<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
<th>Speaker(s)</th>
<th>CME Credit</th>
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<tbody>
<tr>
<td>7:45 – 8:00 am</td>
<td>Welcome &amp; Overview – Alan Ehrlich, MD</td>
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<tr>
<td>8:00 – 8:45 am</td>
<td>COVID-19 Update - Paul D. Simmons, MD, FAAFP</td>
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<tr>
<td>8:45 – 9:45 am</td>
<td>Pulmonary Embolism/Deep Vein Thrombosis and Thrombophlebitis – Alan Ehrlich, MD</td>
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<td>9:45 – 10:00 am</td>
<td>Break</td>
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<td>10:00 – 11:00 am</td>
<td>Acute Markedly Elevated Blood Pressure - Ahmed Mian, MD</td>
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<tr>
<td>11:00 am – 12:00 pm</td>
<td>Fatal Headaches - Vu Kiet Tran, MD, MHSc(Ed), MBA, CHE</td>
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<td>12:00 – 1:00 pm</td>
<td>Lunch on Your Own</td>
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<td>1:00 – 2:00 pm</td>
<td>Respiratory Failure – Maj. Brian Shahan, MD, FAAFP, DFPHM</td>
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<tr>
<td>2:00 – 3:00 pm</td>
<td>Cellulitis and Cellulitis Mimics - Vu Kiet Tran, MD, MHSc(Ed), MBA, CHE</td>
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<tr>
<td>3:00 – 4:00 pm</td>
<td>Sepsis Recognition and ER Management – Ahmed Mian, MD</td>
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<td>4:00 – 4:15 pm</td>
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<tr>
<td>4:15 – 5:15 pm</td>
<td>Dizziness and Vertigo – Alan Ehrlich, MD</td>
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<tr>
<td>5:15 – 6:15 pm</td>
<td>Syncope: An Innovative Approach to Assessment in the ER/UC - Vu Kiet Tran, MD, MHSc(Ed), MBA, CHE</td>
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<td>6:15 pm</td>
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Emerging Infectious Diseases

With Emphasis on COVID-19

Paul D. Simmons, MD, FAAFP
Faculty Physician
St. Mary’s Family Medicine Residency Program
Grand Junction, Colorado
Disclosure

It is the policy of the AAFP that all individuals in a position to control content disclose any relationships with commercial interests upon nomination/invitation of participation. Disclosure documents are reviewed for potential conflicts of interest and, if identified, conflicts are resolved prior to confirmation of participation. Only those participants who had no conflict of interest or who agreed to an identified resolution process prior to their participation were involved in this CME session.

• Vu Kiet Tran, MD, MBA has disclosed a relationship with Elvium on the topic “Acute Pain Management”.

All individuals in a position to control content for this session have indicated they have no relevant financial relationships to disclose.
Learning Objectives

1. Discuss the current understanding of the epidemiology of SARS-CoV-2, risk factors for severe COVID-19 disease, surveillance and reporting, and the effectiveness of various population strategies for prevention of spread.

2. Rationally formulate a strategy for diagnosing patients with COVID-19, including an understanding of test performance and clinical manifestations.

3. Rationally formulate a management strategy for patients with mild-moderate COVID-19 not needing ventilator support, have a basic understanding of initial management of severe disease, and current evidence-based treatment options.
Resistant Organisms

Measles

Chikungunya Fever

CRE

SARS-CoV-2

Pertussis
SARS-CoV-2
AES Question
Question 1

You are evaluating via telemedicine a 45-year-old woman with three days cough, myalgias, and fever symptoms. She usually takes a budesonide/formoterol inhaler daily for asthma, which is well-controlled typically.

Which of the following statements about ICS therapy in COVID-19 illness is true?
A. The inhaler dose and frequency should be increased immediately to decrease the risk of asthma exacerbation.
B. The inhaler should be stopped immediately.
C. The inhaler should be stopped and a LABA should be substituted.
D. The inhaler should be continued for now, and adjusted if symptoms worsen.
AES Question
Question 2

Based on our best available data as of April 2020, the case fatality rate of SARS-CoV-2 infection is:

A. Higher than the “Spanish” influenza epidemic of 1918-1919.
B. Lower than the “Spanish” influenza epidemic of 1918-1919.
C. Higher than the Black Death (bubonic plague) pandemic of 1347-1351.
E. Reportedly higher in most western countries compared to initial reports from China.
How We Got Here

7 Jan – novel CoV identified
11 Jan – first death reported by China
13 Jan – first case outside China, in Thailand
20 Jan - First US case is reported: a 35-year-old man in Snohomish County, WA
30 Jan – WHO declares public health emergency
31 Jan – US bans foreign nationals from entering US if in China prior 2 weeks
8 Feb – US citizen dies in Wuhan
9 Feb – death toll in China surpasses 2003 SARS outbreak
29 Feb – first death on US soil
11 Mar – WHO declares a pandemic
13 Mar – US declares national emergency

Image creative commons from Wikicommons user Banjo, at https://commons.wikimedia.org/wiki/File:Biohazard_black_red.jpg
<table>
<thead>
<tr>
<th>Disease</th>
<th>Attributed Deaths</th>
<th>Years</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plague of Justinian</td>
<td>30-50 million</td>
<td>541-542</td>
<td>Contributed to fall of Western Roman Empire; mortality still debated</td>
</tr>
<tr>
<td>Bubonic Plague (Black Death)</td>
<td>200 million</td>
<td>1347-1351</td>
<td>CFR est. 40-60%</td>
</tr>
<tr>
<td>&quot;Spanish” influenza</td>
<td>40-50 million</td>
<td>1918-1919</td>
<td>CRF &gt; 2.5% overall (variation 0.3% later cases - 33% in Stockholm!)</td>
</tr>
<tr>
<td>“Hong Kong” influenza</td>
<td>1 million</td>
<td>1968-1970</td>
<td>CFR &lt; 0.5% (~5x seasonal flu)</td>
</tr>
<tr>
<td>Ebola (Guinea-Sierra Leone-Liberia)</td>
<td>11,310</td>
<td>2014-2016 largest outbreak to date</td>
<td>CFR 40% this outbreak</td>
</tr>
<tr>
<td>COVID-19</td>
<td>113,300 (12 Apr 20)</td>
<td>2019-present</td>
<td>CFR 3-4% (China), 1.4% or lower overall, higher in elderly (data uncertain) - ?? lower than predicted in US?</td>
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Moderate COVID-19 Illness
Case Study 1

• You see a 65-year-old male smoker who works as a grocery clerk reporting three days of worsening cough, dyspnea at rest, myalgias, and subjective fever. BP = 130/88; pulse = 110 regular; RR = 20.
• He has scattered pulmonary crackles and mild wheezes; SaO2 on RA = 93%.
• The patient is taking metformin 1000 mg BID, lisinopril 40 mg daily, and sildenafil as needed.
• PMH: poorly-controlled T2DM (last A1c 9.3%), well-controlled HTN, erectile dysfunction, smoking.
• How to risk-stratify this patient?
  − Age
  − Comorbidities and control
  − Smoking
  − Medications?

Don’t forget: even in this pandemic, people can still get pneumonia, flu, and COPD exacerbations.
Case Study

Decision-making based on local resources:

- Testing availability in your area?
  
  He is SECOND TIER for testing according to CDC guidelines:
  - Patients in long-term care facilities with symptoms
  - Patients 65 years of age and older with symptoms
  - Patients with underlying conditions with symptoms
  - First responders with symptoms

- Protecting yourself, staff and the public:
  - Procedure mask or N95 and PPE recommended for clinicians contacting COVID-19 PUIs (follow local protocols)
  - Separate entrance, room w door closed, clean thoroughly, room turnover time?
  - N95 if “aerosolizing procedure” (including neb treatment, intubation, suctioning) per CDC
Case Study Evidence

**Testing:** This pt SHOULD be tested per CDC guidelines.

**Disposition:** Case-by-case, but outpatient potential (O2 sat okay; close follow-up by phone/video).

**Should you stop his ACEI?**

**Resources:**


Case Study Recommendations

• **Treatment:** Aggressive supportive care. Smoking cessation. *Avoid steroids.* Close follow-up by phone or video. Consider observation admission. Home pulse oximeter. Consider inhalers.

• **Diagnostic Pearls:** Limited lab testing may be useful for prognosis:
  - Progressive lymphopenia, progressively increasing D-dimer both poor signs
  - If delay in testing results, consider respiratory viral panel, flu test, procalcitonin, and CRP: If other viral testing negative and neg procalcitonin with high CRP → presumed COVID-19.

• **Disposition:** Outpatient or inpatient, case-by-case, depending on local resources.
Case Study 2

• You see a 65-year-old female smoker who works as a flight attendant with three days of worsening cough, dyspnea at rest, myalgias, nausea, and subjective fever symptoms. BP = 110/60; pulse = 110; RR = 22/min.

• She has diffuse crackles and moderate wheezing. Her SaO2 on RA = 88%.

• The patient is taking methotrexate 10mg weekly, folate 1mg daily, and budesonide/formoterol MDI 2 puffs BID.

• PMH: moderate plaque psoriasis; COPD.

How would you **risk stratify** this patient?

• Again: it *could* be an AECOPD, pneumonia, influenza, but…

• She’s in a high-risk age group, has underlying lung disease, smokes, and is immunosuppressed. All bad news.
Case Study

Considerations based on clinical setting / resources:

• **PPE:** As before, protect yourself, your staff, and your patients:
  - Separate entrance, closed door, isolated room, masks for you and staff

• **Testing:** she is high-priority for testing based on age, health condition.
  - Again, *consider* protocol of: 1) flu swab, respiratory panel, procalcitonin, CXR, CRP. 2) if flu, resp panel neg, normal procal, no obvious lobar PNA, and CRP high $\rightarrow$ more likely SARS-CoV-2.
  - Or just get the SARS-CoV-2 test (depending on local protocols and resources).

• **Disposition:** based on her high risk and local resources, consider inpatient (she’s hypoxic already, vitals more deranged)
Case Study Evidence

Pearls based on this case:

• At this time, experts are NOT recommending stopping immunosuppressive medications. At least consult with specialist.

• Inhaled and topical steroids are thought to be safe (not clearly known), but systemic steroids should be avoided unless severely ill.

Resources:

• The American Academy of Dermatology. https://assets.ctfassets.net/1ny4yoiyrqia/PicgNuD0IpYd9MSOwab47/023ce3cf6eb82cb304b4ad4a8ef50d56/Biologics_and_COVID-19.pdf

• American College of Rheumatology. https://www.rheumatology.org/announcements
Case Study Recommendations

Likely would **admit this patient** for supplemental O2 support, lab monitoring, and observation for worsening given her risk factors.

- Prognostic labs: progressively worsening lymphopenia, elevated D-dimer are poor signs
- As above, avoid systemic steroids – UNLESS you clinically suspect underlying, superimposed AECOPD
- Immediate smoking cessation and nicotine support
- Continue immunosuppressive meds and ICS/LABA unless otherwise directed by a specialist
Case Study Resources

Severe COVID-19 Illness
Case Study 3

• You see a 75-year-old man who lives independently, reporting one week of worsening cough, dyspnea at rest, myalgias, and fever symptoms. BP = 110/60 (baseline 150/95), Pulse = 115 regular; RR = 22/min.
• On exam, he appears ill with increased work of breathing. SaO2 on RA = 86%.
• The patient is taking metformin 1000mg BID, atorvastatin, and budesonide/formoterol MDI 2 puffs BID.
• PMH: poorly controlled T2DM (last A1c = 9%), HLD, COPD w 40 pk-yr smoking hx (stopped 3 years ago), has 2 AECOPD per year.

• How would you risk-stratify this patient?
Case Study (cont.)

• Your local hospital has appropriate facilities for admission (isolation possible, respiratory therapy, ventilator and intensive care if needed), so you decide to admit him.
  – What labs or imaging may help you further evaluate and prognosticate?
  – SARS-CoV-2, influenza, resp viral panel, CRP
  – Evidence suggests worsening lymphopenia and D-dimer elevation may suggest higher mortality
  – Of course, VBGs / ABGs, worsening CXR findings...

ARDS CXR from Wikicommons user Samir, at https://commons.wikimedia.org/wiki/File:ARDS_X-Ray.jpg
Some caveats!
- The SARS-CoV-2 test is not flawless: reported sensitivities ~ 75%, with lower sens from nasal → highest sens from BAL samples (intubated). Point being: it’s not perfect – both false + and –
  - Newer tests (as of April 2020) may have much improved performance characteristics…

- WBC can be up or down, though lymphopenia associated w increased mortality

- Procalcitonin generally neg, but can be high in advanced disease and ARDS
Case Study Recommendations

• Patients with COVID-19 can rapidly and dramatically decompensate – **consider transfer** if your facility does not have adequate specialty or equipment support.

• What most COVID-19 patients die of is **ARDS**: follow respiratory status closely, SaO2, serial ABG/VBGs, CXR.

• Resuscitation, HFO2, and intubation are **aerosolizing procedures** – clinicians and staff should be protected with N95 masks and eye protection. [*HHFNC, NIPPV and nebs NOT recommended, but sometimes used!*]

• **Avoid steroids** (unless another indication). There is no good evidence yet for empiric tx with antivirals or other medications (studies ongoing).
A Digression About Medications for COVID-19
Disclaimers as of mid-April 2020

• NONE of the available medications have been proven safe or effective
• Very few randomized controlled trials (if any)
• A lot of data is in vitro or animal data
• In many cases, we are extrapolating from MERS and SARS-CoV-1
• NO MEDICATION IS WITHOUT RISK
Remdesivir

• MOA: Interferes with viral RNA-dependent RNA polymerase; premature termination of viral RNA transcription

• Single Case Report Available
  - 35yo male, recent travel from Wuhan, admitted for cough/fever, found to have Sp02=90%, LLL opacity, started on supportive care and broad spectrum antibiotics
  - Day 11 of illness: remdesivir started, D12: rapid clinical improvement
  

• Several trials ongoing which won’t be completed until May.
Chloroquine and Hydroxychloroquine

• Both are metabolized to the same active metabolite

• MOA
  − Direct Antiviral Activity
    • Intracellular alkalinization inhibits pH-dependent steps of viral replication
    • Impaired viral receptor glycosylation
  − Immune Modification
    • Reduces cytokine production, especially IL-1 and IL-6
    • Inhibit toll-like receptor signaling

• ADRs
  − Short Term: GI upset, ECG abnormalities-QTc prolongation, hypoglycemia, extrapyramidal reactions
  − Long Term: retinal damage (long term, high dose), note that it crosses placenta but has been used for SLE in pregnancy
  − DDIs: Additive toxicity with QTc prolonging medications
Data on Chloroquine/Hydroxychloroquine

• In Vitro
  – Parent compound shows activity against MERS, SARS-CoV-1, active metabolite activity unclear

• Clinical
  – Letter from Chinese physicians: 100 patients: authors claimed that chloroquine was superior to control in shortening disease course, promoting viral clearance, etc, etc
    • *Difficult to interpret-no patient specific information*
  – RCT 3/3/20: 30 patients HCQ (15) vs standard of care (15)
    • *No difference* in conversion rate of COVID-19 PCR on day 7 after randomization
  – RCT 3/27/20: 62 patients HCQ (31) vs standard of care (31)
    • HCQ group showed *shorter time to clinical recovery, fever and cough remission*

• A note on PK/PD
  – EC50 is achieved in infected cells with various dosing regimens, and hydroxychloroquine is shown more potent
  – Presumed lung concentrations achieve EC50 even on Day 10 after all dosing regimens tested, hydroxychloroquine has higher presumed exposure
  – Half life ~ 40 days


Hydroxychloroquine + Azithromycin

- French Study that has gained media attention
- 36 total patients
- Primary outcome = viral suppression 6 days after inclusion
  - 100% were PCR (+) on day 1
  - Controls: (16 pts) - 12% achieved viral suppression
  - HCQ only (14 pts) - 57% achieved viral suppression
  - HCQ + Azith (6 pts) - 100% achieved viral suppression

- Limitations
  - Patients had mild-moderate disease, and 6 of total patients were asymptomatic
  - Patients who got combo had presumably lower viral loads at onset

French Follow-Up

• Subsequently, the same authors reported on 80 patients treated with HCQ/AZ (including the six above):

  – that 83% had negative nasopharyngeal PCR tests for COVID-19 on day seven.

  *Problèmes*: No randomized controls, no intention-to-treat, and non-clinical outcomes (PCR test results).
But another French study fails to duplicate…

Series of 11 patients treated with the same HCQ/AZ protocol and found that 8 of 10 surviving patients still had positive nasopharyngeal PCR tests on day six (Med Mal Infect. 2020 Mar).
Lopinavir/Ritonavir

- Anti-retroviral for HIV
  Lopinavir is a protease inhibitor
  - SARS-CoV-2 contains a chymotrypsin-like protease, potentially inhibited by lopinavir
  Ritonavir is a CYP3A4 inhibitor that boosts levels of lopinavir
  No longer commonly used in HIV due to toxicity, availability of single pill (convenient) regimens

- **In vitro data for SARS-CoV-1 and MERS**
  Huge range of effective concentrations across studies
  Lowered viral loads of MERS-CoV in primates
  We are extrapolating for SARS-CoV-2: no in vitro data exists

- **Clinical Data in SARS-CoV-1**
  Lower rates of intubation and death when LPV/r was combined with ribavirin for initial treatment

- **NEJM 2020: Kaletra in SARS-CoV-2**
  Primary outcome-time to clinical improvement
  - No difference between Kaletra and standard of care
  No difference in decrease in viral load

Tocilizumab (Actemra)

• MOA
  Monoclonal antibody-antagonist of IL-6 receptor: inhibits signal transduction in this inflammatory response pathway
  Theory: cytokine release during viral infections worsens diseases course, may lead to pulmonary fibrosis

• Retrospective review of 21 patients:
  Improved clinical symptoms in patients with severe disease
  Limited information in this review, unclear of timing of drug, many other details missing

• TACOS Trial-pending-Tocilizumab vs CRRT
  Currently is included in China’s SARS-CoV-2 guidelines for patients with extensive and bilateral lung disease and severely ill patients with elevated IL-6 levels (this is a send-out lab)

• $$$$

Effective Treatment of Severe COVID-19 Patients with Tocilizumab; Xu, Wei, et al. 2020
Remdesivir – NEJM April 10, 2020

• 53 pts, 57% on vent, 8% ECMO baseline
• 18 d follow-up, 36/53 improved O2 support
• no control group, not randomized
Other Things Being Studied…

- Anakinra
- Arbidol
- Baricitinib
- Bevacizumab
- Brilacidin
- Convalescent plasma
- Darunavir/cobicistat
- Disulfiram
- Exulizumab

- Favipravir
- Galidesivir
- Griffithsin
- IVIG
- Nelifnavir
- Niclosamide
- Sarilumab
- Sofosbuvir
- Vitamin C
- XueBiJing

And probably CBD.
Back to the Cases…
Case Study 4

• You see an 88-year-old female NH patient with advanced dementia whose care staff are reporting she’s had two days of worsening work of breathing, confusion and agitation, and fevers up to 101.5 F. BP = 95/55, pulse = 130 irreg; RR = 24/min.
• On exam, she has widespread crackles and wheezes, localizes to painful stimuli, but is otherwise obtunded. SaO2 on RA = 78%, SaO2 on 10L by FM = 90%.
• The patient is taking metformin, donepezil, oxybutynin, docusate, and oxycodone.
• PMH is extensive but includes dementia, T2DM, urinary incontinence, constipation, OA.
Case Study (cont.)

• Key points of this case:
  
  − Extremely high risk given age, comorbidities
  − Although the absolute number of hospitalizations among the very elderly are lower (so far), their mortality rate is higher (CDC 3/25 – 10-25% mortality in pts > 85)

  − Family physicians MUST be thinking now about:
    • Advanced care discussions with elderly patients and their families
    • MDPOAs in case of ARDS and intubation
    • Resuscitation wishes and goals of aggressive care
Case Study Evidence

• Online Shared-Decision-Making Tool for COVID-19 from National Hospice and Palliative Care Organization:

• Severe Outcomes in Patients with COVID-19 from CDC MMWR, including updated mortality rates by age:
  https://www.cdc.gov/mmwr/volumes/69/wr/mm6912e2.htm
Case Study Recommendations

• Patients of advanced age and severe illness must be approached with goals of care in mind, realistic expectations, and clear communication with families and caregivers
  – As always, better to have these conversations before the crisis rather than during.

• Keep in mind: resuscitation, suctioning, tracheostomy, and intubation are aerosolizing procedures (HHFNC, NIPPV and nebs NOT recommended in COVID patients, but sometimes used!) – N95s and eye protection minimum; neg pressure room ideal; viral filters on BVMs, tubes

• As of this recording, trials are underway in the West, but there is no good, controlled data to support the empiric use of antivirals or other medications (beyond supportive care) for COVID-19 (see Kalil AC in JAMA 24 Mar 2020 for discussion)
Initial Critical Care for COVID-19
## Critical Care Basics for Mild to Moderate ARDS

<table>
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<tr>
<th>Item</th>
<th>Details</th>
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<tbody>
<tr>
<td>( V_t ) 4-8 mL/kg and ( P_{plat} &lt; 30 \text{ cmH}_2\text{O} )</td>
<td>Consider NMBA and higher PEEP if inadequate</td>
</tr>
<tr>
<td>r/o bacterial infx (pan-cx)</td>
<td>Consider empiric abx</td>
</tr>
<tr>
<td>Target ( S_pO_2 ) 92-96% (or lower!)</td>
<td>Conservative fluid strategy</td>
</tr>
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Source: SCCM
COVID-19 in Pregnancy and Pediatrics
AES Question
Question 3

You are seeing a 19-year-old G1PO at 38 weeks gestation with symptoms consistent with COVID-19. You decide to test her, and it is positive. How does this change your management?

A. The patient may be at slightly higher risk for preterm birth, but this is uncertain.
B. The patient is at much higher risk for COVID-19 complications due to the immunosuppression of pregnancy.
C. She should be induced now as there is clear evidence for intrauterine transmission of SARS-CoV-2.
D. None of the above are true.
Case Study 5

• You see a 26-year-old G2P1001 pregnant patient at 33 weeks of gestation. So far, her prenatal course has been unremarkable. She is now reporting three days of worsening cough, dyspnea with exertion, myalgia and subjective fever symptoms. BP = 125/88, pulse = 105, RR = 22/min.
• The patient is taking a prenatal vitamin.
• She is a generally healthy young woman, had some childhood asthma, and both parents have T2DM.
• How would you risk stratify this patient? Is there anything about pregnancy that is concerning with potential COVID-19?
Case Study Evidence

What is CURRENTLY known (as of early April 2020) about SARS-CoV-2 infection in pregnancy?

First, some good news – in this review of 28 pregnancies, no increased maternal nor neonatal mortality, and no intrauterine transmission:


Second, more good news – pregnancy does not seem to worsen the clinical course of COVID-19:

Case Study Evidence (cont.)

There have been reports of preterm birth associated with pregnant patients with COVID-19, but it is unclear whether the PTB was caused by the illness.

Two excellent resources for FPs practicing OB during this pandemic:

1. ACOG’s COVID-19 guidance, including FAQs, patient info, and evaluation flowcharts: https://www.acog.org/topics/covid-19
2. Society of Maternal-Fetal Medicine guidelines and recommendations, including labor and delivery management: https://www.smfm.org/covid19

Specific issues: delivery or operating room preparation and management if pt is SARS-CoV-2 (+); PPE recommendations; care of the newborn.

• For example: N95s during second stage; avoiding ferning eval in SROM
Case Study Recommendations

Key points about care of pregnant PUIs and COVID-19 patients:

• Protect yourself, your staff, and other patients
• There is some good news so far about SARS-CoV-2 and pregnancy!
• Does not appear to be transmitted intrauterine
• There is a risk of transmission to newborns from infected mothers, but the overall risk of neonatal mortality seems low
• **As far as we now know, SARS-Co-2 is not transmitted via breastmilk, BUT could be transmitted via droplet – so careful prevention measures (hand hygiene, mask, or alternative breastmilk).** [Chen H, et al. Clinical characteristics and intrauterine vertical transmission potential of COVID-19 infection in nine pregnant women: a retrospective review of medical records. Lancet. 2020;395(10226):809.]
Case Study 6

• You see a five-year-old fully immunized boy born at full term whose mother is reporting he’s had three days of worsening cough, wheezing and dyspnea, and myalgia. BP = 90/50; pulse = 130 regular; RR = 30/min.
• On exam, he appears moderately ill with increased work of breathing. T 100.7 F. SaO2 on RA = 94%.
• The patient is taking an albuterol inhaler prn wheezing. His father smokes “outside.”
• He has a history of mild intermittent asthma, but has not been hospitalized nor intubated.

• How would you risk-stratify this patient?
Case Study (cont.)

Approaching children with potential COVID-19 illness:

• Fortunately, SARS-CoV-2 has tended to spare pediatric patients – their illness tends to be milder, and mortality rates have been very low.

• Management is very similar to other respiratory illness – RSV, flu.

• BUT – the additional angle of protection of vulnerable patients, staff, and clinicians.

• AAP is advocating virtual visits, postponing non-immunizing well child care, separate areas of the office for sick vs well, etc. – but NOT neglecting to immunize well children!
Case Study Evidence

• The patient in this scenario has asthma and a parent smoking at home, putting him at higher risk (just as with RSV or influenza) for more serious illness – asthma would make him Tier 2; o/w Tier 4

• There is concern that children may be significant vectors for SARS-CoV-2 spread without manifesting serious illness themselves

• Management approach is similar to what we’re all used to with RSV or influenza – aggressive supportive care, O2 supplementation

• Avoidance of steroids recommended; hospitalization case-by-case.
Case Study Recommendations

• SARS-CoV-2 testing should be considered for this patient depending on local availability, esp given his asthma; but also if he lives or has contact with vulnerable older adults

• Aggressive management of asthma (and parental smoking!) recommended (add a ICS? More frequent SABA? Consider a step-wise home mgmt. plan)

• Consider hospitalization of hypoxia develops, o/w most children can be managed well at home
Case Study Resources


• Also, check the CDC website for specific updates, patient information handouts, and guidance.

Resistant Organisms
Resistant Organisms

**Carbapenem-resistant *Actinetobacter***
- 8500 / year
- 700 deaths in 2017

**Drug-resistant *Candida auris***
- 300-350 / year

**C. difficile**
- 223,900 cases / year
- 12,800 deaths / year
- Case fatality rate 6-30%

**CDC’s Urgent Resistant Organisms**
- Source: www.cdc.gov/drugresistance/
- biggest_threats.html
Resistant Organisms

• CDC’s Urgent Resistant Organisms (continued…)

  • Source: www.cdc.gov/drugresistance/
  • biggest_threats.html

  Carbapenem-resistant Enterobacteriaceae (CRE)
  • 13,000 hospital infections / year
  • 1100 deaths

  Drug-resistant *Neisseria gonorrhoeae* (*N. gonorrhoeae*)
  • 550,000 cases/ year
Mechanisms of Antibiotic Resistance: A Case Study

• **Vancomycin-Resistant Enterococcus (VRE):**

  • Excellent example of the multiple ways bacteria evolve to resist antibiotics
  • It’s not just us…
AES Question
You are seeing an unvaccinated 3-year-old boy who presents with a temperature of 38.3 C, bilateral conjunctivitis and dry cough. Which of the following would NOT be consistent with a diagnosis of measles?

A. Irritability
B. 2 mm whitish elevations with an erythematous base on the oral mucosa
C. An exanthem with discrete vesicular, non-confluent lesions
D. Rhinorrhea and congestion
MEASLES

The “morbilliform” (measles-like) rash starts on the face and head, spreads inferiorly.
Clinical Presentation of Measles:

**PRODROME** (precedes the rash)

The Classic “Three C’s”:
1. Cough (severe, dry, hacking)
2. Coryza
3. Conjunctivitis

Also…
fever, malaise, irritability, photophobia.

**Koplik spots** (3-4 days after rash onset)

Measles is *highly contagious*!
90%+ attack rate
Complications of Measles

Early:
- dehydration
- pneumonitis and pneumonia (3% of adults)
- hepatitis, glomerulonephritis, myocarditis
- encephalitis (4-7 days after rash onset; mortality 10%)

Late and (fortunately) rare:
- subacute sclerosing panencephalitis (8.5 per 1 million cases)

Mortality: 1-2 deaths per 1000 cases
Lab Findings

CBC with leukopenia during prodrome
- Lymphocytes < 2000 is a bad prognostic sign

Respiratory Secretions: multinucleated giant cells
- also seen in buccal or nasopharyngeal mucosa and conjunctivae

Serology:
- anti-measles IgM 4x ULN diagnostic, detectable 3 d after rash onset
- BUT...more false (+) if prevalence low (parvo B19 can cause F+)
- Recommend both acute and convalescent serology (IgM, IgG)
Prevention and Treatment

Prevention: Vaccinate! (12-15 months old)
- efficacy: 95-98% antibody response; secondary vaccine failure reported but rare.
  First dose at 12 mos, second dose >28 d later.
- it’s an *attenuated live virus*, so contraindicated if immunosuppressed or pregnant.

Recommendations of the Advisory Committee on Immunization Practices

Treatment:
- mostly supportive, but vitamin A given in developing countries, and ribavirin has been used but not studied

Report all cases of measles to your Health Department! *Even one case is an outbreak.*
CHIKUNGUNYA
Chikungunya Fever

- Arbovirus, like West Nile, Colorado tick fever, dengue.
- Transmitted by mosquitoes
- 2013: First transmission in the Americas
- 2014: First transmission in the US (Florida)

Image public domain, from 1976, CDC / Dr Thomas Monath
Chikungunya Fever

• Incubation period: 3-7 days after mosquito bite

• Symptoms:
  • Fever, arthralgias, myalgias, joint swelling, rash.

• No specific treatment available.
Timeline of Infection, Symptoms, and Biomarkers.

CARBAPENEM-RESISTANT ENTEROBACTERIACEAE
February 2015

Outbreak of CRE due to contamination of duodenoscopes used for ERCP in LA County hospital
The Culprits

- *Klebsiella pneumoniae* carbapenemase (KPC)
- New Delhi Metallo-beta-lactamase (NDM)
  - Both also reported in *Pseudomonas* (yay!)


Image from Wiki Commons user Gilo1969, creative commons
What To Do With CRE?

• Test for susceptibility to:
  • Colistin, polymyxin B, aztreonam, tigecycline, and fosfomycin.

• Always use 2+ antibiotics.

• Consult an ID specialist!

Pertussis
If only there were a way to prevent it…
Complications of Pertussis

N = 1100 children < 2 years of age hospitalized with pertussis:

- Apnea: 27.1%
- Pneumonia: 9.4%
- Seizures: 2.6%
- Encephalopathy: 0.4%
- Death: 0.9%

2012: 48,277 cases in US, with 20 deaths – the most since 1955!

Pertussis Treatment

Antibiotic Options:
• Erythromycin estolate 1-2 grams in 3 divided doses x 7-14 days
• Clarithromycin 500mg in 2 divided doses for 7 days
• Azithromycin 500mg on day 1, 250 mg daily x 4 days
• TMP/SMX 160/800mg in 2 divided doses x 14 days (only if macrolide-intolerant)

Other treatments:
• Hospitalize infants and severely ill children
• Calm, quiet environment
• Beta-agonists and steroids have tried, but poor evidence
• Cough suppressants unhelpful
• Respiratory isolation until 5 days of abx therapy (or 3 weeks if not treated)
Post-Exposure Prophylaxis for Health Care Workers

- All of us are considered susceptible (waning immunity in adulthood).

- Tx: Azithromycin 500 mg orally on day one followed by 250 mg orally daily on days two to five (trimethoprim-sulfamethoxazole is an alternative)

- Symptomatic? Away from work from **beginning** of catarrhal stage to **third week** after onset of paroxysms…or five days **after** effective therapy

- Asymptomatic? Take the azithromycin and keep working.
Priorities

**Staff and Patient Safety**
- Containment before treatment
- Protect the caregivers, including yourself

**ABCs**
- Fix the physiology
- Treatment comes before diagnosis

**Infection Control**
- Universal precautions
- Isolation

Images all creative commons, from Wiki Commons users Mark Ahsmann, Pollo, Drewk
Immediately Reportable Diseases

- SARS-CoV-2
- Anthrax
- Botulism (clusters/outbreaks)
- Brucellosis (clusters)
- Diphtheria
- Measles
- Novel influenza A (H5N1)
- Poliomyelitis
- Plague (if suspected intentional)
- Rabies in a human or in an animal imported in < 60 days
- Smallpox
- Tularemia (if suspected intentional)
- Viral hemorrhagic fevers
- Yellow fever

From the National Notifiable Diseases Surveillance System (NNDSS)
References (apart from COVID-19)


- Centers for Disease Control and Prevention website: www.cdc.gov


Thank you!
pauldsimmons@gmail.com
Answer Key

1. D
2. B
3. A
4. C
Pulmonary Embolism/Deep Vein Thrombosis and Thrombophlebitis

Alan Ehrlich MD, FAAFP
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University of Massachusetts Medical School
Executive Editor, DynaMed
It is the policy of the AAFP that all individuals in a position to control content disclose any relationships with commercial interests upon nomination/invitation of participation. Disclosure documents are reviewed for potential conflicts of interest and, if identified, conflicts are resolved prior to confirmation of participation. Only those participants who had no conflict of interest or who agreed to an identified resolution process prior to their participation were involved in this CME session.

• Vu Kiet Tran, MD, MBA has disclosed a relationship with Elvium on the topic “Acute Pain Management”.

All individuals in a position to control content for this session have indicated they have no relevant financial relationships to disclose.
Learning Objectives

1. Select the proper diagnostic sequence of tests using validated clinical prediction rules.
2. Select an appropriate therapeutic regimen based on patient characteristics.
3. Manage low risk pulmonary embolism patients in an outpatient setting.
4. Determine the appropriate duration of treatment.
Key Take Home Points

• For DVT and PE, the first step in diagnosis is establishing pretest probability
• Use validated clinical prediction rules to establish the pretest probability
• In most cases, DOACs are the preferred first-line treatment for DVT and PE
• For PE, use risk stratification to guide management
• For Superficial vein thrombosis, size and location guide management
DVT

• DVT is caused by a blood clot obstructing blood flow in the deep venous system
• Incidence about 1.2/1,000 and increases with age
• 90% of DVT occurs in the legs but can occur in the upper extremities or pelvis
• DVT may be provoked or unprovoked
  – Provoked DVT has an identifiable inciting factor such as surgery or bed rest
  – Unprovoked DVT may be
    • Idiopathic
    • Due to inherited or acquired hypercoagulable states such as cancer and pregnancy
Types of DVT

• Distal DVT (calf DVT)
  • Isolated to the deep veins below the knee
  • Paired peroneal veins
  • Posterior tibial vein
  • Anterior tibial vein
  • Calf muscle vein thrombosis

• Proximal DVT
  • 80% of all diagnosed DVT
  • Thrombus extends at least into the popliteal vein
  • Includes ileofemoral DVT

Palareti G Blood. 2014 Mar 20;123(12):1802-9
Image unmodified from: https://commons.wikimedia.org/wiki/File:2136ab_Lower_Limb_Veins_Anterior_Posterior.jpg
AES Question
Question 1
What Test Should You Order?

Rudolph is a 72 year old retired pathologist being treated for pancreatic cancer. He was recently hospitalized for 5 days with pneumonia. He now comes in because of right leg swelling and calf pain. On exam, his right leg is 3.5 cm larger than the left (see image). He otherwise is feeling OK. You suspect he has a DVT.

Which of the following is the best next step?

A. No testing. Begin anticoagulation based on the clinical scenario
B. Order a high sensitivity D-Dimer test
C. Obtain a pulmonary CT angiography scan to rule out a concomitant PE
D. Use a validated tool for estimating his risk for a DVT

Image courtesy of: James Heilman, MD
https://commons.wikimedia.org/wiki/File:Deep_vein_thrombosis_of_the_right_leg.jpg
Virchow’s Triad

- Hypercoagulability
- Hemodynamic changes (stasis, turbulence)
- Endothelial injury/dysfunction

DVTs typically start in venous valve pockets

Risk Factors

- Increasing Age
- Previous DVT
- None (25%-40%)
- Surgery (15%)
  - Orthopedic surgery
  - Cesarean section
- Immobilization (15%)
  - Hospitalization or NH resident
  - Long travel (> 4-6 hours)
  - Stroke
  - Spinal cord injury
- Cancer (20%)

- Hypercoaguable states:
  - Pregnancy/postpartum
  - Oral contraceptive use
  - Sickle Cell Disease
  - Nephrotic syndrome
  - Advanced CKD
  - Factor V Leiden
  - Prothrombin G20210A
  - Antithrombin deficiency
  - Protein C deficiency
  - Protein S deficiency

DVT - Clinical presentation

• May be asymptomatic (especially in hospitalized patients)
• **Unexplained** swelling, pain, warmth, or erythema
• Other possible findings:
  - Popliteal vein tenderness (Pratt’s sign)
  - Pain with dorsiflexion (Homan’s sign)
  - Low grade fever (Michaeli’s sign)
  - Increased heart rate (Mahler’s sign)
• Only half of patients with calf pain and suspected DVT have thrombus

Bauersachs RM. Best Practice & Research Clinical Haematology 25 (2012) 243–251

Image courtesy of: James Heilman, MD
https://commons.wikimedia.org/wiki/File:Deep_vein_thrombosis_of_the_right_leg.jpg
Balancing Goals of Evaluation

Identify Who Needs Anticoagulation

Avoid Unnecessary Testing
Diagnosing DVT Starts With Using A Validated Risk Assessment
Validated Tools For Proximal DVT of LE

- Wells criteria (most commonly used)
- Oudega rule (includes D-dimer)
- CEBI (Center for Evidence-based Imaging)
Wells Criteria

• 1 point each for:
  − Active cancer (treatment ongoing or within previous 6 months or having palliative care)
  − Paralysis, paresis, or recent plaster immobilization of lower extremities
  − Recent episode of being bedridden > 3 days or major surgery within 4 weeks
  − Localized tenderness along distribution of the deep venous system
  − Swelling of entire leg
  − Calf swelling > 3 cm compared with other leg (measured 10 cm below tibial tuberosity)
  − Pitting edema (greater in the symptomatic leg)
  − Collateral superficial veins (nonvaricose)

• Subtract 2 points if alternative diagnosis as likely or more likely than DVT

Rudy’s Score

1 point: Active cancer Paralysis, paresis, or recent plaster immobilization of lower extremities
1 point: Recent episode of being bedridden > 3 days
1 point: Calf swelling > 3 cm compared with other leg (measured 10 cm below tibial tuberosity)

Total score = 3
Interpretation

**3-level**
- Low probability if score of $\leq 0$ (DVT present in 3%)
- Moderate probability if score 1 or 2 (DVT present in 16.6%)
- High probability if score $\geq 3$ (DVT present in 74.6%)

**2-level**
- Unlikely if score is $\leq 1$
- Likely if score is $> 1$

Limitations of Wells Criteria

• Based on data from patients referred to secondary care outpatient clinics
• May not perform as well in:
  − Hospitalized patients
  − Elderly patients
  − Patients with recurrent DVT
  − Patients with cancer
• Cannot safely rule out DVT without further testing
D-Dimer Testing

• Soluble degradation product of crosslinked fibrin
• Usually elevated in acute DVT
• Very sensitive, but not specific
• Elevated with aging, pregnancy, trauma, surgery
• Cut-off for positive result typically ≥ 500 mcg/L (0.5 mg/L)
  - Age-based cutoffs = (Age in years × 10) mcg/L
  - Risk-based cutoffs = 1,000 mcg/L for low risk and 500 mcg/L for others
Compression US

- Imaging modality of choice
- May be proximal (thigh to knee only) or whole leg
- Technique consists of compression of veins by ultrasound probe
  - If no DVT, gentle pressure with the probe causes venous lumen to collapse
  - Lack of compressibility is diagnostic for DVT
- Reported sensitivity and specificity about 95%
  - Less sensitive if: obesity, edema, leg tenderness, hip or knee arthroplasty, cast or overlying bandages
  - May miss pelvic or abdominal thrombosis
  - May have trouble distinguishing acute recurrent DVT from chronic residual thrombus

CT or MRI

• Consider if concern for pelvic or IVC thrombus
• CT usually done with contrast (venography)
• MRI is option for pregnant women and can be used with contrast (venography) or without
Testing for DVT with Low Probability

• Check hs D-Dimer or proximal compression US
  – If negative initial testing, no further testing needed
  – If D-Dimer +, check compression US
  – If compression US + (either as initial or confirming), start treatment

Testing for DVT with Moderate Probability

• Check hs D-Dimer, proximal compression US, or whole leg US
• Negative results
  − If negative D-Dimer or whole leg US, no further testing needed
  − If proximal compression US negative, check D-Dimer or repeat US in 1 week
• Positive results
  − If D-Dimer +, check compression US
  − If compression US + (either as initial or confirming), start treatment
  − If whole leg US + for proximal DVT, start treatment

Testing for DVT with High Probability

• Check proximal compression US or whole leg US
  − Treat if positive
  − Do not use D-Dimer as initial testing if high pretest probability

• Negative results
  − If whole leg US negative, no further testing needed
  − If proximal compression US negative, check hs D-Dimer, whole leg US or repeat US in 1 week
  − If hs-D-Dimer or whole leg US negative, no further testing needed (most of the time)

AES Question
Question 2
Just The Calf

Sheila is a 42 year old accountant who comes in because of pain in her calf. As part of her evaluation she has a compression US of her leg that shows an isolated calf DVT.

Which of the following factors if present, would indicate the need for treatment with anticoagulation?

A. Length of 4 cm
B. A positive D-Dimer
C. Cancer that was last active and treated 1 year ago
D. Recent knee surgery
Isolated Calf DVT

• Diagnosed by whole leg US
• Many resolve spontaneously
• Reported risk of proximal progression and PE is variable
• Risk factors for calf DVT extension warranting anticoagulation
  – Positive D-dimer
  – Close to the proximal veins or extensive thrombosis (> 5 cm in length, involves multiple veins; > 7 mm in maximum diameter)
  – No reversible provoking factor for DVT
  – Active cancer
  – History of VTE
  – Inpatient status

Evaluation of DVT 2-Level Approach
Treatment of DVT

• Goals
  – Prevent PE
  – Prevent recurrence
  – Prevent post-thrombotic syndrome (PTS)
  – Avoid bleeding

• Modalities
  – Anticoagulation
  – IVC filter
  – Thrombolysis or thrombectomy
  – Compression stockings
Other Treatments

• Thrombectomy/Thrombolysis
  − Catheter directed thrombectomy (CDT) may reduce PTS but no difference in QOL
  − CDT-thrombolysis needs to be added to thrombectomy or no benefit
  − Consider for Iliac vein thrombosis

• IVC filter
  − Used if absolute contraindications for anticoagulation
  − Needs to be removed when contraindication to anticoagulation resolved

Anticoagulation

- Treat for 3-6 months
- DOACs preferred to Warfarin

<table>
<thead>
<tr>
<th>Drug</th>
<th>Initial Dosing</th>
<th>Subsequent Dosing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apixaban</td>
<td>10 mg bid for 7 days</td>
<td>5 mg bid</td>
</tr>
<tr>
<td>Rivaroxaban</td>
<td>15 mg bid for 21 days</td>
<td>20 mg qd</td>
</tr>
<tr>
<td>Dabigatrin</td>
<td>LMWH for 5-10 days</td>
<td>150 mg bid</td>
</tr>
<tr>
<td>Edoxaban</td>
<td>LMWH for 5-10 days</td>
<td>60 mg qd (30 if Cr Cl &lt; 30 or use of PPI)</td>
</tr>
<tr>
<td>Warfarin</td>
<td>LMWH for 5-10 days (also start warfarin on day 1)</td>
<td>target INR 2-3</td>
</tr>
<tr>
<td>LMWH</td>
<td>Full strength dosing based on specific LMWH</td>
<td></td>
</tr>
</tbody>
</table>

The SOX trial – Prevention of PTS

- 806 patients with proximal DVT randomized to 30–40 mmHg stockings vs. placebo stocking
- Cumulative 2 years PTS incidence 52.6% vs 52.3% (HR= 1.0)
- No difference in PTS severity or quality-of-life observed
- Compression stockings remain a reasonable option for controlling symptoms of acute proximal DVT

Post-Thrombotic Syndrome

- Pain and disability due chronic venous insufficiency following deep vein thrombosis (DVT)
- Caused by venous hypertension from residual venous obstruction by thrombus
- Develops in 20%-50% following a DVT despite anticoagulant treatment
- Severe PTS in 5%-10% of cases
- Most cases occur within 1 year of acute DVT
- Symptoms similar to those of primary venous insufficiency
Pregnancy Considerations

• First-line imaging is proximal compression US
• D-Dimer rises during pregnancy
• D-Dimer fluctuates during pregnancy
• LMWH is drug of choice

Cancer Patients

• Start those with CrCl > 30 on LMWH; UFH if CrCl < 30
• After 5-10 days of LMWH treatment options include:
  − LMWH
  − Edoxaban
  − Rivaroxaban
• IVC filters rarely indicated

Key NS et al, J Clin Oncol. 2019 Aug 5:JCO190146
Upper Extremity DVT

- Most commonly due to central venous catheter, pacemaker, ICD or cancer
- Constans score can predict risk
  - +1 point if venous material (catheter in a subclavian or jugular vein or pacemaker)
  - +1 point if localized pain
  - +1 point if unilateral pitting edema
  - -1 point if other plausible diagnosis
  - Score < 2 = low risk, 2 = moderate risk, 3 = high risk
- Initial testing is compression US, but consider D-Dimer if low risk
- If US negative but high clinical suspicion options include
  - Moderate or highly sensitive D-dimer
  - Serial ultrasound
  - Venography, computed tomography (CT) venography, or magnetic resonance imaging (MRI) venography

AES Question
Question 3

Sasha

Sasha is a 62 year old woman with a red painful area in her calf for several days. It is not getting worse but not much better either. She has elevated the leg and put warm compresses on it. No recent leg trauma or immobilization but she is not particularly active. She has no chest pain or dyspnea. She has obesity and varicose veins. PMH includes Type II DM and HTN. Her exam is unremarkable except for a red tender area on her left medial calf (see image).

What is her most likely diagnosis?

A. Cellulitis
B. Chronic venous stasis
C. Superficial thrombophlebitis
D. Acute DVT
Superficial Vein Thrombosis (Thrombophlebitis)

- Inflammation and thrombosis of a superficial vein
  - Redness, warmth, and tenderness
  - Cord-like structure of thrombus with surrounding edema
  - May be in varicose vein
- RF same as DVT but also varicose veins
- May appear with DVT or with DVT symptoms

Thrombosis Canada Guidance
Thrombophlebitis Evaluation

- DDX
  - Cellulitis
  - Lymphangitis
  - Chronic dermatitis
  - Erythema nodosum
  - Cutaneous polyarteritis nodosa
- Obtain Compression US to R/O DVT and assess size/location
- Assess for PE symptoms (4% incidence)

Thrombosis Canada Guidance
## Thrombophlebitis Management

<table>
<thead>
<tr>
<th>Size/Location</th>
<th>Treatment</th>
</tr>
</thead>
</table>
| SVT ≤ 3 cm of SFJ | Full anticoagulation  
Compression stocking/bandages  
Encourage ambulation  
Topical NSAIDs if needed for 7-14 days |
| < 4-5 cm in length AND > 3 cm from SFJ | Symptomatic: Warm or cool compresses, topical or oral NSAIDs 7-14 days  
Anticoagulation only if severe sx or major RF for DVT |
| > 5 cm in length and > 3 cm from SFJ | Anticoagulation for 45 days:  
Fondaparinux (2.5 mg subcutaneously per day)  
Rivaroxaban 10 mg po daily  
Prophylactic/intermediate doses of LMWH  
Compression stocking/bandages  
Encourage ambulation  
Topical NSAIDs if needed for 7-14 days |
Pulmonary Embolism

PE - Clinical Presentation

- Risk Factors same as DVT
  - More RF = More risk
  - Up to 40% have no identifiable RF
- Presentation can include
  - Dyspnea/↑ RR
  - Tachycardia
  - Chest pain (especially pleuritic)
  - Pre-syncope or syncope
  - Hemodynamic instability
  - Hemoptysis
  - Hypoxemia – up to 40% have normal O2 sat
  - CXR – Nonspecific changes most common; Hampton hump not sensitive

ECG Changes

- Often normal or nonspecific
- Sinus tachycardia is most common finding

- Findings of RV strain
  - T-wave inversion V1-V4
  - QR pattern in V1
  - S1Q3T3
  - RBBB (may be incomplete)

Determining Pretest Probability

• PERC criteria
• Simplified Wells criteria
• Revised Geneva criteria
• PISA model (requires a specific calculator)
PERC

• Criteria – Low risk if all of the following
  − Age < 50 years
  − Pulse < 100
  − SaO$_2$ > 94%
  − No unilateral leg swelling
  − No hemoptysis
  − No recent trauma or surgery
  − No history of VTE
  − No oral hormone use

• Be careful in deciding when to use PERC

Simplified Wells Criteria

• 1 point for each of the following
  − Previous PE or DVT
  − Heart rate > 100 beats/minute
  − Surgery or immobilization within 4 weeks
  − Hemoptysis
  − Active cancer
  − Clinical signs of DVT
  − Alternative diagnosis less likely than PE

• PE unlikely if ≤ 1, likely if score > 1

Revised Geneva score

- Revised Geneva score
  - Age > 65 years = 1 point
  - Previous deep vein thrombosis (DVT) or PE = 1 point
  - Surgery under general anesthesia or lower limb fracture within 1 month = 1 point
  - Active malignant condition within 1 year = 1 point
  - Unilateral lower limb pain = 1 point
  - Hemoptysis = 1 point
  - Heart rate 75-94 = 1 points, heart rate ≥ 95 = 2 points
  - Pain on lower limb deep venous palpation and unilateral edema = 1 point

- Score Interpretation
  - Low risk if score 0-1
  - Intermediate risk if score 2-4
  - High risk if score ≥ 5

Diagnosing PE

Additional Testing

• Pulmonary angiography – Gold standard but invasive
• ECHO
  – Useful if PE likely but too unstable for CTPA
  – Used for risk stratification after PE diagnosed
• Venous duplex – If clinically suspicious for PE but cannot do imaging, + venous duplex is sufficient for presumptive dx
• Biomarkers: Troponins, BNP, lactate, creatinine
D-Dimer Testing: What Cut-off to Use?

- Standard 500 ng/dL
- Age adjusted: For patients > 50 years old use 10 X Age as cutoff
- Risk Adjusted:
  Intermediate risk – 500 ng/dL
  Low risk – 1000 ng/dL
AES Question
Question 4
Doc Can I Go Home Now?

Peter is a 62 year old lawyer who comes in for evaluation of 12 hours of dyspnea. His ECG had only nonspecific changes and, after having an elevated D-Dimer test he was found to have a PE on CT scanning. He is hemodynamically stable.

Which of the following factors would be enough to preclude outpatient management?

A. Any liver disease
B. Creatinine clearance of 35 mg/dL
C. Positive troponin
D. 4 hours supplemental O2 needed to maintain O2 sat > 93%
Hemodynamic Instability

• CPR needed
• Sustained hypotension
  − Systolic BP < 90 mmHg or systolic BP drop ≥ 40 mmHg
  − Lasting longer than 15 min
  − Not caused by new-onset arrhythmia, hypovolemia, or sepsis
  − Be cautious with fluids (can aggravate RV dysfunction)
  − May need pressors (norepinephrine or dobutamine)
• Obstructive shock
  − Systolic BP < 90 mmHg or vasopressors required to achieve a BP ≥ 90 mmHg despite adequate filling status
  − End-organ hypoperfusion (altered mental status; cold, clammy skin; oliguria/anuria; increased serum lactate)

Assessment if Hemodynamically Stable

• Clinical prediction scores
  – PESI score
  – Simplified PESI score
  – HESTIA score
• RV size/dysfunction/clots on CTPA or ECHO
• Troponin

## PESI vs. s-PESI

<table>
<thead>
<tr>
<th>Parameter</th>
<th>PESI</th>
<th>S-PESI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Age in years</td>
<td>1 point (if age &gt;80 years)</td>
</tr>
<tr>
<td>Male sex</td>
<td>+10 points</td>
<td>–</td>
</tr>
<tr>
<td>Cancer</td>
<td>+30 points</td>
<td>1 point</td>
</tr>
<tr>
<td>Chronic heart failure</td>
<td>+10 points</td>
<td>1 point</td>
</tr>
<tr>
<td>Chronic pulmonary disease</td>
<td>+10 points</td>
<td>1 point</td>
</tr>
<tr>
<td>Pulse rate ≥110</td>
<td>+20 points</td>
<td>1 point</td>
</tr>
<tr>
<td>Systolic BP &lt;100 mmHg</td>
<td>+30 points</td>
<td>1 point</td>
</tr>
<tr>
<td>Respiratory rate &gt;30</td>
<td>+20 points</td>
<td>–</td>
</tr>
<tr>
<td>Temperature &lt;36°C</td>
<td>+20 points</td>
<td>–</td>
</tr>
<tr>
<td>Altered mental status</td>
<td>+60 points</td>
<td>–</td>
</tr>
<tr>
<td>O2 saturation &lt;90%</td>
<td>+20 points</td>
<td>1 point</td>
</tr>
</tbody>
</table>
## Interpreting PESI

<table>
<thead>
<tr>
<th>PESI</th>
<th>s-PESI</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤ 65 = Class I (very low mortality risk)</td>
<td>Low risk</td>
</tr>
<tr>
<td>66-85 = Class II (low mortality risk)</td>
<td></td>
</tr>
<tr>
<td>85-105 = Class III (moderate mortality risk)</td>
<td>High risk</td>
</tr>
<tr>
<td>106-125 = Class IV (high mortality risk)</td>
<td></td>
</tr>
<tr>
<td>≥ 126 – Class V (very high mortality risk)</td>
<td></td>
</tr>
</tbody>
</table>
HESTIA Criteria for Outpatient Treatment (No to all items)

- Hemodynamically unstable
- Thrombolysis or embolectomy needed
- Active bleeding or high risk of bleeding
- Supplemental O2 to maintain oxygen saturation > 90% for > 24 hours
- Severe liver disease

- PE diagnosed during anticoagulant treatment
- Severe pain needing IV pain medication for > 24 h
- Medical or social reason for hospital treatment for > 24 h
- CrCl ≤ 30 mg/dL
- Pregnancy
- History of heparin-induced thrombocytopenia
Risk Stratification

- **High Risk** = Hemodynamically unstable
- **Intermediate Risk** = Hemodynamically stable PLUS ≥ 1 of the following:
  - PESI Class III-V or s-PESI > 0
  - + troponins
  - RV dysfunction
  - HESTIA criteria not met
- **Low Risk** = None of the above (Consider outpatient treatment)

## Risk Stratification

<table>
<thead>
<tr>
<th>Risk Level</th>
<th>Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>High risk</td>
<td>Hemodynamically unstable</td>
</tr>
<tr>
<td>Intermediate risk</td>
<td>Hemodynamically stable PLUS ≥ 1 of the following:</td>
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<tr>
<td></td>
<td>PESI Class III-V or s-PESI &gt; 0</td>
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<tr>
<td></td>
<td>+ troponins</td>
</tr>
<tr>
<td></td>
<td>RV dysfunction</td>
</tr>
<tr>
<td></td>
<td>HESTIA criteria not met</td>
</tr>
<tr>
<td>Low risk</td>
<td>None of the above</td>
</tr>
</tbody>
</table>
High-risk PE Management

Stabilization/Assessment

• Oxygen if pulse ox < 90%
• Start UFH while awaiting diagnostic tests (CTPA)
• ECG: exclude ACS, look for RV strain
• Bedside TEE to exclude alternative cardiac causes, confirm RV dysfunction
• Proceed with CTPA

Definitive Treatment

• Systemic thrombolytic therapy
• Surgical embolectomy if thrombolysis fails
• Consider catheter directed treatment if thrombolysis fails
• If necessary: intubation, mechanical ventilation; consider ECMO if patient unstable needs surgery or CDT

Intermediate- and Low-risk PE Management

- Oxygen if pulse ox < 90%
- If intermediate probability, start anticoagulation pending diagnostic tests
  - LMWH or Fondaparinux preferred over UFH
- DOACs are preferred long-term treatment for most patients
  - If patient has cancer, LMWH preferred followed by VKA
  - Apixaban and rivaroxaban can be started on Day 1
  - Dabigatrin and edoxaban should be started after 5-10 days of LMWH
- Avoid DOACs if CrCl < 30 (25 for apixaban), pregnancy or lactation, or antiphospholipid antibody syndrome
- LMWH preferred for cancer patients
- Routine thrombolysis not indicated, but rescue thrombolysis for hemodynamic deterioration is
Treatment Duration

• Minimum 3 months
• Additional treatment duration based on risk of recurrence vs risk of bleeding
Assessing Bleeding Risks

Risk Factors for Bleeding

- Advanced age (particularly >75 years)
- Previous bleeding (if not associated with a reversible or treatable cause) or anemia
- Active cancer
- Prior stroke, either hemorrhagic or ischaemic
- Chronic renal or hepatic disease
- Concomitant antiplatelet therapy or non-steroidal anti-inflammatory drugs
- Other serious acute or chronic illness

Risk Assessment (Only validated in VKA treated patients)

- HAS-BLED
- VTE-BLEED
- RIETE
Vena Cava Filters

- Use if absolute contraindication to thrombolysis
- Use if recurrence despite adequate anticoagulation

Image courtesy of BozMo
https://commons.wikimedia.org/wiki/File:Inferior_vena_cava_filter.jpg
Practice Recommendations

• For DVT and PE, the first step in diagnosis is establishing pretest probability
• Use validated clinical prediction rules to establish the pretest probability
• In most cases, DOACs are the preferred first-line treatment for DVT and PE
• For PE, use risk stratification to guide management
• In high risk PE, start anticoagulation immediately
• For Superficial vein thrombosis, size and location guide management
Answer Key

1. D
2. B
3. C
4. C
Acute Markedly Elevated Blood Pressure

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Disclosure

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Learning Objectives

1. Provide evidence-based management for severe asymptomatic hypertension.

2. Determine when patients with elevated blood pressure should be admitted to the hospital.
Hypertension (Elevated BP)

• This value is usually a sBP/dBP $\geq$ to 180/120
• MUST have acute end-organ damage = emergency
• Acute effects on brain, heart, aorta, kidneys and/or eyes
• Not always good to bring BP down
• Especially if NO symptoms
• Can develop in anyone NO pre-existing condition needed
• No real threshold for symptoms to manifest

• OFTEN though present with severe asymptomatic hypertension
Hypertensive (Elevated BP)

• Majority of patients already have hx of HTN (90%)
• In patients with known hypertension cerebral autoregulation curve shifts to the right and thus higher arterial pressures are needed to maintain CBF
• This is lost after a reduction of 25% or more of presenting BP
• Thus must use great care in BP reduction
Hypertensive Emergency

• Hypertensive emergencies < 1 percent of all ER visits
• Of these cases…
  • Cerebral infarction (39 percent)
  • Acute pulmonary edema (25 percent)
  • Eclampsia was the least common (2 percent)
AES Question
Question 1

For Hypertensive Emergency You Must Have…

A. Blood pressure >180/110
B. A Headache
C. Evidence of End Organ Damage
D. Elevated Troponin Levels
Clinical Pearl

• If one brings BP down for most HTN emergencies or even asymptomatic hypertensive urgency MUST DO slowly over the first few hours (around 12) and maximum 25% of presenting BP

• Adverse effects may develop such as a stroke/myocardial ischemia
Pathophysiology

• Poorly understood
• Common mechanism believed to be a ‘tipping’ point when vascular resistance increases due to humoral vasoconstrictors
• A critical arterial pressure overwhelms target organ’s ability to compensate for the increased arterial pressure = decrease blood flow
• Wall stress and endothelial injury ultimately = ORGAN HYPOPERFUSION and thus ischemia
Case #1 and Variations

• 67 year old otherwise healthy male presents to your UC/ED with a BP reading of 160/75 while at the local pharmacy he incidentally wanted to take a reading…what would you do?

• Suppose it was 180/80?...still no symptoms

• What if he had a mild headache and known DM/CAD?

• What if it was 180/82 at triage after he was complaining of severe and sudden onset of ‘ripping/tearing’ CP radiating to his back?
A Practical Classification of Common Hypertensive Emergencies

• Microvascular Disorders (small vessel dysregulation, with endothelial damage and local inflammation)
  - Such as encephalopathy, pre-eclampsia/eclampsia

• Macrovascular Disorders
  - Such as CHF, aortic dissection, stroke and subarachnoid hemorrhage
AES Question
Question 2

In most case of Hypertensive Emergencies the BP should be brought down by what maximum % from presenting value?

A. Less than 10%
B. Greater than 40%
C. Less than 25%
D. Greater than 50%
Overarching Treatment Goals

1. BP should be lowered slowly (a bit rapidly in aortic dissection)
   - MANTRA: GO LOW (DOSE) and TITRATE (TO EFFECT)

2. Lower by no more 25% in first couple of hours (15% in first day) to avoid ischemia in organ auto-regulated to higher BP
   - If decrease is too much / too fast can precipitate an ischemic stroke (as vascular beds auto-regulated to higher BP)

3. Therapies that correct the cause (e.g. phentolamine if the BP is elevated by catecholamines) are best

4. Monitor the symptoms to see if BP lowering has had an effect
Evaluation/Diagnosis for Causes of HTN

EMERGENCY

• Neurological symptoms
  – agitation, delirium, stupor, seizures, visual disturbances along with an altered level of consciousness may represent end organ damage which manifests as encephalopathy
  – (nausea/vomiting may be present)
  – whereas any focal deficits may be a sign of an ischemic / hemorrhagic stroke

• Check for acute head injury/trauma

• Look for physical exam signs of increased intracranial pressure: fresh flame hemorrhages, exudates (cotton-wool spots), or papilledema when direct funduscoppy is performed
Evaluation/Diagnosis for Causes of HTN

EMERGENCY

• Chest discomfort or pain may be due to myocardial ischemia or aortic dissection (if a ‘tearing sensation’ that radiates to the back +/- bilateral BP / pulse difference) … symptoms not always ‘textbook’
• Dyspnea may be due to pulmonary edema = CHF
• Pregnancy possible preeclampsia or developing eclampsia
• Sympathomimetic drugs such as cocaine / amphetamine
• Noncompliance / discontinuation of antihypertensive agents
Investigations Based on Presentation

• To evaluate presence of target-organ damage in association with targeted clinical symptoms or signs:
  
  • CXR and ECG
  • U/A
  • Serum electrolytes, creatinine, BhCG, CBC, TSH
  • Cardiac biomarkers (Troponins)
  • CT Head
Treatment Approach Specifics

• Aim is a reduction by maximum 20% within the first day (rarely <130/<80 mmHg)

• Major exceptions to gradual blood pressure lowering over the first day is an acute ischemic stroke

  − BP not lowered unless it is ≥185/110 mmHg in patients who are candidates for reperfusion (thrombolytic) therapy or ≥220/120 mmHg in patients who are not candidates for reperfusion therapy

  − Acute aortic dissection – The systolic blood pressure should be rapidly lowered to a target of 100 to 120 mmHg (to be attained in 20 minutes) to reduce aortic shearing

  − Intracerebral hemorrhage aim is variable depending on ICP often multiple drugs involved and IV BP meds an adjunct in ICU (balance b/w cerebral perfusion vs bleeding)
AES Question
Question 3

An exception to bringing down the BP slowly is in cases where the cause of the HTN is:

A. Due to non compliance with chronic medications
B. Angina
C. Hypertensive Encephalopathy
D. Aortic Dissection
Clinical Features

• Major presentations are reviewed

• Note: Not all cases may actually even present with elevated BP’s thus keep your ‘wits’ about you based on the history, exam and your ancillary testing
### Diseases and the BP levels at Presentation

<table>
<thead>
<tr>
<th>Disease</th>
<th>Threshold Value</th>
<th>% of Patients with Elevated BP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subarachnoid Hemorrhage</td>
<td>&gt; 140 mm Hg sBP</td>
<td>100</td>
</tr>
<tr>
<td>Ischemic Stroke</td>
<td>&gt; 140 mm Hg sBP</td>
<td>76.5</td>
</tr>
<tr>
<td>Hemorrhage Stroke</td>
<td>&gt; 140 mm Hg sBP</td>
<td>75</td>
</tr>
<tr>
<td>Type A vs B Aortic Dissection</td>
<td>&gt; 150 mm Hg sBP</td>
<td>35.7 vs 70.1</td>
</tr>
<tr>
<td>Acute Heart Failure</td>
<td>&gt; 140 mm Hg sBP</td>
<td>&gt; 50</td>
</tr>
<tr>
<td>NSTEMI-ACS</td>
<td>&gt; 140 mm Hg sBP</td>
<td>&gt; 50</td>
</tr>
</tbody>
</table>

Table 61.3 Specific Diseases Associated with Elevated BP’s from Tintinalli’s Chapter 61 Page 443.
Neurological Emergencies

• Severe hypertension with acute neurological signs or symptoms is most challenging due to wider differential diagnosis that vary in goals of treatment (some may not require a low BP)
• Ischemic stroke vs Hemorrhagic stroke (ICH/SDH)
• Head trauma – Increased ICP may = +++ BP and thus treat only if the cerebral perfusion pressure (mean arterial pressure minus intracranial pressure) is >120 mmHg and the intracranial pressure is >20 mmHg
• Hypertensive encephalopathy – In contrast to stroke and head trauma, the signs and symptoms of hypertensive encephalopathy (eg, headache, confusion, nausea, vomiting even seizures) usually abate after BP is lowered and is most often a diagnosis of exclusion
  – Brain CT would be normal
  – Labetolol / Nicardipine IV safe for all neuro related HTN emergencies
  – Precipitous drops always avoided
Cardiac Emergencies

- **Acute heart failure** – LV dysfunction and pulmonary edema give loop diuretics for diuresis (furosemide) AFTER nitroglycerin (spray quickly sublingual then IV drip) to decrease afterload (VITAL)
- Goal is to offset volume overload due to edema
- Drugs that increase cardiac work such as hydralazine or decrease contractility such as labetolol should be avoided
- **Acute coronary syndrome** – IV Nitroglycerin or IV Metoprolol thus decrease myocardial oxygen usage and ischemia = better outcomes
- If in cardiogenic shock then BP would be low
Vascular Emergencies

• Acute aortic dissection – rapidly reduce the blood pressure to a goal systolic of 100 to 120 mmHg within 20 minutes of diagnosis
• 90% abrupt onset of severe pain (often in chest – 78%)
• 25% ECG changes of which 13% will be T wave changes
• Only 31% have a pulse difference (based on BP differentials)
• 17% have some neurological deficit
• CXR abnormal 90% of the time (wide mediastinum) but non specific
• ‘Tearing, ripping and radiating to the back’ is the classic symptom
Vascular Emergencies Continued…

• IV beta blocker (BB) is given first (Esmolol, Labetolol, Propanolol) to reduce the heart rate below 60 beats per minute and decrease shear stress on the aortic wall +/- vasodilator such as NTG
• Anticoagulation is contraindicated (unlike in ACS)
• Be careful in distinguishing the two before starting Tx
Aortic Dissection Types

• The Stanford classification is divided into two groups, A and B, depending on whether the ascending aorta is involved.
• Hypotension is a bad sign.
• **A** – involves ascending aorta and/or aortic arch and possibly the descending aorta. The tear can originate in the ascending aorta, the aortic arch, or more rarely, in the descending aorta. It includes DeBakey types I and II (requires prompt surgical repair).
• **B** – involves the descending aorta or the arch (distal to the left subclavian artery), without the involvement of the ascending aorta. It includes DeBakey type III and often managed medically with HTN agents.
Renal Emergencies

• Severe BP elevation may cause acute injury to the kidneys (acute hypertensive nephrosclerosis)
• May have diffuse edema (anasarca), oliguria and confusion (rare)
• Often find microscopic hematuria * (found in most hypertensive emergencies) and elevated serum creatinine (might be pre-existing thus confirm)
• Hematuria due to fibrinoid necrosis of small arterioles
• Dialysis is rarely needed
• Fenoldopam may temporary improve renal function thus useful in such instances
• Can also use Labetolol
Sympathetic over activity = Hypertension

• Withdrawal of short-acting antihypertensive agents can be associated with severe hypertension
• Severe autonomic dysfunction (eg, Guillain-Barré and multiple system atrophy syndromes or acute spinal cord injury) is occasionally associated with hypertensive emergency
• Ingestion of sympathomimetic agents (amphetamine-like compounds, cocaine) can lead to severe hypertension and end-organ damage
  – IV benzodiazepines and NTG for reduction of drive/comfort
• Pheochromocytoma (periods of normal BP, increase HR, headache)
  – Adrenal tumor resection and pre-treat irreversible alpha blocker such as phentolamine, a combined alpha/beta blocker such as labetolol
No Beta-Blockers

• Unless a beta blocker was recently withdrawn, administration of a beta blocker alone is contraindicated in these settings since inhibition of beta receptor-induced vasodilation can result in unopposed alpha-adrenergic vasoconstriction and a further rise in blood pressure.
Hypertension in Pregnancy

• When BP is elevated and have signs/symptoms along the spectrum of pre-eclampsia, HELLP syndrome and eclampsia
• Such as Proteinuria, hemolysis, elevated liver enzymes, low platelets and +/- seizures

• Often labetolol, hydralazine and methyldopa are used

• NO ACEi has damaging effects on FETUS
AES Question
Question 4

The vast majority of patients presenting with hypertension

A. Have end organ damage
B. Have no history of hypertension
C. Have asymptomatic chronic hypertension
D. Have an intracerebral hemorrhage
Asymptomatic / Benign Presentations

• Often isolated headache, visual changes, chest pain, SOB and dizziness but not correlated with level of elevation
• Often in people already with HTN
• Pain / stress may lead to some elevation in BP
• Often present with epistaxis
• A reading 80 min after arrival / triage may be best indication of chronic HTN
• No standard approach in such cases
• Only 6% had any meaningful test results
• Order only if based on history, complaint and ROS there is a concern
Disposition
…if going home…

• Most patients will go home if not acute life threatening presentation of their hypertension (hypertensive urgency is a clinical presentation with no end organ damage and elevated BP, arbitrarily selected as 180/120 mm Hg)

• If this is the case they must be aware that they have to see their primary care physician to ensure secondary causes vs essential hypertension is confirmed

• Technically the JNC-7 classification refers to hypertension as readings ‘taken in a seated position on 2+ visits’

• Otherwise multiple co-morbidities may result with recurrent ER visits
Treatment

• For true hypertensive emergencies the goal is to safely bring down the BP and thus need parenteral (IV) drugs to titrate.

• Hypertension that is asymptomatic enables one to use oral agents both in the ED and on discharge but would caution on starting these medications.

• If do so please check co-morbidities and medication list thoroughly and give Rx only for 15-30 days until they follow up at a clinic (BP Clinic, Primary Physician or Internist).
Oral Agents

- Prescribe anti-HTN such as a thiazide to start or modify based on comorbidities
- If non compliant on medications may re-instate it
- Increase dosage of current medications being taken for HTN
- Sodium restriction
Acute Treatment

• The drug of choice is often dictated by the type of hypertensive emergency and the local hospital formulary/physician comfortability
• Not much head to head research on outcomes only consensus

• Three main categories
  – A) Beta-Blockers
  – B) Vasodilators
  – C) CCB
Beta-Blockers

• B-blockers: Labetolol is useful for most hypertensive emergencies
• A combined non-selective beta-adrenergic and selective alpha-adrenergic blocker with a rapid onset of action (five minutes or less but weak alpha blockade (7:1) ratio)
• **Labetalol should not be used without prior adequate alpha blockade in patients with hyperadrenergic states since unopposed, inadequately blocked alpha-adrenergic activity can increase blood pressure if beta blockade is not complete**
• Give 20mg IV over 2 minutes to start than can double dose every 10 minutes (maximum 300mg)
• IV infusion may be started as well (0.5 to 2 mg/min)
• Safe most of the time even in pregnancy
• **Do not give in cocaine / meth overdose induced hypertension due to unopposed alpha adrenergic activity**
• Careful if patient has COPD/Asthma (Esmolol is more cardioselective may be a better option in this case as quick onset and short ½ life)
Vasodilators

• Nitroglycerin is great for ACS and pulmonary edema, but arterio-dilates only at high dose
• Therefore for CHF patients, use higher doses to produce afterload reduction
• *Nitroprusside dilates both arteries and veins* but as mentioned has many side effects
• Hydralazine (10-20mg IV Bolus) also dilates arteries (not veins) yet has less predictable effects and raises HR
• **Phentolamine (an alpha blocker) arterio-dilates to counteract catecholamines surge such as in a cocaine (10 to 15 mg every 5 to 15 minutes as necessary) OD or PHEO**
Vasodilators

• Nitroprusside may lead to induced cyanide (metabolite) toxicity
  − Often 4+ hours later with altered mental status/lactic acidosis
  − Increased risk if treatment is over 24 hours, renal impairment and high doses > 2 mcg/kg
  − Can lead to perfusion decrease to brain, heart and kidney

• Nitroglycerin not as effective but better to veno-dilate
  − Less antihypertensive efficacy compared with other drugs used to treat hypertensive emergencies and its effects on blood pressure are variable from person to person and, potentially, from minute to minute. However, it may be useful in patients with symptomatic coronary disease and in those with hypertension following coronary bypass.
  − Initial dose is 5 mcg/min titrate to effect (max 100 mcg/min)
  − Onset in 2 to 5 minutes and lasts 5 to 10 minutes
  − Side effects are headache (due to direct vasodilation) and tachycardia (resulting from reflex sympathetic activation) No cyanide toxicity
CCB

- Nicardipine - works in 5-10 min and may be titrated q15 min and is quite safe and effective in neurological emergencies with favorable effect on myocardial oxygen balance increasing both stroke index and coronary blood flow
  - 5mg/h
  - Works in 5-10 min with vascular selectivity for cerebral/coronary arteries
  - Avoid in decompensated CHF or if on IV Beta Blockers

- Nifedipine is not advocated to be used acutely as multiple side effects
Question 5

Which of the following is not a class of anti-HTN drugs used via an IV to bring BP down acutely?

A. Beta Blockers
B. Diuretics
C. Calcium Channel Blockers
D. Vasodilators
AES Question
Question 6

In cases of Cocaine Toxicity Related Hypertension, which is TRUE

A. One should never bring down the BP
B. One should be careful in using unopposed alpha blockade
C. Only use benzodiazepines for treatment
D. Never give IV fluids
Disposition

• Vast majority of patients will go home
• Level of BP is not relevant it is the symptoms (end organ damage) and etiology of the hypertensive emergency
• If go home and have persistently over 140/90 then should have follow up for possible HTN dx and mgmt at a clinic
• If over 200/120 should start tx to gradually reduce the BP
• Thiazide diruretic or ACEi / Betablocker or CCB depends on patient and physician
• Specific indications of medications can also depend on co morbidities such as if angina/atrial fibrillation then a BB/CCB is good
• CHF then diruetic/BB, Hyperthyroidism/Migraines then BB
Disposition Continued…(Admission)

• High risk patients with chances of a cardiovascular event (DM, CAD, STROKE) should likely be admitted
• Compliance issues or if drop is to be monitored
• Any arrhythmias

• The last thing you want to do is rapidly drop BP below range at which tissue perfusion can be maintained by autoregulation
Practice Recommendations

• Hypertensive Emergency
  • Know the cause behind the BP elevation
  • Ensure no evidence of end organ damage
  • Rule out all of the life threatening causes
  • Titrate the BP down slowly and not more than 25% on the initial value in the first few hours
  • Can use a variety of agents, choose one you are comfortable with and use IV (parental) if possible
  • Monitor the effect and impact on symptoms
  • Disposition accordingly with admission if warranted

• Asymptomatic Hypertension (Urgency)
  • Ensure no symptoms
  • Assess if any investigations need to be done
  • Is this the first presentation?
  • If not is the cause non compliance vs low dosing?
  • If need to start meds ask ‘is it really needed today’?
  • If so then be judicious and start with safe meds and arrange follow up with GP or HYPERTENSION clinic
  • Good disposition instructions
References


• UptoDate: https://www.uptodate.com/contents/search?search=hypertensive%20emergency&sp=0&searchType=PLAIN_TEXT&source=USER_INPUT&searchControl=TOP_PULLDOWN&searchOffset=1&autoComplete=true&language=&max=0&index=2~6&autoCompleteTerm=hyperten

• Emergency Medicine Cases Episode 41 HYPERTENSIVE EMERGENCIES - EMERGENCYMEDICINECASES.COM
Thank-you
Answer Key

1. C
2. C
3. D
4. C
5. B
Fatal Headaches

Vu Kiet Tran, MD, MBA
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Learning Objectives

1. Utilize evidence-based strategies to diagnose patients with headache.
2. Identify red flags for potentially life-threatening causes of headache.
3. Use evidence-based recommendations to prescribe treatment for patients presenting with emergent headache pain.
The ED Headache Challenge!

1-2% → Serious or Life-Threatening
When should I send to the ED for further imaging?
Challenging questions

• If my imaging is negative, when should I push further?

• What percentages of “headaches NYD” are due to benign etiologies vs “malignant” etiologies?
Sandy

• 20 yo woman presents to your UC with a complain of headache and coryza that started this morning.
• She has a feeling of fever but never objectified it.
• She is feeling a bit nauseated but did not vomit.
Sandy

- BP 128/86
- Respiration rate 18
- Pulse 82
- Temperature 38.2 Celsius (100.8 F)
- Cardiovascular exam N
- Neurological exam N
- Neck supple
- Negative Brudzinski and Kernig
How many of you think she needs to go to the ED?
AES Question
Question 1

What would you like to do at this point?

A. Treat from the UC
B. Send to the ED for advanced imaging
Sandy

• What investigations would you do at this point?

• CBC N
• Biochemistries N
• Coagulogram N
• Non contrast CT head N
• LP or not LP?

• Lumbar puncture showed
  – WBC 18 (98% lymphocytes)
  – RBC 26 (1st and 4th tubes)
  – Glucose 4.27 mmol/l (77 mg/dl)
  – Protein 0.55 g/l
AES Question
Question 2

What would you like to do at this point?

A. Admit the patient
B. Keep the patient until the end of your shift and sign-off to your chief
C. Discharge the patient home
D. Call your mother for advice
Sandy

• The following day, she was found to be confused, anxious, agitative, aggressive, with visual hallucination and homicidal ideations
• Temp was 38.7 Celsius (101.66F)
• Multiple generalized convulsions that required IV lorazepam and phenytoin
• Intubated for airway protection
• Admitted to ICU
Sandy

• Her mental status improved and was discharged with levetiracetam 500 mg bid
• The following evening, she awoke after having visions that she would stab and kill her 3yo son
• She was taken back to the hospital for readmission for acute psychosis
Sandy

• CSF analysis were similar to previously obtained
• Bacterial and fungal studies were negative
• Cytology and flow cytology were negative
• Test for Lyme disease, EBV and arboviruses were negative
Sandy

• What else would you do?
Isabelle

• 46 yo female presents to your UC because of headache and malaise
• In town for a urology conference
• Started 8-9 hours ago
Isabelle

- Vital signs normal
- Neurological exam N
- Brudzinski and Kernig negative
- Cardiovascular exam N
AES Question
Question 3

What would you like to do at this point?

A. Send the patient to the ED for CT head
B. Prescribe some acetaminophen and discharge her with proper instructions to return if not feeling better
C. Call your grandmother for advice
Isabelle

• The following day, the patient and her husband were found unresponsive in their hotel room.
• The husband was found VSA
Eric

• 56 yo male presents to your department because he has a headache that started 20 minutes ago.
• He was at the endoscopy clinic upstairs
• Was investigated for esophageal varices
• He is a known cirrhosis patient
• He is not known with same headache previously
• No fever and neck stiffness
• No trauma
• Vital signs are normal
• Physical exam is normal

• What next?
Conditions
Anti-NMDA Receptor Encephalitis
NMDA Receptors

• NMDA receptors are ligand-gated cation channels with crucial roles in synaptic transmission and plasticity
  – NR1 subunit binds Glycine
  – NR2 subunit binds Glutamate
• Overactivity of NMDA receptors is proposed to cause excitotoxicity (epilepsy, dementia, and stroke)
• Low activity of NMDA receptors is proposed to cause Schizophrenia
Anti-NMDA (N-methyl D-aspartate) receptor antibody encephalitis

- Acute form of encephalitis
- Potentially lethal
- High probability for recovery
Pathophysiology

• Caused by an autoimmune reaction primarily against the NR1 subunit of the NMDA receptor
• Associated with tumours (20-57%), mostly teratomas of the ovaries
• Emerging that most cases do not have tumour (41% of cases with no detectable tumour)
## Etiologies

<table>
<thead>
<tr>
<th>Gender</th>
<th>Etiologies</th>
</tr>
</thead>
</table>
| Female | • < 14yo: 9% have ovarian teratoma  
• > 18yo: 50% have ovarian teratoma |
| Male   | • Testicular germ cell tumor  
• Teratoma of the mediastinum  
• Hodgkin’s lymphoma  
• Ovarian cystadenofibroma  
• Neuroblastoma |
Clinical manifestations

• 1-2 weeks of prodromal symptoms
  – Headaches
  – Low grade fever
  – Non-specific viral-like illness
• Decreased level of consciousness
• Seizures
• Progressing to catatonic-like states
  – Akinesis alternating with agitation
  – Mumbling
  – Echolalia
  – Orofacial dyskinesia
  – Eye contact and tracking absent or inconsistent
## Phases of NMDAR encephalitis

<table>
<thead>
<tr>
<th>Phases</th>
<th>Manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Prodromal</strong></td>
<td>• Headache</td>
</tr>
<tr>
<td></td>
<td>• Fever</td>
</tr>
<tr>
<td></td>
<td>• Often misdiagnosed as URTI</td>
</tr>
<tr>
<td><strong>Psychotic/seizure</strong></td>
<td>• Difficulty concentrating</td>
</tr>
<tr>
<td></td>
<td>• Altered mental status</td>
</tr>
<tr>
<td></td>
<td>• Behavioral changes</td>
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<tr>
<td></td>
<td>• Hallucinations</td>
</tr>
<tr>
<td></td>
<td>• Delusions</td>
</tr>
<tr>
<td></td>
<td>• Paranoia</td>
</tr>
<tr>
<td></td>
<td>• Seizures</td>
</tr>
<tr>
<td></td>
<td>• Tonico-clonic</td>
</tr>
<tr>
<td></td>
<td>• Abnormal movements (dyskinesia, myoclonus)</td>
</tr>
<tr>
<td></td>
<td>• Often requiring multiple anti-convulsants and airway management</td>
</tr>
<tr>
<td><strong>Unresponsive phase</strong></td>
<td>• Catatonic state</td>
</tr>
<tr>
<td><strong>Hyperkinetic phase</strong></td>
<td>• Autonomic instability</td>
</tr>
<tr>
<td></td>
<td>• Hyperthermia</td>
</tr>
<tr>
<td></td>
<td>• Tachycardia</td>
</tr>
<tr>
<td></td>
<td>• Bradycardia</td>
</tr>
<tr>
<td></td>
<td>• Hypoventilation</td>
</tr>
</tbody>
</table>
California Encephalitis Project

• Salient features
  – Seizures
  – Movement abnormalities
  – Language and aphasia
  – Autonomic instability
  – Psychiatric manifestations (Hallucinations and psychosis)
California Encephalitis Project

• Female to male 3:1
• 30% of patients are < age of 18
• Rare before the age of 3
Investigations

• Lab tests (NR1 and NR2 subunits)
  – Blood antibodies
  – CSF antibodies
• MRI abnormalities is non-specific at 46%
  – c/t 100% in HSV encephalitis
• CSF shows Pleocytosis
Barriers to Practice Diagnosis

- Characteristic manifestations
- Routine biochemistry unremarkable

- Lab tests (NR1 and NR2 subunits)
  - Blood antibodies
  - CSF antibodies

- Unenhanced CT scan of head unremarkable
- MRI unremarkable in close to 50% of cases
- CSF shows Pleocytosis

Clin Infect Dis. 2012 April 1; 54 (7): 899-904
Lumbar puncture

- CSF shows non-specific abnormality initially
  - Moderate pleocytosis (mainly lymphocytes)
  - Normal or slightly increased protein concentration
  - Negative for bacteria or fungus

- Detection of
  - NR1 and NR2 heteromers

Carr D. et al. CJEM 2013: 1-4
Causes of death

• Cardiac arrhythmias
• Nosocomial complications
• Effects of immunosuppressant therapy
Serotonin Syndrome
Definition

• Potentially fatal condition associated with increased serotonergic activity in the CNS

• Seen in
  – Therapeutic medication use
  – Inadvertent interactions
  – Intentional self-poisoning
Barriers to Practice Diagnosis

• Diagnosis is made purely on clinical grounds.

• Presence of the triad
  − Cognitive effects: delirium, agitation, hallucination, hypomania
  − Autonomic effects: diaphoresis, hyperthermia, tachycardia, diarrhea
  − Somatic effects: hyperreflexia, clonus, tremors
Common pitfalls in management

- Failure to recognize Serotonin Syndrome
- Misdiagnosis
- Failure to understand the potentially rapidly rate of progression
Methemoglobinemia
• Methemoglobin contains iron in the ferric state (Fe$^{3+}$) rather than the reduced ferrous form (Fe$^{2+}$) found in hemoglobin.
  - Causes an alteration in the blood’s ability to bind oxygen
    functional anemia
What is this?

• Occurs when RBCs contain methemoglobin at levels > 1%
• Symptoms are proportional to the methemoglobin level

• Neurologic and cardiac symptoms when levels > 15%
• Fatality > 70%
Colors of MtHB
Pathophysiology

• Congenital
  - 2 forms
• Acquired
  - More common
Predisposing factors

- Mucosal injury
- Methemoglobin reductase enzyme deficiency
- G6PD (Dapsone and Fava beans)
- Family history of methemoglobin reductase enzyme deficiency
- Liver cirrhosis
- PPIs (low gastric acid to maintain low levels of nitrate-reducing bacteria)
Substances causing MtHB

- Copper sulfate
- Organic compounds – Sodium chlorite,
- Recreational drugs – Phenylamine (psychoactive stimulant), cocaine
- Antimalarials – Primaquine, chloroquine,
- Antineoplastic agents – Cyclophosphamide, ifosfamide, flutamide, and 3-aminopyridine-2-carboxaldehyde thiosemicarbazone
- Analgesics and antipyretics – Acetaminophen, and celecoxib
- Zopiclone
- Herbicides and insecticides – Paraquat (dipyridylum), indoxacarb,
- Metoclopramide
- Antibiotics – Sulfonamides, nitrofurans, and para-aminosalicylic acid
- Industrial/household agents – Aniline dyes, nitrobenzene, naphthalene (moth balls), aminophenol, and nitroethane (nail polish remover)
Barriers to Practice
Clinical manifestation

• Has a variable clinical course
• Non-specificity of the clinical findings
  – Fatigue, flu-like symptoms, and headaches
  – Mild cases may go undiagnosed

• The most important aspects of the management are recognition of the condition and prompt initiation of treatment
Clinical manifestation

<table>
<thead>
<tr>
<th>Levels</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>3-15%</td>
<td>Slight discoloration of the skin (asymptomatic)</td>
</tr>
<tr>
<td>15-20%</td>
<td>Cyanosis (pt may be relatively asymptomatic)</td>
</tr>
<tr>
<td>25-50%</td>
<td>Headache, dyspnea, dizziness, weakness, confusion, palpitations, chest pain</td>
</tr>
<tr>
<td>50-70%</td>
<td>Arrhythmias, altered mental status, delirium, seizures, acidosis, coma</td>
</tr>
<tr>
<td>&gt; 70%</td>
<td>Death</td>
</tr>
</tbody>
</table>

Prompt recognition of the condition and initiation of treatment, are critical in the management of methemoglobinemia
Treatment
CO Poisoning
Carbon Monoxide Poisoning

• Constellation of symptoms mimic other illnesses
• 30-50% of all CO-poisoning may be misdiagnosed when presenting to the ED

MMWR. 2005; 54 (2): 36-39
Sources of CO

• Fires: stoves, portable heaters
• Automobile exhaust
• Improperly vented gas water heaters
• Kerosene space heaters
• Charcoal grills, hibachis
• Propane-fueled forklifts
• Gas-powered concrete saws
• Methylene chloride vapors
  – volatile liquid found in degreasers, solvents, and paint removers
• Indoor tractor pulls
• Improperly vented boats and boathouses
Temporal risk factors

- Fall and winter
- Ice storms, hurricanes, flooding
- Ice rinks
- Boat docks

- Portable generators
- Hibachi stoves
- Gas heaters
Symptoms

• The constellation of symptoms do not usually prompt clinicians to think about CO poisoning
• May mimic
  – Food poisoning
  – Migraine
  – Influenza
  – Substance abuse
  – Intracranial bleed
Symptoms

• Severity of symptoms vary with
  – Length of time of exposure
  – CO levels do not necessarily correlate with symptoms
  – Presence of cardiac or respiratory comorbidities
  – Pregnancy
Barriers to Practice
Clinical Presentation

• Misdiagnosis of CO toxicity is common
• History of exposure can be absent
• High index of suspicion - Think about it!
• Making the diagnosis with an ABG (or VBG)
Other conditions to not forget!
Cerebral vasculitis

- Giant cell arteritis
- Takayasu’s arteritis
- Polyarteritis nodosa
- Wegner’s Granulomatosis
- Lupus
- Rheumatoid arthritis

- Headache
- Fatigue/lethargy
- Movement disorders
- Dysesthesia
- Behavioral changes/psychosis
- Skin rash
Carotid/Basilar dissection headache

- Acute headache of SAH intensity - 20%
- Frequently gradual onset HA, however - 80%
- May have antecedent neck trauma (chiropractic manipulation, mild fall low speed MVC, “cracking” neck)
- Might have neck pain, but might not
Cavernous sinus thrombosis

• Predisposed by:
  − 50% facial infection, 30% sphenoid sinusitis, dental infection 10% misc
• Clinical manifestations
  − Cranial nerve findings: CN6 usually first
    • Maybe diplopia (CN 3, 4, 6), unilateral eye swelling
  − Frequently unilateral and misdiagnosed as migraine
Case revisited
Sandy

• 20 yo woman presents to your emergency department with a complain of headache and coryza that started this morning.
• She has a feeling of fever but never objectified it.
• She is feeling a bit nauseated but did not vomit

Anti-NMDAR encephalitis
Isabelle

• 46 yo female presents to your emergency department because of headache and malaise
• In town for a urology conference
• Started 8-9 hours ago

CO poisoning
Eric

• 56 yo male presents to your department because he has a headache that started 20 minutes ago.
• He was at the endoscopy clinic upstairs
• Was investigated for esophageal varices
• He is a known cirrhosis patient
• He is not known with same headache previously
• No fever and neck stiffness
• No trauma
• Vital signs are normal
• Physical exam is normal

Methemoglobinemia
## Fatal headaches with normal imaging

<table>
<thead>
<tr>
<th>Categories</th>
<th>Pathologies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overdoses</td>
<td>• Carbon monoxide</td>
</tr>
<tr>
<td></td>
<td>• Methemoglobinemia</td>
</tr>
<tr>
<td></td>
<td>• Salicylate</td>
</tr>
<tr>
<td></td>
<td>• Amphetamine</td>
</tr>
<tr>
<td></td>
<td>• Cocaine</td>
</tr>
<tr>
<td></td>
<td>• PCP</td>
</tr>
<tr>
<td></td>
<td>• Methanol</td>
</tr>
<tr>
<td></td>
<td>• Cyanide</td>
</tr>
<tr>
<td>Autoimmune</td>
<td>• Anti-NMDA Receptor Encephalitis</td>
</tr>
<tr>
<td></td>
<td>• SLE cerebritis</td>
</tr>
<tr>
<td></td>
<td>• Antiphospholipid syndrome</td>
</tr>
<tr>
<td></td>
<td>• Hashimoto encephalopathy</td>
</tr>
<tr>
<td></td>
<td>• Temporal arteritis</td>
</tr>
<tr>
<td>Drug-related</td>
<td>• Neuroleptic malignant syndrome</td>
</tr>
<tr>
<td></td>
<td>• Serotonin syndrome</td>
</tr>
</tbody>
</table>
# My approach

<table>
<thead>
<tr>
<th>Non-fatal headaches</th>
<th>Fatal headaches</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary headaches</strong></td>
<td><strong>Intracranial pathologies</strong></td>
</tr>
<tr>
<td>Tension headaches</td>
<td>Acute Sinusitis</td>
</tr>
<tr>
<td>Chronic daily headaches</td>
<td>Trigeminal nerve neuralgia</td>
</tr>
<tr>
<td>Migraines</td>
<td></td>
</tr>
<tr>
<td>Pseudo-tumor cerebri</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Space-occupying lesion</td>
</tr>
<tr>
<td></td>
<td>Cerebral venous thrombosis</td>
</tr>
<tr>
<td></td>
<td>Hydrocephalus</td>
</tr>
<tr>
<td></td>
<td>Meningitis</td>
</tr>
<tr>
<td></td>
<td>Encephalitis (viral)</td>
</tr>
</tbody>
</table>
Barriers to Practice
Red flags

• Sudden thunderclap headache
• “Most severe headache of my life”
• Occipital headache
• Headache with exertion or sexual activity
• Fever
• Lateralizing neurological symptoms or signs
• Cognitive or psychiatric features
• Medication and interactions
• Travel
Barriers to Practice
Pitfalls to Avoid

• Too-focused history
• Neglecting a good medication history
• Neglecting to inquire about sick contacts or illness in family members
• Forgetting that fatal systemic conditions also can produce headaches
Think beyond the cranium
Don’t be a vomiting
Review all meds and substances
Collateral history from family
Practice Recommendations

• Must keep Ddx open to avoid the commonly missed fatal headaches
• Inquire about possible *exposures* when assessing atypical/unexplained headaches
• Anti-NMDAR encephalitis deserves a place at the top of encephalitis differential
Answer Key

1. No true answer
2. No true answer
3. No true answer
4. No true answer
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Respiratory Failure

Brian Shahan, MD FAAFP DFPHM
Disclaimer

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• Vu Kiet Tran, MD, MBA has disclosed a relationship with Elvium on the topic “Acute Pain Management”.

All individuals in a position to control content for this session have indicated they have no relevant financial relationships to disclose.
Learning Objectives

1. Apply the most recent evidence for the use of high-flow nasal cannula in clinical practice.
2. Determine patients that have the potential to improve with non-invasive ventilation.
3. Implement best practice therapies for the treatment of respiratory failure due to asthma.
4. Implement evidence-based practices for ARDS and lung protection with mechanical ventilation.
Respiratory Physiology
Respiratory Physiology

Original Image
Respiratory Failure

• Failure of gas exchange

  – Oxygenation
    • Get O2 in
    • Failure = hypoxemia

  – Ventilation
    • Get CO2 out
    • Failure = hypercapnia

Courtesy of Damon Forbes, MD
Respiratory Physiology

- **Oxygenation**
  - Get O2 in
- **Ventilation**
  - Get CO2 out

- FiO2
- PEEP
- Tidal Volume
- RR
AES Question
Question 1

65 yo female with PMH of COPD presents to the ED with tachypnea and cough. ABG on 15L non-rebreather shows of pH of 7.25, PaCO2 of 68, PaO2 of 50, and bicarb of 26. Define the patient’s respiratory status.

A. Acute hypoxemic Respiratory Failure
B. Acute hypercapnic Respiratory Failure
C. Acute hypoxemic and hypercapnic Respiratory Failure
D. Chronic hypercapnic respiratory failure
Respiratory Failure

Hypoxemic (Type 1)
- PaO2 LOW (<60)
- PaCO2 NORMAL/LOW

Hypoxemic and Hypercapnic (Type 2)
- PaO2 LOW
- PaCO2 HIGH (>45)
- pH LOW (if acute)
Acute vs Chronic

- **Acute Resp Acidosis**
  - pH LOW
  - PaCO$_2$ High
  - Bicarb ↑ slightly

  $\text{PaCO}_2 : \text{Bicarb} \, 10:1$

- **Chronic Resp Acidosis**
  - pH NORMAL
  - PaCO$_2$ High
  - Bicarb ↑ High

  $\text{PaCO}_2 : \text{Bicarb} \, 10:4$
A 14 yo female with asthma presents to the clinic with respiratory distress and wheezing. On exam she appears drowsy and you suspect respiratory failure. You order a STAT ABG but your nurse states she is not qualified to perform this test. What can be done instead?

A. Pulse oximetry with venous blood gas  
B. Pulse oximetry alone  
C. VBG only as pulse ox is not accurate  
D. ABG must be done
Blood Gas Analysis

<table>
<thead>
<tr>
<th>Arterial</th>
<th>Venous</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.4</td>
</tr>
<tr>
<td>PaCO2</td>
<td>40</td>
</tr>
<tr>
<td>PaO2</td>
<td>80</td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>24</td>
</tr>
<tr>
<td>Base excess</td>
<td>0</td>
</tr>
</tbody>
</table>
Pulse Oximetry

• Infrared and red light
Pulse Ox > ABG?

• ABG is a painful and expensive test

• 1 ABG will lead to more ABGs

• May be contaminated with venous blood

• Can delay management

• PaO2 measurements are highly variable (+/- 9)

• ABG only measures oxygenation at a single time point

https://emcrit.org/pulmcrit/pulse-oximetry/
SpO2 Pitfalls

- Poor wave form
- Nail polish
- Carboxy Hgb
- Methemoglobin
- P:F ratio
- High altitude

Image from Wikipedia
AES Question
Question 3

You are walking around the hospital ward and a nurse is in distress because her patient, Mr. Jones, has a SpO2 of 77%. You don’t know this patient. What do you do first?

A. Ask the patient his name
B. Trouble shoot the SpO2
C. Call a code blue
D. Start 2L nasal cannula
ACLS First!

• ABCs
  • If patient can talk then airway is clear
    – If nonresponsive then check pulse
AES Question
Question 4

Mr. Jones is able to state his name and says he is short of breath. There is a good waveform on the pulse oximeter which still reads 77%. He appears anxious and the nurse states he is 70 years old and was admitted for PNA. What should be done next?

A. 2L/min O2 via nasal cannula  
B. Non-rebreather at 15 L/min of O2  
C. BiPAP at 10/5 with FiO2 of 100%  
D. Ask the nurse more history
Nasal Cannula

The Good:
• Easily accessible
• Comfortable

The Bad:
• FiO2 28-44%
• Epistaxis
Non-rebreather

The Good:
• Quick access
• High FiO2 (60-80%)

The Bad:
• Mask is one size
• Can suffocate
AES Question
Mr. Jones’ SpO2 has come up to 84% with the non-rebreather. He feels slightly better but still has increased work of breathing. RR is 32, BP is 130/90, HR is 95, Temp is 99.5. He has no contributing PMH and is Full Code. What is the next step in management?

A. Continue non-rebreather as there was good response
B. Recommend intubation
C. Check VBG
D. Initiate BiPAP
E. Initiate HFNC
Mechanical Ventilation

- Respiratory Rate (RR)
- Tidal Volume ($V_t$)
  - $\text{RR} \times V_t = \text{Minute Ventilation} (V_m)$
- Fraction of inspired O2 ($F_iO_2$)
- Positive End Expiratory Pressure (PEEP)

Ventilation (CO$_2$)

Oxygenation
Reasons for Intubation

• Hypercapnic encephalopathy
  – PCO2 likely reassuring if pt is mentating well

• Refractory hypoxemia

• Exhaustion
  – Most common cause!
Hypoxemic Respiratory Failure

Treatment:
↑ FiO2 – if not better:
   Add PEEP!

What form of positive pressure should we use?...
NPPV - CPAP

Continuous Positive Airway Pressure (CPAP):
• Hair dryer in the mouth
• “PEEP” + FiO2
  • Stents open airways

• Indications
  • OSA
  • Neonates with respiratory distress
  • Cardiogenic Pulm Edema
  • Post operative/Atelectasis

Original photo
BiPAP is a brand name
- Hairdryer plus burst of air with inspiration
- Higher “PEEP” + FiO2
  - Recruits more alveoli
  - Decreases work of breathing
  - Can provide some Tv

\[
\frac{IPAP}{EPAP} \quad \text{Start at } \frac{10}{5}
\]
NPPV - Bilevel

Indications for “Rescue BiPAP”
- COPD exacerbation
- Cardiogenic Pulm Edema
- Immunocompromised
- Extubated patients
NPPV - Bilevel

• What about the other causes of Respiratory Failure?
  – Insufficient evidence currently for:
    • Asthma
    • Trauma

  – Recommended against:
    • Undifferentiated acute respiratory failure
    • ARDS

• Don’t use nasal mask for rescue NPPV
NPPV

• Contraindications
  − Critically ill patient
  − Obtunded (unable to protect airway)
  − Vomiting/high volume secretions
  − Upper GI bleeding

• Rules of Thumb
  1. Don’t rescue NIV longer than 24 hours
  2. If not improved in 1-2 hours, intubate!
High Flow Nasal Cannula

- **HFNC**
  - Humidified hairdryer in nose
  - Small “PEEP” + FiO2
  - Decreases dead space
  - Comfortable
  - Improves work of breathing

- **FLORALI trial**
  - Reduced mortality

- **Ann of Em Med**
  - Non-inferior to NIV
HFNC

• Much more comfortable than NIV
• Patients can eat… or vomit
• Decreases dyspnea

• Decreased intubation rate now confirmed with multiple meta-analyses
• Treat as if patient is intubated
AES Question
Mr. Jones was intubated via rapid sequence with confirmed ET tube placement with capnography and chest x-ray. After an hour on the ventilator his ABG shows a pH of 7.55, PaCO2 of 22, PaO2 of 83 and Bicarb 20. Which of the following ventilator settings should be adjusted?

A. Lower respiratory rate
B. Increase Tidal Volume
C. Lower PEEP
D. Increase FiO2
Mechanical Ventilation

- Respiratory Rate (RR)
- Tidal Volume ($V_t$)
  - $\text{RR} \times V_t = \text{Minute Ventilation} (V_m)$
- Fraction of inspired O2 ($F_iO_2$)
- Positive End Expiratory Pressure (PEEP)

Ventilation ($CO_2$)

Oxygenation
Mechanical Ventilation

• Control
• Assist Control
  – Volume
  – Pressure
• IMV/SIMV
• Dual Control Modes
Mechanical Ventilation
Auto Peep/Air Trapping

Original photos
Question 7

Mrs. Jones also presents to the ED with respiratory distress. Her pulse oximeter reads 72% with a good wave form. She is placed on high flow nasal cannula but is soon intubated when her O2 saturations did not improve. Chest x-ray shows →
ABG: pH 7.40 pCO2 40 \( pO2 \) 42 Bicarb 24
Influenza B positive

Courtesy of Damon Forbes, MD
AES Question
Question 7

Which of the following does NOT have evidence supporting the treatment for this condition?

A. Low Tidal Volumes
B. High Peep
C. Prone positioning
D. Neuromuscular paralysis
E. Aggressive fluid resuscitation

ARDS

• Berlin Criteria
  – Occurs within 7 days of pulmonary insult
  – Bilateral chest infiltrates consistent with non-cardiogenic pulmonary edema
  – Severity
    • P:F ratio 200-300
    • P:F ratio 100-200
    • P:F ratio <100

  – Technically all patients with a measured P:F ratio were intubated with a minimum peep of 5
AES Question
Mrs. Jones is 5’2” (158cm) and 330lbs (150kg). Her ideal body weight is 50 kg. What should be her tidal volume when mechanically ventilated with lung protection strategy?

A. 300 cc
B. 400 cc
C. 900 cc
D. 1200 cc
ARDS Treatment

• Paucity of evidence
• Supportive care
  − Treat underlying disorder
  − Support breathing until lungs recover
  − Try not to induce iatrogenic injury

• Purpose of mechanical ventilation
  − “Rest the respiratory muscles while providing adequate gas exchange”

Positive ARDS Evidence

• ACURASYS – Early paralysis

• PROSEVA – Proning

• ARDSNET – Low tidal volumes, High PEEP

• CESAR – ECMO
Negative ARDS Evidence

- FLORALI – HFNC
- ROSE – Early paralysis
- EOLIA – Routine use of ECMO
- OSCAR – Oscillator ventilation technique
OXYGENATION GOAL: \( \text{PaO}_2 \geq 55-80 \) mmHg or \( \text{SpO}_2 \geq 88-95\% \)

Use a minimum PEEP of 5 cm H₂O. Consider use of incremental \( \text{FiO}_2/\text{PEEP} \) combinations such as shown below (not required) to achieve goal.

<table>
<thead>
<tr>
<th>( \text{FiO}_2 )</th>
<th>0.3</th>
<th>0.4</th>
<th>0.4</th>
<th>0.5</th>
<th>0.5</th>
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<th>0.7</th>
<th>0.7</th>
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<tbody>
<tr>
<td>( \text{PEEP} )</td>
<td>5</td>
<td>5</td>
<td>8</td>
<td>8</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td>12</td>
</tr>
<tr>
<td>( \text{FiO}_2 )</td>
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<td>0.9</td>
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<tr>
<td>( \text{PEEP} )</td>
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<td>14</td>
<td>14</td>
<td>15</td>
<td>18</td>
<td>18-24</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Higher PEEP/ lower \( \text{FiO}_2 \)

<table>
<thead>
<tr>
<th>( \text{FiO}_2 )</th>
<th>0.3</th>
<th>0.3</th>
<th>0.3</th>
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<th>0.4</th>
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<tr>
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<td>5</td>
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<td>16</td>
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<tr>
<td>( \text{FiO}_2 )</td>
<td>0.5</td>
<td>0.5-0.8</td>
<td>0.8</td>
<td>0.9</td>
<td>1.0</td>
<td>1.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>( \text{PEEP} )</td>
<td>18</td>
<td>20</td>
<td>22</td>
<td>22</td>
<td>22</td>
<td>22</td>
<td>24</td>
<td></td>
</tr>
</tbody>
</table>

PLATEAU PRESSURE GOAL: \( \leq 30 \) cm H₂O

Check Pplat (0.5 second inspiratory pause), at least q 4h and after each change in PEEP or \( \text{FiO}_2 \).

If Pplat \( > 30 \) cm H₂O: decrease \( \text{FiO}_2 \) by 1 ml/kg steps (minimum = 4 ml/kg).

If Pplat \( > 25 \) cm H₂O and \( \text{FiO}_2 \) \( < 6 \) ml/kg, increase \( V_t \) by 1 ml/kg until Pplat \( > 25 \) cm H₂O or \( V_t = 6 \) ml/kg.

If Pplat \( < 30 \) and breath stacking or dys-synchrony occurs: may increase \( V_t \) in ml/kg increments to 7 or 8 ml/kg if Pplat remains \( < 30 \) cm H₂O.
Plateau Pressure

• Inspiratory hold

• Keep < 30

• PEEP and Tidal Volume
  - Decrease tidal volume if high PEEP is required for oxygenation
ARDS Summary

- Low tidal volume
- High PEEP
- Conservative fluids
- Prone positioning
- Paralysis

- ECMO if still unable to oxygenate

Hardin CC, Hibbert K. ECMO for Severe ARDS. N Engl J Med 378;21
## Review

### Oxygenation (O2 in)

- **FiO2**
- **PEEP**

### Ventilation (CO2 out)

- **Tv**
- **RR**

- **NC/O2 mask** – FiO2 only
- **CPAP** – FiO2 + peep + ↓ WOB
- **HFNC** – FiO2 + peep + ↓WOB + ↓deadspace
- **Bilevel** – FiO2 + PEEP + ↓ WOB + Tv*
- **Mechanical Vent** – FiO2 + PEEP + Tv + RR

*Tv* stands for tidal volume.
AES Question
Fun Questions: Q9

You are on night shift and are called by the nurse because her patient has a pulse ox persistently at 84%. The patient is a 45 yo male with a BMI of 48 who had gastric bypass surgery earlier in the day. His RR is 22 and he appears comfortable with 5L NC. His pulse ox is reading 84% with good wave form and improves to 89% with non-rebreather. Exam shows bilateral decreased breath sounds and intact surgical incisions. CXR shows low lung volumes. VBG showed pH of 7.34 and PaCO2 of 48. What should be done next?

A. CPAP  
B. Albuterol nebulizer  
C. Naloxone  
D. Intubation
Post Op Gastric Bypass

• Atelectasis
  – Hypoventilation
    • Abdominal pain from incision and operation
    • Obesity

• OSA
AES Question
Fun Questions: Q10

14 yo female with PMH of severe asthma presents with dyspnea. She is only able to speak one word at a time and appears drowsy. RR is 35 and bilateral wheezes noted. Pulse ox shows 93% on 2L NC. ABG in ED after 2 albuterol nebulizers in the ambulance shows pH of 7.4 and PaCO2 of 40. What is the next step in management?

A. Intravenous methylprednisolone
B. Continuous nebulizer
C. Admission for observation
D. Intubation
Beware normal ABG in Asthma

• Asthma generally causes hypocapnia
• Eucapnia/hypercapnia suggests fatigue
• Could consider NPPV but low threshold for intubation
AES Question
Fun Questions: Q11

22 yo male with severe asthma was intubated in the ED for respiratory failure. After 10 minutes on the ventilator the nurse reports the patients vitals are worsening. HR is 35, BP 70/30, SpO2 is 72%. What should be done next?

A. Give epinephrine
B. Disconnect the patient from the ventilator
C. Increase PEEP
D. Increase Tidal Volume
Air Trapping

• Causes increased intrathoracic pressure
  − Reduced venous return
  − Mimics tension ptx or cardiac tamponade

• Disconnect ventilator and let pt exhale completely

• Restart ventilations with lower RR and shorter I:E ratio
AES Question
72 yo female with COPD (FEV1 of 25%) and HFrEF (35%) presents with 5 hours of dyspnea and chest tightness. On exam she is anxious appearing. Denies orthopnea. Pulm exam shows faint bilateral expiratory wheezes and faint inspiratory crackles. Temp is 38.0 (100.4), RR 22, HR 110, BP 120/78. SpO2 is 88% on 5L NC after 2 albuterol/ipratropium nebulizers (no home O2 at baseline). CXR demonstrates hyperinflation and cardiomegaly. What is the next step in management?

A. Start levofloxacin
B. Start IV furosemide
C. Obtain CT chest angiogram
D. Give IV methylprednisolone
• Don’t get tunnel vision
• Hypoxemia is normally easily treatable in COPD
• COPD has higher risk of PE
  – Likely due to inactivity and frequent hospitalizations
Practice Recommendations

• High flow nasal cannula may reduce intubation with undifferentiated hypoxemia. (LOE B)

• Bilevel NIV may reduce intubation in cardiogenic pulmonary edema and COPD exacerbation. (LOE B)

• Mechanical ventilation in ARDS should be treated with lung protective strategy with low tidal volume ventilations. (LOE B)
References

• Keenan, S et al. Clinical practice guidelines for the use of noninvasive positive-pressure ventilation and noninvasive continuous positive airway pressure in the acute care setting. CMJA. 2011
• ARDSnet. Mechanical Ventilation Protocol Summary. NIH NHLBI ARDS Clinical Network
• Hardin CC, Hibbert K. ECMO for Severe ARDS. N Engl J Med 378;21
Answer Key

1. C
2. A
3. A
4. B
5. B or E
6. A
7. E
8. A
9. A
10. D
11. B
12. C
AAFP
AMERICAN ACADEMY OF FAMILY PHYSICIANS
STRONG MEDICINE FOR AMERICA
Cellulitis and Cellulitis Mimics

Vu Kiet Tran, MD
Disclosure

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Learning Objectives

1. Perform a differential for cellulitis vs. cellulitis mimics.
2. Outline the 2014 Infectious Disease Society of American Practice guidelines for the management of cellulitis.
3. Determine the appropriate empiric antibiotics regimen (agent and route) to treat cellulitis caused by streptococci, and for cellulitis caused by MDRSOs including MRSA.
AES Question
Question 1
34yo male with redness and pain on left shin that started 36h ago.

Cellulitis?

A. Yes
B. No

https://upload.wikimedia.org/wikipedia/commons/thumb/6/60/Cellulitis3.jpg/1200px-Cellulitis3.jpg
AES Question
Question 2
34yo male with redness and severe pain on right leg that started 4h ago.

Cellulitis?

A. Yes
B. No
AES Question
Question 3
54yo female with redness and pain on left shin that started 48h ago.

Cellulitis?
A. Yes
B. No
AES Question
Question 4
59yo female with redness and pain on left leg that started 48h ago.

Cellulitis?

A. Yes
B. No
AES Question
Question 5
34yo female with redness, swelling and pain on left leg that started 24h ago.

Cellulitis?

A. Yes
B. No
AES Question
Question 6

Cellulitis?

A. Yes

B. No

https://upload.wikimedia.org/wikipedia/commons/thumb/8/8f/Orbital_cellulitis.jpg/300px-Orbital_cellulitis.jpg
AES Question
Question 7
19yo female with redness, itchiness, and mild pain on right thigh that progressively worsened over the last 36h.

Cellulitis?
A. Yes
B. No
AES Question
Question 8
47 yo female with swelling and mild achiness to her left leg that started 48h ago.

Cellulitis?
A. Yes
B. No
Cellulitis
Definition

• Non-necrotizing inflammation of the skin and subcutaneous tissues
• Does not involve the fascia or muscles
• From acute infection, usually follows a breach in the skin
  – Although a portal of entry may not be obvious
  – Breach may involve microscopic skin changes or invasive qualities of certain bacteria
Symptoms

• Associated with the 4 cardinal signs of infection:
  – Erythema
  – Pain
  – Swelling
  – Warmth

• Findings suggestive severe infection:
  – Malaise, chills, fever, and toxicity
  – Lymphangitic spread
  – Circumferential cellulitis
Bacteriology

• In individuals with normal host defenses:
  − Group A streptococci (GAS)
  − S aureus
  − Group B *Streptococcus* (infants < 6 months)
  − Impetigo: *S aureus* and/or *S pyogenes*
  − Erysipelas: streptococcal species such as *S pyogenes*
Bacteriology

- Immunocompromised hosts
  - Gram-negative rods (e.g., *Pseudomonas*, *Proteus*, *Serratia*, *Enterobacter*, *Citrobacter*)
  - Anaerobes
  - Others
    - *Helicobacter cinaedi*
    - *Fusarium* species
    - *Cryptococcus*
    - Herpes simplex virus
Bite wounds

• The infections are usually polymicrobial.
• Dog bites are the most commonly encountered bite wound
• Organisms are of particular interest:
  − *Capnocytophaga canimorsus* (dog)
  − *Eikenella corrodens* (human)
  − *Pasteurella multocida* (dog or cat)
  − *Streptobacillus moniliformis* (rat)
# Antimicrobial therapy

<table>
<thead>
<tr>
<th>Disease entity</th>
<th>Antibiotic</th>
</tr>
</thead>
<tbody>
<tr>
<td>MSSA SSTI</td>
<td>Oxacillin, Cefazolin, Clindamycin, Clindamycin, Dicloxacillin, Cephalexin, Doxycycline, TMP-SMX</td>
</tr>
<tr>
<td>MRSA SSTI</td>
<td>Vancomycin, Linezolid, Clindamycin, Daptomycin, Doxycycline, TMP-SMX</td>
</tr>
</tbody>
</table>

2014 IDSA Guideline for Skin and Soft Tissue Infections
# Antimicrobial therapy

<table>
<thead>
<tr>
<th>Disease entity</th>
<th>Antibiotic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Impetigo (Staph or Strept)</td>
<td>Dicloxacin</td>
</tr>
<tr>
<td></td>
<td>Cephalexin</td>
</tr>
<tr>
<td></td>
<td>Clindamycin</td>
</tr>
<tr>
<td></td>
<td>Amoxicillin-Clavulate</td>
</tr>
<tr>
<td></td>
<td>Mupirocin ointment</td>
</tr>
<tr>
<td></td>
<td>Retapamulin ointment</td>
</tr>
<tr>
<td>Strept skin infection</td>
<td>Penicillin G IV</td>
</tr>
<tr>
<td></td>
<td>Clindamycin IV</td>
</tr>
<tr>
<td></td>
<td>Nafcillin IV</td>
</tr>
<tr>
<td></td>
<td>Cefazolin IV</td>
</tr>
<tr>
<td></td>
<td>Penicillin VK</td>
</tr>
<tr>
<td></td>
<td>Cephalexin</td>
</tr>
</tbody>
</table>
So you think you can diagnose cellulitis?
Distinguishing cellulitis

• Can be challenging but critical if we want to avoid
  − Morbidity and mortality
  − Unnecessary antibiotic use
  − Delay in treatment
Cellulitis or not?

• More than 10% of pts labeled as having cellulitis do not have cellulitis
• Many patients admitted for the treatment of cellulitis actually have stasis dermatitis and lipodermatosclerosis
Mimicking conditions

- 169 patients were referred with the diagnosis of cellulitis
  - 23 patients (13.6%) referred for cellulitis actually had an alternative diagnosis
    - abscess requiring incision and drainage (7 patients)
    - abscess not requiring incision and drainage (6 patients)
    - herpes zoster (2 patients)
    - septic bursitis (2 patients)
    - herpetic whitlow (Herpes simplex virus)
    - gangrenous foot
    - gout
    - foot fracture
    - septic arthritis
    - tinea pedis
Cellulitis or not?

- Cellulitis are overwhelmingly unilateral, with smooth indistinct borders

Bilateral redness/warmth/pain =

Cleveland Clinic Journal of Medicine, Aug 2012, Vol 79(8): 547-552
Cellulitis or not?

• Symptoms and signs *NOT* suggestive of cellulitis
  – Bilateral or symmetrical
  – Lack of pain
  – Mainly pruritic
  – Long-standing course with acute flare
  – Progressive course
  – Non-response to appropriate antibiotic therapy
Stasis Dermatitis
Stasis Dermatitis

• No. 1 cellulitis mimic!

• Earliest cutaneous sequela of chronic venous insufficiency

• *Skin inflammation* caused by blood pooling in the veins in your legs

• Occurs in people over 50.
  − Incidence of this condition may be as high as 20% in those over age 70
  − Women > men (pregnancy-related vein changes)
Stasis Dermatitis - presentation

- Bilateral!
- Swelling in the leg
- Dull aching or heaviness in the leg
- Pain that gets worse when you stand
- Skin can become irritated
  - Red or swollen, crusted, or weepy.
Lipodermatosclerosis
Lipodermatosclerosis

- Refers to a skin change of the lower legs that often occurs in patients who have venous insufficiency
- Two-thirds of affected patients are obese

- It is a type of *Panniculitis*
  - Inflammation of subcutaneous fat
Chronic vs Acute

• May present as an acute or chronic condition
• Acute presentation
  − Generally occurs without any preceding illness or local injury
  − Episodes of painful inflammation, resembling cellulitis
Acute Lipodermatosclerosis

https://www.dermnetnz.org/topics/lipodermatosclerosis/
Acute Lipodermatosclerosis

https://www.dermnetnz.org/topics/lipodermatosclerosis/
Chronic Lipodermatosclerosis

• Chronic lipodermatosclerosis may follow an acute episode or develop gradually
• Common findings include:
  − Pain
  − Hardening of the skin
  − Localized thickening
  − Moderate redness
  − Increased pigmentation
  − Small white scarred areas (atrophie blanche)
  − Edema
  − Varicose veins
  − Leg ulcers
Chronic lipodermatosclerosis

https://www.dermnetnz.org/topics/lipodermatosclerosis/
Contact dermatitis
Contact dermatitis

- Allergic and irritant forms of contact dermatitis are often mistaken for cellulitis
- Can present with intense pruritus and pain
Lymphedema
Presentation

• Localized edema, induration, erythema
• May have chronic changes
  – Hyperkeratosis
  – Dyspigmentation
  – Wart-like architecture
Presentation

- Often presents with unilateral non-pitting edema and erythema
- Absence of systemic symptoms
- Patients are however more susceptible to superficial and deep skin infection
  - Presence of warmth
  - Increased erythema
  - Systemic symptoms (fever, malaise)
Papular Urticaria
Insect bites
Presentation

• Majority of people without sting allergies will show only minor symptoms
  – Redness, swelling, and pruritus can occur 24h later
• Large local reactions may occur in pts allergic to wasp stings, but don’t experience life-threatening symptoms
  – Can include extreme redness and edema that increases for 2-3 days after the sting
Post-bite inflammatory reactions

Pruritus > Pain
Bursitis
Uncomplicated bursitis

• Over 150 bursae (superficial vs deep) in the human body
  – Cushion and lubricate points between the bones, tendons, and muscles near the joints
• Superficial bursa can become inflamed and produce picture of cellulitis at the joint region
  – Movement or pressure is painful
Septic bursitis

• Inflammation of the bursa that is due to infection, resulting from bacterial inoculation
  – Direct (puncture wound)
  – Spread from nearby soft tissues cellulitis
  – Hematogenous (bacterial endocarditis)

• Symptoms of septic bursitis are similar to regular bursitis:
  – Pain, swelling, tenderness of the area immediately above the joint
  – May also have fever/chills or malaise
Hydrofluoric acid burn
Hydrofluoric acid (HF) burn

• HF is one of the strongest inorganic acids
  – Industrial purposes
  – Home rust removers
• Exposure is usually due to inadequate use of protective measures
Hydrofluoric acid (HF) burn

• Dilute solutions (< 7%)
  • Penetrate deeply before dissociating
  • Causes delayed (several hours) symptoms and injuries
  • More severe burn
  • Presents with severe pain without surface abnormality (POOP)

• Concentrated solutions (> 12% solution)
  • Severe superficial burns
  • Involves small areas (digits)
Deadly exposure

• Fluoride ions penetrate and form insoluble salts with calcium and magnesium
  – Depletion of total body stores of calcium and magnesium
    • Cell death
      • Cardiac arrhythmias (hypocalcemia and hyperkalemia)
Management

• Removed soiled clothing
• Decontaminate by irrigation
• Assess and manage life-threatening conditions
• Treatment
  – 2.5% Calcium Gluconate gel to the affected area
  – IV regional Calcium Gluconate (Bier block technique)
  – Intra-arterial Calcium Gluconate
Necrotizing fasciitis
What if it’s worse than cellulitis?

• Necrotizing Soft Tissue Infections
  – Fulminant Tissue Destruction
  – Systemic Toxicity
  – High Mortality (24%)
  – 1% of all Cellulitis/Abscess
Necrotizing Fasciitis: Two Bacteria Etiologies

• Type I → Polymicrobial
  − 1 Anaerobic Species
    • Bacteroides, Clostridium, Peptostreptococcus
    − Streptococcus (Facultative Anaerobe, Not Grp A)
    − Enterobactericiae
      • E. coil, Enterobacter, Klebsiella, Proteus

• Type II → Group A Streptococcus (Beta hemolytic)
  − Can include MRSA
Clinical Features

• **POOP** - Very Tender
• **Extreme rapid progression**
• Swollen
• Warm
• Shiny

3-5 days

- Red-purple → Blue-Gray
- Skin breakdown
- Bullae
- Frank gangrene
- Subcut Emphysema
- Compartment Synd
- Systemic Toxicity

**Anesthesia**
What is concerning?

• Only 57% of EPs suspected the diagnosis!

❖ Median Time to
❖ Antibiotics: 3 hrs 20 mins
❖ Median Time to Surgery: 8.4 hrs
Why did they have so much trouble?

- 15% had normal WBC
- Vast majority are just erythematous
- Only 54% had tenderness
Take-Home Message

• Early Necrotizing Fasciitis is Difficult to Diagnose by appearance alone!
  – Pain Out Of Proportion (POOP)
  – Rapid Clinical Course
Ancillary Investigations

- **Laboratory**
  - Elevated Creatine Kinase (CK)
  - Blood Cultures
  - Aspiration of Skin/Bullae for Gram Stain / Culture

- **Radiology**
  - X-Ray → Gas (formed by Clostridium)
  - CT → Gas highly specific, but often show STS
  - MRI → can overestimate involved tissue
Aggressive Tissue Infections

- Group A Streptococcus
- S. Aureus
- Pseudomonas sp.
- Vibrio Vulnificus
- Clostridium Perfringens
- Pasteurella Multocida
- Aeromonas Hydrophila
- Polymicrobial
POOP

- Necrotizing fasciitis
- Vibrio Vulnificus
- Hydrofluoric acid burn
Question 1

34yo male with redness and pain on left shin that started 36h ago.

Cellulitis?
A. Yes

Cellulitis Left shin

https://upload.wikimedia.org/wikipedia/commons/thumb/6/60/Cellulitis3.jpg/1200px-Cellulitis3.jpg
Question 2

Cellulitis?
B. No

34yo male with redness and severe pain on right leg that started 4h ago.

Necrotizing fasciitis

By Doetsch - Own work, CC BY-SA 4.0, https://commons.wikimedia.org/w/index.php?curid=39487083
54yo female with redness and pain on left shin that started 48h ago.

Stasis Dermatitis
59yo female with redness and pain on left leg that started 48h ago.

Acute Lipodermatosclerrosis

Question 4

Cellulitis?
B. No
Question 5

Cellulitis?
A. Yes

34yo female with redness, swelling and pain on left leg that started 24h ago.

Cellulitis left leg

https://upload.wikimedia.org/wikipedia/commons/5/5c/Cellulitis_Left_Leg.JPG
Question 6

Cellulitis?
A. Yes

Orbital cellulitis

https://upload.wikimedia.org/wikipedia/commons/thumb/8/8f/Orbital_cellulitis.jpg/300px-Orbital_cellulitis.jpg
19yo female with redness, itchiness, and mild pain on right thigh that progressively worsened over the last 36h.

Post-bite inflammatory soft tissue reaction
Question 8

Cellulitis?
B. No

47 yo female with swelling and mild achiness to her left leg that started 48h ago.

Lymphedema
Practice Recommendations

• Cellulitis is rarely bilateral
• Stasis dermatitis is the most common mimic of cellulitis
• Pruritus does not support cellulitis
• If it does not respond to appropriate empiric antibiotic, rethink the diagnosis (not give another antibiotic)
• Inquire about professional/industrial exposure
Answer Key

1. A
2. B
3. B
4. B
5. A
6. A
7. B
8. B
Sepsis Recognition and ER Management

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Medical Director and Chair, Medical Education HRH ED
Investigative Coroner, Province of Ontario
Faculty DFCM/EM University of Toronto and DFM Queens’ University
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Learning Objectives

1. Explain the "spectrum" of SIRS/Sepsis.

2. Point out the variations in vital signs, particularly blood pressure, heart rate, and temperature which may indicate sepsis.

3. Describe the clinical indications for sepsis in special populations (i.e. elderly, those on blood pressure medications, etc.).

4. Provide management of patients in the ER diagnosed with sepsis.
Sepsis

• An evolving definition over the years…

• Providing a mental framework from which to treat patients that may be suspicious of having ‘sepsis’ as early detection really is key

• Consensus for definitions is vital for comparison and analysis of data, research protocols and best practice interventions (treatment)

• Get ready its like a trilogy and like any other ‘blockbuster’ will keep on going with sequels!
Sepsis

• Contributed to almost 20% of all deaths/year in the world, more than 20 deaths every minute – Lancet January 2020

• By far the leading cause of death in the world!

• Very under recognized
  – Identifying sepsis early is important for initiation of life saving treatment

• Any class of microorganisms - most cases are gram +ve/-ve bacteria, can also be fungi, mycobacteria, rickettsia, viruses and protozoans (50% of time no organism identified)
On the Rise!

• Advancing age, immunosuppression drugs and drug resistance
• Up to 85% of cases are in those over the age of 65
• ?More awareness

• Sepsis is a dynamic evolving condition with clinical and laboratory manifestations that change over time not all criteria may not be present at a single time
AES Question
Question 1

Which of the following may lead to an infection causing “Sepsis”?

A. Bacteria
B. Virus
C. Fungi
D. All of the Above
Infection Defined…

• A suspected or proven (positive result on culture, tissue stain or PCR) infection due to any pathogen
• May have evidence of infection includes +ve findings on clinical examination, imaging or lab tests (WBC in sterile fluid, CXR with pneumonia etc)
“Sepsis 1” – 1991 Definition

• A clinical syndrome that has physiological, biologic and biochemical abnormalities caused by a dysregulated inflammatory response to infection (should meet SIRS criteria)*

• Thus life threatening and self damaging ultimately

• When circulatory system collapses it leads to ‘shock’

• This can also lead to multiple organ dysfunction and death
  – Respiratory failure is most common than Renal Failure
Systemic Inflammatory Response Syndrome

• ‘SIRS’
  • Defines the dysregulated response with strict criteria
  • From 1991…

• *Now critics feel too sensitive and not specific enough in identifying sepsis cases in those admitted to ICU
• Does not account for the dynamic time-course of sepsis
• SIRS parameters are arbitrarily simplified
Description of this Inflammatory Process

• Manifested in the first definition of the inflammatory process that is involved in Sepsis (Circa 1991)…**SIRS** came about

• **SIRS + An Infection (real or presumed) = SEPSIS** (“Sepsis 1” Concensus in 1991)

• **SIRS is met when 2 + of the following criteria are met:**

  1. Temperature $\geq 38.3^\circ C (100.9^\circ F)$ or $\leq 36^\circ C (96.8^\circ F)$
  2. HR $\geq 90$ (in absence of external stimulus, long term drug use or painful stimuli)
  3. RR $\geq 20$ or PaCO2 $\leq 32$ mmHg
  4. WBC $\geq 12$ or $\leq 4$ or $\geq 10\%$ Immature Band Forms
“Sepsis – 1” (Visualization of Concept)

Infection
- Bacteria, Fungus, Parasite, Virus etc

SIRS
- Trauma, Burns, Pancreatitis

SEPSIS
SIRS

• Systemic inflammatory response syndrome (SIRS) to identify those with sepsis has fallen out of favor by the new ‘Sepsis 3’ definition of ‘Sepsis’ Circa 2016

• As does not confirm dx of sepsis/infection as features of SIRS can be found in many conditions such at trauma/burns/pancreatitis/vasculitis
• Infection or real/suspected leading to the onset of SIRS
• This part of the Sepsis definition stayed under “Sepsis - 2” in the 2001 consensus
“Sepsis – 1” Continued…

• ‘Severe sepsis’ is when there is lactic acidosis, \( sBP \leq 90 \) or a \( sBP \) drop of \( \geq 40 \) mmHg of normal and complicated by **organ dysfunction**

• Shock is ‘severe sepsis’ with hypotension despite **adequate fluid resuscitation**

• Often the lactate will be up \( \geq 4 \) mmol/L (not necessarily)
“Sepsis – 1”
“Sepsis – 1 and 2”

• Must start with some sort of **infection/bacteremia** which may progress to sepsis eventually

• Sepsis thought of as a spectrum which ranges as follows:

• Infection  Bacteremia  Sepsis/Severe Septic Shock  MODS  Death
Septic Shock – Added in “Sepsis – 2” Was the Key Addition

• A vasodilatory / distributive shock
• Sepsis that has circulatory, cellular and metabolic abnormalities associated with a greater risk of mortality than sepsis alone
• sBP < 90 mmHg or decrease in sBP of > 40 mmHg from baseline in absence of other causes of hypotension
• Often despite adequate fluid resuscitation will need vasopressors for a MAP of more than 65 mmHg
Shock

- Do not forget! Can be for many reasons not only sepsis
- Cardiogenic, Hypovolemic, Anaphylactic, Neurogenic, Obstructive (Pneumothorax/Tamponade) and Endocrine (Adrenal Insufficiency)
AES Question
Question 2

According to the “Sepsis – 1 and 2” definition you must have an Infection + SIRS (2+)…which of the following is not part of the criteria...

A. HR ≥ 90
B. Temperature of ≤ 36C (96.8F)
C. Lactate ≥ 2 mmol/L
D. WBC ≥ 12 or ≤ 4 or ≥ 10% Immature Band Forms
2016 SSCM/ESICM – “Where Sepsis – 3” Started

• Compared SIRS criteria to other methods including SOFA (sequential organ failure assessment) to assess severity of organ dysfunction in a potentially septic patient

• Redefined clinical criteria for identifying genuine sepsis cases and septic shock

• Mortality predictive values were compared
  • SOFA (> 10% will die if meet criteria) superior for in hospital mortality prediction vs SIRS

• The Congress then created a quick modifier qSOFA
SOFA comes along…

• Sequential organ failure assessment score also known as ‘Sepsis Related Organ Failure Assessment score’

• Organ dysfunction is defined as an increase in 2+ of the sequential (sepsis related) organ failure assessment (SOFA) score

• Scale of 0-4

• Score based on six different categories, one each for the respiratory, cardiovascular, hepatic, renal, neurological and coagulation (platelets) systems

• Greater the score = higher risk of death in ICU
SOFA vs SIRS

• Are both essentially predictive scoring methods used to predict mortality

• SOFA is superior to the SIRS criteria in predicting death from sepsis (SOFA does not define sepsis) in the ICU (hospital) setting via organ dysfunction
qSOFA – came out of the 2016 SCCM/ESICM

- Introduced “Sepsis-3” definition in February 2016 (GROUNDBREAKING)
- Simplified version of the SOFA Score as an initial way to identify patients at high risk for poor outcome with an infection = qSOFA
- Only 3 clinical criteria
- Parameters group felt more common in infected patients who may be septic (OUTSIDE ICU) than those with uncomplicated infection
- Serial calculations
- qSOFA has poor sensitivity but good specificity for risk of death
- ? Too late
- ? SIRS better screening tool
qSOFA

• Any score ≥ than 2 = poor outcome for patients outside an ICU environment
• +ve qSOFA score may be due to causes other than Sepsis
• Thus if ≥ 2 then should investigate for organ dysfunction and if possible infection
• Has 3 components:
  − 1. RR ≥ 22
  − 2. Altered Mentation (GCS < 15) and
  − 3. sBP ≤ 100 mmHg
qSOFA continued and 2016 SCCM/ESICM changes…

- For qSOFA complaint is that it may be ‘late in predicting sepsis’
- Other trials have stated SIRS criteria not ideal marker for sepsis (Kaukonen et al) (12% found to have sepsis were SIRS –ve)
- SIRS also present in those hospitalized with no infections or adverse outcomes
- ‘Severe sepsis’ (sepsis with tissue hypoperfusion – lactate, oliguria or organ dysfunction with increased Cr/Coagulopathy) and SIRS are no longer used as the new definitions of sepsis/shock includes tissue hypoperfusion/organ dysfunction
qSOFA

• Eliminated concept of sepsis without organ dysfunction

• Center for Medicare and Medicaid Services (CMS) still supports the SIRS criteria as well as Infectious Disease Society of America (IDSA) as it allows for less overtreatment as more specific
“Sepsis – 3” 2016 Definition Recap

• 2016 International Sepsis Consensus Conference
• From JAMA showed a radical departure from Sepsis 1 (1991), Sepsis 2 (2001) and SSC (2015)
• Felt SIRS criteria has ‘inadequate specificity and sensitivity’
• “Life threatening organ dysfunction due to a dysregulated host response to infection (confirmed or suspected)”
• Abandoned SIRS criteria and eliminated ‘severe sepsis’
• Used SOFA or qSOFA to define organ dysfunction
• Favoured modified SOFA called qSOFA to specifically identify those potentially at risk from dying from sepsis
AES Question
Question 3

The “Sepsis – 3” 2016 Definition led to all of the following EXCEPT:

A. The development of the qSOFA criteria to discern extent of organ dysfunction and thus predict mortality in cases were “sepsis” is proven/suspected
B. The promotion and inclusion of the traditional SIRS criteria in helping to define “sepsis”
C. Removal of the concept of “severe sepsis”
D. Advocating usage of SOFA criteria
Sepsis – 3 Recap

• ‘Septic shock’ very precise definition = persistent hypotension needing vasopressors to maintain a MAP of > 65 mmHg and have a serum lactate > 2 mmol/L despite adequate volume resuscitation
• Got rid of term ‘severe sepsis’ thought to be redundant
• As feel all sepsis has organ dysfunction (can not exist without)
• Disputes the “SIRS + Infection” continuum of 1991…
• Accepted by Society of Critical Care Medicine and others…BUT…the JURY IS OUT STILL!
• Practically a lot of inconsistencies particularly for ICD-10 CM coding
• Does it truly help in the imperative early dx and tx?
Recap...

• Sepsis 1 and 2: Sepsis = Suspected/Confirmed Infection + SIRS

• Sepsis 3: Sepsis = Suspected/Confirmed Infection + qSOFA + SOFA

• qSOFA + SOFA are mortality indicators!!
Please give me something, I want to help my patient...what is Sepsis!!!!...

• Bottom line for front line UC/ER physicians is...keep an open eye out for ‘Sepsis’ when you have concerns about an INFECTION causing physiological havoc on the patient that is supported by deranged clinical and biochemical parameters

• Better to be in tune and early than late!
• Clinical syndrome at the core and at bedside in real time!
• SIRS criteria is helpful to a large extent as well as qSOFA criteria so use both until guidelines evolve
Sepsis Risk Factors

- ICU admission
- Bacteremia (positive blood cultures / MRSA is worse)
- Advanced age (greater than 65) + comorbidities/reserve
- Immunosuppression
- DM/Obesity
- Nosocomial infection have a greater mortality
- Cancer
- Pneumonia (greatest site leading to SEPSIS)
- Hospitalization within 3 months
- Site of infection (UTI’s is the least one leading to SEPSIS)
Mortality

• Very high up to 50%
• With rates increasing based on severity
• SIRS (7%), Sepsis (16%) and Septic Shock (46%)
• Severity of host’s inflammatory response may worsen situation
• Do sepsis ‘bundle’ paths help?...data is not overly convincing
• If admitted into ICU have a higher change to get another subsequent infection (likely confounding)
• 10% often readmitted down the road (CHF, COPD, Recurrent UTIs, C.Difficile)
• Higher over mortality (?Co-morbidities)
Sepsis Clinical Presentation

• Often have **hypotension, tachycardia, fever** (can also be hypothermic), **mental status changes** (confusion, lethargy, agitation, obtunded and comatose)

• With **increasing severity signs of shock** will be evident (cool and clammy skin or cyanosis) and **evidence of organ dysfunction** (such as oliguria/kidney injury)

• Non-specific as other conditions may present as such i.e. pancreatitis and ARDS

• Watch out for age related (neonates) and co-morbid subtleties such as i.e. seniors and immunocompromised that may not mount fever (often hypothermic) or localized source
AES Question
Question 4

Which is not a common clinical sign of sepsis?

A. Altered mental status
B. Only an elevated temperature of > 38.3°C
C. Increased respiratory rate
D. Often will be hypotensive
Clinical Presentation Continued…

• Symptoms and signs of sepsis:
  − Symptoms correlating with an infection source
  − Systolic BP (sBP) < 90mmHg, MAP < 70 or a change in sBP > 40mmHg (may need arterial line for accurate reading)
  − Temp > 38.3°C or < 36°C
  − HR > 90 / min
  − RR > 20 / min (Tachypnea)
  − Signs of end organ perfusion decrease such as cyanosis/mottling, altered mental status/restlessness and or oliguria/anuria
  − Ileus/absent bowel sounds

− Caveat is that these may be modified by pre-existing diseases or medications
Cardiovascular

• Early on will compensate with increased HR (cardiac output and stroke volume are well maintained)
• Decreased peripheral vascular resistance but warm extremities

• Those on Beta-blockers, Calcium Channel Blockers etc may not have an increased HR or if poorly controlled HTN may be ‘relatively hypotensive’!
Respiratory

• Increased RR / Hyperventilation

• But may compensate longer in those with COPD ‘until crash’ as rely on baseline ‘hypoxic drive’ from chronic hypercarbia

• Acute Respiratory Distress Syndrome often develops due to lung edema from alveolar ‘flooding’ and manifested by bilateral diffuse infiltrates on CXR in the absence of pneumonia/CHF
Renal/Hepatic Failure/Endocrine

• Oliguria and Cholestatic Jaundice (from Transaminitis, ALP elevation and hyperbilirubinemia and RBC hemolysis)

• Hyperglycemia due to catecholamine surge and hypoglycemia (rare but common for respiratory ailment related organisms)
Hematologic Abnormalities

• Neutropenia, Thrombocytopenia and the end result of DIC

• BADNESS!!!
Focus on Systems…

- CNS (Meningitis)
- Respiratory (Pneumonia/ILI)
- Intraabdominal (Abscess/Perforated Viscous such As Ruptured Appendix/Tubo ovarian Abscess/Cholangitis/Urosepsis)
- Cellulitis/Necrotizing Fasciitis
- Bones (Osteomyelitis)
What to order...

- CBC, electrolytes, LFT’s, coags, lactate, vbg/abg
- Blood cultures from two sites (aerobic/anaerobic)
- U/A and Urine cultures
- Foley for output measuring
- Cultures from suspected sources (sputum, urine, IV catheter site, wound site or surgical site) do not draw from PICC/CVC site as ports are often colonized with skin flora = false +ves
- Pre antibiotics 31.4% +ve vs. post 19.4%
- Adjunct imaging if relevant to source of infection (often at least CXR)
Laboratory Evidence

- These are really non-specific
- May be associated with abnormalities due to an underlying cause of sepsis or due to tissue hypo-perfusion/organ dysfunction from sepsis

**LACTATE LEVELS ARE KEY FINDING** (> 2 mmol/L)
- WBC > 12 (Leukocytosis) or < 4 (Leukopenia)
- Hyperglycemia in absence of DM ( >140 mg/dL)
- Acute oliguria (urine output < 0.5ml/kg/hour for at least two hours despite adequate fluids)
- Creatinine increase acutely from baseline > micromol/L
- Coagulation abnormalities
- Low platelets (acute)
- Hyperbilirubinemia
- Adrenal insufficiency (low K+/Na+)
CASE: 66YO Male comes to the ER with a diabetic foot ulcer that is oozing along with a cough for two days and a headache...

- His vitals are:
  - HR: 110
  - BP: 95/70
  - Temperature: 40.1°C
  - RR: 24
  - Oxygen Saturation on Room Air: 94%
  - PMHx: Diabetes and COPD

**What are you going to do?**
Early Goal Directed Therapy (EGDT) first 6 Hours

- Famous 2001 Rivers et al Study…early goal directed therapy
- For those with severe sepsis and shock:
  - Mortality was 30% vs 46% not in the EGDT group

- CVP 8-12 mmHg
- MAP $\geq 65 \leq 90$
- ScvO2 $\geq 70$
- Urine Output $\geq 0.5$ cc/Kg/hour
Rivers…EGDT Critiques…

• Staff not blinded to treatment group
• Impossible to determine which interventions were most vital
• The use of ScvO2 and pressure monitoring had not been tested in this population prior and is a continued source of controversy
• The study was single-center
• The control group had an above-average mortality
• Transfusion red blood cells to increase ScvO₂ is controversial as it has been associated with increased mortality in the critically ill
• Equipment provided by Edward Lifesciences and Nova Biomedical
EGDT

- Recent large RCT’s (ProCESS, ARISE, and ProMISe) did not demonstrate a 90-day mortality benefit of early goal directed therapy when compared to standard therapy in severe sepsis.
- But these patients had already been given tonnes of fluids and were not as sick as the patients from the Rivers trial.
- Septic bundles in and of themselves does not lead to any tangible mortality benefits (no consensus on this).
Treatment (Early Goal Directed Therapy)

• What came out of it…

• 1. Optimize oxygen, ventilation and circulation (EDT)
• 2. Start drugs particularly IVF and abx then pressors for MAP > 65 mmHg (To correct low BP and perfusion) via large bore IV/IO then CVC
• 3. Source control

• This was incorporated into the “Surviving Sepsis Campaigns”
Airway Securing as part of Treatment

• Secure airway if needed (usual indications – increased WOB, impending respiratory failure, altered mental status and unable to protect airway)
• May need intubation/mechanical ventilation
• Supplemental oxygen
Bottom Line Treatment

• Established venous access (not necessarily central) but large bore IV’s peripherally as soon as you can...later a central line will have to be placed (CVC) particularly if need for ‘pressors’
• DO NOT delay resuscitation due to a CVC
• **Fluids, fluids, fluids AND Antibiotics** (EMPIRIC based on site or condition)
  - Fluids are crystalloids (normal saline) @ 30cc/kg within first three hours preferably 1.5 hours (or colloids)
  - Rapid infusion
  - Up to 3-5 litres easily (watch out for pulmonary edema)
  - May need to use ‘pressors’ post adequate fluid administration
Treatment

• Surviving Sepsis Campaign 2018 has come up with the ‘1 hour bundle’ from triage and thus want treatment right away

• Best is to keep ‘eyes’ out and have a high index, evaluate early and then continually re-evaluate

• Treatment started early
Fluids, Fluids, Fluids…

- Crystalloids such as Normal Saline or Ringers’ Lactate is fine
- Do not need anything else (no benefit of albumin)
- No role for hypertonic saline

- Over the first 6 hours of hypotension each hour of delay = mean survival decrease of 7.6%-10%
- Whereas survival is near 30% if tx in first 30 minutes of BP drop
Antibiotics

• Antibiotics have a significant impact when administered early as associated with a 50% REDUCTION in mortality particularly if susceptible
• Within first hour that is targeted at site/organism that is suspected
• Greater the delay leads to linear increase in mortality
• If recent abx usage resistance is higher and thus less effective
Antibiotics

• Empiric antibiotics directed towards the site suspected
• Closed space infections such as abscess/empyema should be drained or debrided ASAP
• In shock (especially gram negative, should get combo tx with at least two abx from two different classes)
AES Question
Question 5

The **two most vital components** of treatment for Sepsis are:

A. Vasopressors and placement of a Central Venous Catheter (CVC) "Central Line"
B. Crystalloid Fluids and Transfusion of Red Bloods Cells
C. Acetaminophen and Colloid Fluids
D. Crystalloid or Colloid Fluids and Empiric Antibiotics
Antibiotic Choice

• Tailored to patients history (recent usage) and co-morbidities (diabetes/immunosupression), suspected site, gram stain data, local prevalence and resistance patterns
• Direct empirically wide spectrum for gram +ves/-ves bacteria, fungi and viral (if indicated)
• Typical ones are **Carbapenems/Piperacillin-Tazobactam**
• Can use combination of drugs from two different classes depending on presentation
• Agent chosen based on ‘common’ pathogens
• Such as Klebsiella, E. Coli, S. Aureus, S. Pneumoniae
• If MRSA suspected should add **Vancomycin**
Antibiotics Cont’d…

• Maximum dosing with antibiotics
• Try to find source out ideally within 12 hours thus may need imaging (CT/US) or samples from blood cultures/lavage/aspiration
• Remove any concerning sources such as port-a-cath/foley/percutaneous drain
Antibiotics (Adults-Not Neutropenic)

• If IV drug use then may have MRSA thus add Vancomycin

• Urinary Tract
  – Likely aerobic gram –ve bacilli/enterococcus
  – Levofloxacin 750mg IV q24H or Ceftriaxone 1-2g IV q12-24H

• CNS (Meningitis)
  – Ceftriaxone 2g IV q12H

• Pneumonia
  – Ceftriaxone 1-2g IV q12H + Azithromycin 500mg IV q24H if MRSA add Vancomycin 1g IV q12H

• Biliary Source
  – Aerobic gram –ve bacilli, enterococci
  – Use Pip/Tazo 4.5g IV q6H

• No obvious source of infection
  – Potential Organisms: Gram -ve bacilli, S.Aureus, Strep
  – Use Meropenem 1g IV q8H
If Neutropenic

• Formulary varies but often:

• Ceftazidime 2g IV q8H OR Meropenem 1g IV q8H or Pip/Tazo 4.5g IV q6H
• May add Vancomycin as needed (1g IV q12H)
Antibiotic Choice

• If Pseudomonas is suspected add two of the following with Vancomycin:
  − Antipseudomonal Cephalosporin such as Ceftazidime
  − Antipseudomonal Beta-lactam inhibitor such as Piperacillin/Tazobactam
  − Fluoroquinolone with good anti-pseudomonal activity such as Ciprofloxacin
  − Aminoglycoside such as Gentamycin

− Fungal infections rare and often not in an acute situation but if candida/aspergillus or neutropenic – voriconazole is often appropriate
Vasopressors (“Pressors”)

- If persistently low BP despite adequate fluid resuscitation/antibiotics need to evaluate source again (could have other consequences)
- **Norepinephrine** (NE) is the first line @ 2.5-20 mcg/kg/min and once BP stabilized reduce amount in order to minimize vasoconstriction
- May add **vasopressin 0.03 units/min** (if high HR), **epinephrine** (0.5 mcg/kg/min) and **dopamine** (2-10 mcg/kg/min) (if low HR) as adjuncts
- Phenylephrine for shorter boluses
- CVC for longer administration and if high doses or multiple pressors
Adjuncts

• No role for imaging unless needing it to localize source of an infection
• Also do not need +ve cultures (such as in endocarditis) to confirm Sepsis although can be supportive
• **50% never have +ve cultures!!**
• Try to give abx AFTER cultures are drawn
• Often choice for abx can be evident from the bedside
Signs of Improvement

- Mentation
- BP
- RR
- HR
- Skin Perfusion
- Urine Output
De-escalate

• Reduce all interventions if improvements are noted
• Including narrowing of antibiotics choice i.e. vancomycin if no MRSA isolated from cultures
• Decreasing fluid rate
• Decreasing or discontinue vasopressors
Controversy

• Steroids bolus in refractory cases
  – Back and forth ‘acceptance’
  – Promising evidence in the ADRENAL trial that shows in those patients with septic shock whom are intubated have no mortality benefit but ICU stay decreases along with period of shock
  – Bottom line consider IV hydrocortisone if BP is refractory to fluids/pressors

• RBC’s if very anemic (<70 g/L)
• Glucose levels regulate
• Etomidate is safe
• ?Protein C
• ECMO
Admission

• Ward or ICU
• All depends on whether on mechanical ventilation / “pressors”
• If go to floor need to close monitoring of clinical parameters…especially 6-24 hours
• Can monitor via CVC / arterial line
• CVC if large volumes of fluids/pressors/poor access or monitor CVP
• MAP > 65 mmHg is the best way, UO (0.5cc/kg/hr), HR, RR, O2 and Mental Status
• Serial lactate values generally not helpful to show reperfusion of tissues
Practice Recommendations

• Recognize Sepsis (in any way you can – suggest SIRS/qSOFA)
• Resuscitations with crystalloids (30cc/kg) or colloids (60cc/kg) until perfusion improves
• Antibiotics
• Vasopressors if refractory shock (multiples if needed)
• Steroids if refractory shock
• Keep going DON’T STOP
• Remove any potential source of infection (indwelling catheter/abscess)
• ?ECMO
Practice Recommendations / Blood Cultures

• A note on BC
• No gold standard to interpret or manage +ve cultures but SHOULD NEVER be IGNORED
• Must have knowledge of typical culprits, patients medical hx, immune status, illness at time of draw and condition currently
• Every pathogen should be considered DEADLY until patient re-evaluated and reassessed
• Vital to have 2 draws as may have contaminant and 1 draw can not definitively rule out
Parting Thoughts

• CONCLUDING REMARKS
  • The clinical utility of the new Sepsis 3 definition remains to be seen
  • The lack of a reliable definition of sepsis makes assessment of incidence and changes in outcomes difficult to quantify reliably
  • Value in a screening test for sepsis (highly sensitive) and a confirmatory test (highly specific) rather than ‘all or none’ approach with data
  • Ideally definitions should be useful to both clinicians and researchers
  • Consider sepsis as a possible cause of new organ dysfunction
References


• https://litfl.com/sepsis-overview/ ChrisNickson

• The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3)
  • Mervyn Singer, MD, FRCP; JAMA. 2016;315(8):801-810. doi:10.1001/jama.2016.0287
Answer Key

1. D
2. C
3. B
4. B
5. D
Dizziness and Vertigo

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University of Massachusetts Medical School
Executive Editor, DynaMed
Disclosure

It is the policy of the AAFP that all individuals in a position to control content disclose any relationships with commercial interests upon nomination/invitation of participation. Disclosure documents are reviewed for potential conflicts of interest and, if identified, conflicts are resolved prior to confirmation of participation. Only those participants who had no conflict of interest or who agreed to an identified resolution process prior to their participation were involved in this CME session.

• Vu Kiet Tran, MD, MBA has disclosed a relationship with Elvium on the topic “Acute Pain Management”.

All individuals in a position to control content for this session have indicated they have no relevant financial relationships to disclose.
Learning Objectives

1. Perform a differential diagnosis for dizziness and vertigo to distinguish benign from serious causes requiring urgent evaluation and treatment.
2. Demonstrate physical examination techniques used to determine etiology of dizziness/vertigo, including the use of the HINTS examination, and the Dix-Hallpike maneuver.
3. Perform the Epley maneuver and provider patient education on use of this treatment for symptom relief.
4. Prepare treatment/referral plans for patients diagnosed with BPPH, vestibular neuritis, Meniere disease, vestibular migraine, and vertebrobasilar ischemia.
Your next patient is a 78 year old woman with mild dementia brought in by her daughter because of dizziness.

- They were just getting up from lunch when the older woman complains of dizziness
- No fall but she had to sit down
- Feels better now, but still does not feel quite right

Where do you begin?
Dizzy: A Non-specific Term

"What do you mean by dizzy without using the word dizzy?"
Traditional Classification

- Vertigo
- Presyncope
- Disequilibrium
- Other (Lightheadedness)

- Based on 125 patients at dizziness clinic
- Patients all fluent in English
- Evaluated over 4 half day
- No independent confirmation of Dx
- No brain imaging done

Drachman DA, Hart CW Neurology. 1972 Apr;22(4):323-34
Vertigo

• Hallucination of movement of self or environment
• Associated symptoms
  • Nausea
  • Vomiting
  • Unstable gait
• May be due to peripheral or central causes
  • Vestibular disease
  • CNS structural changes

Image courtesy of Corey Coyle
https://commons.wikimedia.org/wiki/File:Round_Up_-_panoramio_(1).jpg
DDX of Vertigo

Peripheral

• BPPV
• Vestibular Neuritis
• Meniere's disease
• Perilymph leak
• Drugs (ototoxic)
• Otosclerosis

Central

• Migraine
• TIA/Stroke
• MS
• Cerebellopontine angle and posterior fossa tumors (including vestibular schwannoma)
• Drugs (Ethanol, phenytoin, etc.)
• Neurodegenerative (Parkinson, MSA)
Presyncope

- Pallor, diaphoresis, roaring in ears, graying of vision
- Generally diffuse decrease in cerebral blood flow
- No nystagmus
Imbalance/Disequilibrium

- Sensation of loss of balance
- Generally loss of motor control (stroke patients falling to one side)
- Abnormal gait
- No nystagmus

- Cerebellar disease
- Multiple sensory deficits
- Peripheral neuropathy
- Low pressure hydrocephalus
- Parkinson’s
- CVA
Lightheadedness – Just As Clear As Dizziness

• Vague symptoms not easily put into other categories
• Patients may report feeling disconnected from their environment
• No nystagmus
• May be due to psychiatric causes – anxiety, depression
• May be due to multiple sensory deficits
Problems With Traditional Methods

• Patients often have difficulty describing their symptoms
• Patients may give conflicting accounts at different times

• Symptom quality does not reliably predict the cause of dizziness
A New Approach Needed

Time for Change
Key History

- Any concern for general medical problem
  - Fever or other signs of infection
  - Chest pain or dyspnea
  - Abdominal, back or flank pain (aortic vascular issues)
  - Trauma
- Triggers such as change with motion/position
- Timing
- Medications
- Underlying/recent illnesses such as influenza, gastroenteritis, cardiac diseases
- Drug use
Targeted Physical Exam

• Check for hypovolemia, arrhythmias, otitis, etc.

• Look for CNS abnormalities
  – Past pointing (dysmetria)
  – Truncal ataxia with sitting up
  – Assess for nystagmus
  – Romberg (assesses for ataxia, dorsal column dysfunction, peripheral neuropathy)
  – Gait (the more abnormal, the greater risk of CVA)

• For AVS, perform more detailed neuro exam including
  – Motor and sensory exam
  – Cranial nerves
  – Cerebellar function
  – Visual fields (supplied by posterior cerebral artery)

• Perform more extensive neuro and ENT exam when features are atypical for common syndromes
TITRATE (ATTEST)

- **Timing** - onset, duration evolution
  - Acute vs. chronic
  - Continuous vs. episodic
- **Triggers** - actions, motions, situations
  - Distinguish exacerbation from triggering
  - Most dizziness gets worse with movement
- **And Targeted Examination**
  - Physical examination usually normal
  - Check orthostatics
  - Observe gait, check Romberg, Dix-Hallpike
- **Associated symptoms**
- **Timing**
- **Triggers**
- **Exam Signs**
- **Testing**
Acute Vestibular Syndrome

- Dizziness is persistent and present at time of exam
  - Lasts hours to days
  - Present even when lying down
- Symptoms may wax and wane but do not resolve
- Associated symptoms:
  - Nystagmus
  - Nausea or vomiting
  - Gait instability
  - Head motion intolerance
- Movement may \textbf{exacerbate} dizziness but does not \textbf{trigger} it

### 4 Categories

<table>
<thead>
<tr>
<th>Triggered episodic vertigo syndrome (t-EVS)</th>
<th>Spontaneous episodic vertigo syndrome (s-EVS)</th>
<th>Postexposure acute vestibular syndrome (t-AVS)</th>
<th>Spontaneous acute vestibular syndrome (s-AVS)</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Lasts seconds to minutes</td>
<td>• Lasts seconds to days</td>
<td>• Direct trauma or toxic exposure</td>
<td>• Perform HINTS to distinguish peripheral (vestibular neuronitis) from central (CVA) causes</td>
</tr>
<tr>
<td>• Triggered by specific actions</td>
<td>• Cannot be provoked at bedside</td>
<td>• Usually obvious by history</td>
<td>• Rare causes include:</td>
</tr>
<tr>
<td>• Check Dix-Hallpike</td>
<td>• Common causes:</td>
<td>• Common causes:</td>
<td>• Wernicke encephalopathy</td>
</tr>
<tr>
<td>• Common causes:</td>
<td>• Vestibular migraines</td>
<td>• Blunt head trauma</td>
<td>• MS</td>
</tr>
<tr>
<td>• BPPV</td>
<td>• Other causes:</td>
<td>• Drug intoxication</td>
<td>• Cerebellar hemorrhage</td>
</tr>
<tr>
<td>• Orthostatic hypotension</td>
<td>• Meniere disease</td>
<td></td>
<td></td>
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<tr>
<td>• Central paroxysmal positional vertigo (from CVA)</td>
<td>• Panic attack</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• TIA/stroke</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
AES Question
Question 1
The Dix-Hallpike is only positive in BPPV

A. True
B. False
C. No idea
What is Dix-Hallpike and how do you interpret it?
• With the patient sitting on the examination table, facing forward, eyes open, turn the patient's head 45 degrees to the right
• Support the patient's head as the patient lies back quickly ending with the head 20 degrees off the end of the examination table.
• The patient remains in this position for 30 seconds; then the patient returns to the upright position and is observed for 30 seconds
• Repeat with the patient's head turned to the left
• Positive test if any vertigo, with or without nystagmus
### Positive Dix-Hallpike: Peripheral vs. Central

<table>
<thead>
<tr>
<th>Feature</th>
<th>Peripheral</th>
<th>Central</th>
</tr>
</thead>
<tbody>
<tr>
<td>Latency to onset of nystagmus</td>
<td>2-30 sec</td>
<td>None</td>
</tr>
<tr>
<td>Duration</td>
<td>5-90 sec</td>
<td>Persists</td>
</tr>
<tr>
<td>Fatigues on repetition</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Suppression</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Changes with fixation</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Direction of nystagmus</td>
<td>One direction/head position</td>
<td>May change</td>
</tr>
<tr>
<td>Causes</td>
<td>BPPV</td>
<td>CPPV: Intoxication Tumors/CVA</td>
</tr>
</tbody>
</table>
A mistaken belief about what the Dix-Hallpike will show you

- Sensitivity 75%, specificity 79%

- Do not use with AVS: All Vertigo May Get Worse With Head Movement

- Exacerbation of symptoms does not aid in distinguishing causes of vertigo, only triggering does
Head Impulse-Nystagmus-Test of Skew (HINTS)
HINTS Exam

• Only useful in patients with acute vestibular syndrome (continuous dizziness, usually > 24 hours)
• Used to distinguish central problem (CVA) from peripheral (Vestibular neuronitis)
• 95% of patients with AVS have one or the other
HINTS

• 3 oculomotor signs distinguish peripheral from central dysfunction
• Normal vestibuloocular reflex on head impulse test, direction-changing nystagmus, and skew deviation are all characteristic of stroke
  – In acutely dizzy patients (< 48 hours), the presence of any of these findings increased the probability of stroke (LR = 10.8)
  – Absence of all three findings markedly decreased the probability of stroke (LR = 0.02)
  – This LR (0.02) is smaller than the LR for a normal (diffusion-weighted) magnetic resonance image (MRI) (LR = 0.2)
Nystagmus

- Direction named for fast component
  - Start with neutral gaze
  - Then patient should follow examiner's finger as it moves slowly left to right
- Spontaneous unidirectional horizontal nystagmus that worsens when gazing in the direction of the nystagmus suggests a peripheral cause (vestibular neuritis)
- Spontaneous nystagmus that is dominantly vertical or torsional, or that changes direction with the gaze (gaze-evoked bidirectional) suggests a central etiology (stroke)
- Absence of nystagmus makes vestibular neuritis unlikely
Test of Skew

- Patient looks straight ahead
- Cover and uncover each eye
- Vertical deviation of the covered eye after uncovering is abnormal
- Less sensitive for central etiology, but abnormal result is fairly specific for brainstem involvement
Head Impulse Testing

• Grasp the patient’s head with hands over the ears
• Tell patient to focus on your nose with eyes open
• Move the head back and forth through a 10-15º arc on either side
• Rapidly snap the head to the midline
• A lag of the eyes follow by saccade indicates peripheral disease

• Report as saccade present or absent, not positive or negative
Head Impulse Testing

Note a compensation lag when rapidly turning head to patient’s left

Thanks to John Peters for permission to use this video
https://www.youtube.com/watch?v=Wh2oJgbC3I
Carol

Carol is a 66 year old retired florist who comes in complaining of episodic dizzy spells. She feels OK now. She reports feeling unsteady on her feet and the things seem to be moving when this occurs. The episodes last about 5-10 minutes. She cannot recall anything that seems to trigger the spells. She usually will just sit down and rest when this happens and things get better. She reports the first episode about a month ago and she has had 2 in the past week.

She has a history of migraines but she has not had a migraine headache with the spells. Other medical problems include Crohn’s disease and osteoporosis. No current medications. She is not orthostatic and Dix-Hallpike is negative. Normal neuro exam.
AES Question
Question 2
Which of the following is the most likely explanation for his dizziness?

A. Vestibular neuronitis
B. CVA
C. Orthostatic hypotension
D. BPPV
Vestibular Migraine

• Diagnosis requires:
  - ≥ 5 attacks with vestibular symptoms
  - History of migraine headaches
  - Migraine-like symptoms with at least half the attacks (aura, photophobia/phonophobia, pulsing unilateral quality)

• Duration ranges from seconds to days
• Headache is often absent
• When headache present, it may begin before, during, or after the dizziness and may differ from the patient’s other typical migraine headaches
• Nystagmus may be present but often absent

Cerebrovascular Disease

• Posterior circulation TIA
  - Isolated dizziness and vertigo are the most common symptoms
  - May also have CN issues of visual field cuts
  - More frequent in the days to weeks preceding posterior circulation stroke
  - Normal exam in between episodes

• With CVA, almost always other symptoms present and more likely to have abrupt onset of AVS

• If headache and/or neck pain present, think cervical dissection!

Maria

52 year old Hispanic woman, who works as a medical assistant comes in complaining of feeling dizzy. She indicates the feeling comes and goes. Sometimes it seems like it happens with changing positions but not always. It has been present for 2 days. She has not fallen down. She denies hearing loss or tinnitus. No head injury. She has type 1 diabetes and has an insulin pump. She has been well lately with no recent URI.

When you ask her to describe her symptoms without using the word “dizzy” she says “I don’t know, it’s kind of a woozy feeling”. When asked if the room seems to spin or move she says “maybe”
AES Question
Question 3
Which of the following is the most likely explanation for her dizziness?

A. Vestibular neuritis
B. Orthostatic hypotension
C. Meniere disease
D. Posterior circulation CVA
Causes of Hypotension

- Orthostatic changes
  - Hypovolemia
  - Bleeding
  - Medications
- Vasovagal episodes
- Arrhythmias
- Cardiac, cardiac, cardiac

- Postprandial (in elderly)
  - 499 nursing home residents
  - Mean decrease in BP after meals 15 mm Hg
  - 24% had decreases ≥ 20 mm Hg
- Consider hypoglycemia, abnormal lytes in DDX

AES Question
Question 4
Her VS do not show any significant changes. The test most likely to be helpful in establishing a diagnosis is:

A. HINTS testing
B. Dix-Hallpike maneuver
C. Romberg test
D. MRI
Benign Positional Vertigo

- Fairly common
- Generally occurs with rapid movements of head, nausea, vomiting; lasts seconds to minutes.
- Free of vertigo when head is sitting still.
- Dx by Dix-Hallpike maneuver + HX
- Treat with Epley maneuvers
- Medications ineffective in treatment of BPV
John

John is a 62-year-old musician who comes in complaining of dizziness for 2 days. The dizziness is there all the time. He has intermittent nausea and he has vomited 3 times. He had been sick last week and thought he was getting better but since this started he has stayed in bed or on the cough most of the time. He feels unsteady with walking. No change in hearing.

Exam is normal except for head impulse testing which shows a lag with turning to the right. There is no nystagmus.
AES Question
Question 5
Which of the following is the most likely diagnosis

A. Vestibular neuronitis
B. Vestibular migraine
C. Orthostatic hypotension
D. Posterior circulation TIA
Vestibular Neuritis (Vestibular neuronitis)

- Spontaneous vertigo
- Single episodes may last for days
- May follow URI infections
- Commonly affects persons aged 30-50 years
- Nausea, vomiting, instability of gait, but no hearing loss
- May look ill with diaphoresis, nystagmus (70% fine horizontal or rotatory)
- Symptoms improve over a couple of days to weeks
Approach to Dizziness

Does H&P suggest general medical problem?

Yes

Check for:
- Central nystagmus pattern
- Past pointing
- Truncal ataxia with sitting up

No

Treat presumptive diagnosis

Episodic or Continuous and persistent

Episodic

Can dizziness be triggered or provoked in the office?

Triggered

Use PE to distinguish BPPV vs. CPPV vs Orthostatic hypotension

Not triggered

Use history to distinguish Vestibular migraine vs. TIA vs Vasovagal

Continuous

Acute vestibular syndrome

Use HINTS, focused exam, and gait to distinguish Vestibular neuritis vs CVA

Labs

- Most patients with dizziness do not require lab testing
- Blood sugar is the simplest test to check
- Other tests to consider based on history and exam
  - CBC, EKG, electrolytes, BUN, creatinine
  - Holter Monitor
  - Imaging
Imaging
Question 6
Which of the following is true?

A. Up to 20% of patients with *isolated* vertigo have a stroke (proper age, of course)
B. 30% of strokes in the posterior fossa have normal initial DWI-MRIs
C. Ischemic stroke often presents with a seizure.
D. Anyone over 80 presenting with dizziness needs an MRI to rule out stroke
Who needs an MRI?

• 50% of stroke patients have dizziness as a symptom
• 3% of dizzy patients have a stroke
• <1% of patients with a stroke have isolated dizziness
• Up to 25% of patients with AVS may have a CVA

• Consider MRI if central pathology suspected (CVA, tumor)
• Consider based on risk factors, such as age, and abnormal exam

Neurol Clin 2012 30(1): 61-74
CT Does Not Help In Evaluating Vertigo

• 488 consecutive ED patients who had head CTs for dizziness
• Diagnostic yield for head CT for relevant findings was 2.2%
• F/U MRI in 87 patients changed the diagnosis 16 % of the time

• Get an MRI if imaging needed

The MRI Isn’t Always Right

- MRI-DWI is falsely negative in up to 30% in posterior circulation stroke in first 24 hours
- Up to half of small (< 10 mm) strokes may be missed in first 48 hours
- Admission/referral decisions are made on clinical + testing considerations

DWI=Diffusion Weighted Images

Oppenheim AJNR 2000; 21;1434-40
Saber Tehrani AS et al. Neurology 2014 Jun 11
A Word About Cardiac Disease and Dizziness

MI, Hypotension, Syncope

- 63% of such patients have “dizziness”
- 37% have “vertigo” as their only dizziness
- 8% of MI have vertigo
- 17% of syncope have vertigo
- 37% of orthostatic intolerance have vertigo

Newman-Toker. How Often is Dizziness from Primary Cardiovascular Disease True Vertigo? A Systematic Review. 2008
Epley Maneuvers:
Most effective in posterior canal disease.
• 80 patients randomized to Epley in the office and at home or office only.
• Dix-Hallpike negative at 1 week in 90% who did home therapy vs. 72% in office only.

Meniere’s Disease

- Fairly infrequent
- 3rd-4th decade
- Bilateral 45%
- Vertigo
- Hearing loss with discrimination maintained
- Fullness in ear resolves after episode
- Tinnitus (fluctuating)
- Must have ≥ 2 episodes at least 20 min
- Dx by history and audiometry
Vestibular Schwannoma (Acoustic Neuroma)

- Benign tumor arising from CN VIII
- May have tinnitus and hearing loss but discrimination is lost much before hearing loss is complete
- May have associated facial palsy
- Diagnose with MRI
Drugs: Prolong vertigo!

- BPPV should not be routinely treated with vestibular suppressant medications (AAO-HNSF)
- Vestibular training is superior to drugs alone
- Options for medications if needed include
  - Dimenhydrinate (Dramamine)
  - Diazepam/lorazepam
  - Meclizine
  - Metoclopramide
  - Prochlorperazine
Practice Recommendations

• Use symptom pattern rather than symptom quality to diagnose
  - 95% of patients with AVS have either Vestibular neuronitis or CVA
  - For spontaneous episodic vertigo, think Vestibular migraine, Posterior TIA, and Vasovagal causes

• Use Dix-Hallpike and HINTS appropriately

• Don’t do CT on patients with vertigo

• Don’t trust a negative MRI if sx are concerning

• Epley maneuvers are effective for BPPV

• Do not use drugs alone for symptom control
Answer Key

1. B
2. D
3. B
4. B
5. A
6. B
Syncope: An Innovative Approach to Assessment in the ER/UC

Vukiet Tran, CCFP(EM), FCFP, MHSc, MBA, CHE
Staff, Emergency Physician
University Health Network
Disclosure

It is the policy of the AAFP that all individuals in a position to control content disclose any relationships with commercial interests upon nomination/invitation of participation. Disclosure documents are reviewed for potential conflicts of interest and, if identified, conflicts are resolved prior to confirmation of participation. Only those participants who had no conflict of interest or who agreed to an identified resolution process prior to their participation were involved in this CME session.

• Vu Kiet Tran, MD, MBA has disclosed a relationship with Elvium on the topic “Acute Pain Management”.

All individuals in a position to control content for this session have indicated they have no relevant financial relationships to disclose.
Learning Objectives

1. Identify the risk factors for sudden cardiac death.
2. Analyze the evidence surrounding the use of ECG and CT scans in the assessment of the patient with syncope.
3. Perform a differential diagnosis to identify or rule-out cardiac causes of syncope.
75 yo female presents with syncope

Multiple previous episodes
PMH: CAD, CABG, DM
Physical exam normal
ECG: LBBB

She is well in your ED

What will be management?
Case 2
Young female of 28 yo.

Felt weak in the subway station

Then passed out as she tried to get up from her seat

Now in your clinic

What work-up would you like?
Sudden transient loss of consciousness with concurrent diminution in postural tone followed by spontaneous recovery, and absence of neurological sequelae vs pre-syncope (near-syncope)
Definition

• Greek origin “synkoptein” meaning “to cut short”, pause

• Sudden transient loss of consciousness with concurrent diminution in postural tone followed by spontaneous recovery, and absence of neurological sequelae.

vs pre-syncope (near-syncope)
## Syncope and...

<table>
<thead>
<tr>
<th>Syncope</th>
<th>Symptom</th>
<th>Conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Syncope</td>
<td>Chest pain</td>
<td>Aortic dissection</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ruptured AAA</td>
</tr>
<tr>
<td></td>
<td></td>
<td>STEMI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Acute PE</td>
</tr>
<tr>
<td>Syncope</td>
<td>Headache</td>
<td>SAH</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Intra-parenchymal hemorrhage</td>
</tr>
<tr>
<td>Syncope</td>
<td>Shortness of breath</td>
<td>Pneumothorax</td>
</tr>
<tr>
<td></td>
<td></td>
<td>PE</td>
</tr>
<tr>
<td>Syncope</td>
<td>Abdo pain</td>
<td>Ruptured AAA</td>
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<tr>
<td></td>
<td></td>
<td>Ruptured viscous</td>
</tr>
<tr>
<td>Syncope</td>
<td>Bleeding</td>
<td>UGIB</td>
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<tr>
<td></td>
<td></td>
<td>LGIB</td>
</tr>
<tr>
<td>Syncope</td>
<td>Rash</td>
<td>Anaphylaxis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sepsis</td>
</tr>
</tbody>
</table>
Syncope and nothing else…
What is not Syncope!!!

• TIA
• Stroke (ischemic or hemorrhagic)
• Hypoglycemia
Syncope mimics

• Seizures
• Drop-attacks
• Conversion syndromes
• Psychogenic syncope
• Malingering
Aborted Sudden Cardiac Death = Syncope
Sudden Cardiac Death

• Malignant Ventricular arrhythmias
  – WPW
  – Long QT Syndrome
  – Short QT Syndrome
  – Brugada Syndrome
  – ARVC
  – Catecholaminergic VT
A given opportunity to diagnose a potentially fatal disease and prevent sure death in a patient who is currently feeling well and unaware of his fate.
Epidemiology

• 3-5% of ED visits (1-2 million)
• 1-6% of hospital admissions
• Diagnosis in only up to 70-80%
• No cause on initial evaluation 34%
• Most causes are benign
• Mortality low
  – Cardiac origin: 18-33%
Incidence

• Bimodal distribution (10-30yo and > 65yo)
• Rates increase with age (sharp rise at 70 yo)
• Lifetime cumulative incidence (subjects > 65yo): 35-39%
• 80% have their first episode before age of 30y
In General Practice

• Prevalence is 2-9 per 1000 encounters
• Peak ages
  – 10-30yo (women)
  – Age > 65 (both men and women)
• Only a subgroup presents to a medical doctor
  – 44% did not seek medical advice
  – Event rate is 2-4 times higher in the general population than the presentation rate
In General Practice

• More frequent in women
• Young men tend not to visit their GP
• Elderly tend to visit their GP in relation to the younger patient (22 vs 2 visits/1000pt-years)
Incidence doubles with Hx of cardiac disease.

Incidence

Rate per 1000 Person-Years

Age Group (yr) 20-29 30-39 40-49 50-59 60-69 70-79 >80

Men

Women

NEJM 2002; 347: 878-885
Etiologies

• Vasovagal 20%
• Cardiac 13%
• Orthostatic hypotension 9%
• Medications 7%
• Stroke 4%
• TIA 4%
• Other 10%
• Unknown 31%

NEJM 2002; 347: 878-885
My classification

<table>
<thead>
<tr>
<th>Non-fatal</th>
<th>Fatal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vasovagal</td>
<td>Cardiac arrhythmias (and medications)</td>
</tr>
<tr>
<td>Orthostatic hypotension (and medications)</td>
<td>Hemorrhage</td>
</tr>
<tr>
<td>Psychogenic</td>
<td>Sepsis/shock</td>
</tr>
</tbody>
</table>
AES Question
Question 1

What contributes the most to arriving at a diagnosis of causes of syncope?

A. CT scan of the head
B. Holter monitor
C. ECG
D. Lab tests
E. History and physical exam
Question 2

With a good clinical assessment (history and physical exam only), what is your diagnostic yield?

A. 10%
B. 20-25%
C. 45-50%
D. 65-70%
E. I really don’t care. I just want to order that CT head!
History       Physical Exam       ECG
Core work-up

History
Physical exam
ECG
First step

• History, physical exam, and ECG form the cornerstone of initial evaluation
• Diagnostic yield of 45-50%
AES Question
Question 3

Which element of the story in the assessment of syncope is NOT contributory?

A. Medication list
B. Context in which the syncope occurred
C. Family History of sudden death
D. Medical history of Diabetes
E. Persistent of hemi-lateralization
Painful History

• Did the patient have syncope?
  − Dizziness/vertigo?
  − Drop attack? (no LOC)
  − Seizure activity
  − Falls

• Sequence of events:
  − Context
  − Prodrome (and duration of prodrome)
  − During the event
  − After the event

• Neurologic symptoms
History

• Plays a key role in the initial evaluation of syncope
  – Prodromal symptoms
  – Family history
  – Triggers and context
  – Medications

Europace (2009) 11, 937-943
History

• 20 symptoms were assessed
• Outcomes: recurrence of syncope or death
• Factors that *risk stratify*:
  − Age
  − Previous syncopal episodes
  − Psychiatric history
  − Baseline heart disease
  − Abnormal ECG

Ann Intern Med. 1997; 126: 989-996
Historical independent predictors of an abnormal EPS

• Age
• LVEF < 0.40 (CHF)
• Structural heart disease
Age over 65
Congestive heart failure
Existing heart disease
Family history of SCD
Abnormal ECG
ECG

• Low diagnostic yield: 5%
• A normal ECG is highly predictive of benignity
  – In the absence of an abnormal ECG, further cardiovascular testing has little yield
• ECG are non-invasive, easy to perform, and inexpensive
• Abnormal ECG in 82% of patients who died in follow-up

Ann Intern Med, June 15 1997; 126 (12): 989-996
Normal ECG

- Further testing provides very little yield
  - Except for paroxysmal arrhythmias
    - Paroxysmal high grade AV heart blocks (elderly patients)
Things to look for on ECG

- Arrhythmias/blocks
- Ischemias
- PE
- Short PR/LGL/WPW
- Long QT Syndrome
- Short QT Syndrome
- ARVD
- Brugada Syndrome
- HCOM
- Pulmonary hypertension
History and ECG

• ECG in addition to history and physical exam yielded a diagnosis in 76% of cases

Am J Med 2001; 111: 177-184
Basic laboratory testing

• RBW
  - Diagnostic yield: 2-3%
  - usually confirms a clinical suspicion
  - not recommended, should be guided by clinical evaluation

• *Pregnancy test is recommended in all women of child-bearing age*

Ann Intern Med, June 15 1997; 126 (12):989-996
Not so useful labs

• D-Dimer (Euro J Emerg Med 2009. 16: 256-260)
• Myoglobin and CK (Euro J Emerg Med 2009. 16: 84-86)
Cardiac testing

• Diagnostic yield 5-35%
  - Echocardiography
  - Stress testing
  - Holter
  - Loop recorder
  - EPS

Ann Intern Med, June 15 1997; 126 (12): 989-996
Echocardiography

- Low yield 5-7%
- Routine Echo did not establish the cause of the syncope
- Normal Echo for *ALL* patients without a cardiac history and normal ECG
- Important if presence of structural heart disease or abnormal ECG

Ann Intern Med July 1 1997; 127 (1): 76-86
Heart 2002; 88: 363-367
Low yield: < 1%
Indicated in:
  Ischemic heart disease
  Exertional syncope*

Ann Inter Med July 1 1997; 127 (1): 76-86
24 Holter

- Yield of 19%
  - 4% correlation of symptoms with arrhythmia
  - 15% have symptoms without arrhythmia
  - 14% have asymptomatic arrhythmia
- Causal relation between most of these arrhythmias and syncope is uncertain
- A negative holter does not r/o arrhythmogenic etiology
## External Loop recorder

<table>
<thead>
<tr>
<th></th>
<th>24-47%</th>
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</thead>
<tbody>
<tr>
<td>Yield</td>
<td>(highest in patients with palpitations)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Indications</th>
<th>1) Frequent episodes with normal heart</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2) Recurrent events</td>
</tr>
</tbody>
</table>
Implantable Loop Recorder

• Used as an initial strategy (ILR-based strategy)
  − Correlation between syncope and ECG findings in 34% (54% were bradycardia and asystole)
  − In the unexplained syncope, ILR diagnosed an additional 52% (vs 20% by conventional strategy)
  − Overall, yield was 55% vs 19% by conventional strategy
Dx yield of ILP
## Electrophysiology Study

<table>
<thead>
<tr>
<th>Goals</th>
<th>VT, VF, SVT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Risks</td>
<td>PE</td>
</tr>
<tr>
<td></td>
<td>Cardiac perforation</td>
</tr>
<tr>
<td></td>
<td>MI</td>
</tr>
<tr>
<td>Drawbacks</td>
<td>A negative study does not exclude arrhythmogenic cause</td>
</tr>
<tr>
<td></td>
<td><em>Insensitive to detect bradyarrhythmias</em></td>
</tr>
<tr>
<td>Overall</td>
<td>Invasive</td>
</tr>
<tr>
<td></td>
<td>Expensive</td>
</tr>
</tbody>
</table>
Tilt Table Test

- Yield 60%
- Sensitivity 63-83%
- Specificity 90% (0-100%)
- More false-positives in the young
Tilt Table Test

Positive test does not exclude cardiac cause
Neurological testing

• Low yield 2-6%
• Useful if patients have neurological symptoms/signs or carotid bruits
  – Seizures
  – Focal neurological signs
## Neurological testing

<table>
<thead>
<tr>
<th>Test</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>EEG</td>
<td>Studies showed little use in the unselected patient with syncope</td>
</tr>
<tr>
<td></td>
<td>Not recommended as routine workup</td>
</tr>
<tr>
<td>CT and MRI</td>
<td>Yield of 4%</td>
</tr>
<tr>
<td></td>
<td>No use if no neuro symptoms</td>
</tr>
<tr>
<td>Carotid doppler</td>
<td>Usefulness is unknown</td>
</tr>
<tr>
<td>Transcranial doppler</td>
<td>Usefulness in drop attack is unknown</td>
</tr>
</tbody>
</table>
Syncope requires either:
Both hemisphere to be knocked out
The reticular activating system (RSA) to be affected

Syncope is a brain perfusion problem
Why head CTs with syncope

• Strokes and syncope do not typically go together
• Strokes generally do not lead to syncope
• Exceptions:
  – Global bilateral cerebral ischemia or basilar artery disease affecting the RAS (reticular activating system)
  – Vertebrobasilar stroke or migraine
  – Sudden onset of a severe headache in the setting of a possible syncopal event (subarachnoid hemorrhage)
Ictal Syncope

• Temporal lobe seizures can mimic or cause reflex bradycardia or asystole
  − Hypotension and syncope
  − Sudden Unexplained Death in Epilepsy (SUDEP)

Diagnostic yield: 5% (Grossman et al. Intern Emerg med (2007) 2: 46-49)

Diagnostic yield: 3.9% (Al-Nsoor et al. Neurosciences (Riyadh). 2010 (2): 105-109)
Study describes 117 patients who had head CTs following syncope

Zero positive findings

Goyal N et al. Intern Emerg Med. 2006;1;148-150
Patients after a brief syncopal event are unlikely to benefit from a routine head CT.

Intern Emerg Med. 2007 Mar; 2(1): 46–49
Neurosciences (Riyadh). 2010 Apr;15(2):105-9
Strongly discouraged

• EEG, Head CT scan, and MRI are low yield in syncope
• Potential indications
  − Head trauma as a result of syncope
  − Suspicion for subarachnoid hemorrhage
  − History suggestive of focal neurological findings
  − History and physical findings suggestive of temporal seizures

Dis Mon 2009; 55: 532-585
• The evaluation of syncope should include a thorough history and physical exam to identify high-risk clinical predictors for CT head abnormalities.
• In the absence of these predictors, a CT head is unlikely to aid in the management of syncope patients

https://choosingwiselycanada.org/emergency-medicine/
<table>
<thead>
<tr>
<th>Speciality</th>
<th>Tests</th>
<th>Conclusive diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiology</td>
<td>Echo, Holter, EPS, stress test</td>
<td>83%</td>
</tr>
<tr>
<td>Internal medicine</td>
<td>Abdo ultrasound, CT/MRI, miscellaneous</td>
<td>69.5%</td>
</tr>
<tr>
<td>Neurology</td>
<td>EEG, CT/MRI, Tilt test</td>
<td>54.5%</td>
</tr>
</tbody>
</table>

European Heart J 2002 (23); 815-820
Risk stratification based on prognostic factors
Risk stratification 1

- Abnormal ECG
- Age over 65
- Hx of Heart Failure

Overall arrhythmogenic syncope 17-18%

Acad Emerg Med; Dec 2003; 10, 12: 1312-1317
San Francisco Syncope Rule

7-days outcome study
• Sensitivity 96.2%
• Specificity 62%
• NPV 99.2%
• PPV 24.8%

• Decrease admission rate by 10%

• Abnormal ECG
• Shortness of breath
• SBP < 90
• Hct < 30%
• Heart Failure
San Francisco – Validation

**Internal**

30-days outcome study

- Sensitivity 98%
- Specificity 56%
- Potentially decreasing admission by 7%

**External**

7-days outcome study

- Sensitivity 89%
- Specificity 69%

San Francisco – Elderly patients

Application of the rule for pts > 65yo
7-days outcome study

- Sensitivity 76.5%
- Specificity 36.8%
- NPV 87%
- PPV 22.1%

San Francisco
Sensitivity 96%
Specificity 62%
ROC (AUC) 0.92

Clinical Judgment
Sensitivity 94%
Specificity 54%
ROC (AUC) 0.83

Boston Syncope Rule

- Signs and Symptoms of ACS
  - Chest pain or SOB
  - Ischemic ECG changes
  - Other ECG changes

- Worrisome cardiac history
  - CAD
  - HCOM
  - CHF (or low EF)
  - AICD
  - Pacemaker
  - Use of anti-dysrhythmic (excluding BB and CCB)
  - FHx of SCD

- Valvular heart disease (murmur in the ED)
- Signs of conduction disease
  - Multiple syncopal episodes in the last 6 months
  - Palpitation
  - QTc > 500
  - 2\textsuperscript{nd} or 3\textsuperscript{rd} degree HB

- Volume depletion
  - GI bleed by history or hemocult
  - HCT < 30
  - Dehydration

- Persistent abnormal vital signs
- Primary CNS event

JEM. 2007; 33 (3): 233-239
Boston Syncope Rule

- Sensitivity 97%
- Specificity 62%
- PPV 44%
- NPV 99%

Not internally validated
Not externally validated
# Canadian Syncope Score

**Canadian Syncope Risk Score**

<table>
<thead>
<tr>
<th>Category</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Predisposition to vasovagal symptoms (triggered by being in a warm crowded place, prolonged standing, fear, emotion, or pain)</td>
<td>0</td>
<td>-1</td>
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<td>Heart disease history (CAD, afib, flutter, CHF, valvular disease)</td>
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</tr>
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<td>SBP &lt; 90 or &gt; 180mmHg</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Elevated troponin</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Abnormal QRS axis</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>QRS &gt; 130ms</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Corrected QT interval &gt;480ms</td>
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<td>2</td>
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<tr>
<td>Vasovagal syncope (based on clinical impression)</td>
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- Score $< 0$ associated with $< 2\%$ risk of serious adverse event at 30 days.
- Externally validated per data presented at SAEM 2018.
## OESIL risk score

<table>
<thead>
<tr>
<th>Independent Predictors</th>
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</tr>
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<td>Syncope without prodrome</td>
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</table>

European Heart Journal 2003; 24: 811-819
OESIL risk Score

OESIL score > 1 is predictive of mortality

The higher the score, worse is mortality rate
Management should be…

Based on risk and prognosis

and not on diagnosis (if diagnosis is not possible and often difficult to make)
Vu’s Protocol

<table>
<thead>
<tr>
<th>Sarasin et al.</th>
<th>San Francisco</th>
<th>OESIL</th>
<th>Miscellaneous</th>
</tr>
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<tbody>
<tr>
<td>❖ Abnormal ECG</td>
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<td>❖ Abnormal ECG</td>
<td>❖ Exertional syncope</td>
</tr>
<tr>
<td>❖ Age &gt; 65</td>
<td>❖ SOB</td>
<td>❖ Age &gt; 65</td>
<td>❖ Associated CP or SOB</td>
</tr>
<tr>
<td>❖ Hx of CHF</td>
<td>❖ SBP &lt; 90</td>
<td>❖ Cardiovascular disease on Hx</td>
<td>❖ Associated palpitation</td>
</tr>
<tr>
<td></td>
<td>❖ Hct &lt; 30%</td>
<td>❖ Syncope without prodrome</td>
<td>❖ Recurrence of syncope</td>
</tr>
<tr>
<td></td>
<td>❖ CHF</td>
<td></td>
<td>❖ Family history of premature sudden death</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>❖ Drugs that prolong QT</td>
</tr>
</tbody>
</table>
What should we do then?

- Perform a detailed *history* and physical exam
- Routinely obtain an ECG
- Cardiac imaging if clinical suspicion of structural heart disease

- EPS if suspicion of tachyarrhythmias
- Cardiac rhythm monitoring if clinical suspicion of arrhythmia
- No CT scan head if no focal neuro deficit or seizures

Can J Card 27 (2011): 246-253
Cases Revisited
75 yo female presents with syncope

Multiple previous episodes
PMH: CAD, CABG, DM
Physical exam normal
ECG: LBBB

She is well in your ED

What will be management?
Case 1

- Loop recorder placed for 1 month, but was asymptomatic
- Had EPS, normal
- Loop event monitoring again which showed complete AV dissociation
- Pacemaker placement
- No syncope after 2-year f/u
Case 2
Case 2

• Referred to cardiology and admission to CCU.
• A procainamide challenge test was done during EPS.
• Confirmation of Brugada Syndrome.
• Internal defibrillator inserted.
Young female of 28 yo.

Felt weak in the subway station

Then passed out as she tried to get up from her seat

Now in your clinic

What work-up would you like?
Case 3

• B-HCG was positive.
• Pelvic ultrasound showed rupture left ectopic pregnancy with free fluid in the pelvis.
• Transferred care to Gynecology
Vu’s Top 10 take home messages
Message 10

- Diagnose benign causes
Message 9

• EPS in patients with organic heart disease
Message 8

- Holter for patients with heart disease
- Loop monitoring in patients with frequent events and normal hearts
Message 7

• Use clinical decision rules if initial risk is unclear (but know their limitations)
• History, physical examination, and ECG form the cornerstone of the syncope work-up
• Careful (and painful) history give you the diagnosis in almost all cases
Message 5

- **IDENTIFY** high risk criteria
Message 4

• High risk patients should receive cardiac consultation
Message 3

• Patients whom heart disease is known or those with exertional syncope should get cardiac testing (echo)
Message 2

• Do an ECG on all patients
• Have fun assessing the syncopal patient!

(and you will become a Superhero!)
A *given opportunity* to diagnose a potentially fatal disease and prevent *sure death* in a patient who is currently feeling well and *unaware of his fate*.
Answer Key

1. E
2. C
3. D
Syncope: An Innovative Approach to Assessment in the ER/UC

Vukiet Tran, CCFP(EM), FCFP, MHSc, MBA, CHE
Staff, Emergency Physician
University Health Network
It is the policy of the AAFP that all individuals in a position to control content disclose any relationships with commercial interests upon nomination/invitation of participation. Disclosure documents are reviewed for potential conflicts of interest and, if identified, conflicts are resolved prior to confirmation of participation. Only those participants who had no conflict of interest or who agreed to an identified resolution process prior to their participation were involved in this CME session.

• Vu Kiet Tran, MD, MBA has disclosed a relationship with Elvium on the topic “Acute Pain Management”.

All individuals in a position to control content for this session have indicated they have no relevant financial relationships to disclose.
Learning Objectives

1. Identify the risk factors for sudden cardiac death.
2. Analyze the evidence surrounding the use of ECG and CT scans in the assessment of the patient with syncope.
3. Perform a differential diagnosis to identify or rule-out cardiac causes of syncope.
75 yo female presents with syncope

- Multiple previous episodes
- PMH: CAD, CABG, DM
- Physical exam normal
- ECG: LBBB

She is well in your ED

What will be management?
Case 2
Young female of 28 yo.

Felt weak in the subway station

Then passed out as she tried to get up from her seat

Now in your clinic

What work-up would you like?
Sudden transient loss of consciousness with concurrent diminution in postural tone followed by spontaneous recovery, and absence of neurological sequelae vs pre-syncope (near-syncope)
Definition

• Greek origin “synkoptein” meaning “to cut short”, pause

• Sudden transient loss of consciousness with concurrent diminution in postural tone followed by spontaneous recovery, and absence of neurological sequelae.

  vs pre-syncope (near-syncope)
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<tr>
<th>Syncope</th>
<th>Symptom</th>
<th>Conditions</th>
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<td>Syncope</td>
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<td>Aortic dissection</td>
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<td>Ruptured AAA</td>
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<tr>
<td></td>
<td></td>
<td>STEMI</td>
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<td></td>
<td></td>
<td>Acute PE</td>
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<tr>
<td>Syncope</td>
<td>Headache</td>
<td>SAH</td>
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<td>Intra-parenchymal hemorrhage</td>
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<td>Syncope</td>
<td>Shortness of breath</td>
<td>Pneumothorax</td>
</tr>
<tr>
<td></td>
<td></td>
<td>PE</td>
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<tr>
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<td>Bleeding</td>
<td>UGIB</td>
</tr>
<tr>
<td></td>
<td></td>
<td>LGIB</td>
</tr>
<tr>
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<td>Anaphylaxis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sepsis</td>
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</tbody>
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Syncope and nothing else…
What is not Syncope!!!

• TIA
• Stroke (ischemic or hemorrhagic)
• Hypoglycemia
Syncope mimics

• Seizures
• Drop-attacks
• Conversion syndromes
• Psychogenic syncope
• Malingering
Aborted Sudden Cardiac Death = Syncope
Sudden Cardiac Death

• Malignant Ventricular arrhythmias
  – WPW
  – Long QT Syndrome
  – Short QT Syndrome
  – Brugada Syndrome
  – ARVC
  – Catecholaminergic VT
A given opportunity to diagnose a potentially fatal disease and prevent sure death in a patient who is currently feeling well and unaware of his fate.
Epidemiology

• 3-5% of ED visits (1-2 million)
• 1-6% of hospital admissions
• Diagnosis in only up to 70-80%
• No cause on initial evaluation 34%
• Most causes are benign
• Mortality low
  – Cardiac origin: 18-33%
Incidence

• Bimodal distribution (10-30yo and > 65yo)
• Rates increase with age (sharp rise at 70 yo)
• Lifetime cumulative incidence (subjects > 65yo): 35-39%
• 80% have their first episode before age of 30y
In General Practice

• Prevalence is 2-9 per 1000 encounters
• Peak ages
  – 10-30yo (women)
  – Age > 65 (both men and women)
• Only a subgroup presents to a medical doctor
  – 44% did not seek medical advice
  – Event rate is 2-4 times higher in the general population than the presentation rate
In General Practice

• More frequent in women
• Young men tend not to visit their GP
• Elderly tend to visit their GP in relation to the younger patient (22 vs 2 visits/1000pt-years)
Incidence doubles with Hx of cardiac disease

NEJM 2002; 347: 878-885
Mortality according to etiology

NEJM 2002, 47; 878-885
Etiologies

• Vasovagal 20%
• Cardiac 13%
• Orthostatic hypotension 9%
• Medications 7%
• Stroke 4%
• TIA 4%
• Other 10%
• Unknown 31%

NEJM 2002; 347: 878-885
My classification

<table>
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<th>Non-fatal</th>
<th>Fatal</th>
</tr>
</thead>
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<td>Vasovagal</td>
<td>Cardiac arrhythmias (and medications)</td>
</tr>
<tr>
<td>Orthostatic hypotension (and medications)</td>
<td>Hemorrhage</td>
</tr>
<tr>
<td>Psychogenic</td>
<td>Sepsis/shock</td>
</tr>
</tbody>
</table>
AES Question
Question 1

What contributes the most to arriving at a diagnosis of causes of syncope?

A. CT scan of the head
B. Holter monitor
C. ECG
D. Lab tests
E. History and physical exam
AES Question
Question 2

With a good clinical assessment (history and physical exam only), what is your diagnostic yield?

A. 10%
B. 20-25%
C. 45-50%
D. 65-70%
E. I really don’t care. I just want to order that CT head!
Core work-up

History
Physical exam
ECG
First step

• History, physical exam, and ECG form the cornerstone of initial evaluation
• Diagnostic yield of 45-50%

Ann Int Med 1997; 126: 989-996
AES Question
Question 3

Which element of the story in the assessment of syncope is NOT contributory?

A. Medication list  
B. Context in which the syncope occurred  
C. Family History of sudden death  
D. Medical history of Diabetes  
E. Persistent of hemi-lateralization
Painful History

• Did the patient have syncope?
  – Dizziness/vertigo?
  – Drop attack? (no LOC)
  – Seizure activity
  – Falls

• Sequence of events:
  – Context
  – Prodrome (and duration of prodrome)
  – During the event
  – After the event

• Neurologic symptoms
History

• Plays a key role in the initial evaluation of syncope
  – Prodromal symptoms
  – Family history
  – Triggers and context
  – Medications

Europace (2009) 11, 937-943
History

• 20 symptoms were assessed
• Outcomes: recurrence of syncope or death
• Factors that risk stratify:
  – Age
  – Previous syncopal episodes
  – Psychiatric history
  – Baseline heart disease
  – Abnormal ECG

Ann Intern Med. 1997; 126: 989-996
Historical independent predictors of an abnormal EPS

- Age
- LVEF < 0.40 (CHF)
- Structural heart disease
Age over 65

Congestive heart failure

Existing heart disease

Family history of SCD

Abnormal ECG
ECG

• Low diagnostic yield: 5%

• A normal ECG is highly predictive of benignity
  – In the absence of an abnormal ECG, further cardiovascular testing has little yield

• ECG are non-invasive, easy to perform, and inexpensive

• Abnormal ECG in 82% of patients who died in follow-up

Ann Intern Med, June 15 1997; 126 (12): 989-996
Normal ECG

- Further testing provides very little yield
  - Except for paroxysmal arrhythmias
    - Paroxysmal high grade AV heart blocks (elderly patients)
Things to look for on ECG

- Arrhythmias/blocks
- Ischemias
- PE
- Short PR/LGL/WPW
- Long QT Syndrome
- Short QT Syndrome
- ARVD
- Brugada Syndrome
- HCOM
- Pulmonary hypertension
ECG in addition to history and physical exam yielded a diagnosis in 76% of cases.
Basic laboratory testing

• RBW
  − Diagnostic yield: 2-3%
  − usually confirms a clinical suspicion
  − not recommended, should be guided by clinical evaluation

• Pregnancy test is recommended in all women of child-bearing age

Ann Intern Med, June 15 1997; 126 (12):989-996
Not so useful labs

Cardiac testing

• Diagnostic yield 5-35%
  – Echocardiography
  – Stress testing
  – Holter
  – Loop recorder
  – EPS

Ann Intern Med, June 15 1997; 126 (12): 989-996
Echocardiography

• Low yield 5-7%
• Routine Echo did not establish the cause of the syncope
• Normal Echo for ALL patients without a cardiac history and normal ECG
• Important if presence of structural heart disease or abnormal ECG
ECG  Echo

AMERICAN ACADEMY OF FAMILY PHYSICIANS
Exercise stress testing

Low yield: < 1%
Indicated in:
  Ischemic heart
disease
  Exertional syncope*

Ann Inter Med July 1 1997; 127 (1): 76-86
24 Holter

• Yield of 19%
  − 4% correlation of symptoms with arrhythmia
  − 15% have symptoms without arrhythmia
  − 14% have asymptomatic arrhythmia
• Causal relation between most of these arrhythmias and syncope is uncertain
• A negative holter does not r/o arrhythmogenic etiology
## External Loop recorder

| Yield | 24-47%  
(highest in patients with palpitations) |
|-------|----------------------------------|
| Indications | 1) Frequent episodes with normal heart  
2) Recurrent events |
Implantable Loop Recorder

• Used as an initial strategy (ILR-based strategy)
  − Correlation between syncope and ECG findings in 34% (54% were bradycardia and asystole)
  − In the unexplained syncope, ILR diagnosed an additional 52% (vs 20% by conventional strategy)
  − Overall, yield was 55% vs 19% by conventional strategy
Dx yield of ILP
Electrophysiology Study

<table>
<thead>
<tr>
<th>Goals</th>
<th>VT, VF, SVT</th>
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<tr>
<td>Risks</td>
<td>PE</td>
</tr>
<tr>
<td></td>
<td>Cardiac perforation</td>
</tr>
<tr>
<td></td>
<td>MI</td>
</tr>
<tr>
<td>Drawbacks</td>
<td>A negative study does not exclude arrhythmogenic cause</td>
</tr>
<tr>
<td></td>
<td><em>Insensitive to detect bradyarrhythmias</em></td>
</tr>
<tr>
<td>Overall</td>
<td>Invasive</td>
</tr>
<tr>
<td></td>
<td>Expensive</td>
</tr>
</tbody>
</table>
Tilt Table Test

- Yield 60%
- Sensitivity 63-83%
- Specificity 90% (0-100%)
- More false-positives in the young
Tilt Table Test

Positive test does not exclude cardiac cause
Neurological testing

• Low yield 2-6%
• Useful if patients have neurological symptoms/signs or carotid bruits
  – Seizures
  – Focal neurological signs
### Neurological testing

<table>
<thead>
<tr>
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<th>Notes</th>
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<tr>
<td><strong>EEG</strong></td>
<td>Studies showed little use in the unselected patient with syncope</td>
</tr>
<tr>
<td></td>
<td>Not recommended as routine workup</td>
</tr>
<tr>
<td><strong>CT and MRI</strong></td>
<td>Yield of 4%</td>
</tr>
<tr>
<td></td>
<td>No use if no neuro symptoms</td>
</tr>
<tr>
<td><strong>Carotid doppler</strong></td>
<td>Usefulness is unknown</td>
</tr>
<tr>
<td><strong>Transcranial doppler</strong></td>
<td>Usefulness in drop attack is unknown</td>
</tr>
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Syncope requires either:
Both hemisphere to be knocked out
The reticular activating system (RSA) to be affected

Syncope is a brain perfusion problem
Why head CTs with syncope

• Strokes and syncope do not typically go together
• Strokes generally do not lead to syncope
• Exceptions:
  – Global bilateral cerebral ischemia or basilar artery disease affecting the RAS (reticular activating system)
  – Vertebrobasilar stroke or migraine
  – Sudden onset of a severe headache in the setting of a possible syncopal event (subarachnoid hemorrhage)
Ictal Syncope

- Temporal lobe seizures can mimic or cause reflex bradycardia or asystole
  - Hypotension and syncope
  - Sudden Unexplained Death in Epilepsy (SUDEP)

Diagnostic yield: 5% (Grossman et al. Intern Emerg med (2007) 2: 46-49)

Diagnostic yield: 3.9% (Al-Nsoor et al. Neurosciences (Riyadh). 2010 (2): 105-109)
Study describes 117 patients who had head CTs following syncope.

Zero positive findings

Goyal N et al. Intern Emerg Med. 2006;1;148-150
Patients after a brief syncopal event are unlikely to benefit from a routine head CT.
Strongly discouraged

• EEG, Head CT scan, and MRI are low yield in syncope
• Potential indications
  – Head trauma as a result of syncope
  – Suspicion for subarachnoid hemorrhage
  – History suggestive of focal neurological findings
  – History and physical findings suggestive of temporal seizures

Dis Mon 2009; 55: 532-585
Carotid Dopplers
Choosing Wisely Canada

• The evaluation of syncope should include a thorough history and physical exam to identify high-risk clinical predictors for CT head abnormalities.
• In the absence of these predictors, a CT head is unlikely to aid in the management of syncope patients

https://choosingwiselycanada.org/emergency-medicine/
## Coloured-glasses

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<thead>
<tr>
<th>Speciality</th>
<th>Tests</th>
<th>Conclusive diagnosis</th>
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<tbody>
<tr>
<td>Cardiology</td>
<td>Echo, Holter, EPS, stress test</td>
<td>83%</td>
</tr>
<tr>
<td>Internal medicine</td>
<td>Abdo ultrasound, CT/MRI, miscellaneous</td>
<td>69.5%</td>
</tr>
<tr>
<td>Neurology</td>
<td>EEG, CT/MRI, Tilt test</td>
<td>54.5%</td>
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European Heart J 2002 (23); 815-820
Risk stratification based on prognostic factors
Risk stratification 1

• Abnormal ECG
• Age over 65
• Hx of Heart Failure

Overall arrhythmogenic syncope 17-18%

Acad Emerg Med; Dec 2003; 10, 12: 1312-1317
San Francisco Syncope Rule

7-days outcome study
- Sensitivity 96.2%
- Specificity 62%
- NPV 99.2%
- PPV 24.8%

- Decrease admission rate by 10%

• Abnormal ECG
• Shortness of breath
• SBP < 90
• Hct < 30%
• Heart Failure
San Francisco – Validation

Internal

30-days outcome study

• Sensitivity 98%
• Specificity 56%
• Potentially decreasing admission by 7%

External

7-days outcome study

• Sensitivity 89%
• Specificity 69%

San Francisco – Elderly patients

Application of the rule for pts > 65yo
7-days outcome study

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<td>Syncope without prodrome</td>
<td>1.13</td>
</tr>
</tbody>
</table>

European Heart Journal 2003; 24: 811-819
OESIL risk Score

OESIL score > 1 is predictive of mortality

The higher the score, worse is mortality rate
Management should be…

Based on risk and prognosis

and not on diagnosis (if diagnosis is not possible and often difficult to make)
## Vu’s Protocol

<table>
<thead>
<tr>
<th>Sarasin et al.</th>
<th>San Francisco</th>
<th>OESIL</th>
<th>Miscellaneous</th>
</tr>
</thead>
<tbody>
<tr>
<td>❖ Abnormal ECG</td>
<td>❖ Abnormal ECG</td>
<td>❖ Abnormal ECG</td>
<td>❖ Exertional syncope</td>
</tr>
<tr>
<td>❖ Age &gt; 65</td>
<td>❖ SOB</td>
<td>❖ Age &gt; 65</td>
<td>❖ Associated CP or SOB</td>
</tr>
<tr>
<td>❖ Hx of CHF</td>
<td>❖ SBP &lt; 90</td>
<td>❖ Cardiovascular disease on Hx</td>
<td>❖ Associated palpitation</td>
</tr>
<tr>
<td></td>
<td>❖ Hct &lt; 30%</td>
<td>❖ Syncope without prodrome</td>
<td>❖ Recurrence of syncope</td>
</tr>
<tr>
<td></td>
<td>❖ CHF</td>
<td></td>
<td>❖ Family history of premature sudden death</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>❖ Drugs that prolong QT</td>
</tr>
</tbody>
</table>
What should we do then?

- Perform a detailed **history** and physical exam
- Routinely obtain an ECG
- Cardiac imaging if clinical suspicion of structural heart disease
- EPS if suspicion of tachyarrhythmias
- Cardiac rhythm monitoring if clinical suspicion of arrhythmia
- No CT scan head if no focal neuro deficit or seizures

Can J Card 27 (2011): 246-253
Cases Revisited
75 yo female presents with syncope

Multiple previous episodes
PMH: CAD, CABG, DM
Physical exam normal
ECG: LBBB

She is well in your ED

What will be management?
Case 1

- Loop recorder placed for 1 month, but was asymptomatic
- Had EPS, normal
- Loop event monitoring again which showed complete AV dissociation
- Pacemaker placement
- No syncope after 2-year f/u
Case 2
Case 2

• Referred to cardiology and admission to CCU.
• A procainamide challenge test was done during EPS.
• Confirmation of Brugada Syndrome.
• Internal defibrillator inserted.
Young female of 28 yo.

Felt weak in the subway station

Then passed out as she tried to get up from her seat

Now in your clinic

What work-up would you like?
Case 3

• B-HCG was positive.
• Pelvic ultrasound showed rupture left ectopic pregnancy with free fluid in the pelvis.
• Transferred care to Gynecology
Vu’s Top 10 take home messages
Message 10

❖ Diagnose benign causes
Message 9

• EPS in patients with organic heart disease
Message 8

• Holter for patients with heart disease
• Loop monitoring in patients with frequent events and normal hearts
Message 7

• Use clinical decision rules if initial risk is unclear (but know their limitations)
Message 6

• History, physical examination, and ECG form the cornerstone of the syncope work-up
• Careful (and painful) history give you the diagnosis in almost all cases
Message 5

• *IDENTIFY* high risk criteria
Message 4

• High risk patients should receive cardiac consultation
Message 3

• Patients whom heart disease is known or those with exertional syncope should get cardiac testing (echo)
Message 2

• Do an ECG on all patients
• Have fun assessing the syncopal patient!

(and you will become a Superhero!)
A *given opportunity* to diagnose a potentially fatal disease and prevent *sure death* in a patient who is currently feeling well and unaware of his fate.
Answer Key

1. E
2. C
3. D