Objectives: Upon completion of this educational session, the participant will be able to:

(A) Apply the process of differential diagnosis in the examination and management of brainstem and cerebellar infarctions versus acute vestibular pathology.

(B) Identify the constellation of functional impairments associated with specific brainstem, cerebellar and vestibular syndromes.

(C) Utilize the oculomotor examination to identify lesion locations associated with brainstem, cerebellar and vestibular structures.

(D) Determine the long-term outcomes of the brainstem and cerebellar lesions.

Incidence of vertigo and dizziness to Emergency Department. Using data from the National Hospital Ambulatory Medical Care Survey (NHAMCS), vertigo and dizziness accounts for 2.6 million visits annually to US emergency departments. Of all cases of dizziness, 32.9% were due to oto-vestibular causes. Of all oto-vestibular cases, 5.6% were due to acute vestibular syndrome.

Acute vestibular syndrome (AVS) may be peripheral or central in origin.

(A) Symptoms include rapid onset of:

(1) Severe, continuous vertigo or dizziness
(2) Nausea with retching or vomiting
(3) Gait instability
(4) Head motion intolerance
(5) Nystagmus

(B) Duration of symptoms – days to weeks.

(C) Cause: Most cases of acute vestibular syndrome are of benign cause (vestibular neuritis or nonbacterial labyrinthitis), but approximately 25% are caused by brain stem or cerebellar strokes. Half of stroke patients have no focal neurological signs. Imaging not sensitive acutely.

(1) CT scans identify 16% of posterior fossa infarctions acutely.
(2) MRI with diffusion-weighted imaging of the brain identifies only 80% of posterior fossa infarctions in the first day.

(E) Clinical Examination important in identifying patients with acute central vestibulopathologies.

History:

(A) Medical History – Cardiac Risk Factors. Of patients who sustained a brainstem/cerebellar stroke, 30% 1 cardiac risk factor and the others had at least 2 risk factors. 

(1) Arterial hypertension
(2) Diabetes mellitus
(3) Hyperlipidemia
(4) Cigarette smoking
(5) Atrial fibrillation
(6) Hypercoagulable state
(7) Eclampsia
(8) Recent cervical trauma
(9) Prior myocardial infarction
(10) Prior stroke

(B) History of current symptoms of dizziness/vertigo – rapid onset.

(V) **Brainstem anatomy – The rule of 4 of the brainstem.**¹¹ Determine if medial or lateral brainstem based on medial or lateral structures involved. Determine level of lesion based on cranial nerves involved (figure 1).

(A) The 4 medial structures and the associated deficit:

1. The **Motor pathway (corticospinal tract)**: contralateral weakness of the arm and leg.
2. The **Medial Lemniscus**: contra lateral loss of vibration and proprioception in the arm and leg.
3. The **Medial longitudinal fasciculus**: ipsilateral internuclear ophthalmoplegia (failure of adduction of the ipsilateral eye towards the nose and nystagmus in the opposite eye as it looks laterally).
4. The **Motor nucleus and neve**: ipsilateral loss of the cranial nerve that is affected (III, IV, VI, XII)
(B) The 4 lateral structures and the associated deficit:

1. The Spinocerebellar pathways: ipsilateral ataxia of the arm and leg.
2. The Spinothalamic pathway: contra lateral alteration of pain and temperature affecting the arm, leg and rarely the trunk.
3. The Sensory nucleus of the V: ipsilateral alteration of pain and temperature on the face in the distribution of the Vth cranial nerve (this nucleus is a long vertical structure that extends in the lateral aspect of the pons down into the medulla).

(C) There are 4 cranial nerves in the medulla, 4 in the pons, and 4 above the pons (2 in the midbrain).

(D) There are 4 motor nuclei in the medial brainstem (cranial nerve III, IV, VI, and XII) and 4 nuclei in the lateral brainstem (V, VII, IX, and XI)

(VI) Brainstem vascular syndromes.

(A) The blood supply to the brainstem – vertebral basilar system (Figure 2).

1. Paramedian branches (Figure 3) – occlusion of the paramedian branches results in medial (or paramedian) brainstem syndromes (CST involved - motor)
(2) Long circumferential branches (Figure 3 and 4)— occlusion of the long circumferential branches results in lateral brainstem syndromes (spinothalamic tract – sensation) and cerebellar syndromes (ataxia).
(a) Posterior inferior cerebellar artery
(b) Anterior inferior cerebellar artery
(c) Superior cerebellar artery
(2) Maculae of utricle and sacculus. Weighted sensory epithelium consisting of hair cells embedded within the otolithic membrane with calcium carbonate crystals (otoconia) embedded on top.
(1) detect linear acceleration.
(2) Generate translational VOR

(b) CN VIII – vestibular nerve (Figure 7). Innervates hair cells and project to vestibular nuclear complex.
(1) Two branches of the vestibular nerve.
(a) Superior vestibular nerve – fibers originate from lateral

Figure 6. Schematic illustrating differences in the semicircular canal-ocular and short-latency utriculo-ocular connectivity. Blue lines afferent projections, red lines 2nd order connections, and green lines for abducens neuron projections.

Figure 7 Origin (macula and crista ampullaris) and termination (vestibular nuclear complex) of vestibular nerve projections.

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canal, anterior canal, and utricle.
(b) Inferior vestibular nerve – fibers originate from posterior canal and saccule.

(2) Discharge firing rate of vestibular nerve.
(a) At rest, tonic discharge 90 spikes/s, equal on both sides, signally the brain that the head is not moving.
(b) With movement, asymmetry in discharge firing rate. The canal towards which the head is turning increases and the opposite side is inhibited. Firing rate 0-450 spikes/s depending on side.

(2) Central vestibular system
(a) Vestibular nuclear complex (figure 7) - axon projects through medial longitudinal fasciculus.
(1) Superior vestibular nucleus – projects to oculomotor and trochlear nuclei and integrates vertical VOR.
(2) Medial vestibular nucleus – projects to abducens nuclei, oculomotor nuclei and bilaterally to cervical/thoracic spinal cord to integrate horizontal VOR and vestibular colic reflex.
(3) Lateral vestibular nucleus – projects ipsilaterally to spinal cord to integrate lateral vestibular spinal tract (protective extension).
(4) Descending vestibular nucleus – integrates both sides.
(b) Vestibular commissural system
(c) Cerebellum
(1) Flocculus – adjusts and maintains gain of VOR
(2) Paraflocculus – adjusts gain of smooth pursuit
(3) Nodulus – adjusts duration of VOR. Processes otolithic input.

(C) Extrinsic Muscles of Eye and innervation(Figure 8).
(1) Superior oblique – CN IV
(2) Superior rectus – CN III
(3) Inferior oblique- CN III
(4) Inferior rectus- CN III
(5) Medial rectus – CN III
(6) Lateral rectus – CN VI

(D) Direct Vestibulo-Occular Projections:
(1) Anterior Canal
(a) Excitation: ipsilateral superior rectus and contralateral inferior oblique
(b) Inhibition: ipsilateral inferior rectus and contralateral superior oblique

(2) Posterior Canal
(c) Excitation: ipsilateral superior oblique and contralateral inferior rectus
(d) Inhibition: ipsilateral inferior oblique and contralateral superior rectus

(3) Lateral Canal
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(e) Excitation: ipsilateral medial rectus and contralateral lateral rectus
(f) Inhibition: ipsilateral lateral rectus and contralateral medial rectus

(IX) Lesions to the vestibular system – Mechanisms of Compensation.
(A) Inter-relationship of vestibular neurons.
(B) Signs and symptoms of unilateral vestibular deficit.
   (1) Static – At rest, asymmetry tonic firing rate results in:
      (a) Spontaneous nystagmus- lesion AVOR pathway – fast phase away from side of
          lesion. Peripheral lesion spontaneous ny observed with fixation first 5 days and
          without fixation from 5 days – 8 years. Central lesion spontaneous ny observed
          with/without fixation.
      (b) Decreased tone of extensor muscles side of lesion – lesion graviceptive pathway.
   (2) Dynamic – reduced gain of the central vestibular system. Gain = output/input.
      (a) Oscillopsia – an illusion that everything in the environment is moving – lesion
          VOR pathway.
      (b) Postural responses inadequate on side of lesion due to lesion graviceptive
          pathway.

(X) Plane specific classification of central vestibular syndromes based on
oculomotor, postural and perceptual signs.
(A) Planes of motion pitch, roll, and yaw.
(B) Lesion to graviceptive pathways results in vestibular tone imbalance (asymmetrical
resting firing rate), with the head is at rest, resulting in a syndrome consisting of a
perceptual tilt, head and body tilt, vertical misalignment of the visual axes (skew
deviation) and ocular torsion.
(C) Localizing nystagmus in brainstem disorders.\textsuperscript{12,13}

<table>
<thead>
<tr>
<th>Plane</th>
<th>Direction of Nystagmus (Fast Phase)</th>
<th>Location of Lesion</th>
<th>Duration</th>
<th>Side of Lesion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Roll</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>(Frontal)</td>
<td>Contraversive ocular torsion</td>
<td>Pontomedullary</td>
<td>Transient</td>
<td>Unilateral</td>
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<tr>
<td></td>
<td>Contraversive ocular torsion</td>
<td>Interstitial nucleus of Cajal</td>
<td>Permanent</td>
<td>Unilateral</td>
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<tr>
<td></td>
<td>Ipsiversive ocular torsion</td>
<td>Pontomesencephallic</td>
<td>Transient</td>
<td>Unilateral</td>
</tr>
<tr>
<td></td>
<td>Ipsiversive ocular torsion</td>
<td>Rostral interstitial nucleus of the medial longitudinal fasciculus</td>
<td>Permanent</td>
<td>Unilateral</td>
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<tr>
<td>Pitch</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Sagittal)</td>
<td>Upbeat only</td>
<td>Paramedian pontomesencephalalis (ventral tegmental tract, brachium conjunctivum)</td>
<td>Bilateral</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Downbeat only</td>
<td>Flocculus</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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Upbeat or
downbeat or
transitions  Paramedian pontomedullary  Bilateral

**Yaw (horizontal)**  Contraversive Horizontal  Root entry zone of the vestibular nerve, medial and superior vestibular nuclei, and paramedian pontine reticular formation  Unilateral

**Combined syndromes (roll and yaw)**  Torsion and horizontal  pontomedullary syndromes, vestibular neuritis  Unilateral

(D) Signs of plane specific vestibular syndromes.\(^\text{12}\)

| Table 2. Signs of Plane Specific Vestibular Syndromes.\(^\text{12}\) |
|---------------------------------|-----------------|----------------|----------------|----------------|-------------------|
| Plane                                | Vertical  |
|   Misalignment of Visual   | Ocular   |
| Axes (Skew Deviation)          | Head Tilt | Body Tilt | Falls | Perceptual Tilt | Past Pointing |
| Roll                              | x       | x       | x   | Vertical |
| Pitch                             | Forward/ backward | Forward/ backward | Forward/ backward | Horizontal |
| Yaw                               | Rotation/ lateral | Straight ahead | x |

(E) Graviceptive pathway for roll.\(^\text{12}\) Pathway crosses at level of pons. Subjective visual vertical is most sensitive sign in roll plane

| Table 3. Graviceptive pathway for roll.\(^\text{12}\) |
|---------------------------------|-----------------|-----------------|------------------|
| Location of Lesion                     | Arterial Supply | Ocular Tilt Reaction | Subjective Visual Vertical |
|                                     |                 | Ipsiversive | Contraversive | Ipsiversive | Contraversive |
| Peripheral (superior vestibular nerve) | Vertebral | x | x |
| Pontomedullary Junction (medial and superior vestibular nuclei) | Vertebral | x | x |
| Pontomesencephalic from Basilar | Paramedian from Basilar | x | x |
| Thalamus                           | Paramedian Thalamic | None | None | x | x |
| Vestibular Cortex                  | Thalamogeniculate, temporal branches of CMA, deep perforators | None | None | x |
(F) Frequency subjective visual vertical (SVV) tilt, skew deviation, ocular torsion, and ocular tilt reaction (OTR) in acute unilateral brainstem and thalamic infarctions.³

Table 3. Frequency subjective visual vertical, skew deviation ocular torsion, and ocular tilt reaction in acute unilateral brainstem and thalamic infarctions.³

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Patients (no.)</th>
<th>SVV Tilt (%)</th>
<th>Monocular</th>
<th>Binocular</th>
<th>Skew (%)</th>
<th>OTR (%)</th>
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<tbody>
<tr>
<td>Brainstem</td>
<td></td>
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<tr>
<td>Medullary (Wallenberg’s syndrome)</td>
<td>36</td>
<td>94</td>
<td>27</td>
<td>55</td>
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<td>33</td>
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<td>Pontomedullary</td>
<td>13</td>
<td>100</td>
<td>60</td>
<td>20</td>
<td>23</td>
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<td>Pontine</td>
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<td>91</td>
<td>47</td>
<td>33</td>
<td>26.5</td>
<td>12</td>
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<tr>
<td>Pontomesencephalic</td>
<td>12</td>
<td>92</td>
<td>64</td>
<td>18</td>
<td>25</td>
<td>25</td>
</tr>
<tr>
<td>Mesencephalic</td>
<td>16</td>
<td>94</td>
<td>54</td>
<td>38</td>
<td>37.5</td>
<td>25</td>
</tr>
<tr>
<td>Mesodiencephalic</td>
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<tr>
<td>Anterior polar thalamic</td>
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<td>13+</td>
<td>20+</td>
<td>0</td>
<td>0</td>
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<tr>
<td>Paramedian thalamic</td>
<td>14</td>
<td>64</td>
<td>29</td>
<td>43</td>
<td>57</td>
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<tr>
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<td>94</td>
<td>47</td>
<td>36</td>
<td>31</td>
<td>20</td>
</tr>
</tbody>
</table>

(G) HINTS to Diagnose Stroke in the Acute Vestibular Syndrome – Three step bedside examination.¹⁰

1. Examination.
   (a) Head Impulse Test
   (b) Direction-changing nystagmus in eccentric gaze
   (c) Skew deviation (vertical ocular misalignment)

(XI) Clinical Examination:

(A) Neurological screen to identify focal neurological signs.
   (1) Motor screen
   (2) Sensory screen
   (3) Cranial nerves (other than III, IV, and VI)
   (4) Oculomotor examination
      (a) Fixation/alignment
      (b) Extra-ocular movements
      (c) Saccades
      (d) Pursuit
      (e) Gaze holding
      (f) VOR
      (g) Vergence

(B) Disorders of Fixation (Spontaneous nystagmus)
   (1) Downbeating nystagmus:

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a. Most common form of acquired spontaneous nystagmus.
b. Associated with lesions in the pontomedullary tegmentum and flocculus/araflocculus of the cerebellum.
c. Possibly associated with brainstem connects from Anterior Semicircular canal.

(2) Upbeating nystagmus.
   a. Caudal Medulla
   b. Ventral tegmental tract

(3) Torsional nystagmus

(C) Disorders of Alignment: Skew deviation (vertical ocular misalignment).
   (1) Symptom:
      a. Vertical diplopia
      b. Subjective visual vertical
   (2) Differential diagnosis
      a. Graviceptive pathway
      b. CN IV lesion
   (3) Tests: Monocular v binocular
      a. Bucket Test
         i. Ipsiversive v contraversive tilt
      b. Maddox Rods
      c. Upright v Supine Position Test

(D) Disorders of gaze holding
   (1) Peripheral Vestibular Dysfunction
      a. Non-direction changing due to vestibular tone imbalance
      b. Alexanders Law
   (2) Central Vestibular Dysfunction
      a. Direction changing
      b. Neural Integrators
         i. Horizontal
         ii. Vertical

(E) Disorders of the VOR (Head Impulse Test)
   (1) Test of peripheral vestibular function
   (2) Positive in isolated medial vestibular nucleus lesions

(3) References

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