DIZZINESS VERTIGO SYNCOPE AND PRESYNCOPE

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Vertigo and Dizziness

- Prevalence
  - 1 in 10 adults report dizziness in last month
  - Increases in elderly
  - Worsened by decreased visual acuity, proprioception and vestibular input
- Dizziness
  - Non-specific term
  - Different meanings to different people
    - Could mean
      - Vertigo
      - Weak
      - Anemia
      - Syncope
      - Giddiness
      - Depression
      - Presyncope
      - Anxiety
      - Unsteady

Vertigo and Dizziness

- Vertigo
  - Perception of movement
  - Peripheral or Central
- Syncope
  - Transient loss of consciousness with loss of postural tone

Vertigo and Dizziness

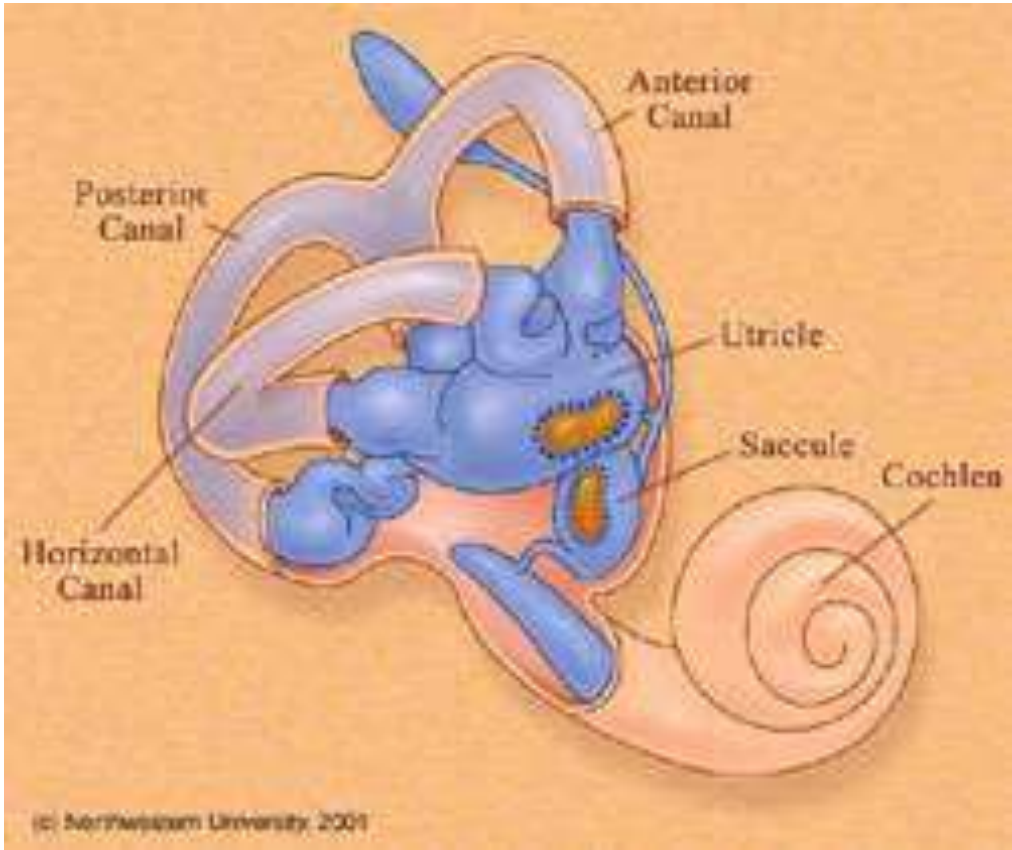
- Presyncope
  - Lightheadedness-an impending loss of consciousness
- Psychiatric dizziness
  - Dizziness not related to vestibular dysfunction
- Disequilibrium
  - Feeling of unsteadiness, imbalance or sensation of “floating” while walking

VERTIGO VS SYNCOPE

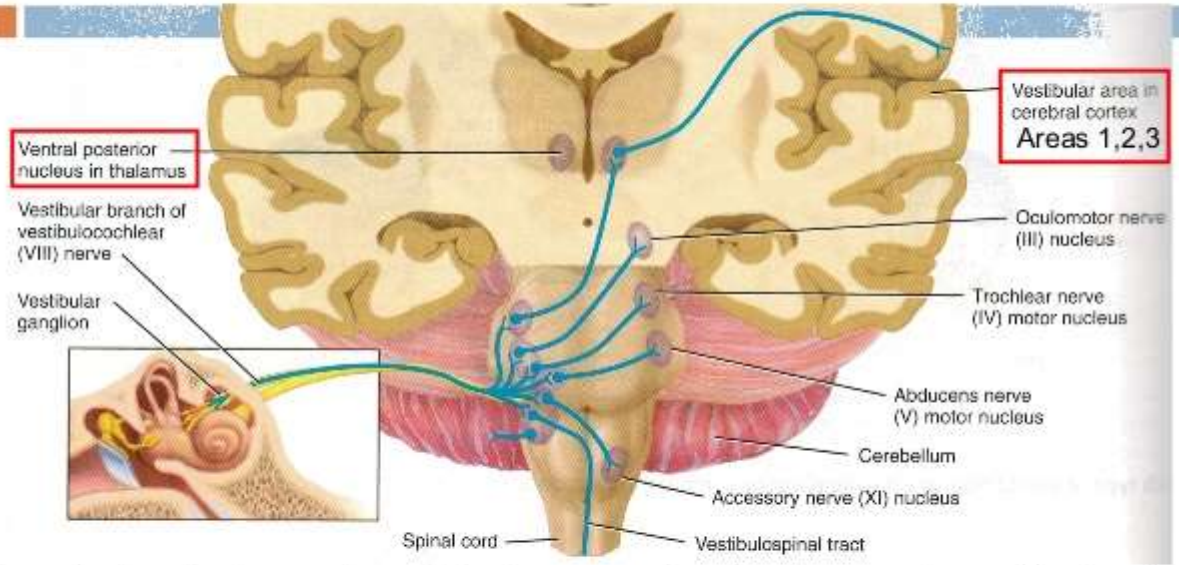
VERTIGO

Vestibular Labyrinth

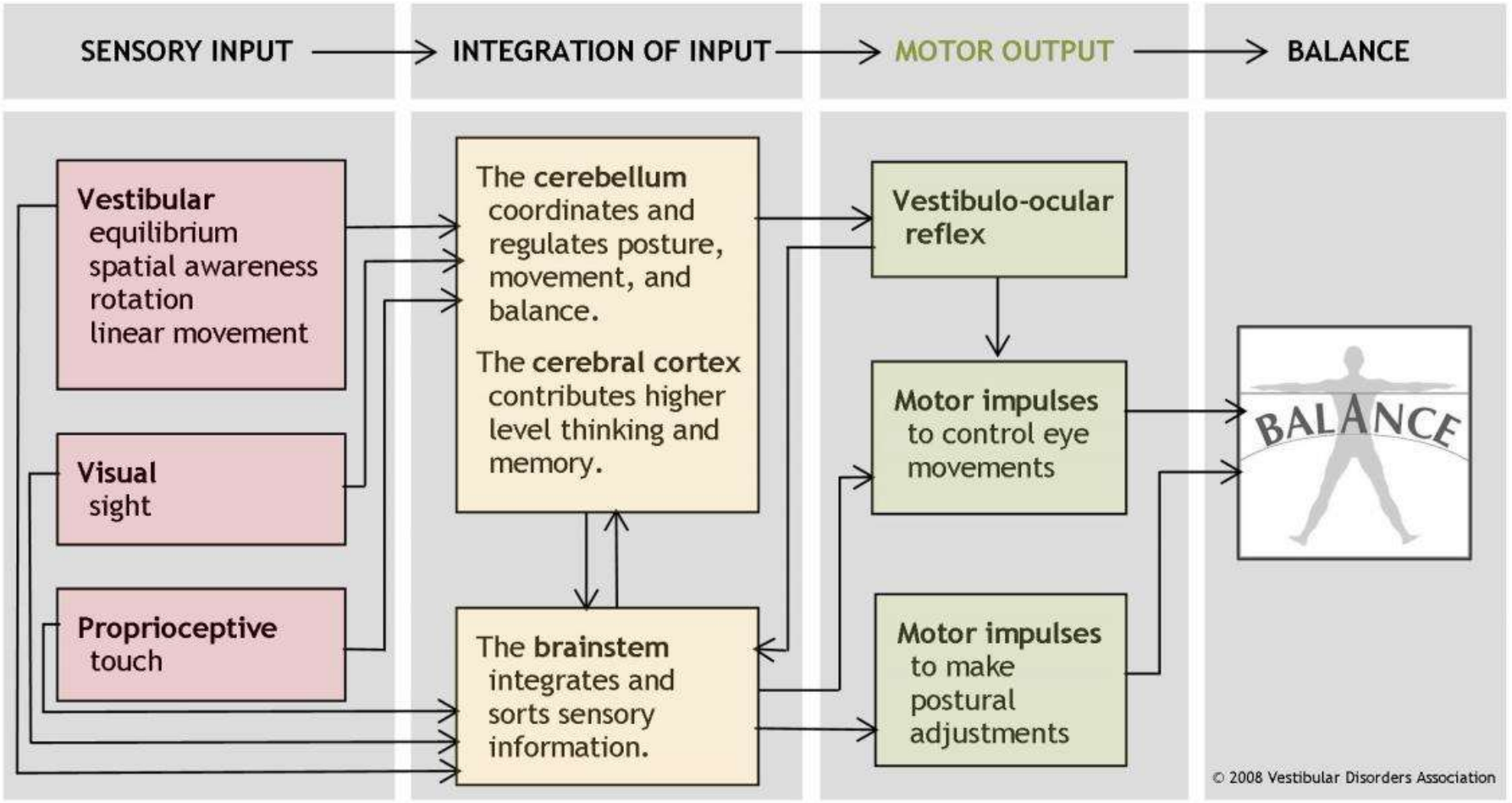
- Pathophysiology
  - Complex interaction of visual, vestibular and proprioceptive inputs that the CNS integrates as motion and spatial orientation
- 3 semicircular canals
  - rotational movement
  - cupula
- 2 otolithic organs
  - utricle & saccule
  - linear acceleration
  - Macula



Vestibular Pathways



- 4.<u>ventral posterior nucleus of thalamus</u> and <u>vestibular area in cerebral cortex</u> (part of primary somatosensory cortex)
  - Conscious awareness of the position and movement of head


SENSORY INPUT
INTEGRATION OF INPUT
MOTOR OUTPUT
BALANCE
Vestibular
equilibrium
spatial awareness
rotation
linear movement
Visual
sight
Proprioceptive
touch
The cerebellum coordinates and regulates posture, movement, and balance.
The cerebral cortex contributes higher level thinking and memory.
The brainstem integrates and sorts sensory information.
Vestibulo-ocular reflex
Motor impulses to control eye movements
Motor impulses to make postural adjustments
BALANCE
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Vertigo-History

- Is it true vertigo?
- Autonomic symptoms?
- Pattern of onset and duration
- Auditory disturbances?
- Neurologic disturbances?
- Was there syncope?
- Unusual eye movements?
- Any past head or neck trauma?
- Past medical history?
- Previous symptoms?
- Prescribed and OTC medications?
- Drug and alcohol intake?

Vertigo-Physical Exam

- Cerumen/FB in EAC
- Otitis media
- Pneumatic otoscopy
- Tympanosclerosis or TM perforation
- Nystagmus
- Fundoscopic exam
- Pupillary abnormalities
- Extraocular muscles
- Cranial nerves
- Internuclear ophthalmoplegia
- Auscultate for carotid bruits
- Orthostatic vital signs
- BP and pulse in both arms
- Dix-Hallpike maneuver
- Gross hearing
- Weber-Rinne test
- External auditory canal vesicles
- Muscle strength
- Gait and Cerebellar function

Dix-Hallpike Maneuver

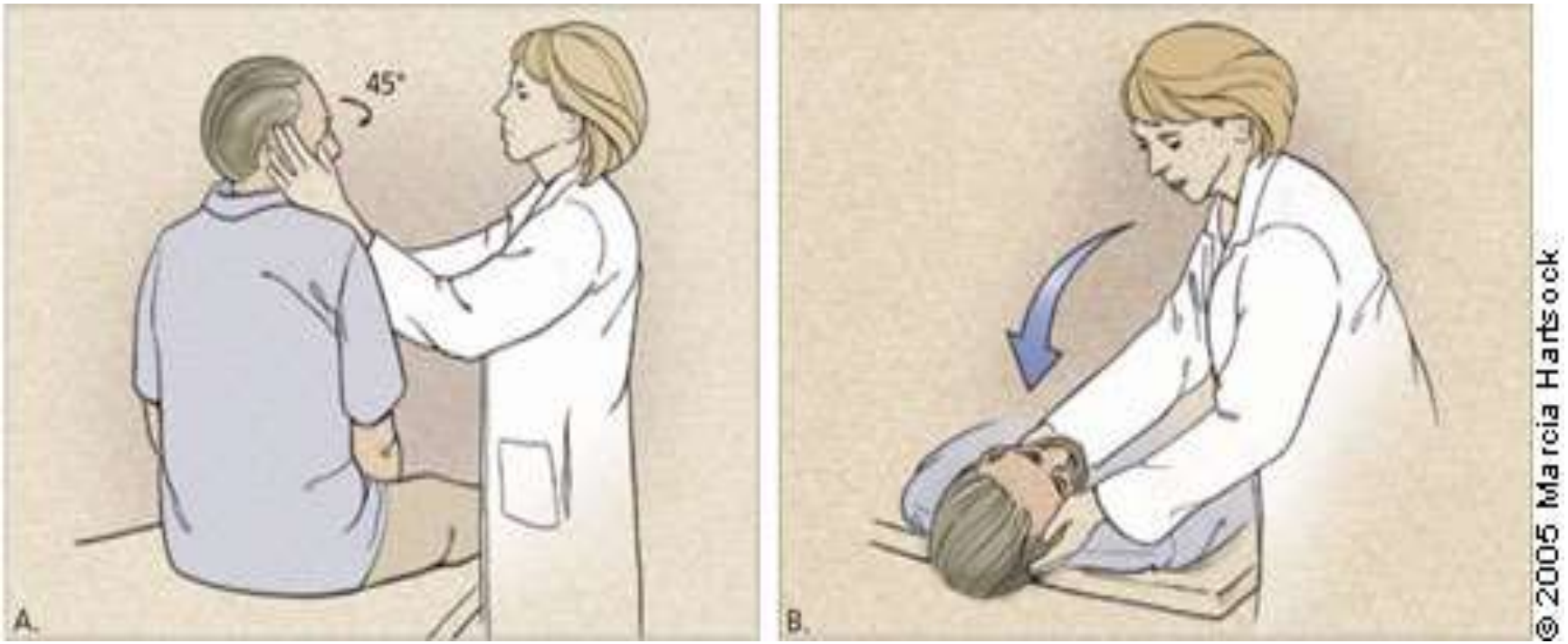
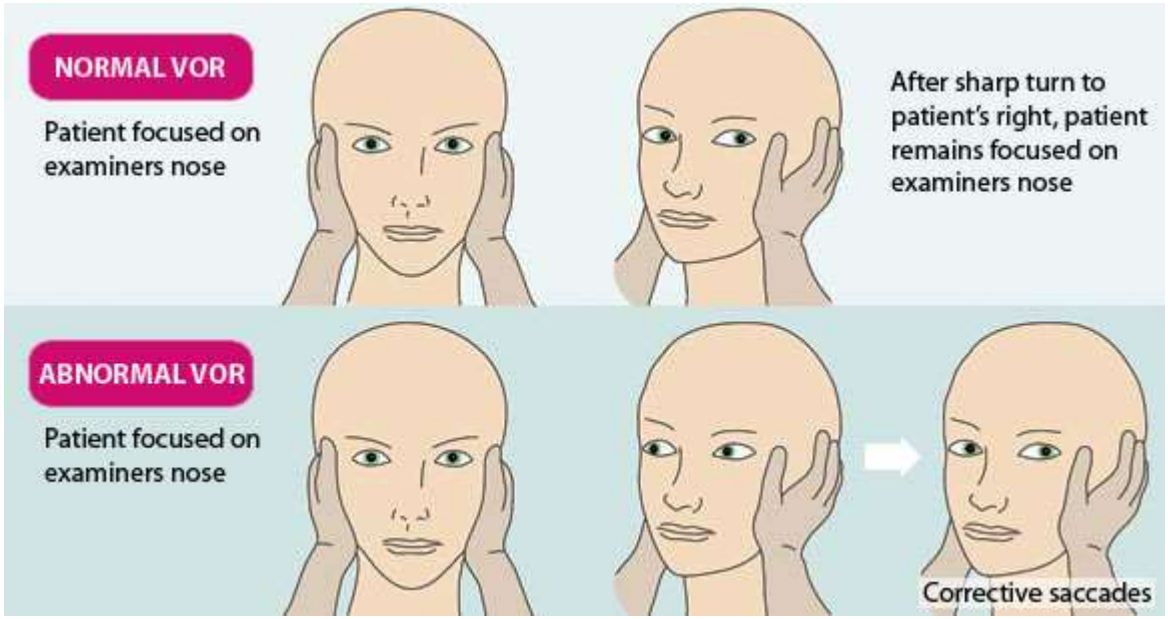


Figure 1. Dix-Hallpike maneuver (used to diagnose benign paroxysmal positional vertigo). This test consists of a series of two maneuvers: With the patient sitting on the examination table, facing forward, eyes open, the physician turns the patient's head 45 degrees to the right (A). The physician supports the patient's head as the patient lies back quickly from a sitting to supine position, ending with the head hanging 20 degrees off the end of the examination table. The patient remains in this position for 30 seconds (B). Then the patient returns to the upright position and is observed for 30 seconds. Next, the maneuver is repeated with the patient's head turned to the left. A positive test is indicated if any of these maneuvers provide vertigo with or without nystagmus.


NORMAL VOR
Patient focused on examiners nose
After sharp turn to patient's right, patient remains focused on examiners nose
ABNORMAL VOR
Patient focused on examiners nose
Corrective saccades

Vertigo-Characteristics

	Peripheral	Central
Onset	Sudden	Usually slow
Severity of Vertigo	Intense	Usually mild
Pattern	Paroxysmal	Constant
Exac. by movement	Yes	Variable
Autonomic	Frequent	Variable
Laterality	Unilateral	Uni or bilat
Nystagmus	Horizontorotary	Any
Fatigable/Fixation	Yes	No
Auditory symptoms	Yes	No
TM	May be abnormal	Normal
CNS symptoms	Absent	Present

Table 1. Differences Between Peripheral and Central Vertigo

Characteristic	Peripheral	Central
Severity of attack	Severe; unable to stand still or walk	Mild to moderate; able to walk
Onset	Sudden	Gradual
Duration	Seconds to minute	Weeks to months
Positional	Yes	No
Associated symptoms	Auditory	Neurologic and visual
Nystagmus	Combined horizontal and rotational	Purely vertical, horizontal, or rotational
Nausea/vomiting	May be severe	Varies

Source: References 3, 7, 12.

Peripheral Vertigo-Differential

- Labyrinthine Disorders
  - Most common cause of true vertigo
  - Five entities
    - Benign paroxysmal positional vertigo (BPPV)
    - Labyrinthitis
    - Ménière disease
    - Vestibular neuronitis
    - Acoustic Neuroma

Benign Paroxysmal Positional Vertigo

- Extremely common
- Otoconia displacement
- No hearing loss or tinnitus
- Short-lived episodes brought on by rapid changes in head position
- Usually a single position that elicits vertigo
- Horizontorotary nystagmus with crescendo-decrescendo pattern after slight latency period
- Less pronounced with repeated stimuli
- Typically can be reproduced at bedside with positioning maneuvers

Labyrinthitis

- Associated hearing loss and tinnitus
- Involves the cochlear and vestibular systems
- Abrupt onset
- Usually continuous
- Four types of Labyrinthitis
  - Serous
  - Acute suppurative
  - Toxic
  - Chronic

Labyrinthitis

- Serous
  - Adjacent inflammation due to ENT or meningeal infection
  - Mild to severe vertigo with nausea and vomiting
  - May have some degree of permanent impairment

- Acute suppurative labyrinthitis
  - Acute bacterial exudative infection in middle ear
  - Secondary to otitis media or meningitis
  - Severe hearing loss and vertigo
  - Treated with admission and IV antibiotics

Labyrinthitis

- Toxic
  - Due to toxic effects of medications
  - Still relatively common
  - Mild tinnitus and high frequency hearing loss
  - Vertigo in acute phase
  - Ataxia in the chronic phase
  - Common etiologies

    -Aminoglycosides
    -Erythromycin
    -Phenytoin
    -Quinidine
    -Alcohol
    -Vancomycin
    -Barbiturates
    -Furosemide
    -Salicylates

Vestibular Neuronitis

- Suspected viral etiology
- Sudden onset vertigo that increases in intensity over several hours and gradually subsides over several days
- Mild vertigo may last for several weeks
- May have auditory symptoms
- Highest incidence in 3rd and 5th decades

Ménière Disease

- First described in 1861
- Triad of vertigo, tinnitus and hearing loss
- Due to cochlea-hydrops
  - Unknown etiology
  - Possibly autoimmune
- Abrupt, episodic, recurrent episodes with severe rotational vertigo
- Usually last for several hours

Ménière Disease

- Often patients have eaten a salty meal prior to attacks
- May occur in clusters and have long episode-free remissions
- Usually low pitched tinnitus
- Symptoms subside quickly after attack
- No CNS symptoms or positional vertigo are present

Acoustic Neuroma

- Peripheral vertigo that ultimately develops central manifestations
- Tumor of the Schwann cells around the 8th CN
- Vertigo with hearing loss and tinnitus
- With tumor enlargement, it encroaches on the cerebellopontine angle causing neurologic signs
- Earliest sign is decreased corneal reflex
- Later truncal ataxia
- Most occur in women during 3rd and 6th decades

Central Vertigo-Differential

- Central Vertigo
  - Vertebrobasilar Insufficiency
    - Atheromatous plaque
    - Subclavian Steal Syndrome
    - Drop Attack
    - Wallenberg Syndrome
  - Cerebellar Hemorrhage
  - Multiple Sclerosis
  - Head Trauma
  - Neck Injury
  - Temporal lobe seizure
  - Vertebral basilar migraine
  - Metabolic abnormalities
    - Hypoglycemia
    - Hypothyroidism

Vertebrobasilar Insufficiency

- Important causes of central vertigo
- Related to decreased perfusion of vestibular nuclei in brain stem
- Vertigo may be a prominent symptom with ischemia in basilar artery territories
- Unusual for vertigo to be only symptom of ischemia

Vertebrobasilar Insufficiency

- Most commonly will also have:

  -Dysarthria -Ataxia -Facial numbness

  -Hemiparesis -Diplopia -Headache

- Tinnitus and hearing loss unlikely

- Vertical nystagmus is characteristic of a (superior colliculus) brain stem lesion

- Up to 30% of TIA's are VBI with pontine symptoms and a focal neurologic lesion

Drop attack

- Abruptly falls without warning, but does not loose consciousness

- Believed to be caused by transient quadraparesis due to ischemia at the pyramidal decussation

Subclavian Steal Syndrome

- Rare, but treatable
- Arm exercise on side of stenotic subclavian artery usually causes symptoms of intermittent claudication
- Blood is shunted away from brainstem into ipsilateral vertebral artery
- Classic history occurs only rarely

Wallenberg Syndrome

- Occlusion of PICA
- Relatively common cause of central vertigo
- Associated Symptoms:

  -nausea -vomiting -nystagmus

  -ataxia -Horner syndrome

  -palate, pharynx and laryngeal paresis

  -loss of pain and temperature on ipsilateral face and contralateral body

Cerebellar Hemorrhage

- Neurosurgical emergency
- Suspected in any patient with sudden onset headache, vertigo, vomiting and ataxia
- May have gaze preference
- Motor-sensory exam usually normal
- Gait disturbance often not recognized because patient appears too ill to move

Vertebral Basilar Migraine

- Syndrome of vertigo, dysarthria, ataxia, visual changes, paresthesias followed by headache
- Distinguishing features of basilar artery migraine
  - -Symptoms precede headache
  - -History of previous attacks
  - -Family history of migraine
  - -No residual neurologic signs
- Symptoms coincide with angiographic evidence of intracranial vasoconstriction

Metabolic Abnormalities

- Hypoglycemia
  - Suspected in any patient with diabetes with associated headache, tachycardia or anxiety
- Hypothyroidism
  - Clinical picture of vertigo, unsteadiness, falling, truncal ataxia and generalized clumsiness

Management

- Based on differentiating central from peripheral causes
- VBI should be considered in any elderly patient with new-onset vertigo without an obvious etiology
- Neurological or ENT consult for central vertigo
- Suppurative labrynthitis-admit and IV antibiotics
- Toxic labrynthitis-stop offending agent if possible

Management

- Severe Ménière disease may require chemical ablation with gentamicin
- Attempt Epley maneuver for BPPV
- Mainstay of peripheral vertigo management are antihistamines that possess anticholinergic properties
  - -Meclizine
  - -Promethazine
  - -Scopolamine
  - -Diphenhydramine
  - -Droperidol

Summary

- Ensure you understand what the patient means by “dizzy”
- Try to differentiate central from peripheral
  - Often there is significant overlap
- Not every patient needs a head CT
- Central causes are usually insidious and more severe while peripheral causes are mostly abrupt and benign
- Most can be discharged with antihistamines

RED FLAGS

Vertigo patients who present with the following signs should go immediately to the emergency room:

- double vision
- headache
- weakness
- difficulty speaking
- difficulty waking up or staying awake
- difficulty walking
- inappropriate actions
- difficulty controlling arms or legs
- abnormal eye movements

Nystagmus, which is characterized by uncontrollable, rhythmic, or jerky eye movements, is one of the signs of BPPV. However, additional diagnostic tests and/or ear, nose, and throat or neurological evaluations should be done in patients who have nystagmus to rule out possibly harmful structural causes.

SYNCOPE

SYNCOPE

- ***Definition***
- ***Mechanism***
- ***Causes***
- ***How to approach***
- ***Algorithm for managing cases in ED***
- ***Risk stratification***
- ***Summary***

Definition

- Syncope is a transient loss of consciousness, associated with loss of postural tone, with spontaneous return to baseline neurologic function requiring no resuscitative efforts

Is it common?

- Accounts for one to three percent of all ED visits and hospital admissions in the United States.
- One-third of individuals are likely to have a syncopal episode during their lifetime.
- The incidence of syncope increased with age, with a sharp rise at age 70 years due to age- and disease-related abnormalities that impair the ability to respond to physiologic stresses that would ordinarily not cause syncope
- cardiac syncope have a high incidence of subsequent cardiac arrest (approximately 24 percent)

Causes of Syncope by Age

Younger Patient

- Vasovagal
- Situational
- Psychiatric
- Long QT*
- Brugada syndrome*
- WPW syndrome*
- RV dysplasia*
- Hypertrophic cardiomyopathy*
- Catecholaminergic VT
- Other genetic syndromes

Older Patient

- Cardiac**
  - Mechanical
  - Arrhythmic
- Orthostatic hypotension
- Drug-induced
- Neurally mediated
- Multifactorial

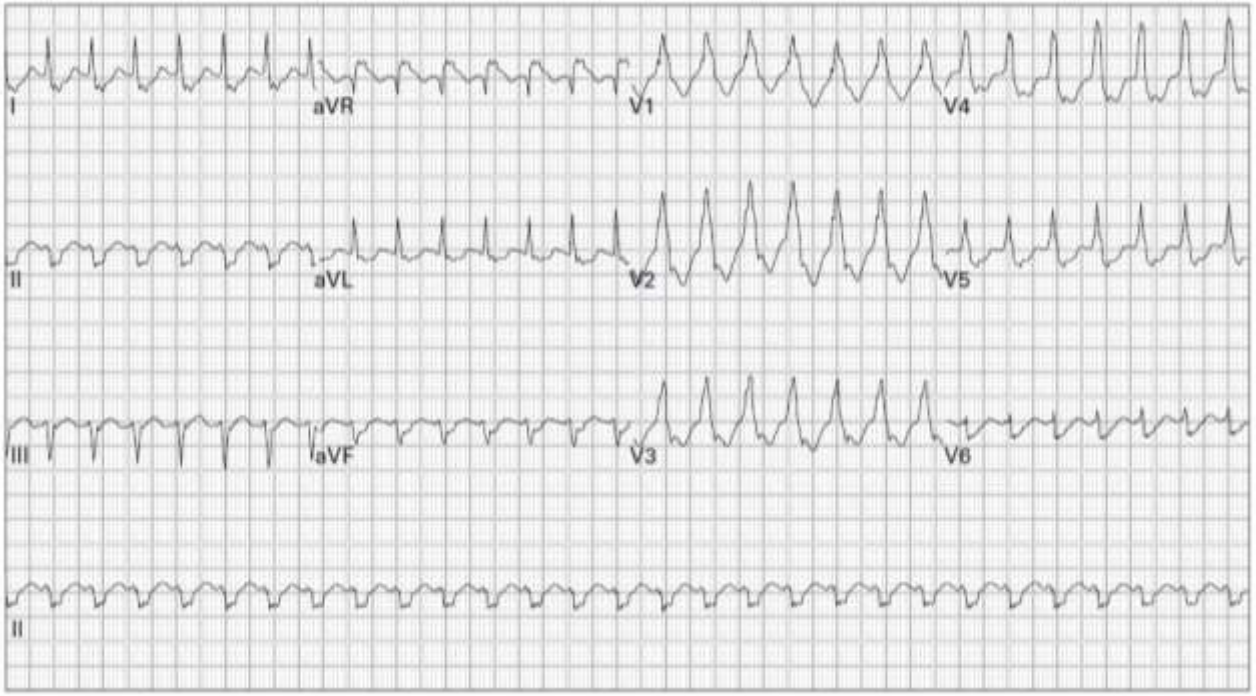
Cardiac syncope

- Cardiac arrhythmias
- Structural cardiac or cardiopulmonary disease
- Ischemia

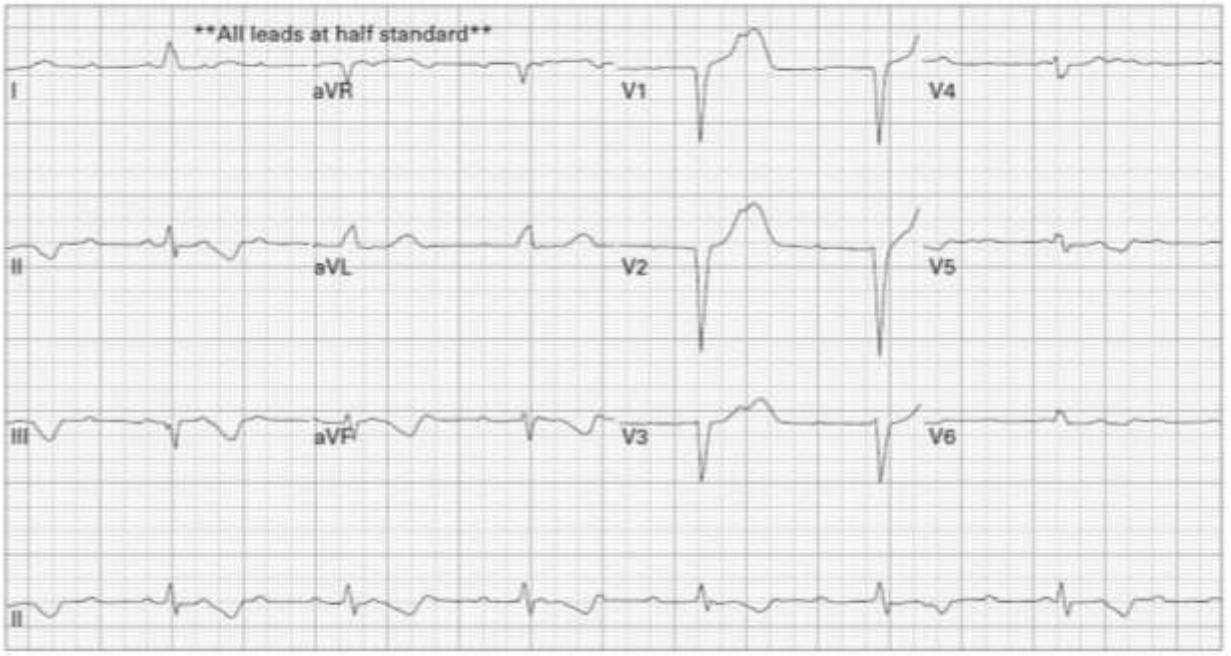
Cardiac syncope

- Cardiac arrhythmias

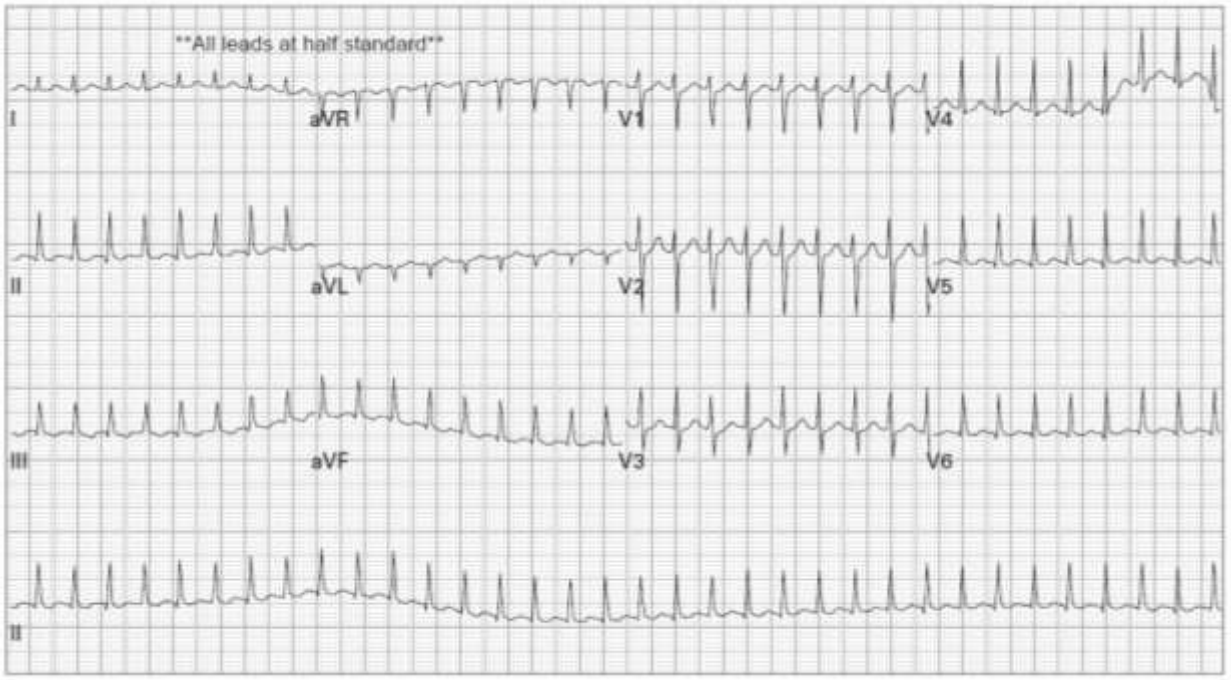
VENTRICULAR TACHYCARDIA



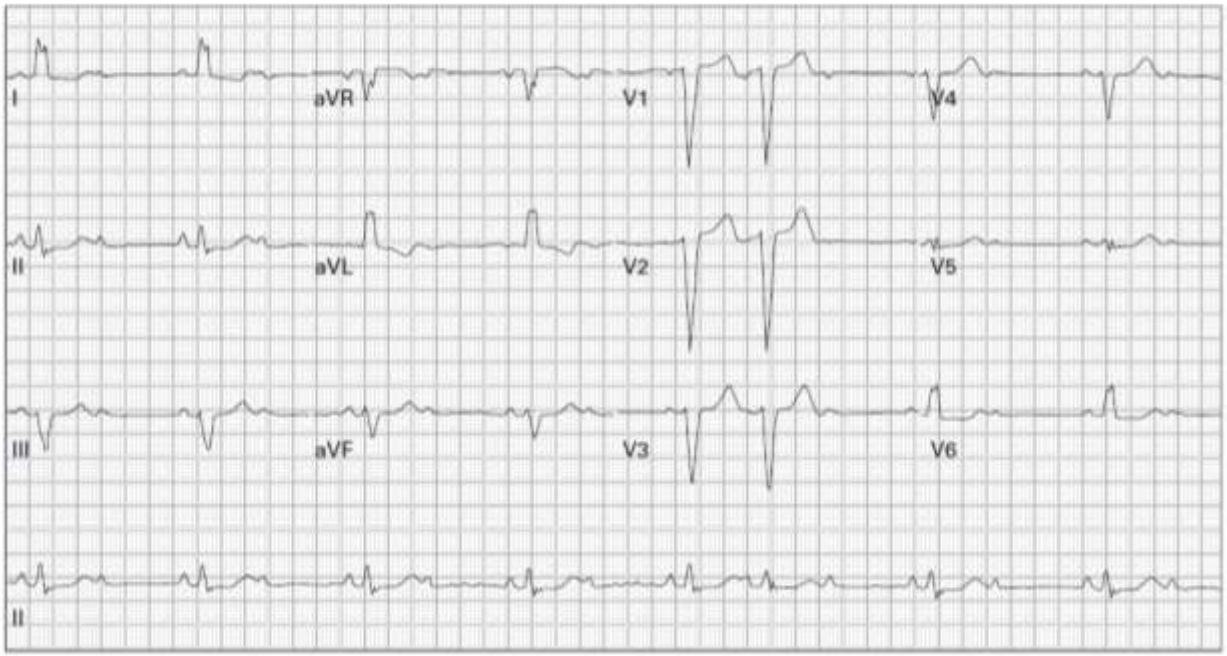
3rd degree heart block



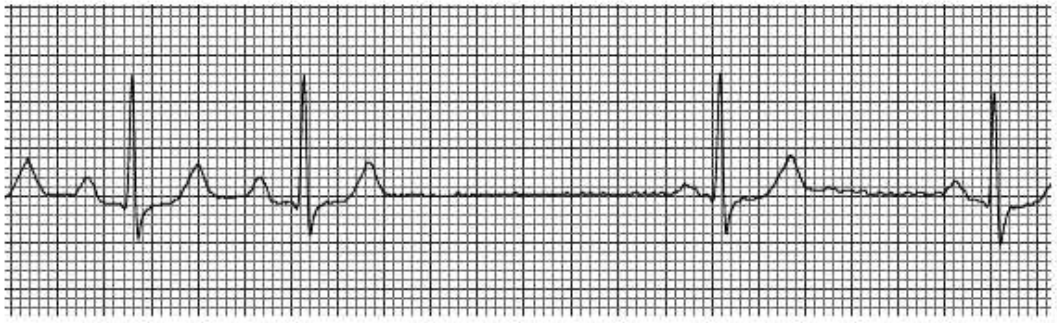
SVT


All leads at half standard
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

Mobtiz type II block



Sinus pause



Cardiac syncope

- Cardiac arrhythmias ⟹ Hemodynamic instability
- Ventricular tachycardia
- Paroxysmal supraventricular tachycardia
- Long QT syndrome
- Bradycardia: Mobitz type II or 3rd degree heart block
- Significant sinus pause >3 seconds & SA node dysfunction
- Implanted device (pacemaker, ICD) malfunction
- Drug-induced proarrhythmias

Cardiac syncope

- Structural cardiac or cardiopulmonary disease:

  → Blood flow obstruction → Abrupt episodic drops in cardiac output

  - Cardiac valvular disease “AS, MS, PS”
  - Obstructive cardiomyopathy
  - Atrial myxoma
  - Acute aortic dissection
  - Pericardial disease/tamponade
  - Pulmonary embolus/pulmonary hypertension

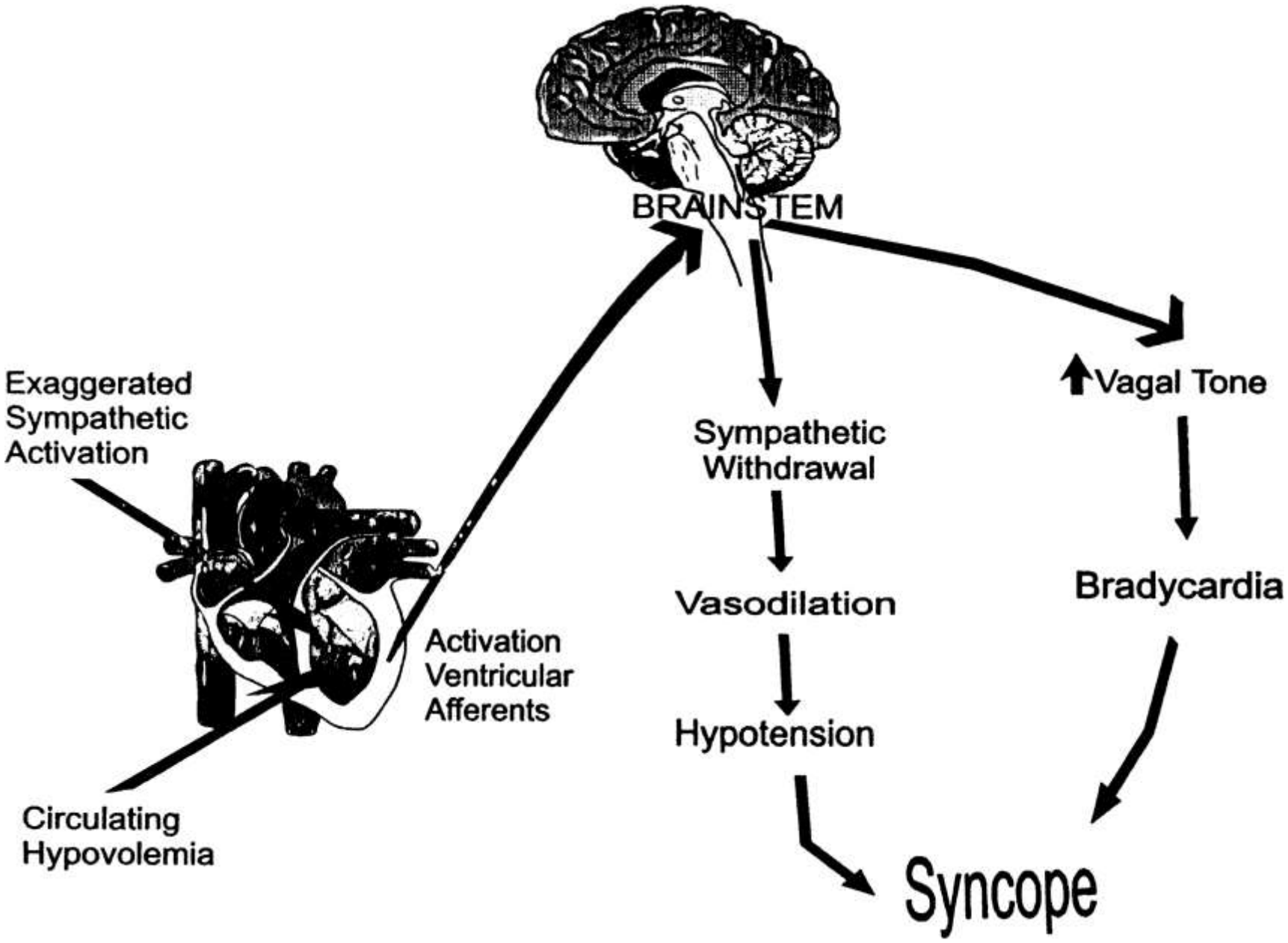
- Acute myocardial infarction/ischemia

NON Cardiac

- Neurocardiogenic syncope “vasovagal or vasodepressor syncope”
- "Classical vasovagal syncope" refers to syncope triggered by emotional or orthostatic stress such as venipuncture (experienced or witnessed), painful or noxious stimuli, fear of bodily injury, prolonged standing, heat exposure, or exertion.
- Situational syncope
- Carotid sinus syncope
- Glossopharyngeal neuralgia

NON Cardiac

- Orthostatic hypotension
- Autonomic failure
- - Primary autonomic failure syndromes (eg, pure autonomic failure, multiple system atrophy, Parkinson's disease with autonomic failure)
- - Secondary autonomic failure syndromes (eg, diabetic neuropathy, amyloid neuropathy)
- - Post-exercise
- - Post-prandial
- Drugs (and alcohol)-induced orthostatic syncope
- Volume depletion
- - Hemorrhage, diarrhea, Addison's disease


BRAINSTEM
Exaggerated
Sympathetic
Activation
Activation
Ventricular
Afferents
Circulating
Hypovolemia
Sympathetic
Withdrawal
Vasodilation
Hypotension
Vagal Tone
Bradycardia
Syncope

NON Cardiac

- Neurovascular syncope:
- Vascular steal syndromes

CAUSES OF SYNCOPE

- Reflex (Neurally-mediated) syncope NMS
  - Vasovagal
    - Emotional stress, fear, pain, blood phobia, instrumentation
    - Mediated by orthostatic stress
  - Situational
    - Cough, sneeze,
    - Gastrointestinal (swallow, defaecation, visceral pain)
    - Post-exercise
    - Post-prandial
    - Others (laugh, brass instrument playing, weightlifting)

CAUSES OF NON-SYNCOPAL ATTACKS

- Episodes that may be confused with syncope include disorders without impairment of consciousness and disorders with partial or complete loss of consciousness .

- ***Seizures*** — They can mimic syncope.

- ***Metabolic or toxic abnormalities*** —. Hypoglycemia and encephalitis can cause coma, stupor, and confusion, but rarely syncope. Nevertheless, if a patient does not recall the history surrounding the event or if the event was unwitnessed, distinguishing coma from syncope

CAUSES OF NON-SYNCOPAL ATTACKS

- ***Neurologic Syncope :***
- subarachnoid hemorrhage
- transient ischemic attack
- complex migraine headache.

- ***Psychiatric syncope***

- ***Drug induced loss of consciousness*** — Drugs of abuse and alcohol may cause a transient loss of consciousness, but generally these patients manifest signs of toxicity, and do not spontaneously return to normal neurologic function immediately after regaining consciousness. Alcohol can also cause symptomatic orthostasis by impairing vasoconstriction.

HOW TO APPROACH A CASE WITH SYNCOPE

HISTORY

- *What actually had happened?*
- *From the patient:*
- *What did he feel just before the collapse?*
- *Was there loss of consciousness?*
- *How long did it take for him to feel back to normal?*
- *From a witness:*
- *Color of the patient during the episode*
- *Seizure activity*
- *Duration of the episode*
- *Speed of recovery*

HISTORY

- *Age:* *Young patients more often experience neurocardiogenic syncope. Emergency clinician must consider the possibility of dysrhythmia particularly if other concerning factors exist (eg, exertional syncope, family history of sudden death).*
- *Associated symptoms and triggers*
- *chest pain, Dyspnea*
- *Palpitations*
- *Headache*
- *Nausea and vomiting, diaphoresis, and pallor*
- *Position*

HISTORY

- Exertional syncope?
- Seizure versus syncope?
- Factors suggestive of seizure include :
- - Prodrome (aura) different from that described for vasodepressor syncope
- - Episode of abrupt onset associated with injury
- - Presence of a tonic phase before the onset of rhythmic clonic activity
- - Head deviation or unusual posturing during the episode
- - Tongue biting (particularly involving the lateral aspect of the tongue)
- - Loss of bladder or bowel control
- - Prolonged postevent (postictal) phase during which the patient is confused.

HISTORY

- Medications
- Previous episodes
- Family history
- Associated injury

PHYSICAL EXAM

- Vital signs
- Irregular pulse
- Low patient oxygen saturation
- Orthostatic vital signs
- Discrepancies between upper extremities in pulse or blood pressure
- General examination
- Intraoral exam
- Neck exam: Carotid bruit, murmurs, JVP
- Injury assessment

PHYSICAL EXAM

- Cardiac examination:
- S3 or S4
- Murmurs
- Lung examination
- Neurologic examination:
- Any focal abnormality?

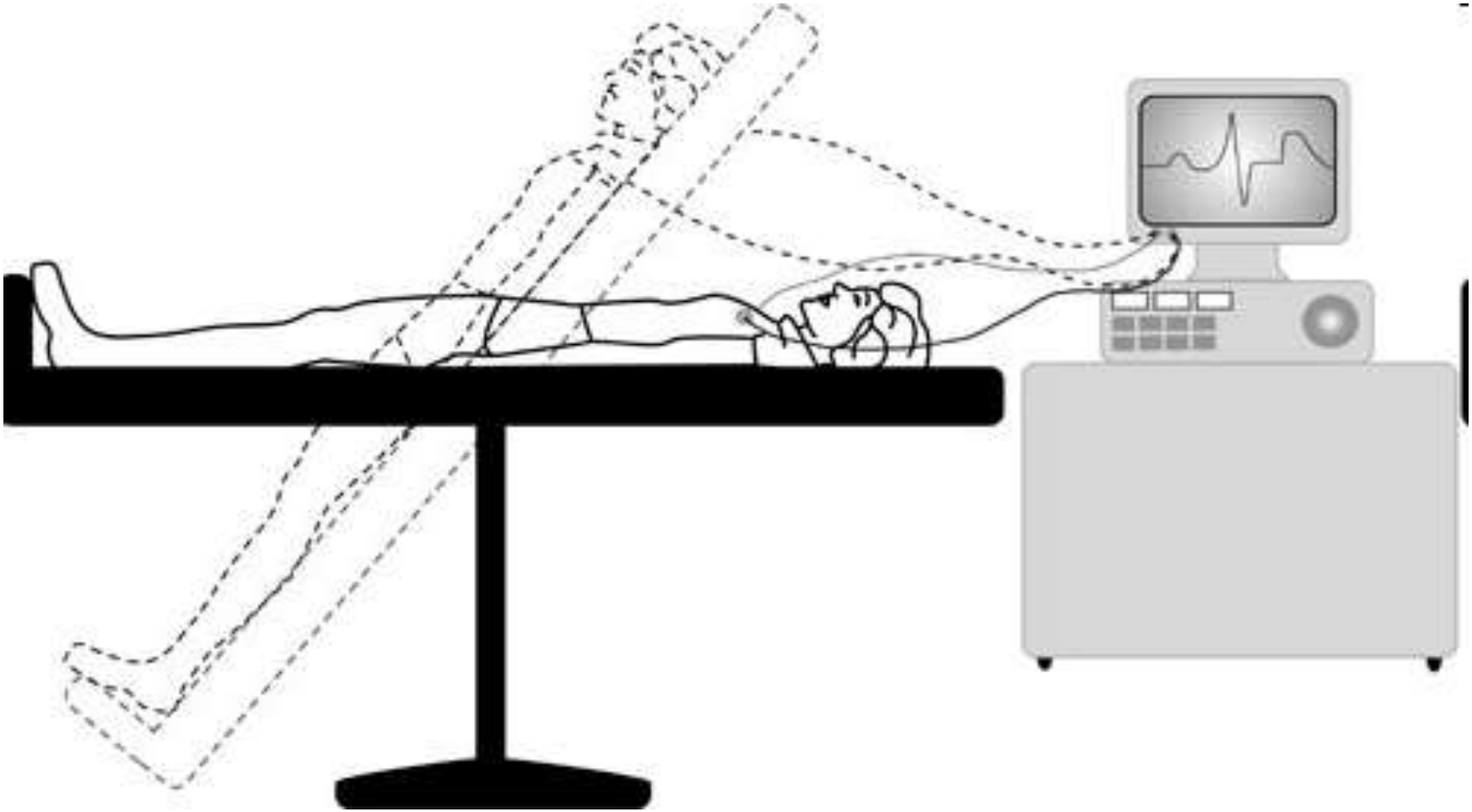
INVESTIGATIONS

- Electrocardiogram : (ECG)
- factors thought to be suggestive of arrhythmia-induced syncope:
- Prolonged intervals (QRS, QTc).
- Severe bradycardia.
- Preexcitation.

INVESTIGATIONS

- Holter monitoring
- Echocardiography
- Neurological:
- CT brain & MRI
- EEG
- Laboratory evaluation :
- FBC & hematocrit
- RBS
- Electrolytes
- Pregnancy test

Tilt table test

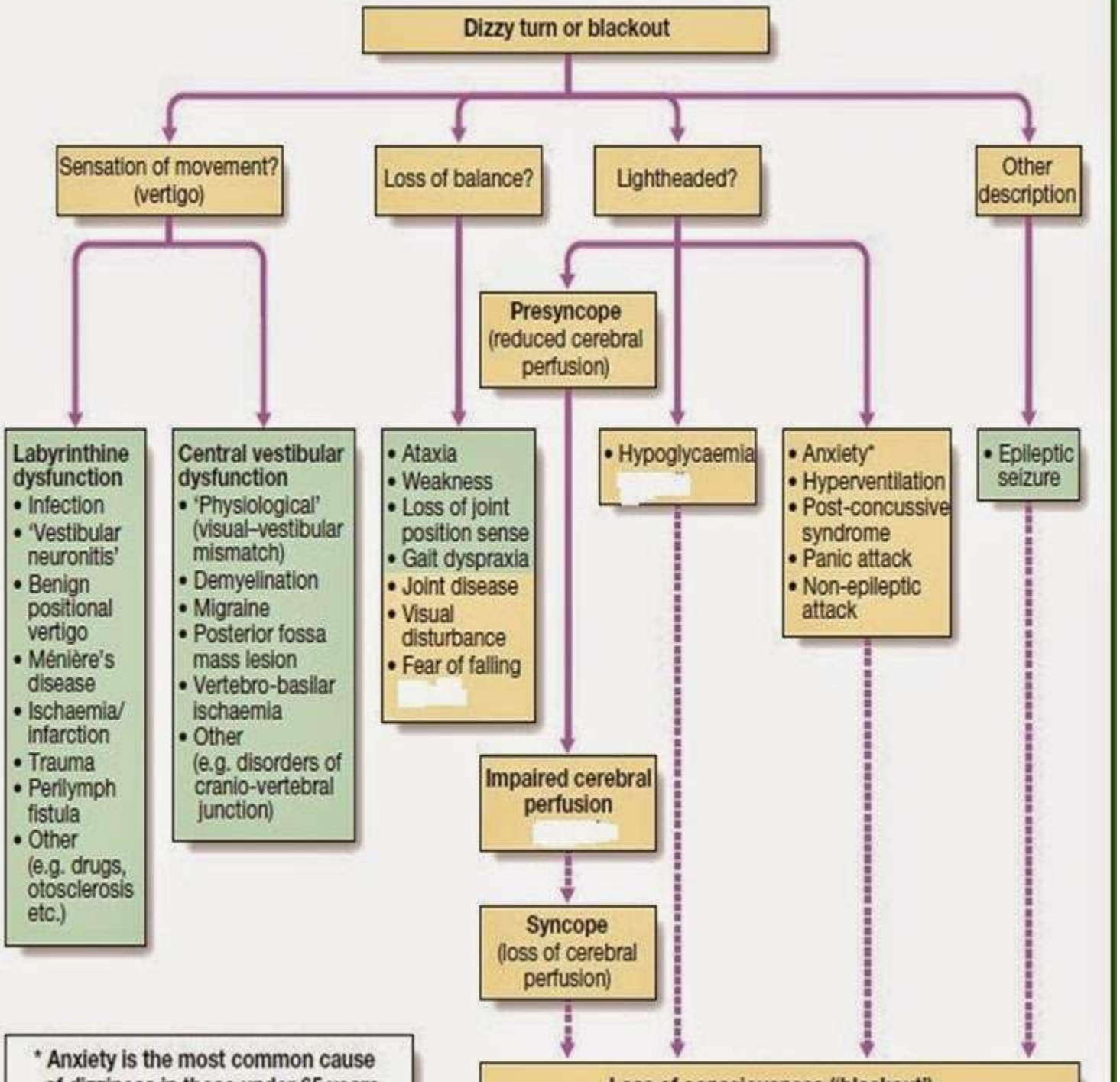


Fainting & Unconsciousness

Treatment

1. When treating the unconscious fainting, the aim is to put back blood to the brain:
   - Check the ABC to find out if the casualty is breathing.
   - Loosen tight clothing.
   - Elevate the person's leg 8 to 12 inches to allow the blood to flow to the brain.
   - Give plenty of fresh air.

The casualty should regain consciousness within 5 minutes


Dizzy turn or blackout
Sensation of movement? (vertigo)
Loss of balance?
Lightheaded?
Other description
Presyncope (reduced cerebral perfusion)
Labyrinthine dysfunction
• Infection
• 'Vestibular neuronitis'
• Benign positional vertigo
• Ménière's disease
• Ischaemia/ infarction
• Trauma
• Perilymph fistula
• Other (e.g. drugs, otosclerosis etc.)
Central vestibular dysfunction
• 'Physiological' (visual–vestibular mismatch)
• Demyelination
• Migraine
• Posterior fossa mass lesion
• Vertebro-basilar ischaemia
• Other (e.g. disorders of cranio-vertebral junction)
• Ataxia
• Weakness
• Loss of joint position sense
• Gait dyspraxia
• Joint disease
• Visual disturbance
• Fear of falling
• Hypoglycaemia
• Anxiety*
• Hyperventilation
• Post-concussive syndrome
• Panic attack
• Non-epileptic attack
• Epileptic seizure
Impaired cerebral perfusion
Syncope (loss of cerebral perfusion)
* Anxiety is the most common cause