NEUROLOGIC EMERGENCIES

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NEUROLOGIC EMERGENCIES

Learning Objectives

Describe the urgent assessment and management of patients with the following neurologic emergencies:

- Depressed consciousness
- Acute ischemic stroke
- Transient ischemic attack
- Intracerebral hemorrhage
- Subarachnoid hemorrhage
- Seizures and status epilepticus
- Acute headache
- Vertigo
- Spinal cord compression/myelitis
- Guillain-Barré syndrome
- Myasthenic crisis
NEUROLOGIC EMERGENCIES
Appropriate Actions by PAs

- Consult Neurology

THE END

Thank you for having me

or...

- Narrow diagnosis yourself and perform appropriate tests, treatment, and consultation

Which do you prefer?
THE CC DILEMMA
Optimal vs. Suboptimal Practices

CC
Chief Complaint/Concern

History

Physical Exam

Diagnostic Tests

Impression

Plan/Recommendations

Optimal—thoughtful investigation & deliberation

Suboptimal—action without thought, often triggered by time pressures & insecurity

Test

Triage

Treat

Avoid “CC & the 3 Ts!”
NEUROLOGIC EMERGENCIES

Patient Assessment

- History
  - Chief complaint
  - *History of present illness*

- Examination
  - Observation
  - *Neurologic exam*

- Tests
  - Brain imaging
  - Blood laboratories
  - Urinalysis & urine cultures
  - Cerebrospinal fluid analysis

When pressed for time, the tendency is to rely on:
- Chief complaint
- Observation
- Brain imaging

The correct neurologic diagnosis is, in fact, usually determined via:
- History of present illness
- Neurologic exam

A few minutes of investigation & reflection is far more effective than routine brain CT scans—and CT may not be the best test.
DIFFERENTIAL DIAGNOSIS PROCESS

Start with a Well-Developed “CC Diff”

Broad differential diagnosis (diff Dx) based on **chief complaint**

Refined diff Dx based on targeted questions in **history**

Refined diff Dx based on focused **exam**

Refined diff Dx based on **tests**

Final impression

In neurology patients, “differential diagnosis” includes syndrome clarification & lesion localization
SYNDROME CLARIFICATION

Step 1 of Neurologic Differential Dx

- **Numbness**
  - Sensory loss
  - Tingling (paresthesia)
  - Weakness

- **Dizziness**
  - Lightheaded (near syncope)
  - Hallucination of movement (vertigo)
  - Imbalance
    - Motor ataxia (cerebellum)
    - Sensory ataxia (proprioception)

- **Speech Difficulty**
  - Aphasia (language)
  - Dysarthria (oral muscles)

- **Confusion/Altered Mental Status**
  - Depressed consciousness
  - Delirium
  - Receptive aphasia
  - Amnesia
  - Hemianopsia
  - Neglect

- **Gait Difficulty**
  - Motor ataxia
  - Sensory ataxia
  - Leg weakness (one or both)
  - Magnetic gait
  - Downgaze palsy
**LESION LOCALIZATION**

*Step 2 of Neurologic Differential Dx*

**Central Nervous System (CNS)**
1. Cerebral cortex
2. Cerebral subcortex
3. Brainstem
4. Cerebellum
5. Spinal cord

**Peripheral Nervous System (PNS)**
1. Nerve root**
2. Nerve plexus
3. Nerve**
4. Neuromuscular junction
5. Muscle

- Every neurologic symptom and sign localizes to one of the specific area(s) of the nervous system on this list
- Localize the “lesion” by:
  - Localizing each individual symptom and sign and
  - Determining where the locations overlap
- One must localize the lesion in the nervous system before considering differential diagnosis or cause

*Neurologic end organs (e.g., eye, ear) are also part of PNS*

**Some nerves (esp. cranial nerves) are both nerve roots & nerves**
### “ALTERED MENTAL STATUS” A Nonspecific, Unhelpful, Confusing Term

#### “AMS” & “CONFUSION” DIFFERENTIAL DIAGNOSIS

<table>
<thead>
<tr>
<th>SYNDROME</th>
<th>DESCRIPTION</th>
<th>LOCALIZATION</th>
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<tbody>
<tr>
<td>DEPRESSED CONSCIOUSNESS</td>
<td>Decreased alertness &amp; awareness</td>
<td>Bicerebral or brainstem</td>
</tr>
<tr>
<td>DELIRIUM</td>
<td>Inattentive, disoriented, abnormal behavior, depressed consciousness</td>
<td>Bicerebral</td>
</tr>
<tr>
<td>RECEPTIVE APHASIA</td>
<td>Nonsensical speech &amp; poor comprehension, but NORMAL attention, behavior, &amp; consciousness</td>
<td>Left temporal or parietal</td>
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<tr>
<td>AMNESIA</td>
<td>Isolated memory deficit for specific period of time</td>
<td>Bithalamic or bitemporal</td>
</tr>
<tr>
<td>HEMIANOPSIA</td>
<td>Unable to find things to the left or right</td>
<td>Occipital</td>
</tr>
<tr>
<td>NEGLECT</td>
<td>Does not acknowledge people to the left; responds inappropriately to voices on left</td>
<td>Right parietal</td>
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LEVEL OF CONSCIOUSNESS

The Spectrum

NORMAL
alert, attentive

DELIRIUM
inattentive, abnormal behavior

LETHARGY / DROWSINESS
purposeful arousal to speech or touch

STUPOR
purposeful arousal to deep pain only

COMA
no purposeful arousal to any stimuli

NOTE: Ethanol (alcohol) intoxication is a common cause of toxic-metabolic encephalopathy that may lead to delirium, lethargy, stupor, or coma
DEPRESSED CONSCIOUSNESS LOCALIZATION

Brainstem vs. Bicerebrum

- **Ascending Reticular Activating System (ARAS)**
  - consciousness center in upper brainstem (midbrain & upper pons)
  - ARAS axons stimulate thalami (T) & cortex of both cerebral hemispheres

- Decreased consciousness is due to ARAS inhibition as a result of either:
  - **Brainstem** lesion (focal)
    - Usually **structural** (stroke or mass)
  - **Bicerebral** dysfunction (diffuse)
    - Usually **physiologic** (e.g., toxic-metabolic encephalopathy)

Brainstem or bicerebral dysfunction causes ↓ consciousness
DEPRESSED CONSCIOUSNESS LOCALIZATION

Eye Movements Are the Key

- Dorsum of upper brainstem contains both:
  - ARAS
  - Nuclei of cranial nerves that control eye movements (3, 4, 6)

- Therefore, in patients with decreased consciousness, **eye movements** help localize lesion:
  - **Brainstem** → **abnormal** eye movements
  - **Bicerebral** → **normal** eye movements

**ARAS & CNs 3, 4, 6**
*are near each other in dorsum of upper brainstem*
ACUTE BICEREBRAL DYSFUNCTION

Etiologies

All of these conditions can cause depressed consciousness or delirium

- **Toxic-metabolic encephalopathy**
- **Intracranial hypertension (↑ICP)**
  - Acute hydrocephalus
  - Subarachnoid hemorrhage
  - Subdural hematoma
  - Intracerebral hemorrhage
  - Tumor (w/ vasogenic edema or bleed)
- **Meningeal irritation**
  - Acute meningitides
  - Chronic meningitides
    - Cancer, lymphoma, leukemia
    - Fungus, syphilis, Lyme
    - TB, sarcoid
  - Subarachnoid hemorrhage
- **Seizure**
  - Complex-partial seizure
  - Postictal state
- **Bilateral ischemic strokes**
  - Embolic shower
  - Watershed (borderzone) ischemia due to hypotension
  - CNS vasculitis (very rare)
- **Hypertensive encephalopathy**
  - = PRES, posterior reversible encephalopathy syndrome
- **Migraine (confusional)**

ICP = intracranial pressure
ACUTE BICEREBRAL DYSFUNCTION

Initial Toxic-Metabolic Workup

First thoughts for patient with depressed consciousness or delirium

- **Drugs**
  - Antihistamines
  - Anticholinergic agents
  - Narcotics
  - Antiepileptic drugs
  - Benzodiazepines
  - Psychoactive drugs
  - Steroids
  - Dopaminergic agents
  - Alcohol
  - Recreational drugs
  - Neuropathic pain drugs

- **Serum chemistries**
  - Sodium
  - Glucose
  - BUN and creatinine
  - Calcium
  - Magnesium
  - Phosphorus
  - Liver enzymes
  - Ammonia

- **Serum CBC**
  - WBC (infection)
  - Hgb / Hct (anemia)
ACUTE BICEREBRAL DYSFUNCTION

If Initial Toxic-Metabolic Workup Is Negative

Other thoughts for patient with depressed consciousness or delirium

- Arterial blood gas (ABG)
  - Hypercapnea
  - Hypoxia
- Medical conditions
  - Thyroid disease
  - Psychiatric disease
  - Epilepsy / seizure
- Toxins
- Nutritional deficiencies (esp. B12 or thiamine)
  - Alcoholism
  - Gastric bypass
- Sleep deprivation or psychosis (esp. ICU)
- CT scan of brain
- MRI scan of brain
- Lumbar puncture
  - WBC
  - RBC & xanthochromia
  - Protein
  - Glucose
  - Gram stain & cultures
  - Other
STROKE DEFINITION & 3 TYPES

Stroke = Sudden Brain Dysfunction Due to Artery Problem

- Ischemic stroke (85%)
  Due to low blood flow (usually blockage by blood clot) in cerebral artery with death of section of the brain (= cerebral infarction)

- Intracerebral hemorrhage (10%)
  Due to bursting of an artery within the brain (= intraparenchymal hemorrhage)

- Subarachnoid hemorrhage (5%)
  Due to bursting of an artery around the brain (in subarachnoid space)
STROKE CENTER LEVELS
Facilitate Time-Sensitive Therapies

Level 1
Comprehensive Stroke Center
- ICH
- SAH
- AIS
- IV tPA
- Hospital Care
- Thrombectomy

Level 2
Primary Stroke Center
- AIS
- IV tPA
- Hospital Care

Level 3
Acute Stroke-Ready Hospital
- AIS
- IV tPA

Thrombectomy
Hospital Care
Hospital Care or Thrombectomy
ACUTE STROKE

Prevent Complications

- Do NOT allow aspiration pneumonia
  - Maintain head of bed elevated at 30 degrees
  - Keep patient NPO (nil per os = nothing by mouth)
    - No food
    - No liquid → liquids are the most difficult consistency to swallow
      (more difficult than solids or purees)
    - No pill

- Do NOT cause urinary tract infection (UTI)

- Do NOT cause hematuria

Do NOT place indwelling bladder catheter
ACUTE ISCHEMIC STROKE—FIRST FEW HOURS
“Time Is Brain: Save the Penumbra”

- **Penumbra** is zone of reversible ischemia (low blood flow) around **core** of irreversible infarction (dead tissue) during first hours to day after ischemic stroke onset

- **Intravenous tPA** (tissue plasminogen activator) given within first 4.5 h, may break blood clot, save penumbra neurons, improve outcome—but may cause dangerous bleeding if given later

- **Mechanical thrombectomy**—removing certain large clots—in addition to tPA within 4.5 h of onset or instead of tPA 4.5-24 h after onset—may save penumbra neurons, improve outcome

*TPA opens up arteries just like Drano opens up clogged water pipes—but large, stubborn blockages may require physical removal (thrombectomy)*
AIS PATHOPHYSIOLOGY

Preserving Penumbra in ED & Stroke Unit

After a few hours, penumbra either returns to normal brain tissue or becomes infarction.

High blood pressure protects penumbra.

Avoid or treat these conditions, all of which damage the penumbra:
- Low blood pressure (hypoperfusion)
- Hyperglycemia (lactic acidosis)
- Fever (↑ metabolic demand)
- Seizure (↑ metabolic demand)

**pen (paene) = almost umbra = shadow**
AIS, TPA, & BLOOD PRESSURE
Give TPA Only if You Can Lower BP “Safely” to 185/110

You may lower BP slightly to give IV tPA, but avoid excessive lowering of BP just to give tPA—“Don’t kill the penumbra to save the penumbra”

- **Clevidipine** (Cleviprex) IV infusion – *top choice*
  - Start 1-2 mg/h, increments 1-2 mg/h q 90 sec, max 32 mg/h

- **Nicardipine** (Cardene) IV infusion – *also excellent choice*
  - Start 5 mg/h, increments 2.5 mg/h q 5 min, max 15 mg/h

- **Labetalol** IV – *third choice*
  - 10-20 mg IV, increments q 10-15 min, max 300 mg

Note Different Target BPs Pre- & Post-tPA
Pre-tPA: < 185/110
During & post-tPA: < 180/105
LEFT MCA THROMBECTOMY SYNDROMES

L MCA Infarctions w/ CT-Exam Correlations

Small-artery occlusion

Unlikely thrombectomy

Subcortex infarction
R hemiparesis (F, A, L)
R hemisensory loss (F, A, L)

(F = face, A = arm, L = leg)

End-of-M1 Occlusion

Possible thrombectomy

Cortex infarction
Aphasia
L gaze preference
R visual field deficit
R hemiparesis (F, A)
R hemisensory loss (F, A)

Beginning-of-M1 Occlusion

Possible thrombectomy

Subcortex + cortex infarction
Aphasia
L gaze preference
R visual field deficit
R hemiparesis (F, A, L)
R hemisensory loss (F, A, L)
RIGHT MCA THROMBECTOMY SYNDROMES
R MCA Infarctions w/ CT-Exam Correlations

Small-artery occlusion
Unlikely thrombectomy

Subcortex infarction
L hemiparesis (F, A, L)
L hemisensory loss (F, A, L)

Cortex infarction
Neglect
R gaze preference
L visual field deficit
L hemiparesis (F, A)
L hemisensory loss (F, A)

Subcortex + cortex infarction
Neglect
R gaze preference
L visual field deficit
L hemiparesis (F, A, L)
L hemisensory loss (F, A, L)

(F = face, A = arm, L = leg)

End-of-M1 Occlusion
Possible thrombectomy

Beginning-of-M1 Occlusion
Possible thrombectomy

Small-artery occlusion
Unlikely thrombectomy

MCA
ACA
PCA
Infarction

OU Neurology
AIS EMERGENCY THERAPY

IV Tissue Plasminogen Activator (TPA)

- Stroke onset = last known well (LKW)
- Must be given **within 4.5 h** of stroke onset, but...
  
  *The earlier you give IV tPA, the better the outcome*

- Disability risk ↓ 30% despite ~5% symptomatic ICH risk

- **There is NO upper age limit**

- You CAN give if taking warfarin & INR ≤ 1.7

- **You SHOULD give if patient has persistent moderate deficits within time window, regardless of improvement after onset**

- Dose is 0.9 mg/kg with maximum dose 90 mg

- Give 10% via bolus, remaining 90% via 1-hour infusion
AIS EMERGENCY THERAPY

IV Tissue Plasminogen Activator (TPA)

**ABSOLUTE CONTRAINDICATIONS**
- Blood glucose < 50
- Blood pressure > 185/110
- Patient took NOAC within 48 h
- Endocarditis
- Aortic dissection
- Abnormal coagulation tests:
  - INR > 1.7, PT > 15, aPTT > 40
  - Platelets < 100k
- Serious head trauma w/in 3 mo
- Intracranial/spinal surgery w/in 3 mo
- Ischemic stroke w/in 3 mo
- History of intracranial hemorrhage (microbleeds NOT contraindicated)
- Intra-axial brain tumor

**RELATIVE CONTRAINDICATIONS**
- Seizure at onset
- Blood glucose > 400
- Major surgery w/in 14 d
- Major trauma w/in 14 d
- Left anterior STEMI w/in 3 mo

Must perform these three tests in all acute stroke patients potentially eligible for tPA or thrombectomy:
1. Fingerstick blood glucose
2. CT head, noncontrast
3. CT angiography, head & neck
ACUTE ISCHEMIC STROKE THERAPY

LKW ≤ 4.5 Hours—Negative CTA

Goal is to reperfuse penumbra.

However, if CTA also shows distal ICA or M1 occlusion...
ACUTE ISCHEMIC STROKE THERAPY

LKW ≤ 4.5 Hours—Positive CTA

Goal is to reperfuse penumbra in patients with large clots blocking middle cerebral artery (MCA)—which are often resistant to IV tPA alone.

Note: In patients presenting in time to receive IV tPA, do NOT obtain CT perfusion prior to thrombectomy.
ACUTE ISCHEMIC STROKE THERAPY
LKW 4.5 to 24 Hours

**Goal is to reperfuse penumbra in patients with large clots blocking middle cerebral artery (MCA) who arrive too late to receive IV tPA & also have evidence of salvageable penumbra on CT perfusion**

- **Last known well (LKW) 4.5-24 h ago**
- **Good preadmission neurologic status (mRS score 0-2)**
- **CT angiography shows MCA blockage**
- **CT perfusion shows small core & big penumbra**
- **Thrombectomy decreases risk of death & disability**
TRANSIENT ISCHEMIC ATTACK (TIA)
Treat as an Emergency!

- Like ischemic stroke, TIA is due to thromboembolism
- In TIA, clot dissolved in time spontaneously with NO infarction and NO permanent deficit
- TIA signals that patient is at risk for ischemic stroke in near future—next time, clot may not dissolve in time
- **Admit patient to hospital, begin aspirin 325 mg daily**
- **Perform diagnostic tests urgently**
  - MRI brain with diffusion-weighted imaging
  - CT angiography head & neck
  - ECG and cardiac monitoring
  - Echocardiography (transthoracic, possibly transesophageal)
- **Based on diagnostic test results, either continue aspirin daily or change to more appropriate regimen**
ISCHEMIC STROKE/TIA MIMICS & CT
Differential Diagnosis of Sudden Onset Focal Neurologic Deficit

HEAD CT SCAN
ABNORMAL
- Intracerebral hemorrhage (ICH)
- Tumor with bleed or seizure
- Abscess with seizure
- Subdural hematoma (esp. acute on chronic)
- Old stroke/brain lesion w/ either:
  - Partial seizure & postictal state
  - Reactivated by toxic-metabolic insult

HEAD CT SCAN
NORMAL
- Hypoglycemia
- Partial seizure (cryptogenic)
- Migraine aura
- Somatic symptoms (psychiatric)

Most commonly fool physicians
RECURRENT SIMILAR SPELLS
Are NEVER due to Stroke or TIA

- No one has the same stroke or TIA over and over
- If a patient has repeat spells that are always the same (i.e., “stereotypical”), one of three things is happening:
  - **Old stroke temporarily “reactivated”**: Patient had a stroke the first time and then had worsening of the same symptoms because of a fever, sedating medication, etc.; MRI scan only shows the old stroke—no new stroke
  - **Seizures from old stroke**: Patient had a stroke the first time and then had “partial” seizures caused by the old stroke; MRI only shows the old stroke—no new stroke
  - **Migraine aura**: Patient is having migraine auras that mimic stroke and never actually had a stroke; MRI scans are always normal—no new or old stroke
INTRACEREBRAL HEMORRHAGE (ICH)

Bleeding INTO Brain

“Hypertensive” ICH

- Due to CHRONIC hypertension for many years with resultant damage to small penetrator arteries in medial brain, including:
  - Basal ganglia – hemiparesis +
  - Thalamus – hemiparesis/sensory loss +
  - Pons – coma
  - Cerebellum – ataxia +

Non-hypertensive ICH

- No history or evidence of chronic hypertension
- Location in outer brain (peripheral or cortical) called *lobar hemorrhage*
- Causes include:
  - Amyloid angiopathy in dementia/elderly patients
  - Ruptured arteriovenous malformation (AVM)
  - Bleeding tumors
  - Bleeding disorders

L = lobar
B = basal ganglia C = cerebellum
T = thalamus P = pons
## THE ICH SCORE

### 4 Factors Associated with Poor Prognosis

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<tr>
<th>FACTOR</th>
<th>CRITERION</th>
<th>POINTS</th>
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<tbody>
<tr>
<td>LOC</td>
<td>GCS 3-4</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>GCS 5-12</td>
<td>1</td>
</tr>
<tr>
<td>AGE</td>
<td>≥ 80 years old</td>
<td>1</td>
</tr>
<tr>
<td>LOCATION</td>
<td>Intraventricular</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Brainstem/cerebellum</td>
<td>1</td>
</tr>
<tr>
<td>VOLUME</td>
<td>≥ 30 cc ((ABC/2))</td>
<td>1</td>
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**Prognosis based on the ICH Score:**
- 1-2 = good outcome
- 3-4 = poor outcome*
- 5-6 = very poor outcome*

*Note: Prognosis better if low GCS is due to hydrocephalus alone*

*Note: Prognosis better if low GCS is due to hydrocephalus alone*
INTRACEREBRAL HEMORRHAGE (ICH)
Emergency Management

ICH Score 1-2
- Maintain SBP 140-160
- Consider neurosurgery
  - External ventricular drain if hydrocephalus
  - Resection, esp. cerebellar hemorrhages
- Admit to stepdown unit or ICU to:
  - Treat blood pressure
  - Prevent complications (aspiration, DVT, etc.)
  - Initiate rehabilitation

ICH Score 3-6*
- Counsel family urgently, preferably before intubation or surgery, to decide treatment options, including possible “comfort care”

*esp. if poor GCS NOT due to hydrocephalus
SUBARACHNOID HEMORRHAGE
Bleeding AROUND Brain (into CSF Spaces)

Nontraumatic SAH is an emergency!

■ Diagnosis
  ➢ Severe, sudden headache; stiff neck
  ➢ Depressed consciousness; nausea, vomiting

■ Etiology
  ➢ Rupture of saccular (= berry) aneurysm
    (outpouching in wall of intracranial artery, typically around circle of Willis) is most common cause of nontraumatic SAH

■ Evaluation
  ➢ Find the blood – by CT head or lumbar puncture
  ➢ Find the aneurysm – by CT angiogram

■ Management
  ➢ Coil (neurointervention) or clip (neurosurgery)
  ➢ Neurosciences ICU
SEIZURE TYPES & LOCALIZATION

Cerebral Cortex – Diffuse, Focal, or Both

1° Generalized Tonic Clonic

Simple Partial

Complex Partial

Partial with 2° Generalization

Diffuse cortex

Focal cortex

Focal cortex

Focal cortex

Focal with 2° Generalization

diffuse with part of opposite hemisphere

thalamus

SEIZURE LOCALIZATION & SEMIOLOGY

Partial Seizures

Simple-Partial Seizures
- Normal consciousness
- Focal symptom that varies based on seizure-focus location
  - Limb shaking
  - Tingling
  - Lights in vision
  - Aphasia
  - Déjà vu
- Postictal focal deficit

Complex-Partial Seizures
- ↓ consciousness (blank staring)
- Aura = focal symptom that varies based on seizure-focus location
- Automatisms – picking, blinking, lip smacking, etc.
- Postictal state
  - Delirium/confusion
  - ↓ consciousness
  - Focal deficit

Seizure focus
Increased electrical activity limited to a focal part of cortex
Seizure focus
Increased electrical activity starts in a focal part of cortex & spreads to a part of contralateral cortex
SEIZURE LOCALIZATION & SEMIOLOGY

Generalized Tonic-Clonic (GTC) Seizures

1° GTC Seizure

- **Tonic phase – STIFF**
  - Diffuse muscle contractions w/ extension of limbs
  - Jaw clenches, limbs stiffen, back arches, abdominal muscles contract—results in tongue biting, abnormal cry or shout, urinary or fecal incontinence

- **Clonic phase – SHAKE**
  - Diffuse, rhythmic, muscle contractions w/ shaking of limbs
  - May result in injury to head & limbs from striking hard surfaces

- **GTC seizure may be primary or secondary**
  - Primary – no aura or postictal focal deficit, due to diffuse brain dysfunction
  - Secondary – starts with aura, ends w/ postictal focal deficit, due to focal brain lesion

Partial Seizure with 2° Generalization

- Increased electrical activity starts simultaneously throughout cortex
- Increased electrical activity starts in focal part of cortex, spreads to thalamus, then spreads to cortex of both hemispheres
SEIZURE
Scary, but Usually NOT an Emergency

■ Partial seizure
  - *In isolation is NOT an emergency*—routine neurology consult suffices
  - *If continuous, is an “urgency,” not an emergency*

■ Generalized tonic-clonic (GTC) seizure
  - *In isolation, is not an emergency*—only concerns are patient injuring him/herself & witness causing harm
  - *Only an emergency in generalized status epilepticus,* which occurs in 1 of 2 settings:
    ▪ Recurrent GTC seizures without return to baseline interictally
    ▪ Prolonged GTC seizure (> 5 minutes) – *time the event*
  - Intubation is only indicated in GTC status epilepticus
GENERALIZED TONIC-CLONIC SEIZURE
What to Do at Bedside

- Remain calm, uncover limbs & observe
  - Each limb
  - Head & eye deviation

- **Protect patient’s head & limbs from injury**

- Do **NOT** put anything into patient’s mouth (increases risk of aspiration and bitten tongues heal well)

- Look at your watch/phone—time the event

- **Do NOT order a benzodiazepine in most cases**
  - Only if shaking > 5 min (time it!)—lorazepam 0.1 mg/kg IV

- **NEVER give a benzodiazepine after shaking stops**

- **Place patient in lateral decubitus position** to avoid aspiration (& elevate head of bed when possible)

Two most common mistakes are:
1. Inappropriate benzodiazepine administration &
2. Inappropriate intubation
GENERALIZED STATUS EPILEPTICUS

Medical Management

- To abort a seizure, give:
  - **Lorazepam** 0.1 mg/kg IV push at < 2 mg/min
  - **ONLY GIVE LORAZEPAM IF PATIENT IS ACTIVELY SEIZING—DO NOT GIVE IF PATIENT STOPPED HAVING A SEIZURE**

- To prevent further seizures, give:
  - **Fosphenytoin** 20 mg PE/kg IV at < 150 mg PE/min (prevents future seizures if therapeutic)
  - Additional fosphenytoin 10 mg PE/kg IV if seizures persist

- Other options include:
  - **Valproic acid** 20-40 mg/kg IV
  - **Levetiracetam** 30-60 mg/kg (max 4500 mg) IV
GENERALIZED TONIC-CLONIC SEIZURE
What to Order

- Evaluation of seizure patient
  - CT brain, noncontrast
  - Laboratories
    - Na, CO₂, glucose, BUN/Cr
    - Mg, Ca, Phos, LFTs
    - CBC, platelets, PT/PTT
    - EtOH level
    - Drug/toxicology screen
    - Antiepileptic drug levels

- Regarding seizure patients in ED
  - Do NOT admit patient with new-onset seizure if not in status, normal CT brain & laboratories, & postictal state resolves
  - If no other reason for admission, discharge w/ follow-up neurology appt & MRI brain w/ contrast

Advise patient re: seizure precautions—no driving, swimming, bathing in closed-drain bathtub, climbing on ladder or other high places until free of seizures for 6 months
HEADACHE DIFFERENTIAL DIAGNOSIS

Primary vs. Secondary Headaches

**Primary Headaches**
- Bothersome but not life-threatening conditions
- Headaches are usually:
  - Intermittent
  - Similar to past headaches
- Scans & tests **not** necessary
- Examples:
  - Migraine / “tension-type”
  - Trigeminal autonomic cephalalgias
    - Cluster
    - Hemicrania

**Secondary Headaches**
- Worrisome, potentially life-threatening conditions
- Headaches are usually:
  - Constant
  - Different from past headaches
- Scans & tests **necessary** (usually)
- Examples:
  - Mass lesion (e.g., brain tumor)
  - Idiopathic intracranial hypertension (IIH)
  - Giant-cell (temporal) arteritis
  - Meningitis
  - Subarachnoid hemorrhage
  - Carotid or vertebral dissection
  - Trigeminal neuralgia
  - Medication-overuse “headache”

*NOTE: Physiologic conditions may trigger migraine (e.g., fever, concussion)*
WHEN TO WORRY ABOUT A HEADACHE

Tests Only Necessary if You Suspect 2° Headache

- Best indicators:
  - First, worst, persistent, or different headache
  - Abnormal neurologic exam

- Other potential indicators:
  - Onset after Valsalva maneuver or head trauma
  - New onset after age 50
  - Exacerbation with head position
  - Lack of migraine-associated features

If history consistent with migraine & neurologic exam is normal, brain scans & other tests are NOT necessary.

In ED patients with headache & normal neurologic exam, CT is noncontributory 99% of the time (67% normal, 32% incidental finding)

HEADACHE MYTHOLOGY

- **High blood pressure NEVER causes headaches**
  - Migraine causes elevated blood pressure as well as headache

- **Sinus headaches are NOT due to sinus infection or allergies**
  - Migraine causes sinus congestion as well as headache

- **Cervical spine disease RARELY causes headaches**
  - Migraine causes neck pain as well as headache

- **Chiari malformation RARELY causes headaches**
  - Chiari malformations are usually incidental findings
  - Migraine is the cause of headache in most patients with this condition
  - Referral to neurosurgery may result in inappropriate brain surgery

- **Eye strain does NOT cause headaches & tension headaches are NOT due to neck muscle contraction or even tension**
  - Tension headaches are actually stress-letdown migraine headaches
MIGRAINE MYTHOLOGY

- Migraine headaches are NOT always severe
  - They can be mild, moderate, or severe

- Migraine attacks do NOT always include headache
  - Migraine attacks without headache are common

- Migraine condition is NOT more common in women
  - Occurs equally among men and women

- Migraine condition occurs in MOST people—and animals
  - Flawed epidemiologic studies only determined the prevalence of a small subset of migraine attacks within the last year, not the overall prevalence of the migraine condition
  - Confers evolutionary advantage in avoiding storms; occurs in animals as well as human beings
MIGRAINE DOES NOT = BAD HEADACHE

Headache Is Tip of the Migraine Iceberg

HEADACHE

Neck/chest pain
Visual floaters
Hemiparesis
Hypertension spells
Constipation
Sinus congestion
Nausea & vomiting
Sensory phobias

Tingling
Aphasia
Diplopia
Ataxia
Hypotension/syncope
Diarrhea
Epistaxis
Motion sickness

“Headache is never the sole symptom of migraine, nor indeed is it a necessary feature of migraine attacks.”

Oliver Sacks, *Migraine, Revised & Expanded*, 1992
MIGRAINE DEFINITION & DESCRIPTION

**Syndrome of “Brain Spells” with Body Symptoms**

**Genetic** condition in which a person has a **predisposition** to suffering recurrent transient episodes (attacks) of **brain dysfunction** with **systemic manifestations** that may include:

- **headache/neck pain** – from mild to severe, variable location
- **focal neurologic symptoms** – mimics stroke/TIA, Meniere
- **GI symptoms (upper or lower)** – equals IBS, mimics gallstones
- **chest pain** – mimics heart attack, equals atypical noncardiac chest pain
- **autonomic dysfunction** – blood pressure changes, pulse/palpitations, sinus congestion, epistaxis, red ear, Horner syndrome, etc.

“triggered” by hormonal or environmental changes or other medical conditions; each episode consists of:

**4 possible phases** (prodrome, aura, pain, postdrome).
THE MIGRAINE ATTACK

Summary

1. PRODROME
Mood changes
Food cravings
Yawning
Eyelid twitching

2. AURA
Visual – lights, spots, zig-zag lines
Sensory – migratory paresthesias
Motor – hemiplegia
Cognitive – aphasia, confusion, etc.
“Brainstem” – vertigo, diplopia, etc.
Autonomic – upper/lower GI sx$s
high/low BP (syncope)
brady/tachycardia

3. PAIN
Headache
Neck pain
Chest pain
Sinus congestion, epistaxis
Sensory phobias:
light, noise, motion,
smell, temperature

4. POSTDROME
Malaise & fatigue
Allodynia (pain to touch)
Difficulty concentrating
“The migraine hangover”

GENETIC PREDISPOSITION

TRIGGER(S)
Barometric pressure (weather/altitude)
Stress changes
Estrogen changes
Thyroid changes
Sleep changes
Hunger, dehydration
Food preservatives
Artificial sweeteners
Alcohol, esp. red wine
Scents, smoke, fumes
Head trauma

OU Neurology
MEDICATION-OVERUSE SYNDROME (MOS)
Common Cause of Status Migrainosus

Also called “analgesic rebound” or “analgesic withdrawal”

History of intermittent headaches or other migraine symptoms with transition to constant headache or other migraine symptoms in association with frequent use of certain medications:

- Analgesic, decongestant, muscle relaxant, or triptan use > 2 d/wk (except prescription naproxen & perhaps diclofenac potassium & meloxicam)
- Any use of ondansetron, proton-pump inhibitor, dipyridamole
- Often perpetuated by physicians, who prescribe offending agents, then accuse patients of drug seeking

Treatment consists of:

- Discontinuation of offending agent(s)
- Prescription naproxen 500 mg BID if no contraindications
- Sedative medications as needed
- Initiation of migraine abortive therapy
MIGRAINE ABORTIVE THERAPY
Hospital/Emergency Department Agents

May repeat these agents q8h PRN:

- Normal saline – 1 L IV bolus (good for pregnant women)
- Magnesium sulfate – 1 g IV
- Valproate (Depacon) – 500 mg IV
- Metoclopramide (Reglan) – 10 mg IV (good for pregnant women—class B)
- Prochlorperazine (Compazine) – 10 mg IV
- Chlorpromazine (Thorazine) – 25 mg IV
- Dihydroergotamine (DHE) – 0.5-1.0 mg IV or IM

Methylprednisolone 500-1000 mg IV is another option

I personally do NOT give ketorolac (Toradol) because many patients have MOS. If you’re CERTAIN patient does NOT have MOS, may give ketorolac 15-30 mg IV q6-8h.
MIGRAINE ABORTIVE THERAPY

Adjunctive Therapy for Nausea, Sedation

- Promethazine (Phenergan) 25 mg q6-8h IV, PO, or topical
- Diphenhydramine (Benadryl) 25-50 mg q6-8h IV or PO
- Hydroxyzine pamoate (Vistaril) 25-100 mg q6-8h PO

*Do NOT use ondansetron (Zofran) since it may trigger migraine*
DIZZINESS

First Clarify What the Patient Means

- **Vertigo**
  - Hallucination of movement of patient or environment
  - Often spinning sensation, but may be side-to-side movement
  - Due to vestibular system dysfunction (inner ear, brainstem, cerebellum or temporal cortex)

- **Ataxia**
  - Gait imbalance or dysequilibrium
  - **Motor ataxia**—not affected by vision, due to cerebellar dysfunction
  - **Sensory ataxia**—worse when vision impaired (e.g., dark room, diabetic retinopathy), due to proprioceptive deficit

- **Lightheadedness**
  - Near syncope or syncope due to hypotension
  - Vibratory or buzzing sensation in head due to migraine
VERTIGO
Management Principles

- Different causes of vertigo are treated differently
- Treating the vertigo symptom alone is insufficient
- Medication for vertigo, itself, is rarely indicated
- Avoid reflexively prescribing a medication without determining or considering cause
- All types of vertigo are worse with head movement
- MRI brain most important if neurologic exam abnormal—don’t forget to assess limb coordination
- Most common causes are:
  - Benign paroxysmal positional vertigo (BPPV)
  - Migraine with vertigo aura

Avoid CC & the 3 Ts!
Meclizine is rarely helpful!
Most common cause of isolated vertigo

Only cause of vertigo that resolves completely when head is still

Nausea and vomiting usually very prominent

No hearing loss, tinnitus, or other neurologic symptoms

Neurologic exam is normal except for nystagmus

Due to canalolithiasis = stimulation of semicircular canal by misplaced “ear rocks” (otoliths)

Diagnosis – Dix-Hallpike maneuver

Management

- Brandt-Daroff exercises
- Epley maneuver (by specialist)

Imaging & drugs less helpful than head-positioning maneuvers
BPPV DIAGNOSIS

Dix-Hallpike (Nylen-Barany) Maneuver

PERFORMANCE
Ask about vertigo, observe for nystagmus

A

Sit pt up, facing forward, eyes open

B

Quickly lie pt back, head turned 45° to L, neck extended over table—observe 30 s

C

Sit pt up, facing forward, eyes open—observe 30 s

D

Quickly lie pt back, head turned 45° to R, neck extended over table—observe 30 s
BPPV DIAGNOSIS
Dix-Hallpike (Nylen-Barany) Maneuver

INTERPRETATION
Ask about vertigo, observe for nystagmus

• Findings suggestive of BPPV on Dix-Hallpike Maneuver
  – Severe vertigo
  – Rotatory nystagmus (clockwise or counterclockwise)
  – Latency (delay in response after assuming position)
  – Fatigue (response lessens as position is maintained)
  – Habituation (response lessens after repeated testing)
BPPV MANAGEMENT
Brandt-Daroff Head-Positioning Exercises

A. Sit up on side of bed
B. Lie on side x 30 s
C. Sit up for 30 s
D. Lie on other side x 30 s
E. Sit up for 30 s

Repeat this cycle 5 times, 3 times/day for 2 weeks.
Most patients experience relief in 10 days.
BPPV MANAGEMENT

Epley Repositioning Maneuver by Specialist

- Performed by trained physician, therapist, or audiologist to move endolymphatic debris out of posterior semicircular canal & into utricle
- May need to repeat until nystagmus abolished
- Patient should avoid supine position x 2 days

A. Sit patient up w/ head turned 45° to R
B. Lower head rapidly below table edge
C. Rotate head rapidly to L at 45° x 30 s
D. Roll patient on L side w/o turning head x 30 s
E. Sit patient up
MIGRAINE WITH VERTIGO AURA
Also Known as Vestibular Migraine

- Most common cause of recurrent vertigo
- *Usually similar episodes in past*
- May have associated migraine features such as:
  - Headache
  - Nausea & vomiting (less prominent than with BPPV)
  - Lower abdominal pain, gas, diarrhea
  - Other auras – syncope, confusion, visual, tingling
  - Sensory phobias – especially light, noise, motion intolerance
  - Identifiable triggers such as stress changes, weather fronts

- Worsens with ondansetron (Zofran)
- *Treat the migraine (not the vertigo)* with migraine abortive & prophylactic agents
ACUTE MYELOPATHY SYNDROME
Spinal Cord Compression or Transverse Myelitis

Key Cervical & Thoracic Dermatomes

- **Weakness** *(quadriplegia or paraplegia)* below level of spinal cord injury
  - Usually with increased reflexes and bilateral Babinski signs

- **Decreased sensation** *(sensory level)* below level of spinal cord injury
  - If transverse myelitis or spinal cord compression, usually involves all sensory modalities (i.e., both pin/temperature AND proprioception/vibration)

- **Urinary incontinence** *(neurogenic bladder)*
  - Acutely, may present with large, distended, atonic bladder
  - Subacutely to chronically, presents with small, hypertrophic bladder with frequency, hesitancy, urgency, small volumes

- **Constipation**

- **Sexual dysfunction**
ACUTE MYELOPATHY MANAGEMENT

Spinal Cord Compression or Transverse Myelitis

- Diagnose with **emergency MRI cervical & thoracic spine**

- For **transverse myelitis**
  - Perform MRI brain, serum oligoclonal bands, & lumbar puncture (including CSF myelin basic protein, IgG synthetic rate, IgG index, & oligoclonal bands)
  - For most causes, give methylprednisolone 1 g IV/d x 3-5 d

- For **spinal cord compression**
  - Due to trauma, consult spine surgeon—do NOT give steroids
  - Due to cancer:
    - Give dexamethasone 10 mg x 1, then 4 mg q6h
    - Consider emergent radiation therapy and/or surgery (neurosurgery or orthopedics for resection of lesion both to decompress spine and for pathologic specimen)
GUILLAIN-BARRÉ SYNDROME (GBS)
Antibodies to Peripheral Nerve Myelin

DIAGNOSIS

- Possible antecedent infection (esp. viral or Campylobacter)
- Symptoms plateau within 4 weeks
- **Ascending paralysis, starting with distal motor deficits**
- **Absent reflexes**
- **Orthopnea due to diaphragm weakness – emergency!**
- Sensory symptoms (esp. tingling & low-back pain), but normal sensory exam
- Cardiac arrhythmias, but normal bladder & bowel function

MANAGEMENT

- **Admit to ICU**
- Monitor cardiac rhythm & BP
- **Elevate head of bed**
  - Gravity helps weak diaphragm
- **Pulmonary mechanics** q 4-12 h
  - FVC = forced vital capacity
  - NIF = negative inspiratory force
- Intubate if:
  - FVC < 1-2 L (< 15-20 ml/kg)
  - NIF less than -25
- Treat with either:
  - IVIG (intravenous immune globulin)
  - PLEX (plasma exchange)

*Diagnostic tests not helpful in first 2 weeks—after 2 weeks:
  - lumbar puncture shows elevated CSF protein with normal CSF WBC,
  - electromyography/nerve conduction study shows demyelinating neuropathy*
MYASTHENIA GRAVIS & MYASTHENIC CRISIS
Antibodies to Neuromuscular Junction Receptors

**DIAGNOSIS**
- **Proximal, symmetric weakness** – “hair, chair, & stair” weakness (when raising arms to do hair, rising from chair, or going up stairs)
- **Fatigable** – worse when tired or at the end of the day
- **Ocular weakness** – common (double vision & ptosis)
- **Oral & facial weakness** – common (nasal speech, dysarthria, dysphagia, chewing, eye-closure, face weakness)
- Normal sensation and reflexes
- Diagnostic tests NOT done urgently:
  - Serum acetylcholine receptor Abs
  - EMG repetitive stimulation

**CRISIS MANAGEMENT**
- Refers to respiratory distress (orthopnea) due to weak diaphragm
- **Admit to ICU**
- **Elevate head of bed**
  - Gravity helps weak diaphragm
- **Pulmonary mechanics** q 2-4 h
  - FVC = forced vital capacity
  - NIF = negative inspiratory force
- Intubate if:
  - FVC < 1-2 L (< 15-20 ml/kg)
  - NIF less than -25
- Treat with cholinesterase inhibitor, pyridostigmine (Mestinon) 60 mg q4h
NEUROLOGIC EMERGENCIES

Learning Objectives

Describe the urgent assessment and management of patients with the following neurologic emergencies:

- Depressed consciousness
- Acute ischemic stroke
- Transient ischemic attack
- Intracerebral hemorrhage
- Subarachnoid hemorrhage
- Seizures and status epilepticus
- Acute headache
- Vertigo
- Spinal cord compression/myelitis
- Guillain-Barré syndrome
- Myasthenic crisis
THE END