Objectives

- Emphasize the importance and ubiquity of acute neurological problems. (e.g., a common medical emergency on commercial airplanes)
- Provide an overview of the most common neuroemergencies, such as acute meningitis, status epilepticus, SAH, cerebral edema, increased ICP, Wernicke’s encephalopathy, acute weakness
- Discuss symptoms and signs that can help identify a neurological emergency
- Discuss steps to manage some common neurological emergencies.

Is It an Emergency?

- Is the acute condition itself life-threatening or disabling if untreated?
- Is there a risk to the community?

Is this an emergency?

You need to get the full H&P!

Or, is this an emergency?

You need to get the full H&P!
How about this one?

You need to get the full H&P!

Basic Algorithm

- Stabilize patient: ABCDs + N + T
- Prevent further injury to patient (or others)
- Identify the physiological process
- Identify pathophysiology (i.e. diagnose)
- Institute treatment or antidote
- Other: get 2 peripheral IVs, stabilize the neck, collect blood and urine samples, consider sending a pregnancy test, give thiamine IV

For the diagnosis of altered mental status, know the VITAMINS!!

- Vascular
- Infectious
- Traumatic, Toxic
- Allergic, Autoimmune
- Metabolic
- Inherited
- Neoplastic, Nutritional
- Seizures

Some Categories

- Increased intracranial pressure (ICP)
- Wenicke’s encephalopathy -- Metabolic dysfunction – IV thiamine
- Neuromuscular failure - EMG
- Acute meningitis - LP
- Status epilepticus - EEG
- SAH - CT
- Acute pain

Increased ICP

- Cushing’s triad: decreased mental status, systemic hypertension and bradycardia – are the consequence of increased ICP and the brain is trying to protect itself by increasing the blood pressure (CPP=MAP-ICP)
- Other symptoms are headache, lethargy, vomiting, progressive neurological deficits
- May be due to many conditions
- Consider hemorrhage, tumor, trauma, hydrocephalus, drugs

Increased ICP Management

- Diagnose
  - non-contrasted CT scan
  - ICP monitor
- Treat
  - Keep neck in neutral position
  - Head of bed 30° up
  - Mannitol – shrinks the normal brain
  - Other: hypertonic saline, barbiturates
  - External ventricular drain (EVD)
  - Large craniotomy
- Steroids for vasogenic cerebral edema (tumor-related)
- Acetazolamide for pseudotumor cerebri
Careful!!!!

- If the cause of the systemic arterial hypertension is increased ICP, you treat the ICP to lower the blood pressure.
- Lowering the blood pressure in the context of increased ICP will render the brain ischemic and aggravate the problems.

Hypertensive Encephalopathy

- Diastolic BP >130 torr, acute end-organ dysfunction
- Average age 40 years, men more often than women
- Some causes include: 1ry hypertension, eclampsia, RAS, pheochromocytoma, drugs (cocaine, others)
- Papilledema, headache, vomiting, visual disturbance, seizures, altered mental status
- Overly aggressive BP Rx can lead to cerebral ischemia; gradually done is the primary Rx
- Admit to ICU, Rx with IV anti-HTN (like esmolol, labetalol, furosemide, nicardipine, hydralazine)

The treatment in hypertensive emergencies is to lower the systemic blood pressure, not to lower the ICP!! Yet, clinically, these 2 emergencies look alike.

Case

45 year old female with history of chronic systemic hypertension is brought to the emergency room complaining for 2-3 days of increasing headaches and blurred vision. Her BP (both arms) is 240/140 torr, HR is 90. On exam her GCS is 13, and she has bilateral papilledema.
- What is the differential diagnosis?
- How would you NOT treat her?

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Cerebral Blood Flow Autoregulation

Wernicke’s Encephalopathy

- A medical emergency!!!
- The common denominator is malnutrition: hyperemesis, bulimia, some alcoholics are malnourished, short-bowel syndrome, cirrhotics, malabsorption syndromes, prolonged NPO states, etc.
- The symptoms and signs are vague, only 10-15 have the “classic” triad of confusion, ophthalmoparesis and gait instability
- Treat upon suspicion, with IV thiamine, 100 mg for at least 3 days, (not PO)
Case
A 53 year old smoker, man comes to the ER complaining that 30 minutes ago, suddenly, he had the onset of headache: “It felt like if truck hit me”. He almost passed out. On exam, GCS 15, his blood pressure is 160/90. He has mild meningeal signs.

• If this is not ICH, venous thrombosis or migraine, what could have caused this problem?
• If the non-con head CT is negative, what would you do?, and when?

Non-traumatic SAH
• Acute basal artery aneurysmal rupture.
• The ruptured aneurysm leaks volume AND pressure
• The pressure “leak” equalizes the MAP and the ICP leading to CPP=0: loss of consciousness
• Initial Hunt-Hess grading helps determine prognosis
• Complications include hydrocephalus, neurogenic stunned myocardium, cerebral vasospasm (leading to strokes)
• Induced arterial hypertension and nimodipine prevent strokes from vasospasm
• Clip or coil the aneurysm - EARLY

Large, fatal, aneurysmal SAH

How about, if you suspect acute SAH, but the CT scan does not show the bleed - what is your next step?

• Hold on to the patient until 6 hours have elapsed from the initial ictus, then do the LP looking for xanthochromia.

Case
55 year old man plantation field worker that you see in the emergency room complaining of few hours of progressive difficulty swallowing and speaking. On exam he has weakness of the bulbar skeletal musculature. He sits forward drooling on the floor.
You call Neurology to come evaluate him for “acute stroke”, right?
Neuromuscular Junction

- Immune-mediated (myasthenia gravis, Lambert-Eaton myasthenic syndrome)
- Genetic
- Infectious (botulism)
- Toxic

Botulism

- Acute onset of dysphagia, dysarthria, ptosis
- Autonomic dysfunction
- Seen in illegal drug users; injuries (sometimes minor)
- Treatment: early diagnostic suspicion, antitoxin, wound debridement, penicillin, supportive therapy (for weeks)

Case

You are volunteering in a hospital in Brazil, when they bring to you a 27 year old farmer, who while at work became acutely confused, mildly weak with diarrhea, urinary incontinence, sweaty and sialorrhea.

His heart rate is 52, BP is 90/50.
Both pupils are small and reactive.
He has prominent rhonchi.
What do you suspect he has?
How should you manage and treat him?

Organophosphate Poisoning

- Diaphoresis, salivation, miosis, lacrimation
- Smooth and skeletal muscle contraction and cramps, defecation
- Seizures, delirium, blurry vision, ataxia
- Bronchospasm and bronchorrhea
- Tachy- or bradycardia, hypo- or hypertension
- Atropine 1-2mg IV (preservative free)
- Prolidoxime (2-PAM) 1-2g/250mL IV over 10 min
- Remove offending agent (e.g. clothes)
- Check RBC cholinesterase

What About Bioterrorism?

- Many terrorist weapons are neurotoxins or neuroinfections
- Including:
  - Organophosphates, nerve gasses
  - Botulism
  - Mycotoxins (trichothecene)
  - Anthrax
  - Viral encephalitis
  - Q fever
- Infectious agents have been developed, but only anthrax has been successfully used in recent history

Case

An 8-year-old boy began this morning complaining of headache, fatigue, sore throat, and generalized muscle pain. While waiting to be seen he experienced a sudden shaking chill, nausea, lightheadedness and increased headache. Over the next 30-45 minutes he became increasingly ill and eventually “delirious” according to the ER nurse, who measured his oral temperature at 39.8 °C. The skin exam reveals the appearance of reddish-purple spots over the patient’s extremities.

What are some diagnostic possibilities?
If it were meningococcemia, what would the risk to the community be?
**Acute Bacterial Meningitis**
- Headache, behavioral change, nuchal rigidity, and fever
- Common agents are: *S. pneumoniae*, *H. influenza*, *Listeria*
- *N. meningitidis* is particularly contagious
- Don’t forget chest x-ray, cultures, underlying conditions
- CSF will show increased cells and proteins with decreased glucose.
- *Hyper-early, appropriate empiric antibiotics therapy is imperative*: ceftriaxone, ampicillin, vancomycin
- Dexamethasone prior to, or with the antibiotics
- Rifampicin prophylaxis for *N. meningitidis* contacts

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**Encephalitis**
- Most commonly is a viral infection, can lead to cerebral abscess, specially with bacterial infections
- HSV-1 - most important virus; others include arboviruses (WEE, EEE, St. Louis, La Crosse), West Nile, HIV, CMV
- Supportive management, hydration, nutrition, ICP monitoring
- CSF, MRI, EEG, biopsy
- Antiviral agents (eg. for HSV use acyclovir)
- For bacteria: ceftriaxone, vancomycin
- Community risk needs to be assessed

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**Acute Ischemic Stroke**
- IV tPA is helpful if administered within the first 3 hours of onset, and if there is no contraindication (READ THESE: eg. NIHSS <6 or >22, CT scan w/o hemorrhage, recent surgery, prior hemorrhage, anticoagulation with heparin or warfarin (INR > 1.7), thrombocytopenia (plt <100k), severe HTN (>165/100), hyper- or hypoglycemia (<50, >400), prior stroke or head injury within 3 months, recent MI, GIB or UG within 3 wks, seizure, rapid recovery)
- Brain injury swell, peak swelling occurs at 3-4 days
- Major acute treatment aim is to prevent a second stroke, and to maximize recovery
- Intra-arterial thrombolytics may be useful in special situations, but there is a high complication rate
**Status Epilepticus**
- Two or more seizures not separated by a period of lucidity
- A B C D + N
- Protect patient and others from physical injury
- Can result in metabolic brain cell death or cardiopulmonary arrest
- Anticonvulsants: lorazepam IV, phenytoin, propofol (requires intubation)
- If in doubt, give thiamine IV
- Labs may include AED levels and drug screen, calcium, magnesium, glycemia, cultures, myoglobin
- Obtain drug history and compliance, also illegal drug use, travel occupational exposures

**Malignant Hyperthermia**
- Unexpected rise in body temperature when anesthetics are administered
- Increased, involuntary skeletal muscle and autonomic hyperactivity
- Suspect an underlying myopathy, esp. central core disease
  - Discontinue anesthesia
  - Dantrolene
  - Hydration
  - EMG?
  - Muscle biopsy?
  - Specific genetic test?

**Questions?**

**Neurological Emergencies**

**References**
4) Harrison’s Internal Medicine, 16th
5) Goez Textbook of Clinical Neurology, 2nd