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Neurologic Emergencies

2nd Annual Inpatient Medicine for NPs & PAs: Hospital Care from Admission to Discharge

Wednesday-Saturday, October 19-22, 2016
Sawgrass Marriott Hotel • Ponte Vedra Beach, Florida
Disclosures

Financial
None

Off-label use
Use of medications for status epilepticus other than lorazepam IV, midazolam IM, and phenytoin/fosphenytoin
Objectives

• At the conclusion of this lecture, the audience should be able to describe and enact:
  • Appropriate management for intracerebral hemorrhage
  • Management for status epilepticus
  • Clinical signs that suggest need for ventilatory support for neuromuscular respiratory failure
  • Evaluation and empiric treatment for bacterial and HSV meningoencephalitis
Case

• 48 year old woman found down and poorly responsive at home in pool of vomit
• Reported to PCP 2 days prior reporting neck pain and headache
• Febrile (38.6 C), tachycardic
• Nuchal rigidity on examination, severely encephalopathic
Case

- 69 year old man, with headache and progressive encephalopathic over 1-2 days
- Today, had a convulsion (seizure) that prompted ED evaluation
- Febrile (38.6°C), without nuchal rigidity; clearly encephalopathic and doesn’t follow commands, but has mildly reduced movement of his right side
These clinical presentations are most consistent with:

A. Stroke
B. CNS infection
C. Status epilepticus
D. Systemic infection with delirium
E. Malingering
These clinical presentations are most consistent with:

A. Stroke

B. CNS infection

C. Status epilepticus

D. Systemic infection with delirium

E. Malingering
What is the most appropriate next step?

A. STAT CT scan prior to LP, then initiate antimicrobial therapy

B. Imaging not necessary; STAT LP, then initiate antimicrobial therapy

C. STAT CT prior to LP, but don’t wait to initiate antimicrobial therapy

D. Imaging not necessary; STAT LP, but don’t wait to initiate antimicrobial therapy
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C. STAT CT prior to LP, but don’t wait to initiate antimicrobial therapy

D. Imaging not necessary; STAT LP, but don’t wait to initiate antimicrobial therapy
Bacterial meningitis

- Decreasing incidence in US over time
- Average age
  - 15 months (1986)
  - 25 years (1998)
- First *Hib* vaccine introduced in US in 1985
  - 20,000 cases (1987)
  - 255 cases (1998)

Bacterial meningitis: Symptoms

- **Classic triad:** Fever, headache, neck stiffness
  - Subacute over hours to <2 days
  - Only about half of patients with all 3 signs

- **Other symptoms**
  - Vomiting
  - Encephalopathy ("altered mental status"), ranging from irritability to coma
  - Light sensitivity
  - Kernig’s sign (K = knee)
  - Brudzinski’s sign
Bacterial meningitis: Risk factors

- Ends of age spectrum (infants and elderly)
- Chronic disease (DM, renal, hepatic)
- Immunocompromise or suppression
- Alcoholism
- Pregnancy (Group B strep, Listeria)
- Crowded living arrangement (N. meningitidis)
Bacterial meningitis: Diagnosis

• Basic labs – CBC, electrolytes, coags

• Blood cultures
  • Up to 50% positive in common meningitis-causing bacteria
  • May be helpful if LP is delayed relative to antibiotic use

• Imaging?
  • Before LP only in certain cases if worried about intracranial pressure
  • Immunocompromised, history of CNS disease, seizure within 1 week, papilledema, decreased consciousness, focal neurologic deficit

Bacterial meningitis: Diagnosis

• Lumbar puncture, including gram stain and culture

<table>
<thead>
<tr>
<th>Meningitis etiology</th>
<th>Opening pressure</th>
<th>Cell count</th>
<th>Glucose</th>
<th>Protein</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt;25 cm H₂O</td>
<td>0-5</td>
<td>60% serum</td>
<td>&lt;40 mg/dL</td>
</tr>
<tr>
<td>Bacterial</td>
<td>Elevated</td>
<td>1000+, neutrophilic</td>
<td>Very decreased</td>
<td>Very elevated</td>
</tr>
<tr>
<td>Viral</td>
<td>Normal to mildly elevated</td>
<td>&lt;500, lymphocytic</td>
<td>Normal to mildly decreased</td>
<td>Normal to mildly elevated</td>
</tr>
</tbody>
</table>
Bacterial meningitis: Treatment (age 2 and up)

• For all:
  • Vancomycin 15-20 mg/kg IV q8-12 hours
  • Ceftriaxone 2 g IV q12 hours
  • Plus: empiric viral coverage…yet to come!

• For age >50 years or immune impaired, add to above:
  • Ampicillin 2 g IV q4 hour

• DO NOT WITHOLD TREATMENT TO WAIT FOR TESTS!
Dexamethasone and *S. pneumoniae*

- *S. pneumoniae* may be more effectively treated if dexamethasone given *before* (or with) initial antibiotic therapy
- Must decide to give before knowing diagnosis!
- Dexamethasone 10 mg IV x 1 prior to abx, then 10 mg q6 hours for 4 days
- Discontinue if CSF returns inconsistent with bacterial meningitis

Viral meningitis

- Majority treated with supportive care
- Most common exception: Herpes simplex virus (HSV)
HSV encephalitis

- May not be associated with meningismus
- Frequently encephalopathic, seizures
- MRI shows characteristic T2 hyperintensity in one or both temporal lobes
- CSF: HSV PCR (may be false negative if <48 hrs)
- Treatment with acyclovir 10 mg/kg IV q8 hours
Approach to suspected CNS infection
Approach to suspected CNS infection

- Labs and treatment performed simultaneously
  - Don’t forget blood cultures!
- LP as soon as possible, combining bacterial and viral studies
- Treat empirically for both bacterial meningitis and HSV until CSF findings confirm etiology
- Tailor treatment as testing comes in

**Basic CSF studies:**
- Cell count and diff
- Glucose
- Protein
- Gram stain and culture
- HSV PCR

**Basic treatment:**
- Vancomycin 15-20 mg/kg IV q8-12
- Ceftriaxone 2 g IV q12
- Acyclovir 10 mg/kg IV q8
- +/- Ampicillin 2 g IV q4 (if risk factors)
- +/- Dexamethasone 10 mg IV (prior to first abx dose if suspect S. pneumoniae)
Case

- 56 year old man presents with acute right-sided weakness
- History of hypertension and atrial fibrillation on warfarin
- BP 180/110 mmHg, pulse 85, O2 sat 96%
- On exam, has dense right hemiparesis. Language is impaired.
- INR = 5
What is the most appropriate next step?

A. Reversal with IV vitamin K alone

B. Reversal with fresh frozen plasma (FFP) alone

C. Reversal with both IV vitamin K and FFP

D. Aggressive blood pressure reduction (SBP <140 mmHg) and reversal with IV vitamin K alone

E. Aggressive blood pressure reduction (SBP <140) and reversal with both IV vitamin K and FFP
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Intracranial hemorrhage

• Many varieties
  • Epidural (surgical emergency)
  • Subdural
  • Subarachnoid
  • Intracerebral

• Will focus on intracerebral since care is often (but not always) non-operative

• Highlights of subarachnoid hemorrhage (SAH)
Intracerebral hemorrhage

• Presentation similar to stroke
• May have associated headache, worsening mental status
• Imaging key to diagnosis
• Risk factors:
  • Hypertension
  • Age
  • Anticoagulation/antithrombotic use

Intracerebral hemorrhage

Goal of early intervention is to prevent expansion!
Intracerebral hemorrhage

- If anticoagulated, REVERSE
  - Vitamin K 5-10 mg IV if warfarin – DOES NOT ACT QUICKLY
  - Fresh frozen plasma or prothrombin complex concentrates
  - Ensure INR stays normalized (repeat!)
- Direct thrombin inhibitors and factor Xa inhibitors
  - Dabigatran (DTI) – idarucizumab
  - Currently investigating options for Xa inhibitors

Intracerebral hemorrhage

- Blood pressure control
- Previously, SBP <180 mmHg
- INTERACT2 trial – trend towards primary outcome, no difference in safety
- Now recommend lowering SBP <140 mmHg in patients without contraindication
What about surgery?

• Cerebellar hemorrhage
  • Not much room to expand
  • Brainstem compression, hydrocephalus, decompensation may occur early
  • Neurosurgeon may decompress at first signs of deterioration

• Cerebral hemorrhage
  • Not usually recommended
  • May intervene if decompensating, comatose, or large hematoma

Cerebellar hemorrhage anatomy
Subarachnoid hemorrhage: Highlights

- Early intervention strategies similar
  - Reverse anticoagulation
  - Control blood pressure (parameters not well defined)

- Angiography to evaluate for aneurysm

- If present and able, secure aneurysm (coiling or clipping)

- Later complications
  - Delayed cerebral ischemia (vasospasm)
  - Cerebral salt wasting
Restarting anticoagulation

- No guidelines regarding timing of restart
- Appears to be safe (and beneficial) in the appropriate context
- Studies confounded
  - Small numbers
  - Non-controlled
  - Primarily warfarin (NOACs small numbers)

Case

• 65 year old woman with history of left hemisphere stroke 6 months prior presents to the ED

• Found unresponsive convulsing on floor, duration unknown at time of discovery

• EMS activated, lorazepam 2 mg IV given en route, brought to ED

• On examination, remains unresponsive with continuous rhythmic clonic jerks of the face and extremities, eye deviation to the right
What is the most appropriate next step?

A. Lorazepam 2-4 mg IV

B. Fosphenytoin 20 PE/kg IV

C. Lorazepam 2-4 mg IV now, and also ordering fosphenytoin

D. Anesthetic induction and intubation

E. Hide in the corner, maybe no one will notice
What is the most appropriate next step?

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# Status Epilepticus

## Definitions of Status Epilepticus.

<table>
<thead>
<tr>
<th>ILAE Definitions of Status Epilepticus</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Time after which if seizures do not terminate patient is considered in status epilepticus ($t_1$)</td>
<td>Time after which ongoing seizures have long term consequences ($t_2$)</td>
</tr>
<tr>
<td>Convulsive status epilepticus</td>
<td>5 min</td>
</tr>
<tr>
<td>Focal status epilepticus with impaired consciousness</td>
<td>10 min</td>
</tr>
<tr>
<td>Absence status epilepticus</td>
<td>10–15 min</td>
</tr>
</tbody>
</table>

## Other Definitions of Status Epilepticus

<table>
<thead>
<tr>
<th>Established status epilepticus</th>
<th>Status epilepticus that persists after treatment with a benzodiazepine (1st line treatment)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Refractory status epilepticus</td>
<td>Status epilepticus that persists after a 1st line agent (benzodiazepine) and 2nd lines agent (additional agent such as levetiracetam, phenytoin, valproic acid) have failed</td>
</tr>
</tbody>
</table>
Status Epilepticus

• Common hospital entity

• High mortality rates
  • Convulsive: up to 27% at 30 days
  • Non-convulsive: up to 65% at 30 days
  • Refractory: up to 61% at “discharge”

• Associated morbidity
  • Non-refractory: 39% return to functional baseline
  • Refractory: 15% independent (still moderately disabled)
Initial management

• Adrenaline runs high
• Remember to stay calm
• First issue: treat the seizures
• Second issue: address the source
  • Treating seizures difficult if etiology not adequately addressed
  • Concurrent with symptomatic treatment
Addressing the source

<table>
<thead>
<tr>
<th>Common Etiologies</th>
<th>Adults</th>
<th>Children</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stroke, including hemorrhagic</td>
<td>20%</td>
<td>10%</td>
</tr>
<tr>
<td>Low antiepileptic drug levels</td>
<td>35%</td>
<td>20%</td>
</tr>
<tr>
<td>Alcohol withdrawal</td>
<td>15%</td>
<td>—</td>
</tr>
<tr>
<td>Drug intoxication (theophylline, imipenem, isoniazid, beta-lactams, clozapine, bupropion, 4-aminopyridine, cocaine, etc) or withdrawal (benzodiazepine, barbiturate, baclofen)</td>
<td>5%</td>
<td>5%</td>
</tr>
<tr>
<td>Anoxic brain injury</td>
<td>15%</td>
<td>5%</td>
</tr>
<tr>
<td>Metabolic disturbances (low glucose, calcium, magnesium, or sodium level; high glucose level; renal failure; liver failure)</td>
<td>15%</td>
<td>5%</td>
</tr>
<tr>
<td>Infection (meningitis, encephalitis, brain abscess, sepsis)</td>
<td>5%</td>
<td>5%</td>
</tr>
<tr>
<td>Traumatic brain injury</td>
<td>2.5%</td>
<td>15%</td>
</tr>
<tr>
<td>Brain neoplasm</td>
<td>5%</td>
<td>0%</td>
</tr>
<tr>
<td>Febrile seizures</td>
<td>—</td>
<td>50%</td>
</tr>
<tr>
<td>Remote brain injury/congenital malformations</td>
<td>20%</td>
<td>40%</td>
</tr>
<tr>
<td>Idiopathic</td>
<td>5%</td>
<td>5%</td>
</tr>
</tbody>
</table>

Addressing the source

- History
- CT head (acutely)
- MRI brain (later on)

Labs
- CBC
- Glucose
- Complete metabolic panel
- Magnesium
- Calcium
- Phosphorus
- Lumbar puncture
- Urine drug screen
- Blood cultures
- Urinalysis and cultures
- AED levels (if on treatment)

- Administer dextrose and thiamine
- Just like ACLS, remember your ABCs: stabilizing the patient is priority
Treatment reality

Lorazepam 2 mg
Fos/phenytoin 1 g
Levetiracetam 1 g
Valproate 1 g
Other IV agent

Lorazepam 2 mg
Other IV agent
Lorazepam 2 mg

Intubation, anesthetic agent
Treatment algorithm

Lorazepam 0.1 mg/kg IV
or
Midazolam 10 mg IM

Fosphenytoin 18-20 PE/kg

Consider intubation, anesthetic agent

Helpful tips

- Lorazepam may be divided into 2 mg increments, but don’t be shy!

- Fosphenytoin (but not phenytoin) may be given IM if no IV access

- If you’re giving lorazepam, you should also be ordering the next line of treatment

Alternatives to fosphenytoin/phenytoin

• Why avoid the only FDA-approved second line agent?
  • If cardiac arrhythmia or hypotension is a problem in an unstable patient
  • Very significant hepatic disease

• __________ – 20-40 mg/kg IV
  • Avoid in hepatic disease

• ______________ – 2.5-4.5 grams IV
  • Requires dose adjustment for renal disease

• Lacosamide – 400 mg IV
  • Avoid in arrhythmia

(But what evidence do we have for these?)

Review

The relative effectiveness of five antiepileptic drugs in treatment of benzodiazepine-resistant convulsive status epilepticus: A meta-analysis of published studies

Zeid Yasiry, Simon D. Shorvon

**Table 1. Event rate and 95% CI**

<table>
<thead>
<tr>
<th>Study name</th>
<th>Event rate</th>
<th>Lower limit</th>
<th>Upper limit</th>
<th>Relative weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aiguabell et al., 2011</td>
<td>0.689</td>
<td>0.500</td>
<td>0.985</td>
<td>5.29</td>
</tr>
<tr>
<td>Alvarez et al., 2011</td>
<td>0.517</td>
<td>0.390</td>
<td>0.642</td>
<td>22.32</td>
</tr>
<tr>
<td>Benign et al., 2009</td>
<td>0.818</td>
<td>0.604</td>
<td>0.930</td>
<td>12.97</td>
</tr>
<tr>
<td>Eue et al., 2011</td>
<td>0.556</td>
<td>0.432</td>
<td>0.673</td>
<td>22.65</td>
</tr>
<tr>
<td>Knafe et al., 2008</td>
<td>0.846</td>
<td>0.549</td>
<td>0.991</td>
<td>8.61</td>
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<tr>
<td>Misra et al., 2011</td>
<td>0.700</td>
<td>0.376</td>
<td>0.900</td>
<td>9.98</td>
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<tr>
<td>Ruegg et al., 2008</td>
<td>0.632</td>
<td>0.403</td>
<td>0.813</td>
<td>15.09</td>
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<tr>
<td>Standish et al., 2010</td>
<td>0.955</td>
<td>0.552</td>
<td>0.997</td>
<td>3.11</td>
</tr>
<tr>
<td>Summary estimate</td>
<td>0.685</td>
<td>0.562</td>
<td>0.787</td>
<td></td>
</tr>
</tbody>
</table>

**Summary**

Event rate and 95% CI

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<th>Study name</th>
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<tbody>
<tr>
<td>Kolwero et al., 2003</td>
<td>0.667</td>
<td>0.376</td>
<td>0.869</td>
<td>33.20</td>
</tr>
<tr>
<td>Malamiri et al., 2012</td>
<td>0.767</td>
<td>0.585</td>
<td>0.884</td>
<td>66.80</td>
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<tr>
<td>Summary estimate</td>
<td>0.736</td>
<td>0.583</td>
<td>0.848</td>
<td></td>
</tr>
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</table>

**Fig. 2. Forest plot for efficacy of phenobarbital; CI: confidence interval.**

**Table 2. Event rate and 95% CI**

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<tbody>
<tr>
<td>Agawal et al., 2009</td>
<td>0.840</td>
<td>0.711</td>
<td>0.918</td>
<td>13.71</td>
</tr>
<tr>
<td>Alvarez et al., 2011</td>
<td>0.568</td>
<td>0.468</td>
<td>0.665</td>
<td>15.37</td>
</tr>
<tr>
<td>Brevard et al., 2006</td>
<td>0.297</td>
<td>0.198</td>
<td>0.419</td>
<td>15.05</td>
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<tr>
<td>Frazier et al., 2006</td>
<td>0.455</td>
<td>0.365</td>
<td>0.569</td>
<td>13.17</td>
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<tr>
<td>Ismail et al., 2012</td>
<td>0.265</td>
<td>0.144</td>
<td>0.435</td>
<td>13.67</td>
</tr>
<tr>
<td>Miyashita et al., 2009</td>
<td>0.029</td>
<td>0.043</td>
<td>0.986</td>
<td>4.02</td>
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<tr>
<td>Ogita et al., 2003</td>
<td>0.364</td>
<td>0.143</td>
<td>0.661</td>
<td>10.60</td>
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<tr>
<td>Tiwari &amp; Savanayavith, 2009</td>
<td>0.459</td>
<td>0.308</td>
<td>0.619</td>
<td>14.40</td>
</tr>
<tr>
<td>Summary estimate</td>
<td>0.602</td>
<td>0.342</td>
<td>0.961</td>
<td></td>
</tr>
</tbody>
</table>

**Summary**

Event rate and 95% CI

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<tbody>
<tr>
<td>Agawal et al., 2009</td>
<td>0.880</td>
<td>0.758</td>
<td>0.945</td>
<td>14.22</td>
</tr>
<tr>
<td>Alvarez et al., 2011</td>
<td>0.746</td>
<td>0.620</td>
<td>0.841</td>
<td>17.00</td>
</tr>
<tr>
<td>Chang et al., 2010</td>
<td>0.588</td>
<td>0.352</td>
<td>0.790</td>
<td>13.08</td>
</tr>
<tr>
<td>Chen et al., 2011</td>
<td>0.500</td>
<td>0.328</td>
<td>0.672</td>
<td>15.65</td>
</tr>
<tr>
<td>Malamiri et al., 2012</td>
<td>0.900</td>
<td>0.732</td>
<td>0.987</td>
<td>10.97</td>
</tr>
<tr>
<td>Olsen et al., 2007</td>
<td>0.730</td>
<td>0.597</td>
<td>0.848</td>
<td>15.55</td>
</tr>
<tr>
<td>Tiwari &amp; Savanayavith, 2009</td>
<td>0.750</td>
<td>0.448</td>
<td>0.917</td>
<td>10.04</td>
</tr>
<tr>
<td>Yu et al., 2003</td>
<td>0.969</td>
<td>0.650</td>
<td>0.998</td>
<td>3.50</td>
</tr>
<tr>
<td>Summary estimate</td>
<td>0.757</td>
<td>0.637</td>
<td>0.848</td>
<td></td>
</tr>
</tbody>
</table>

**Fig. 3. Forest plot for efficacy of phenytoin; CI: confidence interval.**

**Fig. 4. Forest plot for efficacy of valproate; CI: confidence interval.**
Established status epilepticus treatment trial (ESETT)

- Multicenter double-blind RCT
  - Goal 795 patients
  - Intention to treat
  - Enrolled if seizure >5 min after adequate benzodiazepine

- 3 treatment arms, all infused over 10 minutes
  - Fosphenytoin 20 PE/kg
  - ____________ 60 mg/kg
  - Valproate 40 mg/kg

- At 60 minutes post intervention:
  - Seizure
  - Mental status
  - Adverse events

- Chart review for admission status, requirement

Case (Patient 1)

- 54 year old man develops weakness in the feet and trouble with walking → arm weakness following day

- Neurologic consultation initiated:
  - Diffuse moderate weakness, including neck flexors and extensors
  - Short of breath when laying supine
  - Absent reflexes

- Lumbar puncture
  - WBC 3
  - Protein 115
  - Glucose normal
Case (Patient 2)

• 73 year old woman with generalized myasthenia gravis

• For several days, had problems controlling secretions and changes in speech

• Increasing doses of Mestinon not helpful, so prednisone initiated at a high dose

• Presents to the ED with continued speech and swallowing difficulty, now feeling short of breath

• Exam: diaphoretic, chest expansion and neck contraction with inspiration; facial weakness with dysarthria, globally weak
Which patient is clinically most in danger of respiratory failure?

A. Patient 1, because it is more acute in onset
B. Patient 1, because he has orthopnea
C. Patient 2, because she is using accessory muscles to breath
D. Patient 2, because she is older
Which patient is clinically most in danger of respiratory failure?

A. Patient 1, because it is more acute in onset

B. Patient 1, because he has orthopnea

C. Patient 2, because she is using accessory muscles to breath

D. Patient 2, because she is older
Neuromuscular respiratory failure

- Common complication of acute or chronic neurologic disease
  - Acute disease or exacerbation primarily resulting in weakness (AIDP, MG)
  - Decompensation of stable chronic disease during concurrent illness (MG, ALS, etc.)

- Up to 30% of AIDP and MG patients will require mechanical ventilation

Mehta S. *Respir Care* 2006;51:1016.
Symptoms

- Dyspnea
- Orthopnea (SOB when laying flat)
- Tachypnea
- Accessory respiratory muscle use
- Paradoxical breathing

- Dysarthria
- Dysphagia
- Weak cough
- Aspiration
- Hypersomnolence
- Encephalopathy
Paradoxical breathing

I think I got... worse... the last 2 days, particularly... in the... arms.

Forehead sweating

Increased sternomastoid activity

Tachypnea

Abdominal paradox

Mittal MK and Wijdicks EFM. In Critical Care Medicine: Principles of Diagnosis and Management in the Adult. p1121-1129.
Evaluation

• ABCs – stabilize the patient
  • Clinical assessment: Does the patient have impaired consciousness or appear obviously distressed?
  • If the answer is yes, intubate*

• “20-30-40” rule – serial checks
  • Forced vital capacity < 20 ml/kg (or <1 L)
  • Max inspiratory pressure < -30 cm H₂O
  • Max expiratory pressure < 40 cm H₂O

• ABG – but remember that a lack of hypoxemia or hypercapnia does not exclude badness

*In cases of chronic progressive disease like ALS, try to confirm patient goals of care
Evaluation

- Testing to identify a systemic contributors in chronic disease decompensation
  - Infection (CBC, blood cultures, urinalysis)
  - Medication changes or compliance issues
  - Metabolic abnormalities
- Testing to identify a primary neurologic diagnosis (if still needed)
  - Imaging, if indicated
  - LP
  - EMG
  - Myasthenia antibodies
Treatment

- Assistance with ventilation when appropriate
  - Intubation
    - Mortality: 12-20% in AIDP, 4-8% in MG
    - Average duration: 18-29 days in AIDP, 14 days in MG
  - BiPAP – may not perform well if significant bulbar weakness

- Address primary causes
  - Plasma exchange (AIDP, MG)
  - IVIg (AIDP)
  - Medication adjustments (MG)

- Address systemic contributors, if identified
Questions & Discussion