Evaluation of Emergency Department Dizziness Patients: New Concepts

July 9, 2020
(June 17, 2020)

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Erica Scott
Angie Wallace
Stephanie Williford
EB Medicine group.

EB Medicine supported the development of this educational presentation.

FERNE President and Board Chair.
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Also available is the June 2020 podcast, which was presented to participants in 46 countries, and a CME option on the EB Medicine website at www.ebmedicine.net and at https://www.ebmedicine.net/topics/neurologic/dizziness-course
Educational Objectives

• (1) increased competence (knowledge & strategy to use knowledge in patient care)
• (2) improved performance-in-practice
• (3) improved patient outcomes.
Educational Objectives

• (1) Understand dizziness etiologies
• (2) Know how ED dizziness patients present
• (3) Consider new diagnostic approach
• (4) Be able to diagnose and treat ED patients
• (5) Discuss dizziness in COVID-19 setting
• (6) Record key historical questions and exam findings in EMR to exclude posterior stroke Dx
Content Literature

The Timing-and-Triggers Approach to the Patient With Acute Dizziness

Jonathan Edlow, MD, FACEP

• Emergency Medicine Practice
• EB Medicine, December 2019, Vol 21, #12
Dizziness
Etiologies and Diagnoses
Dizziness

- dizzy
- adjective
- diz·zy | \ 'di-zē \ |
- FOOLISH, SILLY
- Having a whirling sensation in the head with a tendency to fall
- Mentally confused

Dizziness Pathologies

- Systemic (fluids, electrolytes, Hb, BP)
- Peripheral (Vestibular dysfunction)
- Central (CNS ischemia, event, lesion)
Normal Vestibular Anatomy and Mechanisms

Peripheral Vestibular Organs

- Figure 2, Page 4 of monograph
- Utricle, saccule
  - Sense vertical and horizontal acceleration
- Three semi-circular canals
  - Sense movement in three directions
- Vestibular nerve
  - Carries input to the brain from periphery
Dizziness
Case Studies

Dizziness Case: 44 yo Dizzy Male

- Sign out. Just check the CT...
- 44-year-old male, previously healthy
- Six hours continuous dizziness
- Unsteadiness. “feeling like I am drunk…”
- Normal VS
- L beating nystagmus with gaze, L gaze
- “If CT is OK, home with meclizine…”
Dizziness Case: Lightheaded 70 yo

- 70-year-old female
- “Lightheadedness” for five days
- Worse with getting out of bed
- The dizziness wakes her out of sleep
- HTN, cholesterol meds
- VS OK, normal neuro exam, no nystagmus
- Should a CT be ordered? Why?

Dizziness Case: Syncopal 58 yo

- 58-year-old male DM, NV x 3
- Syncope reported, but was such bad vertigo he had to lower himself to the ground
- Began 3 hours previously, suddenly
- Nystagmus to R, higher amplitude gaze R
- HIT test +, skew neg, mild unsteady OK gait
- Call the stroke team?
Dizziness History

Dizziness History Overview

• Knowing if the patient is describing lightheadedness, dizziness, or vertigo is less useful than timing, triggers approach

• Patients with vertigo are more likely to confirm the sensation of spinning, like on Tilt-a-whirl, but that doesn’t confirm Dx

• Vertigo: loss of eye control, nystagmus

• Again, no confirmed diagnosis, however
Dizziness History Items

- O  Onset  (When? History same?)
- P  Provocative & Palliative (Head move?)
- Q  Quality  (Lightheaded? Spinning?)
- R  Related Symptoms (ENT, Neuro?)
- S  Severity  (Mild, moderate, severe?)
- T  Temporal (Intermittent? Constant? Now?)
- U  Under care (Clinician? Meds?)

Dizziness Hx Associated Symptoms

- Infection symptoms (ENT, Chest, GI, GU)
- Dehydration symptoms (NV, heat, poor PO)
- Stroke/TIA symptoms
- Cardiovascular symptoms
- GI bleeding, pregnancy symptoms
- Dizziness hx, near syncope, syncope
- MVC, trauma, cervical artery dissection?
Dizziness History
A New Approach

Dizziness History: ATTEST

• Associated symptoms
• Timing
• Triggers
• Examination Signs
• (Confirmatory) Testing
Dizziness
Physical and Neurological Exams
Dizziness General Physical Exam

- VS: Fever, Abnormal VS, HTN, Cushing’s
- Well appearing? AMS? Dehydration?
- Pain or respiratory distress?
- Rash? Skin changes?
- Dysrhythmias?
- Chest, GI or GU infections?

Dizziness H(E)ENT Physical Exam

- H(E)ENT:
  - Sinus and temporal artery tenderness
  - TM and ear canal abnormalities
  - Pharyngitis, retropharyngeal abscess
  - Dental abscess
  - Nasal polyps, green discharge
  - Neck swelling, tenderness, bruits,
  - Nystagmus
Dizziness Eye Physical Exam

- Eye exam:
  - Visual acuity? (Vision loss, peripheral loss)
  - Visual fields? (Field cut, tunnel vision)
  - Diplopia? (Vertical or horizontal gaze)
  - PERRL? (Marcus Gunn pupil)
  - EOMI? (Cranial nerve III, IV, VI deficits)
  - Lid lag? (CN III palsy)
  - Fundus ok? (Discs sharp or papilledema)

Dizziness Neurological Exam

- AVPU, Mental status
- Speech (Aphasia or dysarthria)
- Motor functioning face and extremities
- Dysmetria (Finger to nose, heel to shin)
- Truncal ataxia, rhomberg, gait
- Abnormal reflexes
- Neuropsychiatric assessment
Dizziness
Nystagmus Exam

Dizziness and Nystagmus Exam

- Central, peripheral structures keep eyes aligned
- CNS: Occipital cortex, cerebellum, brain stem
- Peripheral: Vestibular system
- Eyes synchronously follow head movement
- Allows for the body to move without dizziness
- Nystagmus: abnormal eye movement
- Dizziness/Vertigo can result, with sensation of room spinning and/or lightheadedness
Nystagmus Exam

• Eye normally remain central, coordinated
• If one side of vestibular system is diseased and is stimulated, eyes will slowly drift to that side
• Drift to affected side with a quick snap back to correct location is called nystagmus
• Nystagmus named for stimulation (ie lateral gaze), and direction of quick snap back response
• Example: Gaze to R, eyes drift right, with fast horizontal (lateral) movement to L (L nystagmus)

Nystagmus Background

• Eye normally remain central, coordinated
• Nystagmus causes pathological drift to side of abnormal semicircular canal stimulation
• Nystagmus comes from Greek work meaning “to drop off to sleep”
• When nystagmus occurs without any stimulation of the labyrinth system, think central etiology
Nystagmus Exam

• (1) Have patient look at you, watch for drift, and quick fix response to the nystagmus drift
  – Note any vertical or rotational nystagmus
• (2) Ask patient to fix central gaze on your nose, see if nystagmus improves or worsens with gaze
• (3) Move eyes in all directions, note CN palsies
  – Note if bidirectional horizontal, vertical, or rotational nystagmus seen with eye movement
• (Some patients do have physiologic nystagmus)
Dizziness History: ATTEST

- Associated symptoms
- Timing
- Triggers
- Examination Signs
- (Confirmatory) Testing

Clinical Pathway for the ATTEST Approach to Emergency Department Patients With Acute Dizziness

1. Does the history and physical examination suggest a general medical cause?
   - YES
     - Diagnose AVS vestibular neuritis versus posterior circulation stroke (Class II)
     - Go to “Clinical Pathway for Diagnostic Evaluation of Patients With an Acute Vestibular Syndrome,” page 11
   - NO
     - Evaluate and treat presumed diagnosis

2. Has the dizziness been continuously present, and does it persist at the time of evaluation?
   - YES
     - Diagnose t-EVS vertebral migraine versus TIA
   - NO

3. Is the dizziness triggerable?
“BAD” Nystagmus (Central?)

- These nystagmus findings are “BAD” only in the setting of Acute Vestibular Syndrome (AVS)

- Acute, severe persistent vertigo at presentation
  - Pure lateral (horizontal) nystagmus, no gaze*
  - Vertical, torsional (rotational) nystagmus
  - Worse nystagmus with central gaze
  - Lateral nystagmus that changes fast component direction with gaze to both lateral sides (R nystagmus with L gaze, L nystagmus with R gaze)

BPPV Important Nystagmus Note!

- In posterior canal BPPV, the nystagmus is vertical and rotational (torsional)
- In horizontal canal BPPV, the nystagmus is horizontal and direction changing with gaze
- These are benign findings in t-EVS BPPV, but are considered worrisome in AVS
- Therefore nystagmus interpretation depends on presumed AVS or t-EVS BPPV Dx
Nystagmus Exam Requirement

- Must establish if the nystagmus exam is being done in the setting of either suspected acute vestibular syndrome (AVS) or triggered episodic vestibular syndrome (t-EVS)
- In AVS, “BAD” nystagmus signs worrisome
- In t-EVS, these same “BAD” nystagmus signs are benign due to peripheral (vestibular) etiology in BPPV (due to otolith problem)
- Need to determine likely etiology first

“BAD” Nystagmus (Central?) in BPPV

- BPPV presentation (t-EVS):
  - Pure lateral (horizontal) nystagmus, no gaze
  - Vertical, torsional (rotational) nystagmus
  - Worse nystagmus with central gaze
  - Lateral nystagmus the changes fast component direction with gaze to both lateral sides
- This may be the only nystagmus observation that is worrisome in BPPV
Nystagmus Central Gaze Key Concept

• In general, nystagmus that is related to a peripheral vestibular problem will improve with fixed central gaze

• Ask patient to look at your nose from a comfortable distance right in front of them

• If with fixed central gaze at your nose the nystagmus does not improve or it worsens, then suspect a central etiology of the nystagmus, again because peripheral causes generally improve with central gaze fixation

Dizziness Neuroimaging
Non-contrast Head CT

- Low yield (< 10%)
- High clinical utility (accurate, disposition)
- In general, NCCT is initial test of choice in all ED patients that require neuroimaging
- Useful to exclude SAH, hemorrhage, abscess, space occupying lesions, edema, mass effect, midline shift

Dizziness Non-contrast Head CT

- 1.6% positive for findings related to sx
- ICH rarely presents as isolated AVS
- In dizziness patients, intracerebral hemorrhage only seen in 0.5% of cases
- If headache, CPPV findings, consider CT
Dizziness MRI Neuroimaging

- DWI MRI can miss stroke in first 48 hours
- In acute ischemic stroke, negative DWI MRI in 7% of patients, most often posterior
- In AVS, DWI MRI negative in 12-18% of patients when performed in first 72 hours
- In small strokes, DWI MRI neg in 50% of pts
- Large vessel disease should be considered

Dizziness & Neuroimaging

Which statement about brain imaging in patients with acute dizziness is TRUE?

a. In pts with BPPV, a brain MRI can help establish the diagnosis.
b. In posterior circulation stroke, CT is diagnostic in > 75% of cases.
c. In patients with posterior circulation stroke, early MRI with diffusion-weighted images establishes the diagnosis in nearly all pts.
d. In pts with vestibular migraine, neither CT nor MRI is indicated.
Dizziness & Neuroimaging

Which statement about brain imaging in patients with acute dizziness is TRUE?

• Answer: D. Neither CT nor MRI is indicated.

• Explanation:

• A key point is that not only is imaging not required but, in fact, has serious limitations in patients with a posterior circulation stroke. The “reassurance” that a negative CT scan in a dizzy patient excludes a central cause is false reassurance. CT scanning is notoriously unreliable for any acute ischemic stroke and is even worse in the posterior circulation. Importantly, early MRI, and even diffusion-weighted imaging (DWI)-MR, can miss stroke in these patients if it is performed in the first 48 hours. In a meta-analysis of 3236 patients with acute ischemic stroke, nearly 7% had a negative DWI-MR, and this was strongly associated with a posterior circulation location.

• Section: Diagnostic Studies, page 15.

Dizziness Exam

A New Approach Explained
Dizziness History: ATTEST

- Associated symptoms
- Timing
- Triggers
- Examination Signs
- (Confirmatory) Testing
ATTEST Evaluation: Three Steps

• Medical screening exam normal?

• Is the dizziness now constant and continuous?

• If intermittent dizziness, is it spontaneous or triggered?
**ATTEST Evaluation**

- General medical etiologies screening

- **Constant dizzy:** Acute Vestibular Syndrome (AVS)

- **Intermittent dizzy:** t-EVS or s-EVS
  - t-EVS: Triggered episodic vestibular syndrome
  - s-EVS: Spontaneous episodic vestibular syndrome

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**Clinical Pathway for the ATTEST Approach to Emergency Department Patients With Acute Dizziness**

1. **Does the history and physical examination suggest a general medical cause?**
   - YES
   - NO

2. Diagnostic “STOP” (Class II):
   - Worsening dizziness?
   - Arm asymmetry?
   - True deficit sitting up?

3. Evaluate and treat presumed diagnosis

4. **Has the dizziness been continuously present, and does it persist at the time of evaluation?**
   - YES
   - NO

5. Diagnose AVS vestibular neureitis versus posterior circulation stroke (Class II)
   - Go to “Clinical Pathway for Diagnostic Evaluation of Patients With an Acute Vestibular Syndrome,” page 11

6. **Is the dizziness triggerable?**
   - YES
   - NO

7. Diagnose t-EVS BPPV versus orthostatic hypotension (Class I)

8. Diagnose s-EVS vestibular migraine versus TIA (Class II)
EMR: Medical Sx, Stroke Signs?

- General medical cause sx, signs are positive
- No vertical, rotational nystagmus
- No arm dysmetria, weakness
- No vision, speech, swallow, sensory changes
- No truncal ataxia or gait abnormalities
- (Therefore stroke symptoms negative)
- **Then treat the presumptive medical cause**
EMR: Dizziness Persistence

- No general medical cause sx, signs

- Is the dizziness constant and persistent?
  - If yes: diagnose *Acute Vestibular Syndrome*
  - If no: then establish if dizziness triggerable
Dizziness Triggerable?

- If no AVS diagnosis, and therefore episodic dizziness, consider the trigger question
- Episodic with focus on “Is dizziness triggered?”

  - **If yes:** Triggered Episodic Vestibular Syndrome
    - t-EVS (triggered)
  
  - **If no:** Spontaneous Episodic Vestibular Syndrome
    - s-EVS (spontaneous)
ED Dizziness Patients: Timing Triggers
Edward P. Sloan, MD, MPH, FACEP

June 17, 2020
EB Medicine

ATTEST Summary

• Gen medical etiology, no stroke symptoms: Rx
• Constant dizzy: Acute Vestibular Syndrome (AVS)
• AVS: Vestibular Neuritis/Labyrinthitis or Posterior Stroke
• Intermittent dizzy: t-EVS or s-EVS
• t-EVS: BPPV or orthostatic hypotension
• s-EVS: Vestibular migraine or TIA (spells)
• Three diagnostic strata, six main diagnoses
**ATTEST Diagnoses**

- Gen med etiology, no stroke symptoms: Rx
- Constant dizzy: Acute Vestibular Syndrome (AVS)
- AVS: Vestibular Neuritis or Posterior Stroke
- Intermittent dizzy: t-EVS or s-EVS
- t-EVS: BPPV or orthostatic hypotension
- s-EVS: Vestibular migraine or TIA
- Three diagnostic strata, six main diagnoses

**Dizziness Characteristics**

Patients with an acute onset of dizziness that is persistently present and present at the time of examination in the ED have which of the following vestibular syndromes?

- a. Acute vestibular syndrome
- b. Triggered episodic vestibular syndrome
- c. Spontaneous episodic vestibular syndrome
- d. Chronic vestibular syndrome
Dizziness Characteristics

Patients with an acute onset of dizziness that is persistently present and present at the time of examination in the ED have which of the following vestibular syndromes?

- **Answer:** A. Acute vestibular syndrome
- **Explanation:**
  - Patients will have one of several syndromes: (1) the acute vestibular syndrome (AVS), (2) the spontaneous episodic vestibular syndrome (s-EVS), and (3) the triggered episodic vestibular syndrome (t-EVS). Patients with the AVS have sudden or rapid onset of dizziness that is continuously present; associated with nausea, head motion intolerance, and often (but not always) nystagmus.23 Patients with the s-EVS have intermittent episodes of dizziness that come “out of the blue,” without any trigger at all. Patients with the t-EVS have very brief episodes of dizziness that are reliably triggered by something; most often, this is movement of the body (such as standing up) or movement of the head (such as turning over in bed).10.

Acute Vestibular Syndrome: Bedside Diagnostic Testing
AVS Posterior Stroke Findings

- Looking for stroke signs on exam
- Five tests are performed
- If any of these specific findings are noted, then a stroke must be considered
- Abnormal nystagmus, vertical skew deviation, not a positive HIT exam, stroke findings, or ataxia all suggest a central etiology
- Any of these suggest posterior circulation stroke
AVS Specific Stroke Findings

- Central pattern nystagmus
- Skew deviation with vertical correction noted
- Negative, no unilateral, correct saccade on HIT
- CNS signs on neurological exam
- Patient is unable to sit or walk unassisted
- Any of these suggest posterior circulation stroke

Table 2. Summary of Usual Physical Examination Findings in Symptomatic Patients With the Acute Vestibular Syndrome

<table>
<thead>
<tr>
<th>Examination Component</th>
<th>Peripheral (ALL must be present to diagnose vestibular neuritis)</th>
<th>Central (ANY ONE of these findings suggests posterior fossa stroke)</th>
</tr>
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<tbody>
<tr>
<td>Nystagmus (straight-head gaze and rightward and leftward gaze)</td>
<td>Dominantly horizontal, direction-lead, beating away from the affected side</td>
<td>Dominantly vertical and/or torsional or dominantly horizontal, direction-changing on left/right gaze</td>
</tr>
<tr>
<td>Test of skew (alternate cover test)</td>
<td>Normal vertical eye alignment and no corrective vertical movement (ie. no skew deviation)</td>
<td>Slow deviation (small vertical correction on uncovering the eye)</td>
</tr>
<tr>
<td>Head Impulse test</td>
<td>Unilaterally abnormal with head moving toward the affected side (presence of a corrective saccade toward the normal side)</td>
<td>Usually bilaterally normal (no corrective saccade)</td>
</tr>
<tr>
<td>Targeted neurological examination (see text, page 13)</td>
<td>No cranial nerve, brainstem, or cerebellar signs</td>
<td>Presence of limb ataxia, dysarthria, dysphagia, paresis, anhidrosis, facial sensory loss (pain/temperature), unilateral decreased hearing</td>
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<tr>
<td>Gait and truncal ataxia</td>
<td>Able to walk unassisted and to sit up in stretcher without holding an or leaning against bed or rails</td>
<td>Unable to walk unassisted or sit up in stretcher without holding on or leaning against bed or rails</td>
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</table>

Note: Nystagmus, vestibular neuritis, and skew deviation are typically present with downward torsional nystagmus, but this is a rare disorder. From the emergency medicine perspective, nystagmus in a patient with an acute vestibular syndrome should be considered to be central (i.e., stroke).

- Most central posterior fossa strokes will have direction-led horizontal nystagmus that, alone, cannot be distinguished from that typically seen with vestibular neuritis.
- Many patients with posterior circulation strokes will have no skew deviation; so, on this criterion alone, cannot be distinguished from vestibular neuritis.
- Skew in the anterior inferior cerebellar artery territory may produce an unilaterally abnormal head impulse test that mimics vestibular neuritis, but skew deviation is usually present as a clue. If a patient has bilaterally abnormal head impulse testing, this is also suspicious for a central lesion if nystagmus is present (as may be seen in Wernicke syndrome).

Table 2. Summary of Useful Physical Examination Findings in Symptomatic Patients With the Acute Vestibular Syndrome

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1 Inferior branch vestibular neuritis will present with downbeat torsional nystagmus, but this is a rare disorder. From the emergency medicine perspective, vertical nystagmus in a patient with an acute vestibular syndrome should be considered to be central (a stroke).
2 More than half of posterior fossa strokes will have direction-fixed horizontal nystagmus that, alone, cannot be distinguished from that typically seen with vestibular neuritis.
3 Many patients with posterior deceleration strokes will have no skew deviation; so, on this criterion alone, cannot be distinguished from vestibular neuritis.
4 Strokes in the anterior inferior cerebellar artery territory may produce a unilateral abnormal head impulse test that mimics vestibular neuritis, but hearing loss is usually present as a clue. If a patient has bilaterally abnormal head impulse test, this is also suspicious for a central lesion if nystagmus is present (as may be seen in Wernicke syndrome).

AVS Nystagmus: Peripheral vs Central

- **Peripheral** etiology:
  - Horizontal nystagmus with stimulation
  - Fixed horizontal movement (fast correction in one direction, suggesting one side with illness)

- **Central** etiology:
  - Horizontal nystagmus without any stimulation
  - Vertical or torsional nystagmus
  - Direction changing horizontal fast movement (fast correction in both directions due to central disease)

Test of Skew

- Cover, then uncover one eye at a time alternately
- **Peripheral** etiology: No vertical skew deviation
- No vertical correction as eye is uncovered
- **Central** etiology: Positive skew deviation
- Vertical correction as eye is uncovered to get both pupils to be aligned on vertical axis
- Similar to vertical nystagmus, bad finding in AVS
**Head Impulse Test**

- Only verified useful in patients with nystagmus
- Looking for asymmetry in vestibular response
- Hold head 15° to each side, quick move to center
- Peripheral etiology: Unilaterally correct saccade
- There is a unilateral fast correcting saccade to center seen with eye mvmt (vestibular problem)
- Central etiology: Bilaterally no center correction
- No unilateral correcting saccade, eyes centered


**Figure 5, page 12 of monograph**

- Shows how eyes follow rotation to the diseased side with correcting saccade to the center
- A unilateral compensatory saccade suggests peripheral vestibular etiology of dizziness
- If there is no compensatory saccade bilaterally, and eyes remain fixed centrally, the vestibular system is working normally (think central)

https://www.sralab.org/rehabilitation-measures/head-impulse-test-head-thrust-test
Head Impulse Test Language

- Only performed in patients with nystagmus
- Normal pts: no correcting fast saccade
- Central Ds pts: no correcting fast saccade
- *Unilateral* corrective saccade to center with turn suggests *vestibular* etiology (one diseased side)
- *Unilateral eye saccade is correct, good when vestibular dx is the likely etiology of dizziness*
- No saccade bilaterally is worrisome, not correct, in dizzy AVS patients (think central etiology)

Neurological Exam Findings

- *Peripheral* etiology: No findings
  - No cranial nerve, brainstem, or cerebellar findings
- *Central* etiology: Limb ataxia, dysarthria, hear loss
  - Pain, temperature sensory loss
  - Diplopia, ptosis, anisocoria
Truncal Ataxia and Gait Test

- **Peripheral** etiology: No findings
- Able to sit without holding on, able to walk

- **Central** etiology: Unable to sit without holding on
- Unable to walk unassisted

Dizziness and Nystagmus

In patients with the *acute vestibular syndrome*, nystagmus findings that suggest a central cause include all of the following EXCEPT:

a. Uni-directional horizontal nystagmus
b. Torsional nystagmus
c. Vertical nystagmus
d. Direction-changing horizontal nystagmus (gaze-evoked)
Dizziness and Nystagmus

In patients with the *acute vestibular syndrome*, nystagmus findings that suggest a central cause include all of the following EXCEPT:

• Answer: A. Uni-directional horizontal nystagmus

• Explanation:
  • If a patient looks forward and his eyes drift to the left, then snap back to the right, he has a *right-beating horizontal nystagmus*. This is usually very easy to see, especially in the first 2 to 3 days of the patient’s onset of symptoms. Next, ask the patient to follow the examiner’s finger, going 30° to 40° to the right, then to the left. This is called *gaze-evoked nystagmus*. Also look for *vertical or pure torsional nystagmus*. In patients with the AVS, nystagmus that is vertical, torsional, or that changes direction with the direction of gaze is *central*.

• Section: Test 1: Nystagmus Testing, page 10.

Acute Vestibular Syndrome: EMR Statements
Acute Vestibular Syndrome 5 Q

- Is there central pattern nystagmus?
  - Vertical, rotational, pure lateral with no stimulation, lateral nystagmus that changes direction with gaze
- Is there skew deviation? (Like vertical nystagmus)
- Is HIT negative, not OK? (No unilateral correction)
  - Unilateral, correcting fast saccade is good, positive
- Are there CNS signs on neurological exam?
- Is the patient unable to sit, walk unassisted?
AVS 5 Statements for EMR

- No central pattern nystagmus findings seen.
- No vertical skew deviation observed.
- *Positive (good) unilateral correcting* HIT seen.
- No stroke CNS signs on neurological exam.
- Patient is able to sit and walk unassisted.
- These findings allow for **Vestibular Neuritis / Labyrinthitis** diagnosis in AVS, as posterior stroke is reasonably excluded.

Three Categories:

*Specific Diagnoses*
Table 1. Timing-and-Trigger-Based Vestibular* Syndromes in Acute Dizziness and Their Corresponding Differential Diagnosis

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Description</th>
<th>Common Benign Causes</th>
<th>Common Serious Causes</th>
<th>Important Rare Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>AVS</td>
<td>Acute, continuous dizziness lasting days, accompanied by nausea, vomiting,</td>
<td>Vestibular neuritis</td>
<td>Posterior circulation</td>
<td>Multiple sclerosis</td>
</tr>
<tr>
<td></td>
<td>nystagmus, head-motion intolerance, and gastrointestinal discomfort</td>
<td>Labyrinthitis</td>
<td>ischemic stroke</td>
<td>Malignant hypertension</td>
</tr>
<tr>
<td>s-EVS</td>
<td>Episodic dizziness that occurs spontaneously, is not triggered, and usually</td>
<td>Vestibular migraine</td>
<td>Posterior circulation</td>
<td>Cardiac dysrhythmia</td>
</tr>
<tr>
<td></td>
<td>lasts minutes to hours</td>
<td>Meniere disease</td>
<td>TIA</td>
<td>Pulmonary embolism</td>
</tr>
<tr>
<td>h-EVS</td>
<td>Episodic dizziness triggered by a specific, obligate trigger (typically</td>
<td>BPPV</td>
<td>Cerebrovascular</td>
<td>Chorea</td>
</tr>
<tr>
<td></td>
<td>change in head position or standing up), and usually lasting less than</td>
<td>Orthostatic hypotension</td>
<td>stroke</td>
<td>Panic attack</td>
</tr>
<tr>
<td></td>
<td>minute.</td>
<td>caused by benign</td>
<td>due to serious medical</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>problems</td>
<td>illness</td>
<td></td>
</tr>
</tbody>
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Note: This table lists the most common diseases causing these presenting syndromes and is not intended to be exhaustive.

*The word vestibular here connotes vestibular symptoms (dizziness or vertigo or imbalance or light-headedness, etc.), rather than underlying vestibular disease (e.g., BPPV or vestibular neuritis).

Dizziness is considered (not dizzy at baseline; dizziness develops with movement), as position vertigo due to BPPV. This must be distinguished from dizziness that is ‘worsened’ (dizzy at baseline, worsens with movement); such exacerbations are common in AVS, whether peripheral (neuritic) or central (stroke).

Abbreviations: AVS, acute vestibular syndrome; BPPV, benign paroxysmal positional vertigo; CPPV, central paroxysmal positional vertigo; s-EVS, spontaneous episodic vestibular syndrome; h-EVS, triggered episodic vestibular syndrome; TIA, transient ischemic attack.

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Acute Vestibular Syndrome
Table 1. Timing-and-Trigger-Based Vestibular* Syndromes in Acute Dizziness and Their Corresponding Differential Diagnoses

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Description</th>
<th>Common Benign Causes</th>
<th>Common Serious Causes</th>
<th>Important Rare Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>AVS</td>
<td>Acute, continuous dizziness lasting days, accompanied by nausea, vomiting, nystagmus, head-motion intolerance, and gait unsteadiness</td>
<td>Vestibular neuritis</td>
<td>Posterior circulation ischemic stroke</td>
<td>Neurosurgery, aneurysm, subarachnoid hemorrhage, other medical causes</td>
</tr>
<tr>
<td>s-EVS</td>
<td>Episodic dizziness that occurs spontaneously, is not triggered, and usually lasts minutes to hours</td>
<td>Vestibular migraine</td>
<td>Posterior circulation TIA</td>
<td>Seizures, brain tumors, meningioma</td>
</tr>
<tr>
<td>h-EVS</td>
<td>Episodic dizziness triggered by a specific, definite trigger (typically a change in head position or standing up), and usually lasting less than 1 minute</td>
<td>BPPV</td>
<td>Cerebrovascular disease due to serious medical illness</td>
<td>Stroke, subarachnoid hemorrhage</td>
</tr>
</tbody>
</table>

Note: This table lists the most common diseases causing these presenting syndromes and is not intended to be exhaustive.

*Dizziness is triggered (not dizzy at baseline, dizziness develops with movement), as in position vertigo due to BPPV. This must be distinguished from dizziness that is “exacerbated” (dizzy at baseline, worse with movement); such exacerbations are common in AVS, whether peripheral (neuritis) or central (stroke).

Abbreviations: AVS, acute vestibular syndrome; BPPV, benign paroxysmal positional vertigo; CPPV, central paroxysmal positional vertigo; s-EVS, spontaneous episodic vestibular syndrome; h-EVS, triggered episodic vestibular syndrome; TIA, transient ischemic attack.


Vestibular Neuritis / Labyrinthitis

- Peripheral, benign etiology
- Most often a viral etiology, but other etiologies:
  - Bacterial middle ear infection
  - Meningitis, CNS infection
  - Injury, tumor
  - Medicine side effect
Labyrinthitis

- Peripheral, benign etiology
- Inflamed labyrinth (inner ear), which transmits abnormal input to the brain thru normal (?) vestibular nerve
- Please note that both vestibular nerve and labyrinth can be inflamed at one time
- It may be hard to distinguish one from other

Vestibular Neuritis

- Peripheral, benign etiology
- Inflamed vestibular nerve, which transmits input from the labyrinth (inner ear) to the brain
- Most often a viral etiology, but other etiologies:
  - Bacterial middle ear infection
  - Meningitis, CNS infection
  - Injury, tumor
  - Medicine side effect
Vestibular Neuritis / Labyrinthitis Rx

- Steroids to reduce inflammation
- Antihistamines for symptoms relief
- Antivirals?
- Appropriate home support, close follow-up
- Hospital admission for those with questionable signs and symptoms
- Posterior circulation Sx can quickly progress

Posterior Circulation Stroke Sx

- Central, serious etiology
- Most important dizziness etiology
- Dizziness/vertigo
- N/V (seen with vertigo also)
- Nystagmus (seen with BPPV also)
Posterior Circulation Stroke Sx

- Headache
- Dim vision, diplopia
- Limb weakness/dysmetria
- Sensory changes (pain, temperature)
- Dysarthria, dysphagia
- Truncal ataxia, ataxic gait
- These are unique posterior circulation ischemia Sx, Sx not seen in benign BPPV diagnosis

Rare AVS Diagnoses

- Multiple sclerosis
- Wernicke’s encephalopathy
- Drug effects or toxicities
**Spontaneous, Episodic Vestibular Syndrome**

**A Note on “Spells”**

- Recurrent similar non-provoked episodes or “spells” should be addressed systematically
- Consider seizure etiology
- Complex partial seizures may cause sx
- Patients with seizure disorder may have disconjugate gaze at baseline (autism)
- CNS ischemia may also be the cause
- Posterior circulation ischemia common if dizzy
Vestibular Migraine

- At least 5 episodes of mod-severe symptoms
- Between 5 min and 72 hours
- History of migraine headaches
- Migraine headache features with at least 50% of vestibular migraines
  - Headache with migraine characteristics
  - Photophobia or phonophobia
  - Visual aura

(Table 3, page 13)
### Meniere’s Disease

- Peripheral, benign etiology
- Vertigo, drop attacks related to
- Tinnitus, hearing loss
- Ear pressure or pain
- Usually unilateral
- Rx with salt and fluid control
- External devices, surgery may be required

### Posterior Circulation TIA

- Central, serious etiology
- Up to half of posterior circulation TIAs have dizziness as only symptom
- Symptoms related to long brainstem tracts
- Cranial nerve dysfunction
- Visual field cuts (posterior cerebral artery ischemia of visual cortex)
- May be high risk TIAs vs anterior circulation
Rare s-EVS Diagnoses

- Cardiac dysrhythmia
- Pulmonary embolism
- Seizure disorder
- Panic attacks and anxiety disorders

Triggered, Episodic Vestibular Syndrome
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<td>Episodic dizziness that occurs spontaneously, is not triggered, and usually lasts minutes to hours</td>
<td>Vestibular migraine</td>
<td>Meningeal disease</td>
<td>Drug/medication side effects or toxicity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Meniere disease</td>
<td></td>
<td>Vasovagal syncope</td>
</tr>
<tr>
<td>h-EVS</td>
<td>Episodic dizziness triggered by a specific, obnoxious trigger (typically a change in head position or standing up), and usually lasting less than 1 minute</td>
<td>BPPV</td>
<td>Orthostatic hypotension caused by benign problems</td>
<td>Gastroesophageal reflux disease</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Orthostatic hypotension due to serious medical illness</td>
<td></td>
<td>Postural orthostatic tachycardia syndrome</td>
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<td></td>
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<td></td>
<td></td>
<td>Panic attack</td>
</tr>
<tr>
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<td></td>
<td>Vestibular artery rotation (slow-Hunter syndrome)</td>
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• Dizziness is "triggered" (not dizzy at baseline, dizziness develops with movement), as in position vertigo due to BPPV. This must be distinguished from dizziness that is "exacerbated" (dizzy at baseline, worsen with movement); such exacerbations in common in AVS, whether peripheral (neuritis) or central (stroke).

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**Triggered EVS (t-EVS) Diagnoses**

- Specific trigger induces symptoms
- Change in head position or standing up
- Usually lasts less than one minute
- Panic attacks and anxiety disorders
- Not dizzy at baseline
- (Exacerbated or worsened baseline dizziness more common in AVS)
Benign Paroxysmal Positional Vertigo

- Peripheral, benign etiology
- Very brief episodes of dizziness
- Episodes that wake patients up from sleep
- Dizziness occurs with lying down or turning in bed: Positive Likelihood Ratio = 60 (Huge)
- Most often posterior canal
- Dix-Hallpike test or supine head roll test

BPPV Important Nystagmus Note!

- In posterior canal BPPV, the nystagmus is vertical and rotational!
- In horizontal canal BPPV, the nystagmus is horizontal and direction changing with gaze!
- These are benign findings in t-EVS BPPV, but are worrisome in AVS
- Nystagmus interpretation depends on if the symptoms suggest AVS or t-EVS BPPV
Central Paroxysmal Positional Vertigo

- Central, serious etiology
- Structural lesions adjacent to 4th ventricle
- Tumor, MS plaque, small brainstem stroke
- Headache, diplopia, frequent recurrence
- Abnormal cranial nerve or cerebellar function
- Down beating* or non-extinguishing nystagmus
- Nystagmus in isolation without dizziness
- Poor response to positional maneuvers

Orthostatic Hypotension

- Peripheral, benign or serious etiologies possible
- Orthostatic VS, sx suggest hypovolemia
- (May be optimized if done after treatment)
- Stand for 2-3 minutes prior to VS testing
- HR increase of 20-30 BPM
- Decline in SBP ≥ 20 mm Hg
- Decline in DBP ≥ 10 mm Hg

https://www.ahrq.gov/professionals/systems/hospital/fallptoolkit/fallptk-tool3f.html
Rare t-EVS Diagnoses

- Superior canal dehiscence
- Postural tachycardia syndrome
- Panic attack
- Vertebral artery rotation
  (Bow Hunter’s syndrome)

Benign Paroxysmal Positional Vertigo (BPPV)
BPPV

- Posterior canal in 80-85%
- Dix-Hallpike maneuver is diagnostic
- Up-beating and torsional nystagmus
- Epley maneuver treats this type of dizziness
- It repositions otoliths which have moved into not normal location
BPPV Pathophysiology

- Dislodged otoliths from the utricle migrate into one of the semicircular canals
- Posterior canal is most dependent, common
- Otoliths cause motion to be simulated
- “Heavy cupula” simulates motion as otoliths become stuck in cupula
- Disruption of vestibular-ocular reflex (VOR), which maintains gaze with movement

Dix-Hallpike Test

- Patient lies on exam table, outstretched legs
- Head turned 45º to one side, then head dropped down 30º below table
- Examine for nystagmus for 30 seconds
- If maneuver causes symptoms with nystagmus to be seen, Epley maneuver may help to reposition semicircular canal crystals
Dix-Hallpike Test

- Head turned and dropped down
- Assess for symptom of vertigo (dizziness)
- Examine for nystagmus
- If both positive over 30 seconds, this test confirms the BPPV diagnosis

Epley Maneuver

- Turning the head in all three planes
- Changing head position to allow for the otoliths to return to their normal location away from the semicircular canals
- Cure may require repeated Epley Maneuver sessions, much like physical therapy
- There are ENT specialists that perform this maneuver on a regular basis in vertigo patients.
Epley Maneuver

• Pt is positioned lying flat, with the head hanging over the end of the bed, turned 45° away from the midline
• Turn the patient’s head 90° to the contralateral side, approximately 45° past the midline for 30 seconds
• While maintaining the position of the patient’s head, ask the patient to roll onto their shoulder on the side their head is currently turned towards


Epley Maneuver

• Once the patient is on their side, rotate the patient’s head so that they are looking directly towards the floor. Maintain this position for 30 seconds to a minute.
• Sit the patient up sideways, while maintaining head rotation.
• Once the patient is sitting upright, the head can be re-aligned to the midline and the neck can be flexed so that the patient is facing downwards (chin to chest). Maintain this position for 30 seconds.

In a patient with possible BPPV, the most likely physical examination maneuver to help confirm the diagnosis is:

a. Head impulse test
b. Skew deviation
c. Finger-to-nose testing
d. Dix-Hallpike maneuver

Answer: D. Dix-Hallpike maneuver

Explanation:

• In patients with suspected BPPV, bedside testing can confidently establish the diagnosis. The most commonly affected canal is the posterior canal (pc-BPPV) which is usually tested by the Dix-Hallpike maneuver. If this test is negative on both sides, then the horizontal canal (hc-BPPV) is tested by the supine head roll test. In pc-BPPV, the nystagmus is typically up-beating and torsional, and in hc-BPPV it is horizontal and direction-changing.

• Section: Triggered Episodic Vestibular Syndrome, page 14.
Dizzy Patient Risk Management Pitfalls

Risk Management Pitfalls for Dizziness in the Emergency Department
(Continued on page 17)

1. “I thought that because the dizziness got worse with head movement, it had to be peripheral.” This is a common misconception. Dizziness at rest in a patient with a cerebellar stroke or tumor often intensifies with head motion. It is crucial to distinguish dizziness that is triggered by movement (no dizziness at rest, but dizziness develops with movement) versus dizziness that is exacerbated by movement (dizziness is present at rest, but worsens with head movement).

2. “The negative CT ruled out a stroke!” Brain CT is a great test for hemorrhage, but a terrible test for posterior circulation infarction. Hemorrhage is a very uncommon cause of isolated dizziness without other symptoms or signs. One should never rely on a negative head CT to exclude a cerebellar or brainstem infarct.

3. “I ruled out a posterior circulation TIA because isolated dizziness is never due to ischemic other brainstem findings will always be present.” This is a misconception that stems from old expert opinion dating back to the mid-1970s. Nearly all studies make it clear that isolated dizziness is the most common transient symptom that precedes posterior circulation stroke and occurs in approximately 8% of these patients.

4. “The patient had acute dizziness for 24 hours. The neuroradiologist read the MRI with diffusion-weighted images as negative, so there is no way this is a stroke.” It is important to emphasize that in the first 6 to 72 hours after a posterior circulation ischemic stroke that presents with isolated dizziness, diffusion-weighted MRI will miss as many as 1 in 5 patients. There is a reluctance by physicians, in general, to accept that an MRI can be negative in any acute stroke, but the data are clear.

5. “The patient had a bad headache and said he had some transient double vision, but the Dix-Hallpike test was positive on both sides. I gave him meclizine for his BPPV.” There are some symptoms that never occur with BPPV—including headache and double vision. One can never make a diagnosis of BPPV in a patient with severe headache or diplopia (even if transient). As well, the treatment for BPPV is a canalith repositioning maneuver such as the Epley maneuver, not meclizine.
6. “The patient had both hearing loss and dizziness, so it has to be a peripheral problem – right?”
The classic teaching that coexistence of an acute hearing loss plus dizziness is always a peripheral lesion is wrong. A stroke of the AICA territory or of the labyrinthine artery can cause a stroke of the lateral pons or of the vestibular labyrinth and cause both hearing and balance findings.

7. “The patient was only 32 years old with no vascular risk factors, so there’s no way that this is a stroke.”
Young patients presenting with orofacial symptoms are more common in young stroke patients than in vestibular neuritis patients, including anterior cerebral and carotid territory strokes (especially through a patient foramen ovale), but they also can have large-artery disease. Young age is associated with stroke misdiagnosis.

8. “The patient felt very dizzy on gait examination, but had no nystagmus at all, so I ruled out cerebellar stroke.”
Only about half of patients with cerebellar strokes have nystagmus, so its absence in no way rules it out. In fact, the absence of nystagmus makes acute cerebellar strokes less likely and probably increases the probability of stroke in a patient with an ACS without nystagmus.

9. “I know HINTS testing and the HT was unequivocally positive. I saw a corrective saccade, so the problem must be vestibular neuritis.”
No single component of the HINTS testing rules out stroke. Although the HT is the most sensitive of all the components of HINTS, it still only has a sensitivity of about 85%. It is also important to recognize that this sensitivity was done in studies by neuro-otologists, and the sensitivity in routine emergency medicine practice is not known.

10. “The HINTS testing was worrisome, but the neurologist said to do a MRI and if it was negative, to discharge the patient and he would see him on Monday.”
Neurologists are the usual consultant for an acutely dizzy patient, but many neurologists still use the outdated “symptom-quality” approach to dizziness and some are unfamiliar with newer data about the HINTS testing being more sensitive than early MRI. If the physical examination suggests a central cause, it trumps a “negative” MRI.
Dizziness Case: 44 yo Dizzy Male

- Sign out. Just check the CT...
- 44-year-old male, previously healthy
- Six hours continuous dizziness
- Unsteadiness. “feeling like I am drunk…”
- Normal VS
- L beating nystagmus with gaze, L gaze
- “If CT is OK, home with meclizine…”

Dizziness Case: 44 yo Dizzy Male

- “If CT is OK, home with meclizine…”
- Wait!
- Negative brain CT does not reliably exclude a posterior circulation event
- Acute vestibular syndrome
- Need to complete the 5 step exam
- Worrisome presentation, admitted for obs
Dizziness Case: Lightheaded 70 yo

- 70-year-old female
- “Lightheadedness” for five days
- Worse with getting out of bed
- The dizziness wakes her out of sleep
- HTN, cholesterol meds
- VS OK, normal neuro exam, no nystagmus
- Should a CT be ordered? Why?

Dizziness Case: Lightheaded 70 yo

- 70-year-old female
- Consider BPPV diagnosis:
  - Intermittent dizziness
  - Dizziness wakes out of sleep
  - Sx at rest, no nystagmus
  - Vertigo not necessarily reported
- Dix-Hallpike test +, Rx Epley maneuver
Dizziness Case: Syncopal 58 yo

- 58-year-old male  DM, NV x 3
- Syncope reported, but was such bad vertigo he had to lower himself to the ground
- Began 3 hours previously, suddenly
- Nystagmus to R, higher amplitude gaze R
- HIT test +, skew neg, mild unsteady OK gait
- Call the stroke team?

Dizziness Case: Syncopal 58 yo

- 58-year-old male  DM, NV x 3
- AVS requires good exam, 5 questions
- Nystagmus to R, no skew deviation, corrective HIT saccade, no other neuro findings, and no ataxia all reassuring
- Less likely presentation of posterior stroke
- Rx with steroids, anti-histamines
Dizziness and COVID-19 Infection

COVID-19 CNS Mechanisms

- COVID-19 virus uses ACE2 receptor for access
- ACE2 receptors in neurological tissues
- Brain access thru nasal olfactory epithelium and thru CNS capillary endothelial lining
- Potential for causing cerebral edema prior to diffuse organ system loss of homeostasis

Baig Targeting CNS Mech ACS Chem Neurosc March 2020
COVID-19 CNS Mechanisms

• Direct infection injury via blood neurons
• Hypoxic injury (given respiratory pathogen)
• ACE2 receptor cellular access
• Immune injury (SIRS)
• Infectious toxic encephalopathy, viral encephalitis, acute cerebrovascular disease

Wu Nervous System Involve Brain Beh Imm March 2020

COVID-19 CNS Mechanisms

• Viral infections can cause seizures/SE, encephalitis, neuromyopathy, Guillain-Barre, transverse myelitis, flaccid myelitis
• Coronavirus, influenza, RSV, hMPV, enterovirus
• Direct neurotropism of these viruses
• Affinity for localizing selectively in nerve tissue

Robinson Neuro Viral Contagions Crit Care Expl Apr 2020
COVID-19 CNS Complications

- Myalgia and headache common
- Direct viral invasion of all neurological tissues
- Myopathy, polyneuropathy, rhabdomyolysis
- Meningitis and encephalitis
- Anosmia, ageusia (loss of smell and taste)
- Post-viral autoimmune processes
- Encephalomyelitis, Guillain-Barre

Berger Nervous System J NeuroVir Apr 2020

COVID-19 Neuro Symptoms

- Observational case series, Wuhan, China
- 214 patients, 41% severe (more neuro comps)
- 36% neuro complications in 3 categories
- CNS (dizzy HA, AMS, CVA, ataxia, seizure)
- Peripheral (taste, smell, vision, nerve pain)
- Skeletal muscle injury (neuromuscular)

Mao Neuro Comps Wuhan China JAMA Neuro May 2020
COVID-19 and Headache

• MEDLINE search
• 41,000 patients described
• Headache in 8-12%
• HA in two disease phases:
  • Acute headache in viral infection phase
  • HA with hypoxia and with cytokine storm

Belvis COVID 19 Headache Case Headache May 2020

COVID-19 Headache

• HA reported in 11-34% of hospital COVID-19 pts
• New, sudden-gradual onset, moderate-severe, throbbing or pressure, poor analgesic response
• Bilateral temporoparietal, frontal, or periorbital
• High relapse rate in active infection phase
• Possible peripheral trigeminal nerve endings or pro-inflammatory hypoxia and/or cytokines

Bolay COVID 19 Headache Headache May 2020
COVID-19 Meningo-Encephalitis

• 64-year-old woman, no psych hx
  – Psych ward, then tonic-clonic seizure (focal SE)
  – MRI normal, LP cw meningo-encephalitis
  – Treated with acyclovir, improved over 4 days

• 67-year-old woman, COVID positive for 17 days
  – Intense wake up HA, confused, bathroom syncope
  – Left sided stroke symptoms, MRI OK, CSF pleocytosis
  – Rx acyclovir, anbx, discharged after 4 days

Bernard-Valnet Mening-Enceph Eur J Neuro May 2020

COVID-19 Meningitis

• 24-year-old nine days into fever, HA, fatigue
• Unresponsive, emesis, lying on floor
• Transient generalized tonic-clonic seizures
• CSF COVID-19 detected
• MRI positive in lateral ventricle, temporal lobe, hippocampus suggestive of meningitis

Moriguchi Meningitis Encephalitis Int J Inf Sx Mar 2020
COVID-19 Orbital Cellulitis

- 12-year old Egyptian male, 3 days orbital swelling
- 15-year old male, 3 days orbital swelling
  - Ceftriaxone and vancomycin
  - CT: Ipsilateral pansinusitis, orbital cellulitis, epidural abscess
  - Case 1: Surgical intervention, anbx, improvement
  - Case 2: Surgery, persistent fever, complicated course

Turbin Orbital Cellulitis Sinusitis CNS Orbit Apr 2020

COVID-19 CNS Venous Thrombosis

- 59-year-old, 4 days fronto-temporal headache
  - DM, HTN, persistent, severe HA, fever x 1
  - CT superior sagittal, transverse, sigmoid sinus and IJ vein densities suggestive of thrombus, CTV negative
  - Represented 4 days later with stroke sx (unilateral weakness, slurred speech, expressive aphasia)
  - CTV re-read: sigmoid and transverse sinus defects
  - LMWH, NIHSS from 10 to 4 at 24 hours, d/c home

Hughes Cereb Venous Sinus Thromb Eur J Int Med 2020
COVID-19 CNS Thrombosis

- 64-year-old male, wake up stroke, L hemiparesis, SOB, no tPA
  - 16 days prior fever, myalgias, COVID 19
  - Tachycardia, HTN, hypoxic respiratory failure, ETI
  - CT: hypo-attenuation, loss gray-white differentiation
  - CTA: ICA high-grade stenosis; no IC occlusion
  - Repeat CT at 24 hours: cerebral edema, mass effect, R MCA and ACA territories

Goldberg CV Disease AJNR May 2020

Dizziness and COVID-19

- COVID-19 virus is neurotropic, toxic
- COVID-19 can cause dizziness via:
  - Direct labyrinth, vestibular nerve effects
  - CNS, head and neck infections
  - Dehydration, hypovolemia, orthostasis
  - CNS venous thrombosis
  - CNS posterior circulation thrombosis
Dizziness Patient Conclusions

• Acute, severe, continuous sx: AVS
• Intermittent sx: triggered or spontaneous
• Orthostasis, BPPV, posterior stroke top Dx list
• Five exam findings help exclude posterior stroke
• BPPV Dx with Dix-Hallpike; Rx with Epley
• Caution in setting of COVID-19 pandemic
• EMR templates, dot phrases help make exam process systematic and easily accomplished

Dizziness Pt Recommendations

• Understand dizziness pathologies, 3 diagnostic strata, 6 diagnoses, and (3) treatments
• Study nystagmus findings, significance, and BPPV maneuvers online and in monograph
• Create templates, dot phrases to exclude posterior stroke findings in dizzy patients
• Explain Dx, provide patients follow-up
• Caution with dizzy patients in COVID-19 setting
Questions?
ferne.org@gmail.com