NEUROLOGIC EMERGENCIES

R. Keith Bailey, MD, MPAS
VAPAA Conference
Houston Texas
2017
Points To Ponder

- PANRE may test on any patient condition
- However, there are no reported unconscious patient situations on test
Neurologic Emergency Outline

• Change in Mental Status / Coma
• Stroke/TIA Syndromes
• Seizure & Status Epilepticus
• Head Trauma
• Infectious
• Vertigo/Headaches
• Peripheral Neuropathies
The Neurologic Exam

• KEY!! Must do a complete thorough neuro exam to properly identify and diagnose any neurologic abnormality.

• Exam should include 5 parts:
  – Mental status, level of alertness (GCS)
  – Cranial nerve exam
  – Motor / Sensory exam
  – Reflexes
  – Cerebellar
  – Consider ; MMSE if Psych components
Define Mental Status

State of consciousness: If not fully awake suspect metabolic disorder or space occupying lesion
Orientation: person, place, time  AOx3
New memory: recall 3 or 4 unrelated items in 5 min.
Knowledge of current events
Old Memory: correct info about distant past
Calculation: simple math 2+2 = 4 (before)
Abstract thought: Ball and Orange alike
Judgment: What would he do if he spotted a fire in a theatre
Picture interpretation: focus on one tiny spot
What is mood: Cheerful, fearful, paranoid
Change in Mental Status / COMA

- Potential Causes – ‘AEIOU TIPS’
  - A = Alcohol (Drugs & Toxins)
  - E = Endocrine, Exocrine, Electrolyte
  - I = Insulin
  - O = Opiates, OD
  - U = Uremia
  - T = Trauma, Temperature
  - I = Infection
  - P = Psychiatric disorder
  - S = Seizure, Stroke, Shock, Space occupying lesion
• **Temperature**
  - Hypothermia: causes coma when Temp<32.0°C/89.6°F
  - Hyperthermia: causes coma when Temp>42.0°C/107.6°F

• **Infection**
  - Meningitis, Encephalitis, Sepsis

• **Endo/Exocrine, Electrolyte**
  - Hypo/Hyperglycemia
  - Hypo/hyperthyroidism
  - Hypo/hypernatremia
  - Hepatic encephalopathy

• **Opioids/ OD / Alcohol**
  - Heroin, Psych Meds (TCA’s, SSRI’s)
Change in Mental Status/Coma

- T for Trauma
S for Stroke / Space Occupying Lesions
Space Occupying Lesion
• Always attempt to get a complete history!!
• LOOK at your patient!
  – Smell the breath (ketones, alcohol, fetid)
  – Observe respiratory rate & patterns (Cheyne-Stokes)
  – Look for abnormal posturing.
    • Decorticate (Flexion of UE with Extension of LE)
    • Decerebrate (Extension of all Ext.)
  – Look for needle marks, cyanosis, signs of trauma
• Obtain GCS Score!  E4 V5 M 6
  – If less than 8, IMMEDIATE airway stabilization FIRST priority!!
Glasgow COMA Scale

- Scores range from 3 (Worst) – 15 (Best)
- Important for classifying degree of alteration. (Head Trauma)
- GCS < 8 = INTUBATE!!
- EYE Opening Response
  - 4 = Spontaneous
  - 3 = To Voice
  - 2 = To Pain
  - 1 = None
- Remember as “4 eyes”
Glasgow COMA Scale

- **Verbal Response**
  - 5 = Oriented and converses
  - 4 = Confused but converses
  - 3 = Inappropriate words
  - 2 = Inappropriate sounds
  - 1 = None
Glasgow COMA Scale

- **Motor**
  - 6 = Obeys commands
  - 5 = Localizes pain
  - 4 = Withdraws to pain
  - 3 = Decorticate (flexes to pain)
  - 2 = Decerebrate (extends to pain)
  - 1 = None
AMS / COMA Essential Stabilization & Assessment Measures

• Always assess & stabilize ABC’s first
  – special attention to airway with C-Spine immobilization / protection. Oxygenate!
  – IV line, fluids, Thiamine 100mg IV, 1 amp D 50, & Narcan (if needed) 0.4mg increments until response.

• Complete history and physical exam after stabilization

• Radiographic clearance of C-Spine

• Labs / CT as indicated
Anatomy of Cerebral Blood Flow

- Anterior Circulation: 80% of cerebral blood flow originates from the carotids which supplies the
  - Frontoparietal lobes
  - Anterior temporal lobes
  - Optic nerve and retina

- Posterior Circulation: 20% of cerebral blood flow which originates from the vertebrobasilar arteries
  - Thalamus & Brainstem
  - Occipital cortex and Cerebellum
  - Upper Spinal cord & Auditory and Vestibular functions in ear

- Circle of Willis: connects the Anterior and Posterior circulations
Ischemic Strokes: (thrombi or emboli)

- Cerebral Thrombi may result from:
  - Atherosclerosis (#1 cause)
  - Infective arteritis
  - Vasculitis
  - Hypercoagulable states
  - Post traumatic carotid or vertebral artery dissections

- Cerebral emboli may result from:
  - Mural thrombus from heart (#1 cause)
  - Aortic plaques
  - Endocarditis
  - Long bone or Dysbaric injuries (fat / air emboli)
Hemorrhagic Strokes result from
- Spontaneous rupture of berry aneurysm or AV malformation (Subarachnoid hemorrhage)
- Rupture of arteriolar aneurysms secondary to:
  - Hypertension
  - Congenital abnormality
  - Blood dyscrasia / Anticoagulant usage
  - Infection
  - Neoplasm
- Trauma (Epidural / Subdural Hematomas)
- Hemorrhagic transformation of embolic stroke
Type of Stroke (rule of 2/3’s)
- 2/3 of ALL Strokes are ISCHEMIC
  - 2/3 of these are thrombotic
  - Therefore thrombotic, ischemic strokes most common.
- Incidence of Stroke
  - Biggest Risk Factors
    - Prior TIA (30% will have stroke in 5 years)
    - HTN
    - Atherosclerosis
    - DM
    - Hyperlipidemia
    - Smoking
Ischemic Stroke Syndromes: Thrombotic vs. Embolic

- **Thrombotic Syndromes**
  - Usually slow, progressive onset
  - Sx develop shortly after awakening and are progressive

- **Embolic Syndromes**
  - Usually abrupt onset with maximal deficit that tends to improve over time as the embolus breaks up.
Middle Cerebral Artery Occlusion (MCA)

- # 1 type
- Contralateral hemiplegia, hemianesthesia, and homonymous hemianopsia
- Upper extremity deficit >> Lower extremity
- Aphasia (if dominant hemisphere involved)
- Conjugate gaze impaired in the direction of the lesion
Ischemic Stroke Syndromes

- **Anterior Cerebral Artery Occlusion (ACA)**
  - Contralateral leg, arm, paralysis
  - Lower Extremity deficit >> Upper extremity
  - Loss of frontal lobe control
    - Incontinence
    - Primitive grasp and suck reflexes enacted

- **Posterior Cerebral Artery Occlusion (PCA)**
  - Ipsilateral CN III palsy, visual loss
  - Contralateral hemiparesis and hemisensory loss
  - Memory loss
Vertebrobasilar Artery Occlusion (VBA)

Hallmark: Crossed Neurological Deficits

CN AND Cerebellar deficits that affect BOTH sides of the body, with contralateral pain and temperature deficits.

- Contralateral hemiplegia
- Ipsilateral CN III palsy with Cerebellar findings.
  - Nausea/Vomiting
  - Vertigo, Nystagmus,
  - Ataxia, Dysarthria
  - Tinnitus, deafness
TIA’s (Transient Ischemic Attacks)

- Definition: A temporary loss of neurologic function, that resolves completely < 24 hours.
- Clinically;
  - Arm numbness, weakness, HA
  - Facial droop, slurred speech
  - Sx resolved, or improve over time
- Main point: These patients at high risk for stroke if:
  - > 50
  - HTN, DM, Smoker, Prior TIA in last month
  - Any prior CVA…… ADMISSION IS THE RULE!!
- Treat as CVA: Head CT (CVA protocol)
- ASA 81-325mg po
- Oxygen, 2L NC
- If cardiac arrhythmia (atrial fibrillation) present, consider Heparin ONLY after Head CT and Neuro consultation.
• Subarachnoid Hemorrhage
  – Highest incidence in 35-65 year old.
  – Usually from the rupture of a berry aneurysm
  – Clinically:
    • abrupt onset of “worst headache of life”
    • Nuchal rigidity, photophobia, vomiting, retinal hemorrhages.
  – Diagnosis: CT + LP!!!!
    • CT only 92% sensitive within 24 hours of event, loses sensitivity >24 hours out from headache.
    • 72 hours out CANNOT r/o without LP!
  – Management:
    • Consider adding Nimodipine 60 mg Q6 to reduce vasospasm
Hemorrhagic Stroke Syndromes

• Intracerebral
  – Hypertensive intracerebral hemorrhage MOST common cause.
  – Traumatic, contusion, coup/contracoup
  – Rupture of small blood vessels with bleeding inside the brain parenchyma
    • Putamen
    • Cerebellar
    • Thalamus
    • Pontine (3 P’s – pinpoint pontine pupils)
Intracerebral Hemorrhage
Treatment of Stroke

• AS ALWAYS – ABC’s FIRST
• What’s the Serum Glucose??
  – Consider Thiamine 100mg IV, D 50 bolus if hypoglycemic.
  – Treat Hyperglycemia if Serum Glucose > 300mg/dl
• Protect the brain
  – Keep SBP >90mm Hg
  – Goal keep CPP > 60mm Hg (CPP=MAP-ICP)
  – Treat Fever (Mild Hypothermia beneficial)
    • Acetaminophen 650mg po or pr, cooling blanket
  – Oxygenate (Keep Sao2 >95%)
  – Elevate head of bed 30 deg. (Clear c-spine)
• Frequent repeat Neuro checks!! Reassess GCS!
Treatment of Stroke

- What type of stroke is Present??
  - Hemorrhagic vs Ischemic
    - Any signs of shift herniation?
    - Neurosurgery evaluation or transfer necessary?
- Other management adjuncts:
- **Ischemic** strokes
  - ASA 81-325mg
  - Patients with Systolic BP >220, Diastolic >120 need BP control with Nitroprusside or Labetolol.
  - DO NOT OVERTREAT BP or risk extending the infarct.
  - Heparin not shown to be of benefit in recent studies, however, still frequently used.
    - Consult Neurologist before use
    - If used, No bolus, just infusion.
    - Risk of hemorrhagic transformation.
Treatment of Strokes

- **Strokes with Edema, Mass Effect or Shift**
  - Load with Phosphenytoin 1000mg for seizure prophylaxis
  - Acute seizure prophylaxis still of benefit.
  - Mannitol, Decadron??
    - Recently shown to be of NO benefit, some Neurosurgeons still advocate, so consult first.
  - Hyperventilation??
    - NOT beneficial and perhaps harmful, don’t do it!

- **Thrombolytics??**
  - Ischemic strokes ONLY with large deficit NOT improving.
  - Time from symptom onset <3 hours
  - No ABSOLUTE Contraindications!!
  - Inclusion and Exclusion Criteria
  - Benefit Questionable
Thrombolytic Therapy for Acute Stroke Checklist

- Answer to ALL must be **YES**:
  - Age 18 or older
  - Clinical diagnosis of Acute Ischemic Stroke causing a measurable NON improving neurologic deficit.
  - NO high clinical suspicion for SAH
  - Time of onset to treatment is <180 minutes.
Thrombolytic Therapy for Acute Ischemic Stroke Checklist

- Answer to ALL MUST be NO:
  - Evidence of hemorrhage on CT
  - Active internal bleeding (GI/GU) within last 21 days.
  - Known bleeding diasthesis:
    - Platelets<100,000
    - Heparin within last 48 hours with elevated PTT
    - Warfarin use with PT > 15 seconds
  - Within 3 months of IC injury, prior surgery or prior ischemic stroke.
  - Within 14 days of serious trauma, major surgery
  - Recent AMI, arterial puncture/LP within 7 days
  - History of prior ICH, AVM, tumor, or aneurysm or seizure at stroke
  - Systolic BP >185mmHg, or Diastolic BP >110Hg
Seizures & Status Epilepticus

• Background:
  – 1 – 2% of the general population has seizures
  – Primary
    • Idiopathic epilepsy: onset ages 10-20
  – Secondary
    • Precipitated by one of the following:
      • Intracranial pathology
        – Trauma, Mass, Abscess, Infarct
      • Extracranial Pathology
        – Toxic, metabolic, hypertensive, eclampsia
**Seizure Types**

- **Generalized Convulsive Seizures (Grand Mal):**
  - Tonic, clonic movements, (+) LOC, apnea, incontinence and a post-ictal state

- **Non Convulsive Seizures (Petit Mal):**
  - Absence seizures - “blank staring spells”
  - Myoclonic - brief contractions of selected muscle groups

- **Partial Seizures**
  - Characterized by presence of auditory or visual hallucinations
  - Simple = somatic complaints + no LOC
  - Complex = somatic complaints + AMS or LOC
Approach for 1st Seizure, New Seizure, or Substance/ Trauma Induced Seizure

- As always ABC’s First
- IV, O2, Monitor.
  - Send blood for CBC, Chem 20, Tox screen as appropriate
  - Anticonvulsant levels
  - Prolactin levels / Lactate level
- CXR / UA/ Head CT
- Is patient actively seizing? Post ictal? Pseudoseizure?
  - Consider treatment options
- Complete History and Physical Exam
  - Including detailed Neuro Exam
  - Repeat Neuro evaluations a must!
Guidelines for Postictal Head CT Scans

- Status Epilepticus (a true emergency)
- Abnormal Neuro findings
- No return to GCS 15
- Prolonged HA
- History of malignancy
- CHI (Closed Head Injury)
- HIV infection or high risk for HIV
- Anticoagulant use
- Age > 40
As Before, But History, History, History!!

Main causes of Breakthrough Seizure:
- Noncompliance with anticonvulsant regimen
- Start of new medication (level alteration)
  - Antibiotics, OCP’s
- Infection
  - Fever
- Changes in body habitus, eating patterns
- Supratherapeutic level
**Status Epilepticus**

- **Definition**: operationally defined as seizure lasting greater than 5 minutes OR two seizures between which there is incomplete recovery of consciousness.

- **Treatment algorithm**:
  - As before ABC’s
  - IV, O2, Monitor
  - Consider ALL potential causes
    - INH (pyridoxime/B-6 deficiency)
    - Eclampsia
    - Alcoholic (thiamine/B-1 deficiency)
    - Other Tox ingestion (TCA’s, sulfonylurea OD)
    - Trauma
Status Epilepticus Treatment

- **FIRST LINE TREATMENT**
  - Lorazepam (Ativan) 2mg/min IV up to 10 mg max. OR Diazepam (Valium) 5mg/min IV or PR up to 20mg

- **SECOND LINE TREATMENT**
  - Phenytoin or Fosphenytoin (Cerebyx):
    - 20mg/kg IV at rate of 50mg/min

- **THIRD LINE TREATMENT**
  - Get Ready to intubate at this point!!
  - Phenobarbital 10-20mg/kg @ 60 mg/min
Status Epilepticus Treatment

• FINAL TREATMENT
  – Barbiturate Coma
    • Pentobarbital 5mg/kg @ 25 mg/min
    • Stat Neurology consult for evaluation and EEG
    • Pentobarbital titrated to EEG response.

• Always get a through HISTORY
  – Possible trauma
  – Medications in house
  – Others sick, symptomatic
  – Overall appearance of patient
Status Epilepticus Adjunctive Treatment by History

- **Thiamine 100mg IV, 1-2 amps D 50**
  - If suspect alcoholic, malnourished, hypoglycemia
- **Magnesium Sulfate 20cc of 10% solution**
  - As above of if eclampsia (BP does NOT have to be 200/120!!)
- **Pyridoxine 5 gms IV**
  - INH or B-6 deficiency
Closed Head Injury

- **Definitions:**
  - Concussion/TBI: refers to a transient LOC following head injury. Often associated with retrograde amnesia that also improves.
  - “Coup” = injury beneath the site of trauma
  - “Countrecoup” = injury to the side polar opposite to the traumatized area.
  - Diffuse Axonal Injury: tearing and shearing of nerve fibers at the time of impact secondary to rapid acceleration/deceleration forces. Causes prolonged coma, injury, with normal initial head CT and poor outcome.
The single most important factor in the neurologic assessment of the head injured patient is level of consciousness. (LOC)

Always assume multiple injuries with serious mechanism.
- ESPECIALLY C-SPINE!!!!
- Unless hypotensive WITH bradycardia and WARM extremities (spinal cord injury); hypotension is ALWAYS secondary to hypovolemia from blood loss in the trauma patient!

The most common intracranial bleed in CHI is subarachnoid hemorrhage.
Closed Head Injuries with Hemorrhage

- Cerebral Contusion
  - Focal hemorrhage and edema under the site of impact.
  - Susceptible areas are those in which the gyri are in close contact with the skull
    - Frontal lobe
    - Temporal lobes
  - Diagnostic Test of Choice: NC Head CT
  - Treatment: Supportive with measures to keep ICP normal. Repeat Neuro checks. Repeat Head Ct in 24 hours. Good prognosis.
Cerebral Contusion
Subdural Hematoma

- Occurs secondary to acceleration/deceleration injury with resultant tearing of the bridging veins that extend from the subarachnoid space to the dural sinuses.
- Blood dissects over the cerebral cortex and collects under the dura overlying the brain.
- Patients at risk:
  - Alcoholics
  - Elderly
  - Anticoagulant users
- Appears as “sickle shape” and does not extend across the midline
Subdural Hematoma
Epidural Hematoma

- Occurs from blunt trauma to head especially over the parietal/temporal area.
- Presents as LOC which then patient has lucid interval then progressive deterioration, coma, death. (Patient talks to you & dies!)
- Commonly associated with linear skull fracture
- Mechanism of bleed is due to tear of artery, usually middle meningeal.
- PE reveals ipsilateral pupillary dilatation with contralateral hemiparesis.
- CT Scan: a BICONVEX (lens) density which can extend across the midline
Epidural Hematoma
Management of Closed Head Injuries

- As always ABC’s with C-Spine precautions
- IV, O2, Monitor.
- Stabilize and resuscitate
  - Sao2>95%
  - SBP>90
  - Treat Fever
- Head of Bed 30% (once C-Spine cleared)
- Stat Head CT with Stat Neurosurgical evaluation for surgical lesions.
- Repeat Exams, looking for signs of herniation.
Signs of Herniation / Increased ICP

- Headache, nausea, vomiting
- Decreasing LOC
- Sixth nerve paresis (one or both eyes adducted)
- Decreased respiratory rate
- Cushing reflex (hypertension/bradycardia/bradynpea)
- Papilledema
- Development of signs of herniation
  - Fixed and dilated pupil
  - Contralateral hemiparesis
  - Posturing
Herniation Syndromes

- CPP = MAP – ICP: Must keep CPP >60 mm Hg
- Uncal Herniation:
  - Occurs when unilateral mass pushes the uncus (temporal lobe) through the tentorial incisa, presenting as:
    - Ipsilateral pupil dilatation
    - Contralateral hemiparesis
    - Deepening coma
    - Decorticate posturing
    - Apnea and death
Herniation Syndromes

• Cerebellar Herniation
  – Downward displacement of cerebellar tonsils through the foramen magnum.
  – Presents as:
    • Medullary compression
    • Pinpoint pupils
    • Flaccid quadriplegia
    • Apnea and circulatory collapse
Infectious Emergencies

- Meningococcemia
Meningitis: inflammation of the meninges

History:

- **Acute Bacterial Meningitis:**
  - Rapid onset of symptoms <24 hours
    - Fever, Headache, Photophobia
    - Stiff neck, Confusion
  - Etiology By Age:
    - 0-4 weeks: E. Coli, Group B Strep, Listeria
    - 4-12 weeks: neonatal pathogens, S. pneumo, N. meningitides, H. flu
    - 3mos – 18 years: S. pneumo, N. menin., H. flu
    - >50/ alcholics: S. pneumo, Listeria, N. menin., Gram(-) bacilli
Lymphocytic Meningitis (Aseptic/Viral)
- Gradual onset of symptoms as previously listed over 1-7 days.
- Etiology:
  - Viral

Atypical Meningitis
- History (medical/social/environmental) crucial
- Insidious onset of symptoms over 1-2 weeks
- Etiology:
  - TB(#1)
  - Coccidiomycosis, crytococcus
Physical Exam Pearls
- Infants and the elderly lack the usual signs and symptoms, only clue may be AMS.
- Look for papilledema, focal neurologic signs, ophthalmoplegia and rashes
- As always full exam
  - Checking for above
  - Brudzinski’s sign
  - Kernigs sign
- KEY POINT: If you suspect meningococcemia do NOT delay antibiotic therapy, MUST start within 20 minutes of arrival!!!!!
Emergent CT Prior to LP
- Those with profoundly depressed MS
- Seizure
- Head Injury
- Focal Neurologic signs
- Immunocompromised with CD4 count <500

DO NOT DELAY ANTIBIOTIC THERAPY!!
Lumbar puncture
**Meningitis**

- **Lumbar Puncture Results**

<table>
<thead>
<tr>
<th>TEST</th>
<th>NORMAL</th>
<th>BACTERIAL</th>
<th>VIRAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pressure</td>
<td>&lt;170</td>
<td>&gt;300</td>
<td>200</td>
</tr>
<tr>
<td>Protein</td>
<td>&lt;50</td>
<td>&gt;200</td>
<td>&lt;200</td>
</tr>
<tr>
<td>Glucose</td>
<td>&gt;40</td>
<td>&lt;40</td>
<td>&gt;40</td>
</tr>
<tr>
<td>WBC’s</td>
<td>&lt;5</td>
<td>&gt;1000</td>
<td>&lt;1000</td>
</tr>
<tr>
<td>Cell type</td>
<td>Monos</td>
<td>&gt;50% PMN’s</td>
<td>Monos</td>
</tr>
<tr>
<td>Gram Stain</td>
<td>Neg</td>
<td>Pos</td>
<td>Neg</td>
</tr>
</tbody>
</table>
Meningitis Management

- **Antibiotics By Age Group**
  - Neonates (<1 month) = Ampicillin + Gent. or Cefotaxime + Gent
  - Infants (1-3 mos) = Cefotaxime or Ceftriaxone + Ampicillin
  - Children (3 mos-18 yrs) = Ceftriaxone
  - Adults (18 yr-up) = Ceftriaxone + Vancomycin
  - Elderly/Immunocomp = Ceftriaxone + Ampicillin + Vancomycin
• Steroids
  – In children, dexamethasone has been shown to be of benefit in reducing sensorineural hearing loss, when given before the first dose of antibiotic.
  – Indications:
    • Children > 6 weeks with meningitis due to H. flu or S. pneumo.
    • Adults with positive CSF gram stain
  – Dose: 0.15mg/kg IV
Encephalitis

- Always think of in the young/elderly or immunocompromised with FEVER + AMS
- Common Etiologies:
  - Viral
    - West Nile
    - Herpes Simplex Virus (HSV)
    - Varicella Zoster Virus (VZV)
    - Arboviruses
      - Eastern Equine viruses
      - St. Louis Encephalitis
Encephalitis

• Defined as: inflammation of the brain itself
• Most cases are self limited, and unless virulent strain, or immunocompromised, will resolve.
• The ONLY treatable forms of encephalitis are:
  – HSV
  – Zoster
Management:

- Emergent CT: As indicated for meningitis
- ABC’s with supportive care.
- Lumbar puncture:
  - Send for ELISA and PCR
- Acyclovir 10 mg/kg Q 8 hours IV for HSV and Zoster
- Steroids not shown to be of benefit.
Headache & Vertigo

- Headache
- Types of Headache:
  - Migraine
    - With aura
    - Without aura
  - Cluster Headache
  - Subarachnoid hemorrhage
  - Temporal arteritis
Migraine
- Now thought to be due to neurogenic inflammation and abnormalities of serotonergic transmission.
- Symptoms:
  - Severe headache either preceded by a visual “aura” (scintillating scotoma or VF cut) or motor disturbance.
  - Nausea, vomiting, light sensitivity, sound sensitivity

Factors that may provoke an attack include:
- Menstruation, Sleep/food deprivation
- Physical activity or certain foods (chocolate)
- Contraceptive estrogens
Migraines

- History & PE
  - CRUCIAL to obtain HA history from patient
    - Is this HA similar to others or is it “worst HA of life”
    - Prior workups
    - Medications
    - Foods
    - Menses
  - FULL PE including Neuro exam
Migraines

• **Management**
  - Place patient in cool, quiet, dark environment
  - IV fluids if dehydrated
  - Abortive therapy:
    • Phenothiazines (antimigraine and antiemetic)
    • DHE (vaso/venoconstrictor) + antiemetic
    • Sumatriptan (5-HT agonist)
    • Opiods as LAST RESORT!!
Headaches

- Cluster Headaches
  - Classically as boring headache on one side of face behind the eye.
  - May be associated with signs of facial flushing, tearing, nasal stuffiness

TX: 100% O2 by N/C at 6-8 l/min
  - If no relief, Sumatriptan
Subarachnoid hemorrhage
  - Clinically: Abrupt onset of severe thunderclap “worst HA of life”.
  - Usually associated nausea and vomiting
  - Nonfocal neurologic exam (usually)

Etiology: usually due to leaking berry aneurysm.

DX: CT +LP A MUST
  - If CT (-), MUST perform LP
  - LP (+) if (+) xanthrochromia OR failure of CSF to clear RBC’s by tube #4
• Subarachnoid Hemorrhage
  – Management
  – ABC’s as always
  – IV, O2, Monitor
  – Head of bed @ 30 degrees
  – Prophylax patient for seizures with Dilantin load.
  – Ca Channel blocker (nimodipine) 60 mg Q6 h to prevent vasospasm, and rebleeding
• Temporal Arteritis
  – Etiology: a granulomatous inflammation of one or more of the branches of the external carotid artery
  – Clinically presents as:
    • Severe unilateral HA over Temporal area
    • Usually in middle aged females.
    • PE reveals: a tender, warm, frequently pulseless temporal artery, with decreased visual acuity on the affected side.
• Temporal Arteritis
  – DX: Clinically + ESR elevation, usually >50
  – Confirm with biopsy of artery
  – TX: HIGH dose steroids are VISION SAVING!
    • Start on prednisone IMMEDIATELY once suspected
      – Prednisone 60 – 80 mg Q day
  – Stat Neurology Consult
Vertigo

• History and PE exam again CRUCIAL!!
  – History:
    • Truly a vertiginous complaint?
      – r/o syncope / near syncope??
    • Acute onset of severe symptoms or more gradual course
  – PE:
    • Full exam paying particular attention to:
      – HEENT : Eyes, TM’s
      – Neuro : Cerebellar function
Peripheral Vertigo

History:
- Acute onset of severe dizziness, nausea, vomiting.
- May be a positional worsening of symptoms
- Recent history of URI or similar episodes in past which resolved.

PE Pearls:
- Horizontal nystagmus which fatigues
- Possible TM abnormality
- Normal Neuro exam with normal cerebellar function and gait.
- Reproduction of symptoms with Hallpike maneuver
Vertigo

• Peripheral
  – Common Causes:
    – Labrynthitis
    – Cerumen Impaction
    – OM
    – OE
    – URI
    – Menieres Disease (tinnitus, hearing loss, vertigo)

• TX: Symptomatic and treat underlying cause:
  – Antivert 25 mg Q6h
  – Neurology / ENT follow up
Vertigo

- Central Vertigo
  - Due to lesions of brainstem or cerebellum
  - 10 - 15% of cases
  - Signs & Symptoms:
    - Gradual onset of mild disequilibrium
    - Mild nausea and vomiting
    - Nonfatigable nystagmus (any direction)
    - Associated neurological abnormalities:
      - Ptosis
      - Facial palsy, dysarthria
      - Cerebellar findings, ataxia
Vertigo

- **Central**
  - **Causes:**
    - Brainstem ischemia or infarction
    - Cerebellar hemorrhage
    - Vertebrobasilar insufficiency
    - MS
  - **Diagnosis:**
    - Thorough Neurologic exam
    - Head CT with Posterior fossa thin cuts
  - **Management:**
    - Neuro consult
    - Admit and workup depending on etiology
Emergent Peripheral Neuropathies

- Acute Toxic Neuropathies
  - Diptheria (Corynbacterium diptheriae)
    - Acutely ill patient with fever, in a dPT deficient patient.
    - Membranous pharyngitis that bleeds
    - Powerful exotoxin produces widespread organ damage.
      - Myocarditis/AV Block, Nephritis, Hepatitis.
      - Neuritis with bulbar and peripheral paralysis.
      - (ptosis, strabismus, loss of DTR’s)
  - TX: Parenteral PCN or Erythromycin
    - Horse Serum antitoxin
    - Respiratory isolation and admission the rule.
Botulism (Clostridium botulinum toxin)

- Earliest finding (90%) = Blurred vision, diplopia, ophthalmoplegia, ptosis
- Neurologic abnormalities descend and will lastly involve the respiratory musculature and cause respiratory paralysis and death with 6 hours if not treated!
- Mentation and sensation are normal.
- Remember in infants with FTT (failure to thrive)
  - Raw honey contains C. botulinum
- Tx: Aggressive airway stabilization!
- Trivalent serum antitoxin
- Lastly, there have been some recently reported cases of hypersensitivity to “Bo-tox”
  - So ……..
Tetanus

- Symptoms 4 “T”s
  - Trismus, Tetany, Twitching, Tightness
  - Risus sardonicus / opisthotonus
  - Signs of sympathetic overstimulation.
    - Tachycardia, hyperpyrexia, diaphoresis.

- Management:
  - Human Tetanus Immunoglobulin (HTIG)
  - dT Toxoid
  - Metronidazole
Guillain-Barre Syndrome
- Most common acute polyneuropathy.
- 2/3’s of patients will have preceding URI or gastroenteritis 1-3 weeks prior to onset.
- Presents as: paresthesias followed by ascending paralysis starting in legs and moving upwards.
  - Remember Miller-Fischer variant: has minimal weakness and presents with ataxia, arreflexia, and ophthalmoplegia.
- DX: LP will show cytochemical dissociation.
  - Normal cells with HIGH protein.
- TX: Self limiting, Early and aggressive airway stabilization.
Myasthenia Gravis
- Most common disorder of neuromuscular transmission.
- An autoimmune disease that destroys acetylcholine receptors (AchR) which leads to poor neurotransmission and weakness.
- Proximal >> Distal muscle weakness
- Commonly will present as:
  - Muscle weakness exacerbated by activity, and is relieved by rest
- Clinically: ptosis, diplopia and blurred vision are the most common complaints. Pupil is spared!
?
THE END