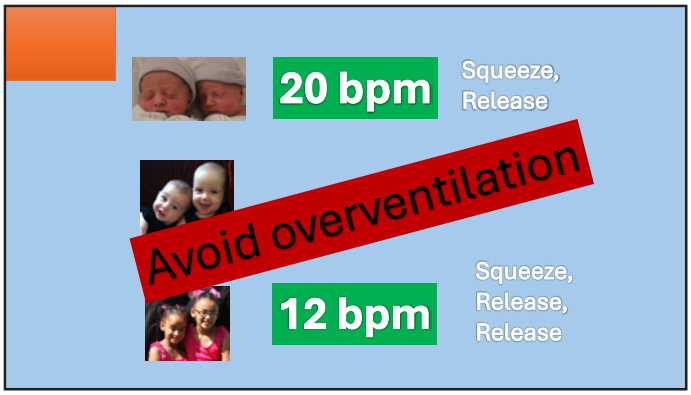
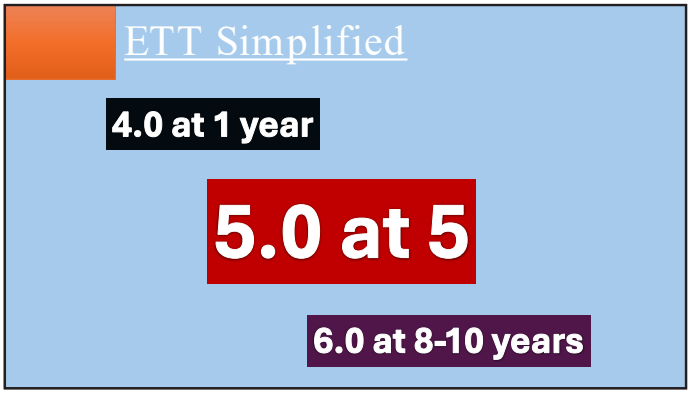
Older child/adult

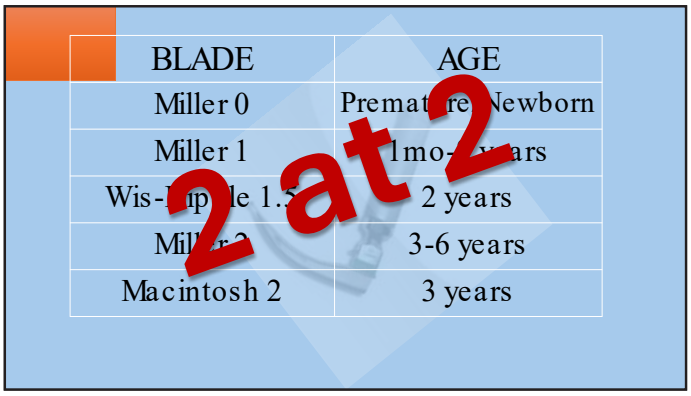

10 mL/kg Air
Adult
800-1200 mL
Infant
290-500 mL
Pediatric
700 mL
Neonatal
80-120 mL


10 mL/kg Ai
Bag to chest rise
Adult
700 mL
Neonatal
80-120 mL





BLADE	AGE
Miller 0	Premature/Newborn
Miller 1	1mo-2 years
Wis-Hipple 1.5	2 years
Miller 2	3-6 years
Macintosh 2	3 years



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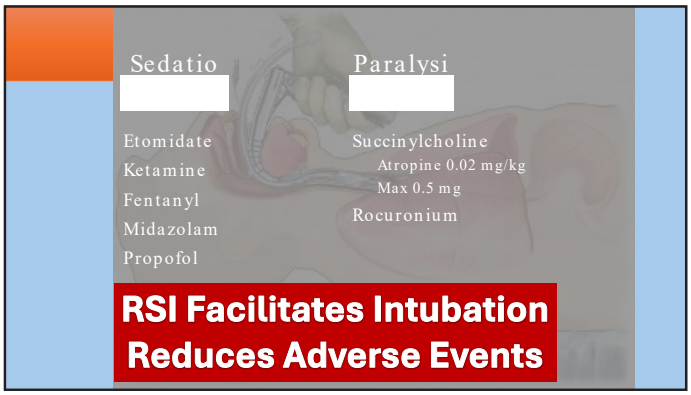
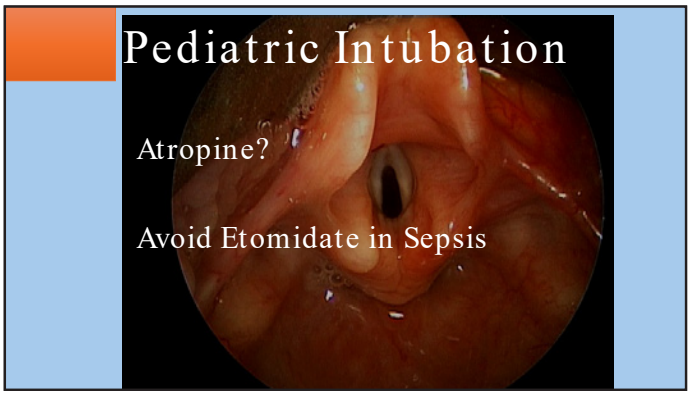
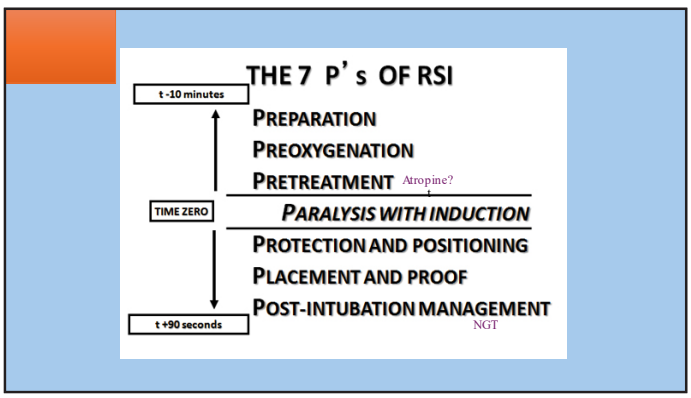
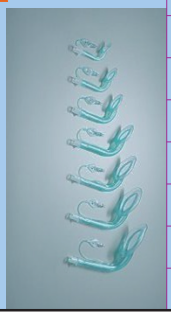


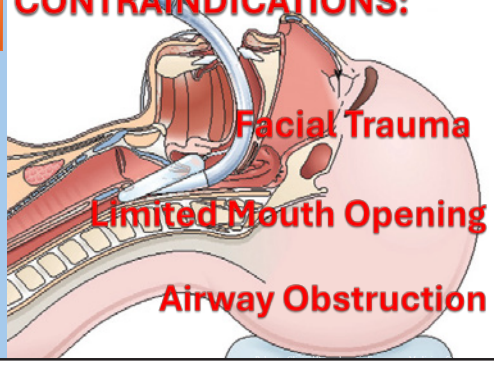






LMA SIZE	WEIGHT (KG)
1	≤ 5
1.5	5-10
2	10-20
2.5	20-30
3	30-50
4	50-70
5	70-100
6	>100

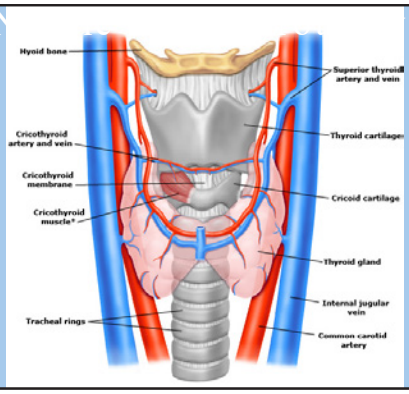
CONTRAINDICATIONS:

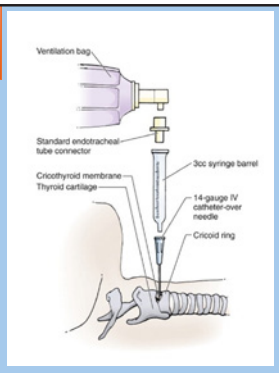


Facial Trauma

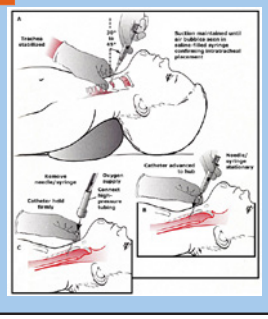
Limited Mouth Opening

Airway Obstruction





Needle Cricothyrotomy

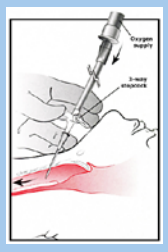


10-12 L/min
Flow Rate

Percutaneous Transtracheal Ventilation

1:4 I:E Ratio
10-12 Breaths/min

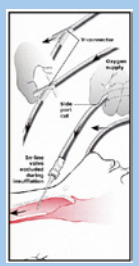
Passive
Exhalation



Percutaneous Transtracheal Ventilation

Airway
Obstruction

1:8-10 I:E Ratio
RR 5-6 Breaths/Min



Complications to Needle Cric

- Unsuccessful
- Barotrauma
- Posterior Tracheal Injury
- Esophageal Perforation
- Laryngeal/Vocal Cord Damage
- Infection
- Alternative: CERTAIN DEATH

Resuscitation Mechanics

Compressions

Hard (1/3 AP diameter) and Fast (100-120/min)

End-tidal CO2 →monitor CPR quality. No established values in children

Ventilation

15:2 if no advanced airway

1 breath/2-3 sec (20-30 breaths/min) with continuous chest compressions *2020 update*

Resuscitation Mechanics

Compressions

Hard (1/3 AP diameter) and Fast (100-120/min)

End-tidal CO2 →monitor CPR quality. No established values in children

Ventilation

Conventional CPR better

15:2 if no advanced airway

1 breath/2-3 sec (20-30 breaths/min) with continuous chest compressions *2020 update*

PALS

Consider reversible cause of asystole and PEA

Shock with 2-4 J/kg (max 10 J/kg) in VF/VT

Children <8 years:

AED with peds dose attenuating system (if possible)

Can move pads A-P to separate

Venoarterial ECMO

Improved survival in reversible underlying disease processes

Post-Arrest Care

Avoid Hyperthermia

Normoxia (>94%)

Maintain SBP>5th %ile for age

(parenteral fluids and/or inotropes/vasoactive drugs)

Neonatal Resuscitation

85% initiate spontaneous respirations within 10-30 seconds

10% respond to drying and stimulation

3% Need PPV

2% Intubation

0.1% Require chest compressions

Neonatal Resuscitation

PPV if HR <100

Compressions if HR <60

Neonatal Resuscitation

PPV if HR <100

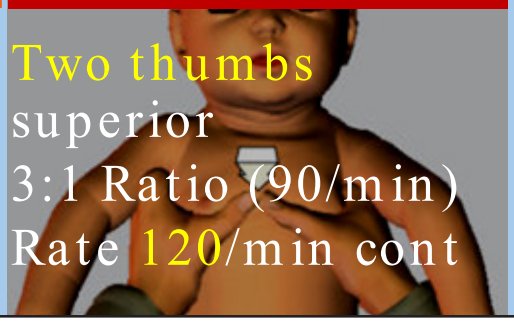
40-60 bpm (30 bpm with compressions)

Room Air if >35 weeks

Compressions if HR <60

100% O2 with compressions

Neonatal Resuscitation



Two thumbs superior

3:1 Ratio (90/min)

Rate 120/min cont

Neonatal Resuscitation

Intubation

Epinephrine

IVF/Blood

Withholding Support

<23 Weeks

<400 Grams

Severe Congenital Anomalies

DISCONTINUE RESUSCITATION AFTER 10 MINUTES

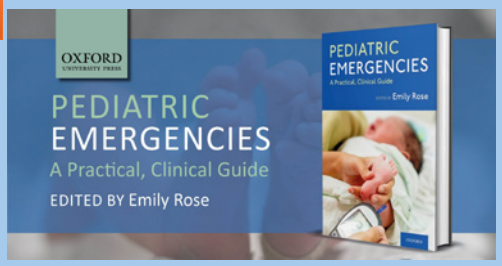
If no HR

Neonatal Resuscitation

- Room Air and Titrate Up
- Ventilation is the priority
- Compressions if HR<60
- Discontinue Resuscitation after 10 minutes

Summary

- Kids don't get hypotensive easily
- 95% cardiac arrest in kids is of respiratory etiology
- Go crazy with IVF in sepsis (unless you're in Africa)
- Cold shock is more common in kids→epi +/- milrinone
- Neonatal Resus—PPV when HR <100; Compressions if HR<60
- Asthma still kills kids. Educate!!



SELF EVALUATION

Pediatric Resuscitation Essentials

1. A baby is born without a pulse. Despite active resuscitation, no pulse is regained. How long should the resuscitation continue per the NALS guidelines?

a. 10 minutes
b. 20 minutes
c. 30 minutes
d. 60 minutes

2. What is the pressor of choice in most cases of pediatric sepsis?

a. Dopamine
b. Epinephrine
c. Norepinephrine
d. Milrinone

3. A 3-year-old child is found unresponsive and not breathing normally. Which of the following is the most appropriate first action?

a. Check for a pulse for 10–15 seconds
b. Start chest compressions immediately
c. Deliver two rescue breaths and then reassess
d. Open the airway and check for responsiveness and normal breathing

4. When performing CPR on a pediatric patient with a single rescuer, what is the correct compression-to-ventilation ratio?

a. 15:2
b. 30:2
c. 5:1
d. 3:1

5. What is the initial recommended dose of energy for defibrillation in a pediatric patient with ventricular fibrillation?

a. 2 J/kg
b. 4 J/kg
c. 6 J/kg
d. 10 J/kg

Answer Key: 1. A, 2. B, 3. D, 4. B, 5. A

Cardiac Arrest: Beyond the Guidelines

Michael W. Donnino, MD

DISCLOSURES

1. RESEARCH GRANTS: NIH, DAY ZERO DIAGNOSTICS, FOUNDATIONS, PHILANTHROPY

OBJECTIVES

1. TO EXAMINE CHALLENGING CASES IN CARDIAC ARREST MANAGEMENT
2. TO EXPLORE OPTIMAL AIRWAY MANAGEMENT STRATEGIES DURING CARDIAC ARREST
3. TO EVALUATE CURRENT THERAPIES FOR CARDIAC ARREST AND POST-ARREST MANAGEMENT

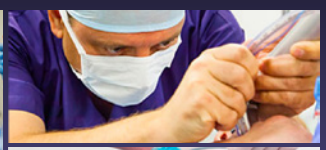
TO INTUBATE OR NOT TO INTUBATE?

YOU ARE CALLED TO ROOM 21 AND FIND A 65 YEAR OLD MALE IN CARDIAC ARREST....

WHAT NEXT?

1. INTUBATE AS SOON AS POSSIBLE
2. DO NOT INTUBATE UNTIL ROSC
3. GIVE COUPLE ROUNDS OF CPR/ACLS AND THEN CONSIDER INTUBATION
4. I HAVE NO IDEA
5. SOME OTHER OPTION NOT MENTIONED

PERHAPS..."IT DEPENDS"



Variable	Jabre et al	Wang et al	Benger et al
Location	France & Belgium	USA	England
Number of Subjects	2,043	3,004	9,296
Intervention (vs. ETI)	BVM	SGA (King LT)	SGA (iGel)
Operator	Paramedic (w/ ED MD) or ED MD	ALS (or specially trained BLS)	ALS
Outcome	No difference in survival (RR 1.02) or good neurological outcome (1.03)	Higher survival (RR 1.34) and good neurological survival (RR 1.42) for SGA group	No difference in survival (RR 0.95) or good neurologic outcome (RR 0.96) at hospital discharge

Variable	Jabre et al	Wang et al	Benger et al
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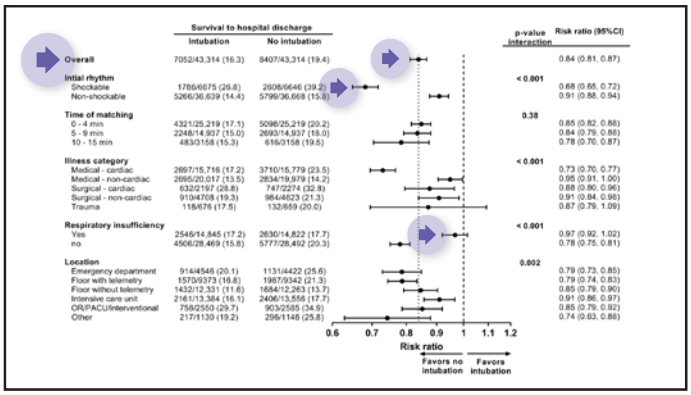
Intervention (vs. ETI)	BVM	SGA (King LT)	SGA (iGel)
Operator	Paramedic (w/ ED MD) or ED MD	ALS (or specially trained BLS)	ALS
ETI Success Rate	98%	52% (vs 81% for LT)	69% (vs 85.4% for iGel)
Outcome	No difference in survival (RR 1.02) or good neurological outcome (1.03)	Higher survival (RR 1.34) and good neurological survival (RR 1.42) for **SGA** group	No difference in survival (RR 0.95) or good neurologic outcome (RR 0.96) at hospital discharge

WHAT ABOUT IN-HOSPITAL ARREST?

- 86,628 PTS - PROPENSITY SCORE WITH RISK SET MATCHING
- SURVIVAL WAS LOWER FOR THOSE INTUBATED COMPARED TO THOSE NOT INTUBATED (16 VS 19%)

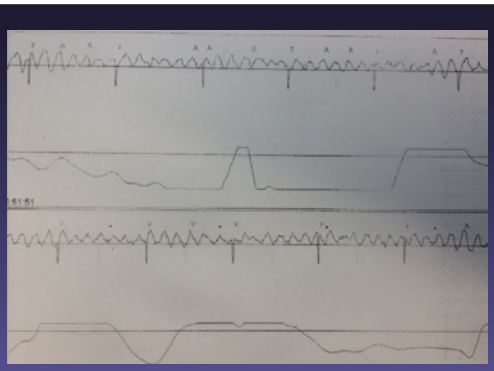


CURRENT RECOMMENDATIONS

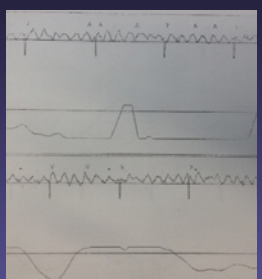
- EITHER BAG-VALVE MASK OR ADVANCED AIRWAY STRATEGY MAY BE CONSIDERED
- IF ADVANCED AIRWAY IS USED, EITHER AN ENDOTRACHEAL TUBE OR SUPRA-GLOTTIC AIRWAY CAN BE USED DEPENDING ON EXPERTISE
- SMALL PRINT: ULTIMATE DECISION SHOULD BE BASED ON HOST OF PATIENT AND PROVIDER CHARACTERISTICS

71 YEAR OLD POST-OP DAY #5 FROM CABG IS NOW IN CARDIAC ARREST

WHAT DO YOU WANT TO KNOW NEXT???



APART FROM CPR, WHAT NEXT??



1. DEFIBRILLATE
2. GIVE EPINEPHRINE 1 MG IV
3. GIVE AMIODARONE 300 MG IV
4. CALL SURGEON FOR THORACOTOMY
5. SOMETHING ELSE

WHICH ANTI-ARRHYTHMIC DO YOU REACH FOR FIRST IN PATIENTS WITH PERSISTENCE OF THIS RHYTHM DESPITE DEFIBRILLATION?

1. LIDOCAINE
2. AMIODARONE
3. EITHER LIDOCAINE OR AMIODARONE
4. NEITHER LIDOCAINE OR AMIODARONE AS THEY REALLY DON'T WORK

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The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Amiodarone, Lidocaine, or Placebo in Out-of-Hospital Cardiac Arrest

P.J. Kudenchuk, S.P. Brown, M. Daya, T. Rea, G. Nichol, L.J. Morrison, B. Leroux, C. Vaillancourt, L. Wittwer, C.W. Callaway, J. Christenson, D. Egan, J.P. Ornato, M.L. Weisfeldt, I.G. Stiell, A.H. Idris, T.P. Aufderheide, J.V. Dunford, M.R. Colella, G.M. Vilke, A.M. Brienza, P. Desvigne-Nickens, P.C. Gray, R. Gray, N. Seals, R. Straight, and P. Dorian, for the Resuscitation Outcomes Consortium Investigators*

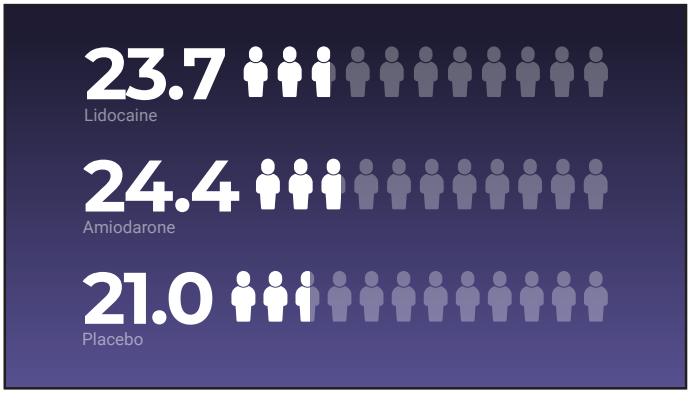
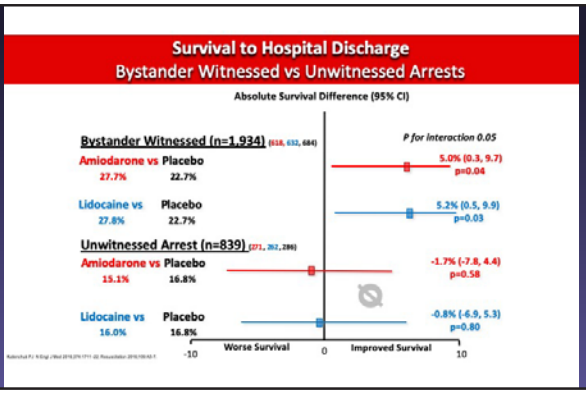


Table 3. Outcomes According to Trial Group in the Per-Protocol Population.*

Outcome	Amiodarone (N=974)	Lidocaine (N=993)	Placebo (N=1059)	Amiodarone vs. Placebo		Lidocaine vs. Placebo		Amiodarone vs. Lidocaine	
				Difference (95% CI)	P Value	Difference (95% CI)	P Value	Difference (95% CI)	P Value
				percentage points		percentage points		percentage points	
Primary outcome: survival to discharge — no./total no. (%)†	237/970 (24.4)	233/985 (23.7)	222/1056 (21.0)	3.2 (-0.4 to 7.0)	0.08	2.6 (-1.0 to 6.3)	0.16	0.7 (-3.2 to 4.7)	0.70
Secondary outcome: modified Rankin score ≤3 — no./total no. (%)‡	182/967 (18.8)	172/984 (17.5)	175/1055 (16.6)	2.2 (-1.1 to 5.6)	0.19	0.9 (-2.4 to 4.2)	0.59	1.3 (-2.1 to 4.8)	0.44
Mechanistic (exploratory) outcomes									
Return of spontaneous circulation at ED arrival — no./total no. (%)	350/974 (35.9)	396/992 (39.9)	366/1059 (34.6)	1.4 (-2.8 to 5.5)	0.52	5.4 (1.2 to 9.5)	0.01	-4.0 (-8.3 to 0.3)	0.07
Admitted to hospital — no. (%)	445 (45.7)	467 (47.0)	420 (39.7)	6.0 (1.7 to 10.3)	0.01	7.4 (3.1 to 11.6)	<0.001	-1.3 (-5.7 to 3.1)	0.55
Modified Rankin score in all patients‡	5.0±1.9	5.1±1.8	5.2±1.8	-0.14 (-0.30 to 0.02)	0.09	-0.06 (-0.22 to 0.10)	0.45	-0.08 (-0.24 to 0.08)	0.34



EPINEPHRINE

"PATIENTS RECEIVING ADRENALINE DURING CARDIAC ARREST HAD NO SIGNIFICANT IMPROVEMENT IN THE PRIMARY OUTCOME OF SURVIVAL TO HOSPITAL DISCHARGE ALTHOUGH THERE THERE WAS A SIGNIFICANTLY IMPROVED LIKELIHOOD OF RETURN OF SPONTANEOUS CIRCULATION"

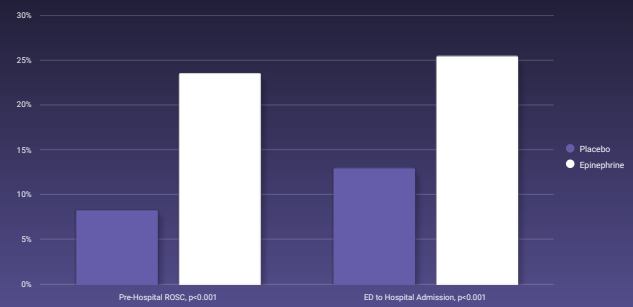

Resuscitation

Effect of adrenaline on survival in out-of-hospital cardiac arrest: A randomised double-blind placebo-controlled trial ☆

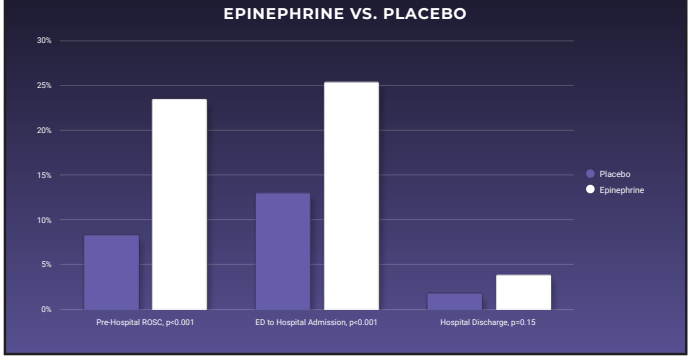
Abstract

Background

EPINEPHRINE VS. PLACEBO



EPINEPHRINE VS. PLACEBO



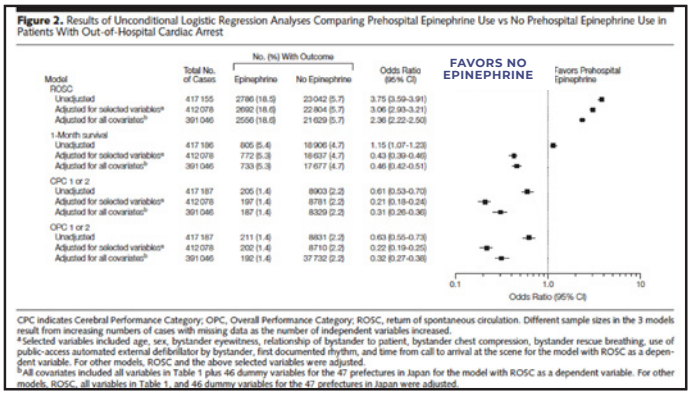
Prehospital Epinephrine Use and Survival Among Patients With Out-of-Hospital Cardiac Arrest

Akihito Hagihara, DMSc, MPH
Masaru Hasegawa, MD
Takeru Abe, MA
Takashi Nagata, MD
Yoshifumi Wakata, MD
Shogo Miyazaki, PhD

Figure 2. Results of Unconditional Logistic Regression Analyses Comparing Prehospital Epinephrine Use vs No Prehospital Epinephrine Use in Patients With Out-of-Hospital Cardiac Arrest

Model	Total No. of Cases	No. (%) With Outcome: Epinephrine	No. (%) With Outcome: No Epinephrine	Odds Ratio (95% CI)
ROSC				
Unadjusted	417 155	2786 (18.5)	23 042 (5.7)	3.75 (3.59-3.91)
Adjusted for selected variables[a]	412 078	2692 (18.6)	22 804 (5.7)	3.06 (2.93-3.21)
Adjusted for all covariates[b]	391 046	2566 (18.6)	21 629 (5.7)	2.36 (2.22-2.50)
1-Month survival				
Unadjusted	417 186	805 (5.4)	18 906 (4.7)	1.15 (1.07-1.23)
Adjusted for selected variables[a]	412 078	772 (5.3)	18 637 (4.7)	0.43 (0.39-0.46)
Adjusted for all covariates[b]	391 046	733 (5.3)	17 677 (4.7)	0.46 (0.42-0.51)
CPC 1 or 2				
Unadjusted	417 187	205 (1.4)	8903 (2.2)	0.61 (0.53-0.70)
Adjusted for selected variables[a]	412 078	197 (1.4)	8781 (2.2)	0.21 (0.18-0.24)
Adjusted for all covariates[b]	391 046	187 (1.4)	8329 (2.2)	0.31 (0.26-0.36)
CPC 1 or 2				
Unadjusted	417 187	211 (1.4)	8831 (2.2)	0.63 (0.55-0.73)
Adjusted for selected variables[a]	412 078	202 (1.4)	8710 (2.2)	0.22 (0.19-0.25)
Adjusted for all covariates[b]	391 046	192 (1.4)	37 732 (2.2)	0.32 (0.27-0.38)

CPC indicates Cerebral Performance Category; OPC, Overall Performance Category; ROSC, return of spontaneous circulation. Different sample sizes in the 3 models result from increasing numbers of cases with missing data as the number of independent variables increased.
[a] Selected variables included age, sex, bystander eyewitness, relationship of bystander to patient, bystander chest compression, bystander rescue breathing, use of public-access automated external defibrillator by bystander, first documented rhythm, and time from call to arrival at the scene for the model with ROSC as a dependent variable. For other models, ROSC and the above selected variables were adjusted.
[b] All covariates included all variables in Table 1 plus 46 dummy variables for the 47 prefectures in Japan for the model with ROSC as a dependent variable. For other models, ROSC, all variables in Table 1, and 46 dummy variables for the 47 prefectures in Japan were adjusted.


BMJ

RESEARCH

Evaluation of pre-hospital administration of adrenaline (epinephrine) by emergency medical services for patients with out of hospital cardiac arrest in Japan: controlled propensity matched retrospective cohort study

OPEN ACCESS

Shinji Nakahara *assistant professor*[1], Jun Tomio *assistant professor*[2,3], Hideto Takahashi *associate professor*[4], Masao Ichikawa *professor*[4], Masamichi Nishida *director*[5], Naoto Morimura *professor*[6], Tetsuya Sakamoto *professor*[7]

Abstract

Objectives To evaluate the effectiveness of pre-hospital adrenaline (epinephrine) administered by emergency medical services to patients with out of hospital cardiac arrest.

Design Controlled propensity matched retrospective cohort study. In which pairs of patients with or without control, adrenaline were created

Intact survival (6.6% v 6.4%; 1.01, 0.78 to 1.30) among those with VF/VT, and higher proportions of overall survival (4.0% v 2.4%; odds ratio 1.72, 1.45 to 2.04) and neurologically intact survival (0.7% v 0.4%; 1.87, 1.04 to 2.37) among those with non-VF/VT.

Conclusions Pre-hospital administration of adrenaline by emergency medical services improves the long term outcome in patients with out

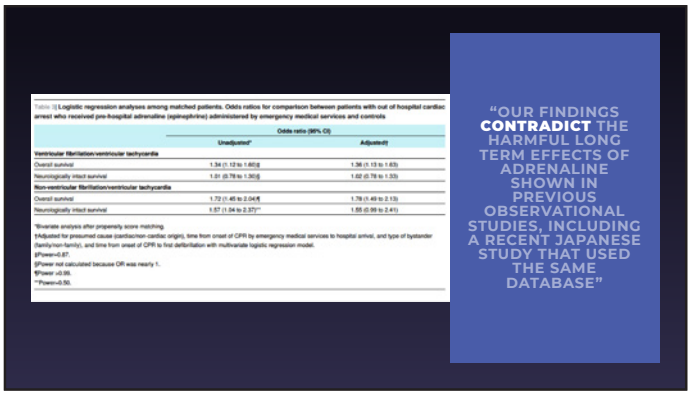
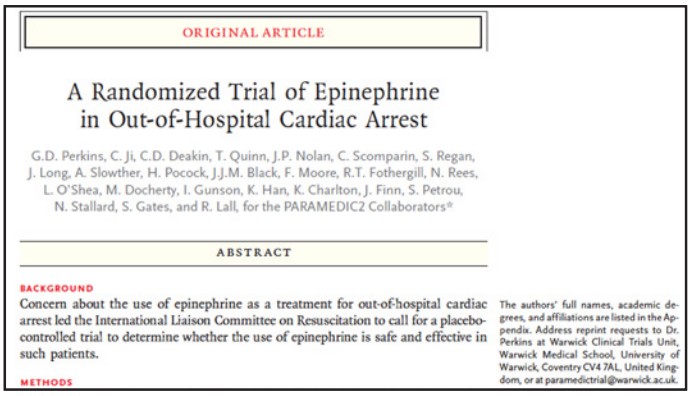


Table 3. Logistic regression analyses among matched patients. Odds ratios for comparison between patients with out of hospital cardiac arrest who received pre-hospital adrenaline (epinephrine) administered by emergency medical services and controls

	Odds ratio (95% CI)	
	Unadjusted*	Adjusted†
Ventricular fibrillation/ventricular tachycardia		
Overall survival	1.34 (1.13 to 1.60)‡	1.36 (1.13 to 1.63)
Neurologically intact survival	1.01 (0.78 to 1.30)‡	1.02 (0.78 to 1.33)
Non-ventricular fibrillation/ventricular tachycardia		
Overall survival	1.72 (1.45 to 2.04)¶	1.78 (1.49 to 2.13)
Neurologically intact survival	1.57 (1.04 to 2.37)**	1.55 (0.99 to 2.41)

*Bivariate analysis after propensity score matching.
†Adjusted for presumed cause (cardiac/non-cardiac origin), time from onset of CPR by emergency medical services to hospital arrival, and type of bystander (family/non-family), and time from onset of CPR to first defibrillation with multivariate logistic regression model.
‡Power=0.87.
§Power not calculated because OR was nearly 1.
¶Power =0.99.
**Power=0.50.

"OUR FINDINGS **CONTRADICT** THE HARMFUL LONG TERM EFFECTS OF ADRENALINE SHOWN IN PREVIOUS OBSERVATIONAL STUDIES, INCLUDING A RECENT JAPANESE STUDY THAT USED THE SAME DATABASE"



ORIGINAL ARTICLE

A Randomized Trial of Epinephrine in Out-of-Hospital Cardiac Arrest

G.D. Perkins, C. Ji, C.D. Deakin, T. Quinn, J.P. Nolan, C. Scomparin, S. Regan, J. Long, A. Slowther, H. Pocock, J.J.M. Black, F. Moore, R.T. Fothergill, N. Rees, L. O'Shea, M. Docherty, I. Gunson, K. Han, K. Charlton, J. Finn, S. Petrou, N. Stallard, S. Gates, and R. Lall, for the PARAMEDIC2 Collaborators*

ABSTRACT

BACKGROUND

Concern about the use of epinephrine as a treatment for out-of-hospital cardiac arrest led the International Liaison Committee on Resuscitation to call for a placebo-controlled trial to determine whether the use of epinephrine is safe and effective in such patients.

METHODS

The authors' full names, academic degrees, and affiliations are listed in the Appendix. Address reprint requests to Dr. Perkins at Warwick Clinical Trials Unit, Warwick Medical School, University of Warwick, Coventry CV4 7AL, United Kingdom, or at paramedictrial@warwick.ac.uk.

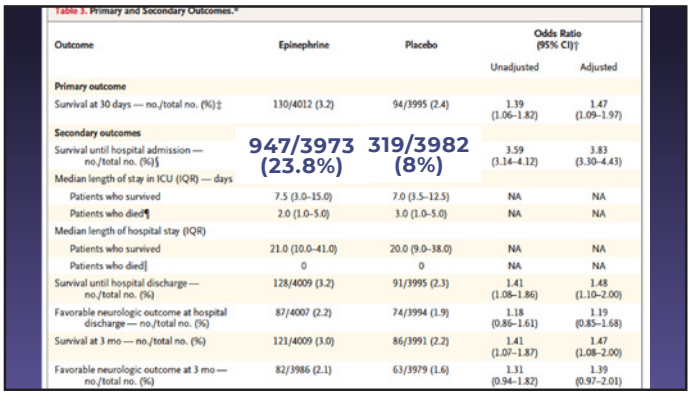
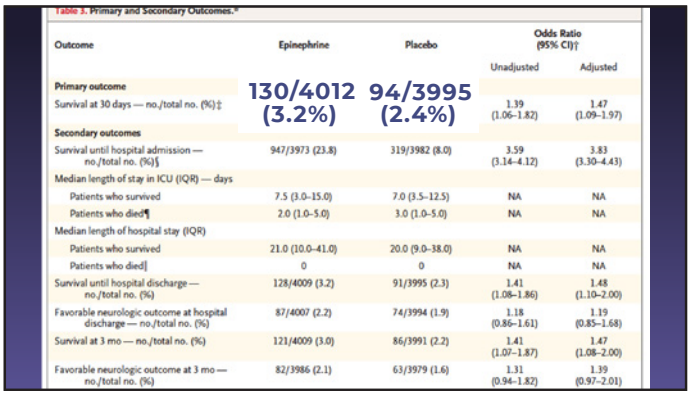


Table 3. Primary and Secondary Outcomes.*

Outcome	Epinephrine	Placebo	Odds Ratio (95% CI)† Unadjusted	Odds Ratio (95% CI)† Adjusted
Primary outcome				
Survival at 30 days — no./total no. (%)‡	130/4012 (3.2)	94/3995 (2.4)	1.39 (1.06–1.82)	1.47 (1.09–1.97)
Secondary outcomes				
Survival until hospital admission — no./total no. (%)§	947/3973 (23.8)	319/3982 (8%)	3.59 (3.14–4.12)	3.83 (3.30–4.43)
Median length of stay in ICU (IQR) — days				
Patients who survived	7.5 (3.0–15.0)	7.0 (3.5–12.5)	NA	NA
Patients who died¶	2.0 (1.0–5.0)	3.0 (1.0–5.0)	NA	NA
Median length of hospital stay (IQR)				
Patients who survived	21.0 (10.0–41.0)	20.0 (9.0–38.0)	NA	NA
Patients who died‖	0	0	NA	NA
Survival until hospital discharge — no./total no. (%)	128/4009 (3.2)	91/3995 (2.3)	1.41 (1.08–1.86)	1.48 (1.10–2.00)
Favorable neurologic outcome at hospital discharge — no./total no. (%)	87/4007 (2.2)	74/3994 (1.9)	1.18 (0.86–1.61)	1.19 (0.85–1.68)
Survival at 3 mo — no./total no. (%)	121/4009 (3.0)	86/3991 (2.2)	1.41 (1.07–1.87)	1.47 (1.08–2.00)
Favorable neurologic outcome at 3 mo — no./total no. (%)	82/3986 (2.1)	63/3979 (1.6)	1.31 (0.94–1.82)	1.39 (0.97–2.01)



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Patients who survived	21.0 (10.0–41.0)	20.0 (9.0–38.0)	NA	NA
Patients who died‖	0	0	NA	NA
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Favorable neurologic outcome at hospital discharge — no./total no. (%)	87/4007 (2.2)	74/3994 (1.9)	1.18 (0.86–1.61)	1.19 (0.85–1.68)
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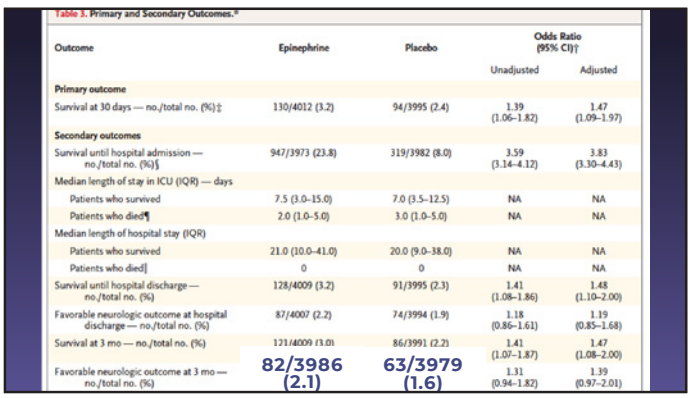
Favorable neurologic outcome at 3 mo — no./total no. (%)	82/3986 (2.1)	63/3979 (1.6)	1.31 (0.94–1.82)	1.39 (0.97–2.01)



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Secondary outcomes				
Survival until hospital admission — no./total no. (%)§	947/3973 (23.8)	319/3982 (8.0)	3.59 (3.14–4.12)	3.83 (3.30–4.43)
Median length of stay in ICU (IQR) — days				
Patients who survived	7.5 (3.0–15.0)	7.0 (3.5–12.5)	NA	NA
Patients who died¶	2.0 (1.0–5.0)	3.0 (1.0–5.0)	NA	NA
Median length of hospital stay (IQR)				
Patients who survived	21.0 (10.0–41.0)	20.0 (9.0–38.0)	NA	NA
Patients who died‖	0	0	NA	NA
Survival until hospital discharge — no./total no. (%)	128/4009 (3.2)	91/3995 (2.3)	1.41 (1.08–1.86)	1.48 (1.10–2.00)
Favorable neurologic outcome at hospital discharge — no./total no. (%)	87/4007 (2.2)	74/3994 (1.9)	1.18 (0.86–1.61)	1.19 (0.85–1.68)
Survival at 3 mo — no./total no. (%)	121/4009 (3.0)	86/3991 (2.2)	1.41 (1.07–1.87)	1.47 (1.08–2.00)
Favorable neurologic outcome at 3 mo — no./total no. (%)	82/3986 (2.1)	63/3979 (1.6)	1.31 (0.94–1.82)	1.39 (0.97–2.01)

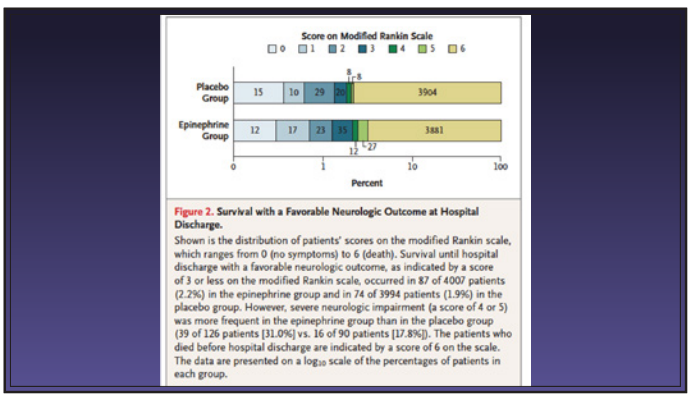
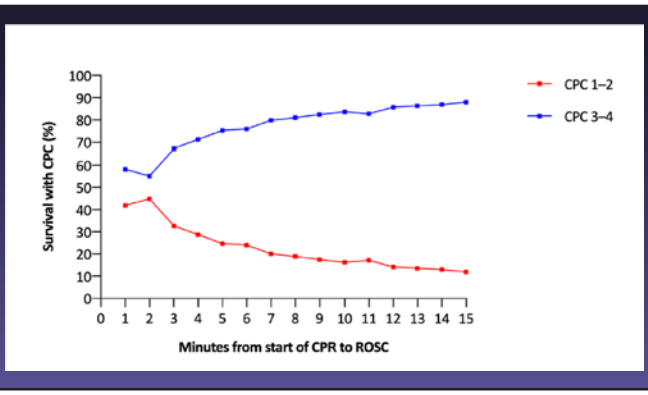
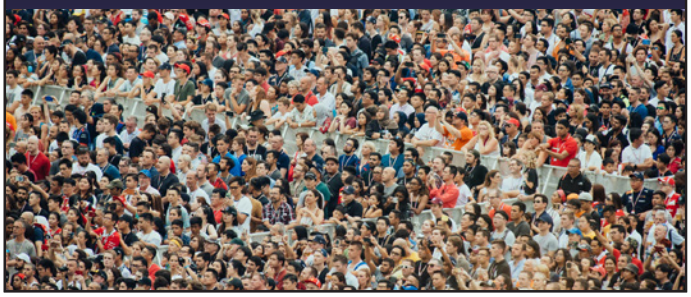


Figure 2. Survival with a Favorable Neurologic Outcome at Hospital Discharge.

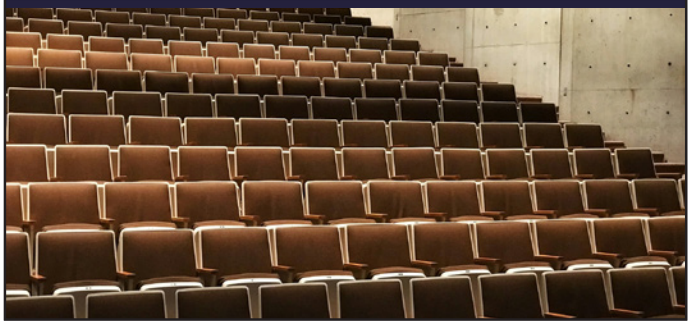
Shown is the distribution of patients' scores on the modified Rankin scale, which ranges from 0 (no symptoms) to 6 (death). Survival until hospital discharge with a favorable neurologic outcome, as indicated by a score of 3 or less on the modified Rankin scale, occurred in 87 of 4007 patients (2.2%) in the epinephrine group and in 74 of 3994 patients (1.9%) in the placebo group. However, severe neurologic impairment (a score of 4 or 5) was more frequent in the epinephrine group than in the placebo group (39 of 126 patients [31.0%] vs. 16 of 90 patients [17.8%]). The patients who died before hospital discharge are indicated by a score of 6 on the scale. The data are presented on a $\log_{10}$ scale of the percentages of patients in each group.



HOW TO REDUCE A LARGE CROWD AT THE CODE...



I FEEL A PULSE!



IMMEDIATE POST-ARREST

1) ANTICIPATE HYPOTENSION AND TREAT PROACTIVELY (LITERALLY, KEEP A FINGER ON THE PULSE)

2) BE READY TO REACT TO A RE-ARREST (IE, KEEP PADS IN PLACE)

3) GAIN ADEQUATE ACCESS (VENOUS AND ARTERIAL)

TARGETED TEMPERATURE MANAGEMENT (HISTORY)

2002- TWO "BREAKTHROUGH" RCTS PUBLISHED IN NEJM (VF/VT) SUPPORTING INDUCED HYPOTHERMIA TO 33-34C

- Small sample sizes, sub-optimal randomization in one (every other day), other issues

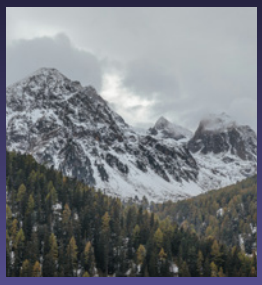
2010- TTM#1: 33 VERSUS 36C - NO DIFFERENCE

- Strength: Large, well-done trial
- Criticism/viewpoint: Did not test "normothermia"

2016- TTM#2: 33 VERSUS 37C - NO DIFFERENCE

- Strength: Large, well-done trial
- Criticism: Did not represent the patient profile we see in the US (I do not agree with this critique)

CURRENT TTM RECOMMENDATIONS



ILCOR: KEEP TEMP < 37.5

AHA: SELECT AND MAINTAIN A CONSTANT TEMP BETWEEN 32 AND 37.5

OTHER POST-ARREST INTERVENTIONS

OXYGEN: INITIALLY PROMISING TO AGGRESSIVELY LOWER BUT RCT DATA HAS NOT SUPPORTED

PCO2: NO DIFFERENCE IN RCT DATA

KEEPING THE DOCTORS AWAY MIGHT BE ONE OF THE BEST INTERVENTIONS FOR POST-CARDIAC ARREST MANAGEMENT

1) AT LEAST 72 HOURS POST-TTM THOUGH THIS IS A MINIMUM

2) PROGNOSTICATION CAN BE CHALLENGING


THANK YOU!

SELF EVALUATION

Cardiac Arrest: Beyond the Guidelines

1. T/F - Supraglottic airways (SGAs) such as the King LT or iGel have shown similar or better outcomes compared to endotracheal intubation in some cardiac arrest studies.

2. In-hospital data from over 80,000 patients showed survival was:

a. Higher for those intubated during arrest
b. Lower for those intubated during arrest
c. The same regardless of intubation
d. Dependent only on patient age

3. T/F - Both amiodarone and lidocaine were shown to improve survival to hospital discharge compared to placebo in witnessed cardiac arrests.

4. What is the main effect of epinephrine in cardiac arrest according to large trials?

a. Improves long-term neurological outcomes
b. Increases return of spontaneous circulation (ROSC) but not survival to discharge
c. Decreases chance of ROSC
d. Has no effect at all

5. T/F - Current targeted temperature management (TTM) recommendations allow any constant temperature between 32–37.5°C.

6. In the TTM trial comparing 33°C vs 36°C after cardiac arrest, the main result was:

a. Clear benefit of 33°C
b. Clear benefit of 36°C
c. No significant difference in mortality or outcomes
d. Harm in both groups

Answer Key: 1. T, 2. B, 3. T, 4. B, 5. T, 6. C

Learning from Diagnostic Errors: A Clinician's Perspective

Jonathan A. Edlow, MD

Epidemiologist's View

- Millions of patients & billions of dollars
- 10s-100s of thousands (estimates up to 400,000/y) deaths (and other adverse events) attributable to diagnostic error
- Occur with high-risk conditions: SAH, stroke, MI, appendicitis, PE, spinal cord compression & others

Clinician's View

- Self analysis of my own errors from selected cases over the years that stand out in my mind
- How our culture views errors
- Potential strategies to minimize the frequency of errors

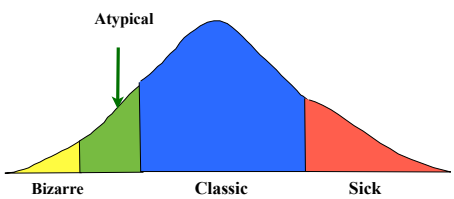
To err is human - how our culture processes errors

June 2010

Reasons for diagnostic errors

- Knowledge gaps
- Intrinsic variability of biological systems
- Cognitive issues (anchoring, premature closure, etc)
- Cultural issues
- Communication issues
- System issues

Intrinsic variability of biological systems



Diagnostic Errors . . .

. . . we ALL make them

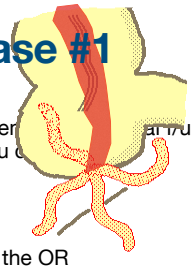
- Abdominal pain
- Chest pain
- Headache

- **Sailors run aground**
- **Quarterbacks throws interceptions**
- **Financial experts buy stocks that go down**
- **Generals lose battles by choosing the wrong attack plan**
- **Military intelligence if often wrong (WMD in Iraq)**

Abdominal Pain case #1

- Young man with 2d of prominent R mid/RUQ pain, exactly like 4 or 5 prior episodes over the prior 9 months, each lasting a few days, then completely resolving. No anorexia, nausea, vomting or fevers. Never been to the ED for this, but this time, "wanted some answers"
- Afebrile, very mild R-sided abdominal tenderness
- I thought it was Crohn's. It was a mid-week day, so I had an attending surgeon I was going to refer him to see him in the ED. He saw the patient and agreed

Abdominal Pain case #1



- Scheduled an outpatient barium ener[illegible] and told him, "the one thing I'm sure you [illegible] appendicitis"
- Returns worse at 0300 and goes to the OR and has a very long, retrocecal, upwards pointing (towards RUQ) appendix that showed both acute and chronic inflammation

Lessons learned

- Knowledge gaps & misconceptions
  - Appendicitis doesn't present with intermittent symptoms over months
  - Appendicitis patients always have nausea & loss of appetite
  - I was somewhat enamored with my diagnosis of Crohn's disease - the hubris of youth

Fool me once . . .

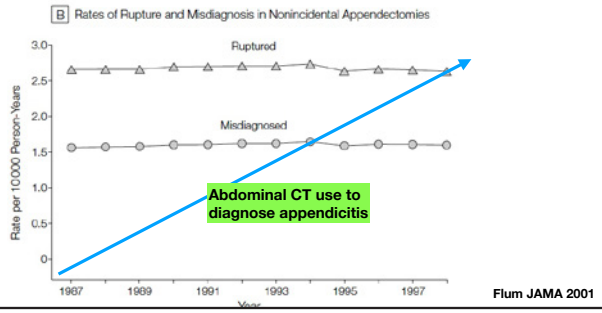
. . . fool me twice

- **Patient sent in to the ED by his PCP for ? appendicitis**
- **55-year old man with mild mid-abdominal pain after eating a lot of potato chips with Olestra. He said, "I was a pig and ate the whole bag." He had no anorexia, nausea, vomiting, fevers or chills.**
- **VS were normal. There was NO abdominal tenderness on palpation, but he did have very mild and to some extent, delayed (by 30 seconds) discomfort when one released abdominal pressure, but not typical rebound tenderness. WBC was normal.**

. . . fool me twice

- **I didn't think he had appendicitis but I was seeing the patient with a 3rd year EM resident, who felt that we should get a CT. I acceded to his request, in part to facilitate his learning. I was going off shift and the resident was going to be in the ED for several more hours. I gave the resident my cell phone number and told him, somewhat facetiously, "call me at home if the CT is positive."**
- **Several hours later, I got the call**

Will more testing help?



Flum JAMA 2001

Lessons learned

- **When another clinician sends in a patient to have "X", and you don't think "X" is necessary, call them and have a discussion. There was some reason why they thought "X" and maybe they have some information you don't. A discussion will be had and one of us will convince the other of their position.**
- **Understand the limitations of *every test* we have . . . including history and physical exam**
- **Communicate uncertainty to the patient. In these days of "shared decision-making" communicate ambiguity in situations where there is ambiguity.**
  - **Risks v benefits of CT scan.**
  - **Depends on a patient's values.**
  - **If no CT, share the diagnostic uncertainty with the patient to emphasize the need to and threshold for returning for re-evaluatoin**

Since then . . .

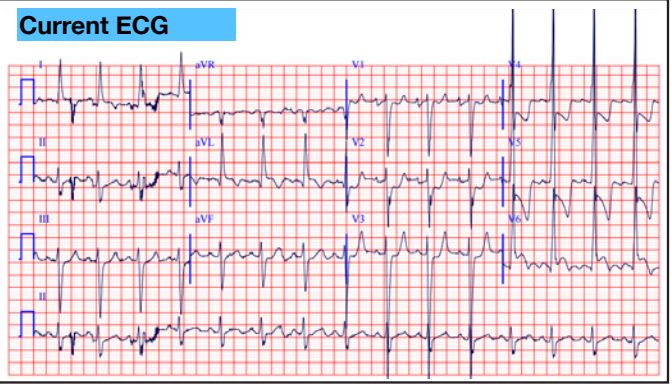
- I have not knowingly missed a diagnosis of appendicitis
- My threshold for doing a CT has decreased
- My appreciation for the wide spectrum of presentations has matured

Pleuritic chest pain - the Jedi mind trick

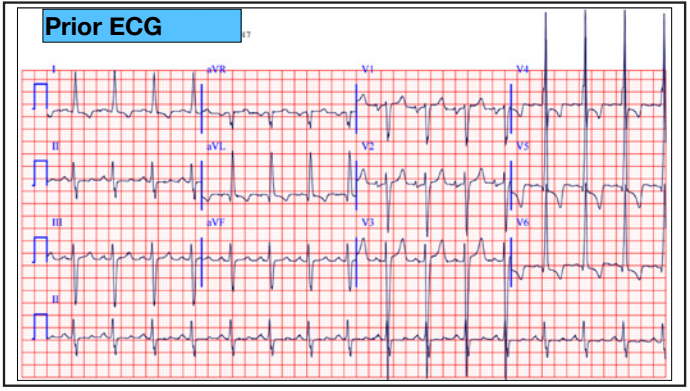
Chest pain

- 45-year old ♂ has sudden left-sided ***pleuritic*** CP, severe SOB starting 2 hours ago. The patient was very emphatic that this was very "similar to but slightly worse than a recent heart failure exacerbation". No back pain, fever, cough.
- PMH: COPD, HF (EF 30%), CKD, hypertension and s/p AICD
- HR 90 BP 168/113 R 44 O2sat 99% on 3L, normal cardiopulmonary and leg exams
  - Normal CXR
  - Troponin 0.02 and D-dimer 2100
  - BNP 4600 (lower than prior values) Creat 2.6

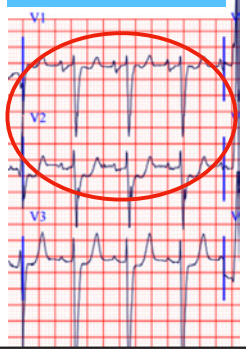
Current ECG



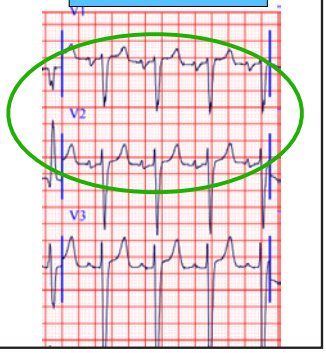
Prior ECG



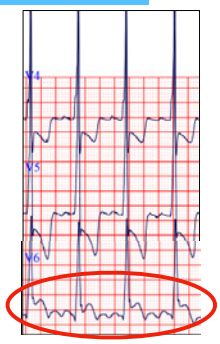
Current ECG V1-3



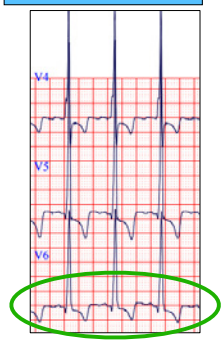
Prior ECG V1-3



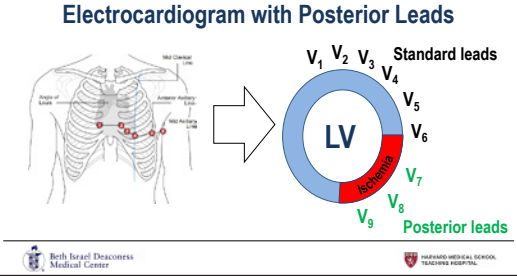
Current ECG V4-6



Prior ECG V4-6



Diagnosed with posterior MI



Over the next 11 hours, a shift change, he was seen by 8 doctors before the correct diagnosis was made

- "Curbside" (partial) cardiology consult in ED (did not see ECG)
- Heparinized & admitted for V/Q (LENI negative in ED & no CTA due to creatinine of 2.6
- Formal echo shows new postero-lateral LV hypokinesis, repeat troponin rises to 6.33
- Goes to Cath lab the next day; occluded obtuse marginal coronary artery which is stented
- At discharge, he had a measurable decrease in baseline EF

Decision-making

- Study done in the Israeli parole board (consisting of a judge, a criminologist and a social worker) system
- Prisoners in jail who had served at least 2/3rds of their sentences who are up for parole
- They analyzed 1112 individual cases over 50 different days over a 10-month period from 2 different parole boards

Decision-making

- Case 1 (0850) - an Arab Israeli serving a 30-month sentence for fraud
- Case 2 (1510) - a Jewish Israeli serving a 16-month sentence for assault
- Case 3 (1625) - an Arab Israeli serving a 30-month sentence for fraud

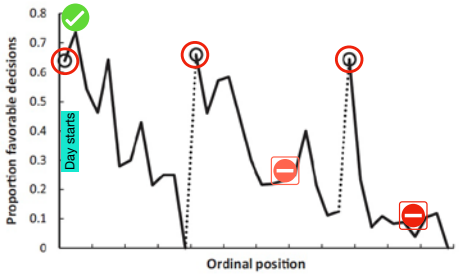
What factors influenced the board's decisions most?

- Ethnicity (Arab or Jew)
- Amount of time already served
- Number of prior convictions
- Time of day that the case was heard
- Gender of prisoner
- Gender of the judge on the board

Decision-making

- Case 1 (0850) - an Arab Israeli serving a 30-month sentence for fraud
- Case 2 (1510) - a Jewish Israeli serving a 16-month sentence for assault
- Case 3 (1625) - an Arab Israeli serving a 30-month sentence for fraud

Decision Fatigue



Post-car crash confusion

- 50 year-old woman involved in a single car crash, low mechanism ~ 24 hours prior. She was the restrained driver and hit a parked car, very minimal damage to either vehicle. No LOC. She did not seek medical care after the event, and had a mild HA & mild neck pain.
- She slept a good part of the next day and on awakening, she feels "in a fog" and intermittently asking non-sensical questions. She described the onset as "gradual"
- BP - 184/92, otherwise normal VS. No midline C-spine tenderness. She is fully oriented and conversational; the cranial nerves, cerebellar function and gait were normal. The only finding was that she was a bit tearful and both concerned and frustrated by her perceived confusion.
- Brain CT (looking for a hemorrhage) was read as, "no mass, edema or bleed, no territorial infarction. A small focus of hypo-density in the genu of the left internal capsule likely an infarct of undetermined age No bony abnormalities." (I was called and knew the full report)

Post-car crash confusion - "unknown unknowns"

Post-car crash confusion

- Given the normal neurological exam (which I repeated after seeing the CT report), and was again, completely normal and given her description of the mood symptoms starting gradually, I diagnosed a post-concussive syndrome and referred her to our neurology-run concussion clinic.
- It did not occur to me that she might have had a stroke that led to the car crash and which also caused a pseudo-bulbar affect.

Post-car crash confusion

- Two days later, she sees her PCP, with persistent but improving mood symptoms. Because of the CT report, the PCP orders a MRI which confirms that the hypodensity on the CT was in fact a sub-acute stroke

Post-car crash confusion

Lessons learned

- A knowledge gap related related to atypical presentations of acute stroke - the crash (which was likely precipitated by the stroke) and the confusion (that developed gradually) and her description of a "gradual onset" threw me off
- Atypical acute stroke presentations can present with psychiatric conditions, cranial neuropathy, monoplegia (even isolated hand weakness) or dysarthria, mimicking many different diagnoses. Sudden onset of neurological symptoms is usually an important clue in these cases.
- Considerations of the "single car crash"

Can clinical decision rules, guidelines, algorithms & pathways help?

"Not part of our agreement"

Who says we are going to use this rule or that rule (or any rule at all)? In what situation(s)? For which patients? But given the medico-legal environment, if you stray from usual practice or a clinical decision rule, document the explicit reason why.

"You have to be a pirate for the pirate's code to apply and you're not"

So defining the right diagnosis or the proper situation for the "rule" to apply" is KEY. No matter what the rule is, we still need to to use the right "rule" in the right patient in the right situation.

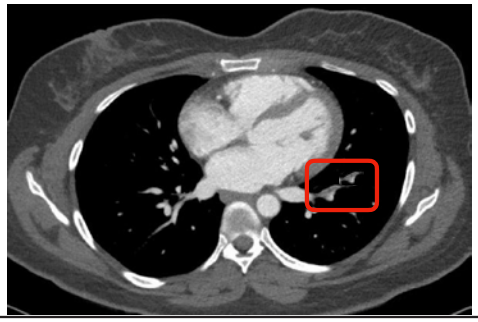
"Not so much a code as a 'guideline'"

Even if there is a rule (or pathway or guideline), it's still really just a suggestion. We have to be careful in crafting these "suggestions" and clever as to when and how we use them in clinical practice.

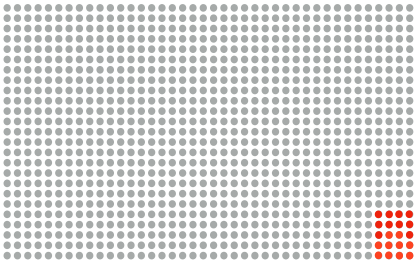
Testing threshold

- 39-year old woman with 10 days of bilateral pleuritic CP
  - s/p ovarian cystectomy **8 weeks** PTA (she was discharged on POD#1)
- She is PERC negative (PERC = surgery **< 6 weeks**)
  - What would you do?
  - The PCP did a CXR & ECG (both normal) and a D-dimer = 3500

A PERC negative PE

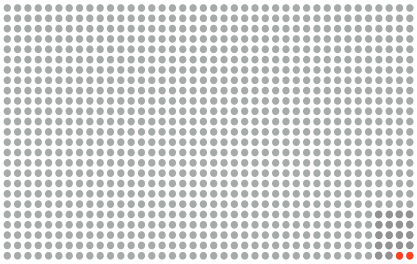


Testing Threshold - PERC for PE



PERC misses ≤ 2% of PEs (20/1000)

Testing Threshold - 6-hour CT for SAH



Upper limit of 95% CI = 2/1000

Headache

- 25 year-old man with 36 hours of 8/10 R frontal throbbing HA with "aura" - seeing a red ball. He has had many similar HAs for ~ 10 years including colorful visual auras (though less intense) but has never seen a neurologist nor been imaged.
- BP 180/119, full neuro exam normal including visual fields and optic fungi

Migraine HA

- Other than the high BP and the "greater intensity" of the visual aura, presentation was exactly like multiple prior HAs. (However, he had never come to the ED for a HA)
- Because of the high BP, I did a CT which was normal, treated him for migraine and discharged him the headache improved
- His blood pressure decreased (though not to normal) as his headache improved

Medical decision-making in my chart

"This is most likely migraine. I will treat him with Benadryl, Zofran and Compazine. Given the elevated BP, I will CT him. I am also going to refer him to neurology urgent care because he has never had any workup for these headaches. Given the duration of his episodes, I am fairly certain that this is a primary headache syndrome, and not something requiring further imaging or LP."

Medical decision-making in my chart

Serial Exam at 18:22 "He's feeling better and the CT is negative. I will discharge with close neuro/PCP f/u."

My ED Diagnosis 1: Likely migraine headache

"Remember that patient you saw . . ."

- Returns the next day after a seizure. He c/o "blind drunkenness", and confusion & was found to have a creatinine of 11 and urinary obstruction
- CT now shows multi-focal cortical edema involving both occipital lobes - posterior reversible encephalopathy syndrome (PRES)
- Evaluation shows metastatic pelvic rhabdomyosarcoma

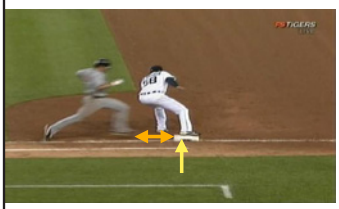
Missed PRES

- Balance between over- and under-resource utilization
  - Should I have asked about urinary function as part of a ROS?
  - Should I have gotten labs?
  - Should I have gotten a consultation or MRI?
  - Was I overly focused and anchored on his self-diagnosis of migraine?

Cultural Issues in the ED

- How are diagnostic errors
  - recognized?
  - analyzed?
  - processed to avoid repetition

Medical errors - QA system: get the data!



But where is the ball?

How clincians feel after a diagnostic error

But the system, in this case, Bud Selig, MLB commissioner decided ***NOT*** to reverse the call, citing 'the human factor' (thus this was NOT officially a perfect game) despite the evidence. But partly due to this play & this call, MLB expanded the use of instant replay use in 2014 (***4 years*** after the incident).

Doctors, at least historically, don't like 'check-lists' or 'cookbook medicine' & prefer the 'art' of medicine

Communication Issues

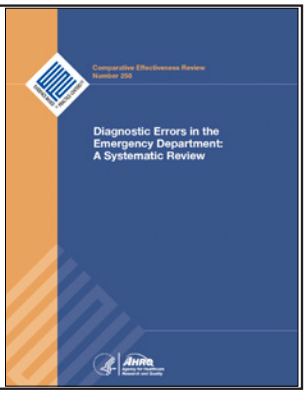
- Closed loop communications
- Clear & unambiguous language
- Do we understand each other's words?
- Do we really know what we think we know?

Clear & unambiguous language (cauda equina)

Note that all of these mistakes which we just discussed relate to a misdiagnosis

The 2022 AHRQ report

744 page document



The 2022 AHRQ report major findings in the 4-page executive summary

- **5.7% of ED patients were misdiagnosed**
- **2% suffered harm related to the misdiagnosis**
- **0.3% had serious harm (death or permanent disability)**

The 2022 AHRQ report

- **Given 130 million ED visits per year in the US, these percentages translate into:**
  - **7.4 million patients misdiagnosed**
  - **2.6 million with misdiagnosis-related harm**
  - **370,000 with serious misdiagnosis-related harm**

The 2022 AHRQ report

- 5 conditions accounted for 38% of all errors
  - Stroke
  - MI
  - Aortic dissection or aneurysm
  - Spinal cord compression or injury
  - Venous thromboembolism

The 2022 AHRQ report methodological concerns

- Three non-US-based studies formed the basis for the numbers
  - Canary Island, Switzerland and Canada
- The one death (an aortic dissection) was from a 2004 Canadian study of 503 ED patients (forming the basis for 0.2% death rate related to misdiagnosis)
- The Swiss study was of 755 consecutive patients admitted to an Internal Medicine unit. A misdiagnosis was defined as a discrepancy between the ED diagnosis and the hospital discharge diagnosis.

The 2022 AHRQ report methodological concerns

- So in the Swiss study, if I admitted a patient "upper GI bleed" and the discharge diagnosis was "bleeding peptic ulcer", that would count at a misdiagnosis.
- But Emergency Medicine is all about safe disposition rather than making a specific diagnosis.
- Diagnosing a hypotensive trauma patient with hemopertoneum by POCUS and getting the patient to the OR is high quality emergency care, even if the discharge diagnosis is ruptured spleen. It may be a diagnostic "discrepancy" but it's good care

However, despite the methodological concerns

- Everyone would acknowledge that misdiagnosis remains an important issue
- And that at least some of it is not preventable, nor necessarily even desirable (PERC with a 2% miss rate, the hypotensive trauma patient with hemoperitoneum of ? cause)
- But that some of it is and we should work to minimize preventable diagnostic errors to as low a rate as possible

Potential solutions to misdiagnosis

- Individual clinician level
- Departmental cultural level
- Systems level

Individual clinician level

- Education about reasons for misdiagnosis; remember the bell-shaped curve
- Close knowledge gaps; pursue life-long education
- Try to avoid premature closure/diagnostic anchoring
- Combine our Type 1 and Type 2 thinking

Departmental cultural level

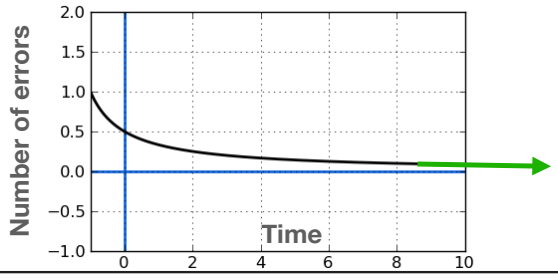
- Create a blame-free culture for reporting misdiagnosis; institute a QA committee and M&M conference
- Minimize interruptions in work-flow
- Encourage questioning or healthy dose of skepticism of diagnoses as a normal part of the sign-out process, both within the ED and at sign-out to an inpatient service
- Create checklists and/or clinical pathways or guidelines
- Work with hospital administration to minimize ED crowding and maximize availability of lab, radiology and consultants

Systems level

- National level effort to reduce ED crowding
- Create clinical pathways or guidelines
- Maximize EHR's ability to present data to the clinician in a digestible way (e.g., highlighting abnormal results)
- Intelligent use of AI to push data and diagnostic possibilities to clinicians
- Create systems-engineering solutions to measure diagnostic errors and reduce their frequency

Goals & philosophy: consider the asymptote

"constantly approaching but never reaching"



SELF EVALUATION

Learning from Diagnostic Errors: A Clinician's Perspective

1. Some of the reasons for diagnostic errors include knowledge gaps, the intrinsic variability of biological systems and systems issues.

2. Appendicitis always presents with nausea and anorexia.

3. The increased availability of abdominal CT scanning for appendicitis during the decade between 1987 and 1997 led to a decrease in appendicitis misdiagnosis.

4. Using a well-studied clinical decision rule, such as the PERC criteria to exclude a pulmonary embolism, will eliminate misdiagnosis.

5. Potential solutions to reducing the misdiagnosis rate include efforts aimed at individual clinical education, improvements in the ED culture and system, or engineering fixes.

Answer Key: 1. T, 2. F, 3. F, 4. F, 5. T

FACULTY

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You may contact Dr. Grushky with your comments or questions at algrushk@umich.edu.

Radiology for the Emergency Department

Alexander D. Grushky, MD

Alexander Grushky, M.D.

- **Board Certified Diagnostic Radiologist**
- **Fellowship trained in Musculoskeletal Radiology at Corewell Health Royal Oak (Formerly Beaumont)**
- **Emergency Radiology Division at Michigan Medicine**

1

Objectives

Briefly discuss the role of imaging in the emergency setting

Identify opportunities to optimize the interdisciplinary relationship between Emergency Physicians and Radiologists

2

Imaging in the ED

- Imaging can play a significant role in providing expedient and efficacious care in the emergency setting
- Appropriate imaging tests can aid in diagnosis and help direct treatment
- Unnecessary imaging can lead to excess radiation exposure, increased length of stay, and increased costs[1]

3

Imaging in the ED

- We're getting busier!
- Emergency room use has increased[2]
  - 140,000,000 ED visits in US in 2021
  - 43 visits per 100 people
  - Prevalence of ED visits increased from 17.2 (1999) to 21.7 (2019)
- Imaging utilization has increased[3]
  - Level 1 Trauma center from 2014-2021 saw volume increases in chest X-Ray (5.5%), CT (35.5%), and MRI (56.3%)
  - Total wRVU per ED encounter increased 34.9%

4

Imaging in the ED

- A good working relationship between the ED and Radiology is crucial

5

Topics

01	02	03	04	05
Appropriate Use	Importance of Clinical Information	Importance of Protocol	Artifacts and limitations of imaging	Operational Issues

6

Appropriate Use

- Depending on clinical scenario, imaging may be warranted
- Multiple different modalities are available in the emergent setting
  - Availability varies between institutions
- What modality to use?

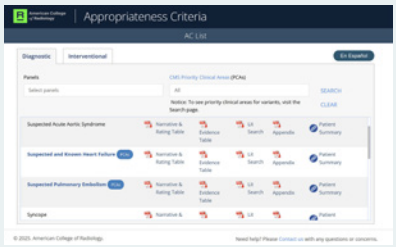
7

ACR Appropriateness Criteria[4]

- American College of Radiology has developed a series of imaging algorithms based on various clinical scenarios
- Available on the ACR website[4]
- Includes evidence-based suggestions on which imaging modality best fits a clinical scenario
- Example case: Suspected Pulmonary Embolism[5]

8

ACR Appropriateness Criteria[4]



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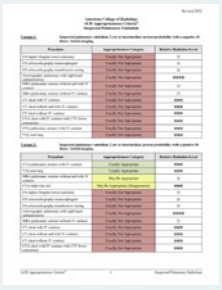
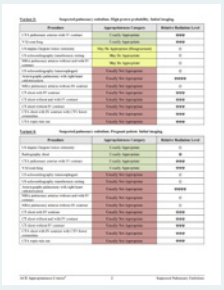
ACR Appropriateness Criteria[5]





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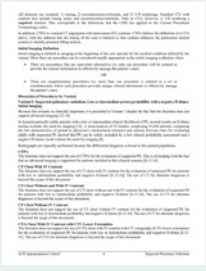
ACR Appropriateness Criteria[5]

Variant 2: Suspected pulmonary embolism. Low or intermediate pretest probability with a positive D-dimer. Initial imaging.

Procedure	Appropriateness Category	Relative Radiation Level
CTA pulmonary arteries with IV contrast	Usually Appropriate	☢☢☢
V/Q scan lung	Usually Appropriate	☢☢☢
MRA pulmonary arteries without and with IV contrast	May Be Appropriate	O
CTA triple rule out	May Be Appropriate (Disagreement)	☢☢☢
US duplex Doppler lower extremity	Usually Not Appropriate	O
US echocardiography transesophageal	Usually Not Appropriate	O
US echocardiography transthoracic resting	Usually Not Appropriate	O
Arteriography pulmonary with right heart catheterization	Usually Not Appropriate	☢☢☢☢
MRA pulmonary arteries without IV contrast	Usually Not Appropriate	O
CT chest with IV contrast	Usually Not Appropriate	☢☢☢
CT chest without and with IV contrast	Usually Not Appropriate	☢☢☢
CT chest without IV contrast	Usually Not Appropriate	☢☢☢
CTA chest with IV contrast with CTV lower extremities	Usually Not Appropriate	☢☢☢

11

ACR Appropriateness Criteria[5]



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ACR Appropriateness Criteria[5]

Variant 2: Suspected pulmonary embolism. Low or intermediate pretest probability with a positive D-dimer. Initial imaging.

CTPA

CTPA is a first-line diagnostic imaging tool after the D-dimer examination and is routinely performed in this clinical scenario. CTPA is highly sensitive and specific [20-24]. CTPA may occasionally demonstrate pathology other than PE that may be responsible for the patient's symptoms [22].

CT Chest With IV Contrast

There is no relevant literature to support the use of CT chest with IV contrast to assess PE in patients with low or intermediate probability with positive D-dimer.

CT Chest Without and With IV Contrast

There is no relevant literature to support the use of CT chest without and with IV contrast to assess PE in patients with low or intermediate probability with positive D-dimer. When IV contrast is given during the CT acquisition, the study should be performed as a CTPA.

CT Chest Without IV Contrast

There is no relevant literature to support the use of CT chest without IV contrast to assess PE in patients with low or intermediate probability with positive D-dimer.

CTA Chest With IV Contrast with CTV Lower Extremities

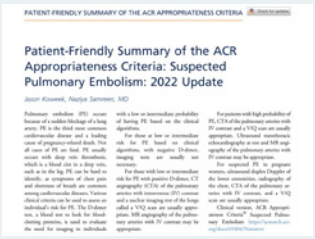
Older literature shows that the field of view for CTA can be extended to include the lower extremities so that both the pulmonary arteries and the deep veins of the leg can be imaged during the same imaging session [25,26]. However, this protocol is very rarely used at present, owing to the accuracy of performing US for DVT and the increased burden of contrast and radiation for the associated extended craniocaudal field of view [27].

13

ACR Appropriateness Criteria[5]

14

ACR Appropriateness Criteria: Patient Summary[6]


PATIENT-FRIENDLY SUMMARY OF THE ACR APPROPRIATENESS CRITERIA

Patient-Friendly Summary of the ACR Appropriateness Criteria: Suspected Pulmonary Embolism: 2022 Update

Jason Kovacek, Nadya Samreen, MD

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Case 1

- Transient Ischemic Attack, facial droop, right hand numbness
- Initial imaging is CT head without contrast
  - Rapid acquisition
  - Assess for hemorrhage prior to thrombolytic therapy
- CTA head and neck help identify vascular occlusion or injury
- MRI has better detection of early ischemia[7]

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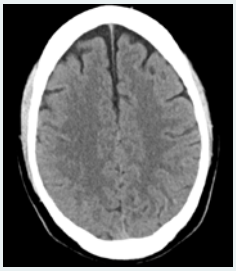
Case 1



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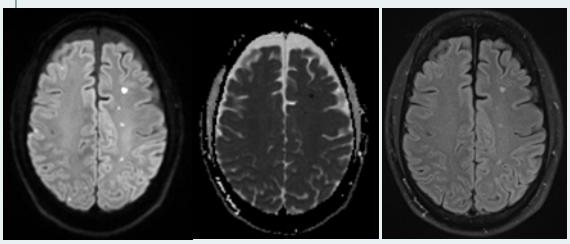
Case 1



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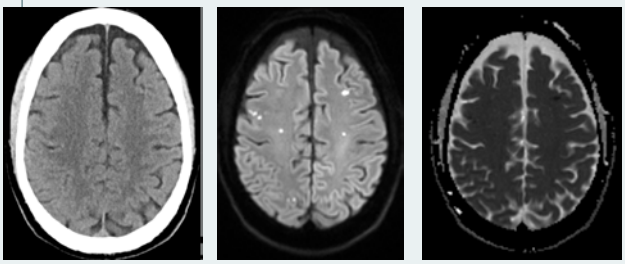
Case 1



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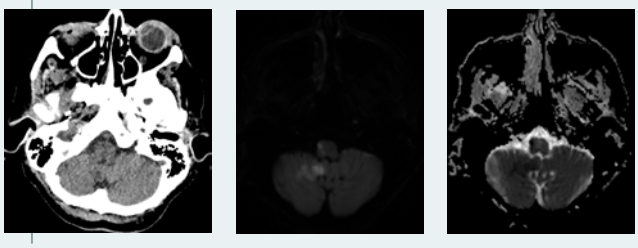
Case 1a: Neuro deficit, acute stroke suspected



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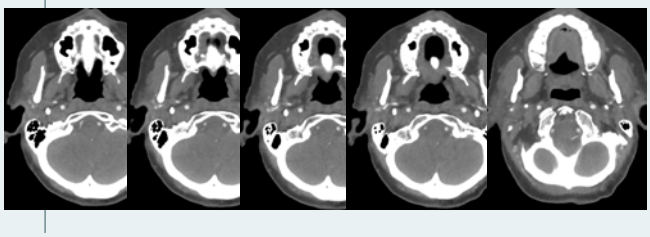
Case 1a: Neuro deficit, acute stroke suspected



21

Case 2

- Fall off bicycle onto right side, acute pain
- Initial imaging is X-Ray
- Can be limited in the setting of osteopenia
- CT can help identify occult fractures and characterization
  - Newer CT scanners can be as accurate as MRI[8]
- MRI useful in cases of high clinical suspicion and negative XR/CT[9]

22

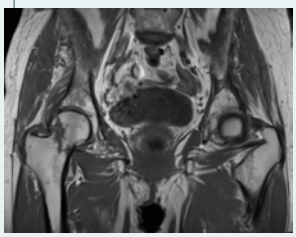
Case 2

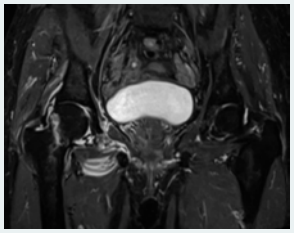
23

Case 2

24

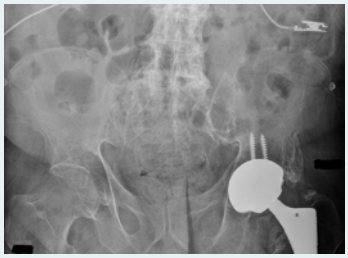
Case 2





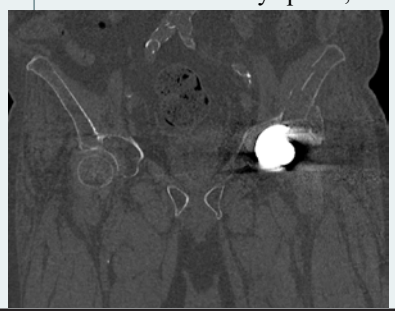
25

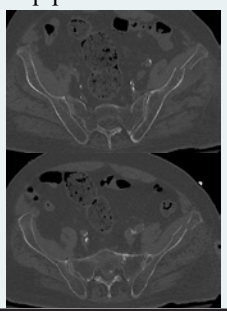
Case 2a: Fall 2 days prior, left hip pain



26

Case 2a: Fall 2 days prior, left hip pain





Topics

01	02	03	04	05
Appropriate Use	Importance of Clinical Information	Importance of Protocol	Artifacts and limitations of imaging	Operational Issues

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Importance of Clinical Information

- The provided clinical history or reason for exam helps guide the radiologist in their search pattern
  - Also allows for more accurate ICD-10 coding
- The more descriptive the better!
- Localization of symptoms is helpful
- McBee, M. et. al. describe the "What-When-Where" method[10]
  - E.g. Fall off monkey bars yesterday with left wrist pain

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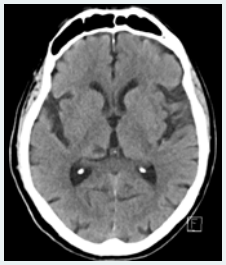
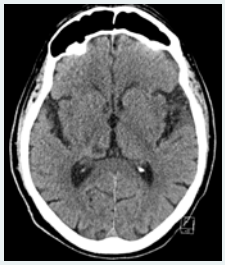
Importance of Clinical Information

- "Comparison with prior imaging would be of benefit"
  - Sometimes to most important image is the prior one!
  - Helps stabilize seemingly worrisome or chronic findings
- Direct comparison is optimal
  - Prior reports may mention, but best to have direct comparison with prior images
- Can avoid unnecessary and costly follow up

30

Case 3: Fall, left leg weakness





6 months prior

31

Case 4: Shortness of breath

Present on prior 6 month ago and older

32

Case 4: Shortness of breath

33

Topics

01	02	03	04	05
Appropriate Use	Importance of Clinical Information	Importance of Protocol	Artifacts and limitations of imaging	Operational Issues

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Importance of Protocol

- Not all CT's and MRIs are created equal!
- Contrast vs. non contrast
- Single vs. multiphase CT
- MRI brain vs MS protocol
- Call radiologist if questions!

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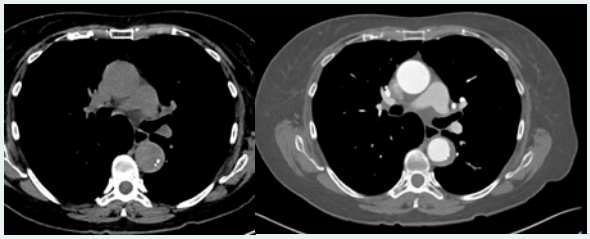
Case 5: Chest pain, acute aortic syndrome suspected



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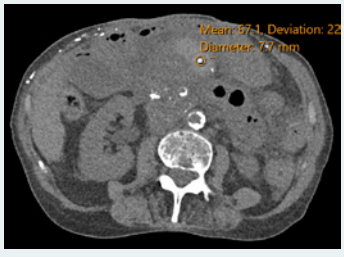
Case 6: History of Whipple, GI bleed, abdominal pain



37

Case 6: History of Whipple, GI bleed, abdominal pain

38

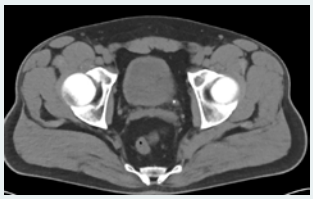
Case 7: History of diverticulitis, rectal bleeding

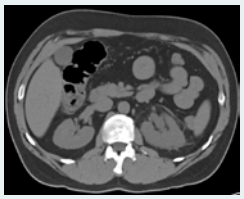
39

Case 7: History of diverticulitis, rectal bleeding

40

Case 8: Colicky abdominal pain





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Topics

01	02	03	04	05
Appropriate Use	Importance of Clinical Information	Importance of Protocol	Artifacts and limitations of imaging	Operational Issues

42

Artifacts and Limitations of Imaging

- Artifact on imaging arise from many different sources
- Can decrease diagnostic value of imaging

43

Artifacts and Limitations of Imaging

- CT
  - Streak/beam hardening
  - Motion
  - Timing of contrast
- MRI
  - Motion
  - Metal related artifact

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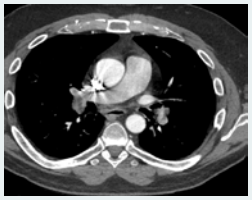
CT artifact

- Streak/beam hardening artifact



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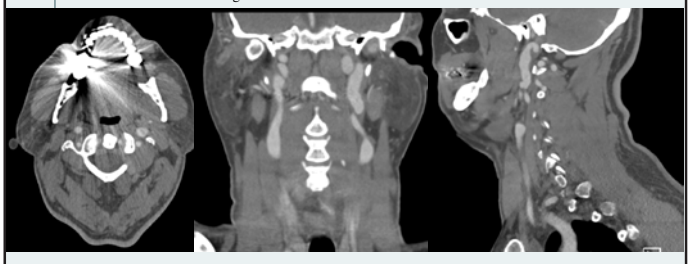
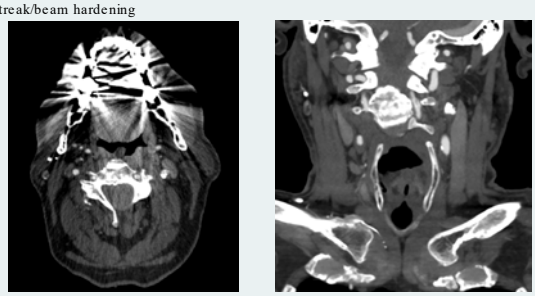
CT artifact

- Streak/beam hardening



CT artifact

- Streak/beam hardening



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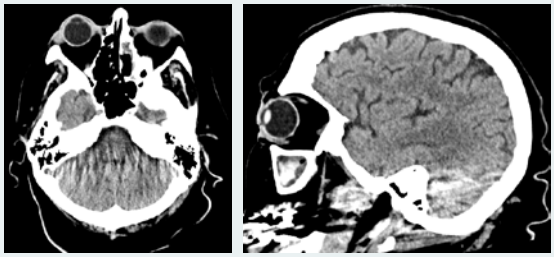
CT artifact

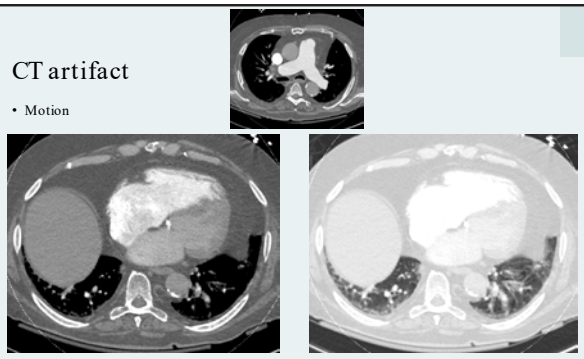
- Streak/beam hardening



48

CT artifact

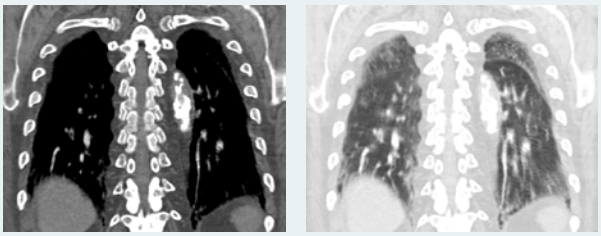
- Motion



49

CT artifact

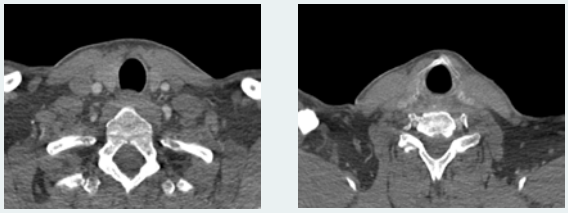
- Motion



50

CT artifact

- Motion



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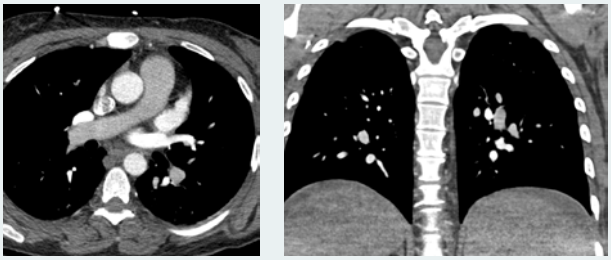
CT limitations

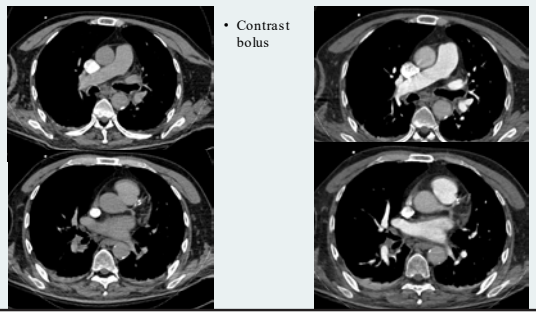
- Contrast bolus



52

CT limitations

- Contrast bolus



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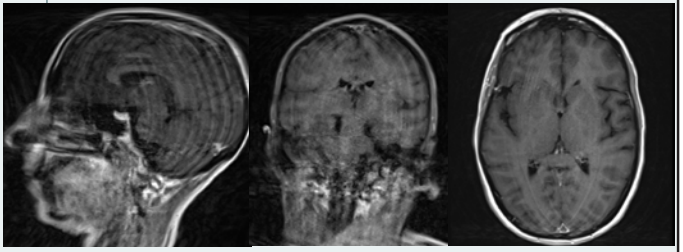
MR motion artifact



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MR metal related artifact

Topics

01	02	03	04	05
Appropriate Use	Importance of Clinical Information	Importance of Protocol	Artifacts and limitations of imaging	Operational Issues

Operational Issues

- Imaging Triage
  - Varies by institution
  - Stroke, trauma, acute aortic syndrome vs. atraumatic joint pain, chronic cough
  - Can result in longer turn around for lower acuity cases

Operational Issues

- Time from scan to final read
  - Multiple barriers
    - Patient transport
    - Imaging backlog/triage
    - Protocoling
  - Varies by modality
    - CT average turn-around-time at one institution is 4.7 hours[11]
  - Patients can return before imaging is available to read

Operational Issues

- Importance of final read
  - CT/CTA head and neck for stroke
    - Call for bleed vs. no bleed
  - Final read can have nonemergent findings requiring follow up
    - Apical lung nodule
    - Thyroid nodule

Topics

01	02	03	04	05
Appropriate Use	Importance of Clinical Information	Importance of Protocol	Artifacts and limitations of imaging	Operational Issues

Final Thoughts

- A good relationship between the Emergency and Radiology departments is essential
- Clear communication goes a long way
- Can always call with questions

Citations

1. Mills AM, Raja AS, Marin JR. Optimizing diagnostic imaging in the emergency department. Acad Emerg Med. 2015 May;22(5):625-31. doi: 10.1111/acem.12640. Epub 2015 Mar 2. PMID: 25731864; PMCID: PMC4523049.
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SELF EVALUATION

Radiology for the Emergency Department

1. T/F - Emergency room utilization and utilization of radiology examinations has remained stable over the past decade?

2. T/F - The initial imaging for a suspected pulmonary embolism with low or intermediate pretest probability with a positive D-Dimer is a CT chest with and without contrast?

3. Which of the following would be the most useful clinical history to provide for a radiology exam?

a. Pain
b. Trauma
c. Injury
d. Fall down stairs 1 day prior with acute right hip pain

4. T/F - A non-contrast CT abdomen has no role in the evaluation of suspected gastrointestinal bleeding

5. Which of the following are potential barriers to the finalization of an imaging study?

a. Patient transport
b. Imaging Backlog
c. Protocol issues
d. Imaging triage
e. All of the above

Answer Key: 1. F, 2. F, 3. D, 4. F, 5. E