Neurologic History and Examination

Ivo Bekavac, MD, PhD
History

• The same as any history
• Age
• Chief complaint
• History of present illness
• Past medical history
• Social history
• Family history
• Review of systems
General physical examination

• Vital signs (BP, HR, RR, temp)
• Skin
• HEENT (Head, Eyes, Ears, Nose & Throat)
• Check for CAROTID and OCULAR BRUITS
• Chest and cardiovascular (Heart murmur, rhythm)
• Abdomen (bruit, organomegaly)
• Extremities (atrophy, cold, pulses)
• Rectal
The detailed neurological examination in adults
### Organization of the Neurologic Examination

#### A. Mental Status
1. Level of alertness
2. Language
   - Fluency
   - Comprehension
   - Repetition
   - Naming
3. Memory
   - Immediate
   - Short-term
   - Long-term
   - Recent (including orientation to place and time)
   - Remote
4. Calculation
5. Construction
6. Abstraction

#### B. Cranial Nerves
1. Olfaction (CN I)
2. Vision (CN II)
   - Visual fields
   - Visual acuity
   - Funduscopic examination
3. Pupillary light reflex (CNs II, III)
4. Eye movements (CNs III, IV, VI)
5. Facial sensation (CN V)
6. Facial strength
   - Muscles of mastication (CN V)
   - Muscles of facial expression (CN VII)
7. Hearing and vestibular function (CN VIII)
8. Palatal movement (CN IX, X)
9. Dysarthria (CNs IX, X, XII)
10. Head rotation (CN XI)
11. Shoulder elevation (CN XI)
12. Tongue movements (CN XII)

#### C. Motor
1. Gait
2. Coordination
3. Involuntary movements
4. Pronator drift
5. Individual muscles
   - Strength
   - Bulk
   - Tone (resistance to passive manipulation)

#### D. Reflexes
1. Tendon reflexes
2. Plantar responses
3. Superficial reflexes
4. "Primitive" reflexes

#### E. Sensory
1. Light touch
2. Pain/temperature
3. Joint position sense
4. Vibration
5. Double simultaneous stimulation
6. Graphesthesia
7. Stereognosis

A SCREENING NEUROLOGIC EXAMINATION

For screening purposes in patients without neurologic complaints

• MENTAL STATUS
• CRANIAL NERVES
• MOTOR SYSTEM
• REFLEXES
• SENSATION
MENTAL STATUS EXAMINATION

• Level of consciousness (arousal)
• Attention and concentration
• Memory (immediate, recent and remote)
• Language
• Visual spatial perception
• Executive functioning
• Mood and thought content
• Praxis
• Calculation
Arousal

While taking the history and examining the patient observe:

• Alert
• Attentive
• Sleepy
• Unresponsive
Attention and concentration

Attention – the ability to focus and direct cognitive processes and to resist distraction

Concentration – the ability to focus and sustain attention over a period of time.
Impaired memory is a common complaint which may be presented by patients and/or family members.

MULTIPLE DIMENSIONS:
• Immediate and working
• Recent
• Remote
Language

- Fluency
- Content (paraphasic errors and neologisms)
- Repetition (“no ifs, ands or buts”)
- Naming
- Comprehension
- Reading
- Writing
Visual spatial perception

- History: losing objects, getting lost, difficulty navigating
- Perceptual and constructional abilities (copying, drawing)
- Clock drawing
Praxis

- Praxis (ideomotor) – performance of learned motor movement in the absence of primary deficit in motor and spatial abilities.
- Difficulty dressing, feeding and bathing.
- Ideational praxis – “take this piece of paper, fold it in half, and place it in the envelope”
- Relatively common feature of corticobasal degeneration
Calculations

• “serial seven”
• Sensitive to impaired attention and performance (test), anxiety and to the patient’s educational level
• Acalculia
• Left angular gyrus lesion (Gerstmann syndrome), dementia
Executive functioning

• Complex set of capacities including planning, purposive action and effective performance that enable a person to engage successfully in appropriate, goal-directed, socially responsible, and self-serving behavior.

• Prefrontal cortex and their connections through the caudate nuclei

• Impairments in insight and judgment are early indicators of executive dysfunction

• Evaluation is difficult, neuropsychological testing
Mood and thought content

- Mood and emotional state have a strong impact on mental status and cognitive functioning.
- Depression
- Apathy
- Pseudobulbar palsy
Others

• Right-left orientation, finger agnosia (Gerstmann syndrome – in addition to dysgraphia, acalculia) – posterior left hemisphere-angular gyrus

BRIEF COGNITIVE ASSESSMENT

• Mini-mental status examination (MMSE) – 0-30
• Clock drawing
CRANIAL NERVE EXAMINATION
Olfaction (CN I)

• Almost never tested
• If no history of olfactory problem, no need to test it.
• Test: occlude one nostril (coffee, peppermint, cinnamon)
Vision (CN II)

• Visual fields
• Acuity
• Fundoscopic examination
Visual fields

- Confrontation
- Humphrey visual field
## Anatomy of the visual pathways and visual field correlation (view of underside of brain)

<table>
<thead>
<tr>
<th>Location</th>
<th>Field Defect</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Left Optic Nerve</td>
<td></td>
<td>No light perception left eye</td>
</tr>
<tr>
<td>2 Chiasm</td>
<td></td>
<td>Bitemporal hemianopsia</td>
</tr>
<tr>
<td>3 Right Optic Tract</td>
<td></td>
<td>Incongruous left homonymous hemianopsia</td>
</tr>
<tr>
<td>4 Left Lateral Geniculate Nucleus</td>
<td></td>
<td>Right homonymous (lateral choroidal artery) or incongruous right homonymous hemianopsia</td>
</tr>
<tr>
<td>5 Left Temporal Lobe</td>
<td></td>
<td>Right homonymous upper quadrant defect (&quot;pie in the sky&quot;)</td>
</tr>
<tr>
<td>6 Left Parietal Lobe</td>
<td></td>
<td>Right homonymous defect, denser inferiorly</td>
</tr>
<tr>
<td>7 Left Occipital Lobe (upper bank)</td>
<td></td>
<td>Right homonymous lower quadrantanopsia (macular sparing)</td>
</tr>
<tr>
<td>8 Left Occipital Lobe (lower bank)</td>
<td></td>
<td>Right homonymous upper quadrantanopsia (macular sparing)</td>
</tr>
<tr>
<td>9 Right Occipital Lobe</td>
<td></td>
<td>Left homonymous hemianopsia</td>
</tr>
</tbody>
</table>

The patient presented acutely with painless loss of vision in the left eye. Visual acuity was 20/20 OD, 1/200 OS, and there was a dense left relative afferent pupillary defect. The fundus shows retinal whitening (edema of the nerve fiber layer) in the macular region (arrow), with a "cherry-red" spot in the fovea, the classic appearance of a central retinal artery occlusion.
Visual fields: Complete Homonymous Hemianopia

Humphrey visual field showing a left complete homonymous hemianopia.

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Right occipital infarction

Axial FLAIR MRI images showing a right occipital infarction.
Visual fields: Congruous incomplete homonymous hemianopia

Humphrey visual field (24-2) showing a right congruous incomplete homonymous hemianopia.
Visual fields: Incongruous incomplete homonymous hemianopia

Humphrey visual field showing a right incongruous incomplete homonymous hemianopia secondary to a left optic tract tumor.

Bilateral homonymous hemianopias

Humphrey visual field (24-2) showing bilateral homonymous hemianopias; there is a complete right homonymous hemianopia and an incomplete congruous left homonymous hemianopia. This patient had bilateral occipital infarctions.
<table>
<thead>
<tr>
<th>Etiology</th>
<th>Typical duration</th>
<th>Pattern of visual loss</th>
<th>Associated symptoms and signs</th>
<th>Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Monocular ischemia, carotid disease, other</td>
<td>1 to 10 minutes</td>
<td>Monocular, rapid onset, altitudinal onset</td>
<td>Hollenhorst plaque, hemispheric symptoms</td>
<td>Retinal embolism (usually)</td>
</tr>
<tr>
<td>embolic source</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Giant Cell arteritis</td>
<td>Variable</td>
<td>Usually monocular</td>
<td>Headache, neck pain, jaw claudication</td>
<td>Ischemia of optic nerve</td>
</tr>
<tr>
<td>Papilledema</td>
<td>Seconds</td>
<td>Monocular graying or blurring</td>
<td>Headache, diplopia</td>
<td>Elevated intracranial pressure</td>
</tr>
<tr>
<td>Idiopathic retinal vasospasm</td>
<td>5 to 60 minutes</td>
<td>Monocular positive or negative symptoms</td>
<td>Transient retinal arterial narrowing, headache</td>
<td>Vasospasm</td>
</tr>
<tr>
<td>Migraine</td>
<td>10 to 30 minutes</td>
<td>Usually binocular, positive symptoms with spread</td>
<td>Usually followed by migraine headache</td>
<td>Spreading cortical depression, possibly retinal vasospasm</td>
</tr>
<tr>
<td>Vertebral arterial ischemia</td>
<td>1 to 10 minutes</td>
<td>Homonymous hemianopia</td>
<td>Isolated or accompanied by other brainstem deficits</td>
<td>Embolic</td>
</tr>
<tr>
<td>Seizure: Ictal</td>
<td>3 to 5 minutes</td>
<td>Binocular, lateralized, positive phenomenon common</td>
<td>Altered consciousness, motor symptoms</td>
<td>Epileptic discharge</td>
</tr>
<tr>
<td>Postictal</td>
<td>20 minutes, longer</td>
<td>Binocular visual field loss</td>
<td>Preceding ictus</td>
<td>Cortical inhibition</td>
</tr>
</tbody>
</table>
Acuity

- Hand-held visual acuity card - 14 inches in front of the patient’s eye
- 20/20 normal
Fundoscopic examination

• Right eye examine with your right eye
• Reduce the room illumination
• Ask the patient to look straight ahead
• Optic disc (clarity of the disc outline, venous pulsations, retinal vessels)
Normal fundus appearance
Ischemic optic neuropathy in a patient with giant cell arteritis who lost vision abruptly four days prior to this examination. The optic disc is swollen and its margins are blurred.

Courtesy of Gene Hunder, MD.
Central retinal artery occlusion with macular sparing

Fundus photograph of a central retinal artery occlusion with sparing of the macular region due to the presence of a cilioretinal artery.
Central retinal vein occlusion

Intraretinal hemorrhages in all four quadrants on funduscopic examination.
Pupillary light reflex (CN II and III)

• Shine a penlight on the bridge of the patient’s nose
• Swinging flashlight
• Afferent pupillary defect
## Functions of the vertical and oblique extraocular muscles

<table>
<thead>
<tr>
<th>Functions</th>
<th>Superior rectus</th>
<th>Inferior rectus</th>
<th>Superior oblique</th>
<th>Inferior oblique</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary</td>
<td>Elevation</td>
<td>Depression</td>
<td>Incyclotorsion</td>
<td>Excyclotorsion</td>
</tr>
<tr>
<td>Secondary</td>
<td>Incyclotorsion</td>
<td>Excyclotorsion</td>
<td>Depression</td>
<td>Elevation</td>
</tr>
<tr>
<td>Tertiary</td>
<td>Adduction</td>
<td>Adduction</td>
<td>Abduction</td>
<td>Abduction</td>
</tr>
</tbody>
</table>
Eye movements (CN II, IV and VI)
Normal eyelid position

The upper lid covers 1 to 2 mm of the upper limbus. The lower lid covers the lower limbus minimally. The central light reflex can be seen within the pupil. The margin reflex distance is measured from this reflex to the upper eyelid margin.

Courtesy of Michael S Lee, MD.
Parasympathetic innervation and the pupillary reflex pathway

Sympathetic pathway for pupillary innervation

Müller's muscle
Sudomotor and vasoconstrictor fibers to forehead
Hypothalamus
Ophthalmic artery
Pons
Medulla
Long ciliary nerve
Nasociliary nerve
Carotid plexus
Sudomotor and vasoconstrictor fibers to face
Superior cervical ganglion
Internal carotid artery
External carotid artery
Preganglionic neuron
Inferior cervical ganglion
Subclavian artery
Lung
C1, C2
C8, T1, T2
Central neuron
Postganglionic neuron

* Trigeminal ganglion.

Flowchart explaining the approach to a patient with anisocoria

Patient with anisocoria

Relevant history and examination with specific attention to:
- History of ocular trauma
- Check old photographs (ptosis, ocular deviation, long standing anisocoria)
- Use of topical medications
- Exposure to toxins and drugs
- Associated ocular and neurological symptoms/signs

Which pupil is abnormal?
Examine pupils in light and dark

Anisocoria more in dark (small pupil abnormal)
- Dilation lag
- Ptosis
- Test with 0.3 to 1 percent Apraclonidine
  - Small pupil dilates (anisocoria reversal)
  - Horner syndrome

Anisocoria equal in light and dark
- Brisk reaction to light

Anisocoria more in light (large pupil is abnormal)
- Isolated
- Sluggish to light
- Light near dissociation
- Use 0.1 percent Pilocarpine
  - Third nerve palsy
- Ptosis/ophthalmoplegia

Large pupil constricts
- Adie’s tonic pupil
- Physiologic anisocoria

Large pupil does not constrict
- Minimal/no constriction
- Pharmacologic anisocoria
A) Normal pupillary reactions. Both pupils are symmetric in the light and dark. B) The small pupil is abnormal. The right pupil does not dilate well in the dark. C) The large pupil is abnormal. The right pupil does not react well to light. D) Physiologic anisocoria. The amount of anisocoria is the same in light and dark.
During horizontal saccades, the paramedian pontine reticular formation (PPRF) burst cells innervate the abducens nucleus, which contains two distinctive sets of neurons. Axons from the abducens motor neurons innervate the ipsilateral lateral rectus muscle, while the axons of the abducens internuclear cross the midline to become the medial longitudinal fasciculus (MLF) and subsequently innervate the medial rectus subnucleus of the oculomotor complex (cranial nerve nucleus III).

Internuclear ophthalmoplegia (INO) is characterized by slowing and/or limitation of the adducting eye during horizontal saccades and is the result of damage to the MLF within the dorsomedial pontine or midbrain tegmentum, adjacent to the fourth ventricle and cerebral aqueduct, respectively.

Internuclear ophthalmoplegia (INO)

This patient exhibits a left INO characterized by adduction slowing without ocular limitation. The top picture (A) depicts the patient in a primary gaze position, while the bottom picture (B) depicts the point at which the right eye reaches a 20 degree target while saccading to the right. Note that the right eye reaches the target prior to the left eye, as demonstrated with the midpupillary markers shown. (Note that the left eye has not changed position off the midpupillary marker). At saccade end, both eyes achieve target fixation (as shown in the oculogram).

### Possible etiologies of internuclear ophthalmoplegia

- Multiple sclerosis (commonly bilateral)
- Brainstem infarction (commonly unilateral)
- Brainstem and fourth ventricle tumors
- Arnold-Chiari malformation
- Infection: bacterial, viral, and other forms of meningioencephalitis
- Hydrocephalus, subdural hematoma, supratentorial arteriovenous malformation
- Nutritional disorders: Wernicke's encephalopathy and pernicious anemia
- Metabolic disorders: hepatic encephalopathy, maple syrup urine disease, abetalipoproteinemia, Fabry's disease
- Drug intoxications: tricyclic antidepressants, phenothiazines, narcotics, lithium, barbiturates, propranolol
- Cancer
- Head trauma
- Degenerative conditions: progressive supranuclear palsy
- Syphilis
- Pseudointernuclear ophthalmoplegia of myasthenia gravis and Fisher's syndrome
<table>
<thead>
<tr>
<th>Cause</th>
<th>Levator function</th>
<th>Eyelid crease margin</th>
<th>Other clinical features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congenital abnormality of the levator muscle*</td>
<td>Reduced</td>
<td>Crease often absent</td>
<td>Often unilateral. Many patients also have amblyopia, strabismus.</td>
</tr>
<tr>
<td>Aponeurotic ptosis</td>
<td>Normal</td>
<td>Often increased</td>
<td>Uni- or bilateral. Isolated finding of ptosis.</td>
</tr>
<tr>
<td>Cranial nerve 3 palsy</td>
<td>Reduced</td>
<td>Normal</td>
<td>Impaired extraocular movement in ipsilateral eye. If ipsilateral pupil dilated, urgent evaluation for aneurysm is required.</td>
</tr>
<tr>
<td>Horner’s syndrome</td>
<td>Normal</td>
<td>Normal</td>
<td>Ipsilateral miotic pupil.</td>
</tr>
<tr>
<td>Myasthenia</td>
<td>Reduced</td>
<td>Normal</td>
<td>Uni- or bilateral. Variable and fatigable. Diplopia and extraocular movement abnormalities often present.</td>
</tr>
<tr>
<td>Muscle disease</td>
<td>Reduced</td>
<td>Normal</td>
<td>Orbicularis oculi, other extraocular or bulbar muscles may be affected</td>
</tr>
</tbody>
</table>

* Other, less common causes of congenital ptosis are discussed in text.
Patient with ptosis of the right upper lid secondary to levator dehiscence

Note the absence of the right upper lid crease. The patient is raising the right eyebrow to try and compensate for the ptosis. The left lid shows pseudoretraction because of Hering's law of equal innervation.

Courtesy of Michael S Lee, MD.
Patient with neurofibromatosis 1 and mechanical ptosis secondary to a plexiform neurofibroma on the right upper lid.

Patient with a mechanical ptosis of the right upper lid secondary to a plasmacytoma.

Courtesy of Michael S Lee, MD.
Partial third nerve palsy

This patient has ptosis of the right upper eyelid. Note that the right eye does not elevate well, indicating a superior division third nerve palsy.

Courtesy of Michael S Lee, MD.
Horner's syndrome

In dim light, the anisocoria is accentuated with the right pupil more miotic. The right upper lid is ptotic by 1.5 mm.

*Courtesy of Michael S Lee, MD.*
Chronic progressive external ophthalmoplegia

This patient has profound ptosis and ophthalmoplegia in all directions of gaze.

Courtesy of Michael S Lee, MD.
Nystagmus

Ocular oscillations (can occur in normal subjects, drug induced, vestibular or cerebellar dysfunction)

Jerk nystagmus – repetitive, quick movement of the eyes in the opposite direction, several times in a raw. Slow and fast phase.

Pendular nystagmus – slow sinusoidal oscillation of the eyes that may occur in any direction, without fast phases.
## Classification of jerk nystagmus

<table>
<thead>
<tr>
<th>Primary position</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Pure directions</td>
<td></td>
</tr>
<tr>
<td>Upbeat</td>
<td></td>
</tr>
<tr>
<td>Downbeat</td>
<td></td>
</tr>
<tr>
<td>Torsional</td>
<td></td>
</tr>
<tr>
<td>Horizontal</td>
<td></td>
</tr>
<tr>
<td>Peripheral vestibular</td>
<td></td>
</tr>
<tr>
<td>Congenital</td>
<td></td>
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<tr>
<td>Periodic alternating</td>
<td></td>
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<tr>
<td>Latent</td>
<td></td>
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<tr>
<td>Epileptic</td>
<td></td>
</tr>
<tr>
<td>Mixed directions (peripheral vestibular)</td>
<td></td>
</tr>
<tr>
<td>Horizontal/torsional</td>
<td></td>
</tr>
<tr>
<td>Vertical/torsional</td>
<td></td>
</tr>
</tbody>
</table>

### Gaze-evoked

- Gaze holding
- Rebound
- Peripheral vestibular
- Brun’s

### Positional

- Paroxysmal positional
- Posterior canalithiasis
- Anterior canalithiasis
- Horizontal canalithiasis
- Static positional

### Dissociated

- Internuclear ophthalmoplegia

### Other

- Convergence-retraction nystagmus
Facial sensation (CN V)

- V₁ – ophthalmic
- V₂ – maxillary
- V₃ – mandibular
- Corneal reflex (V₁ & CN VII)
The sensory distribution of the trigeminal nerve (cranial nerve V) and its three divisions (V1, V2, V3) is shown along with branches of the cervical spinal nerves that innervate cutaneous regions of the head and neck.
Facial strength

• Muscles of mastication (CN V)
• Muscles of facial expression (CN VII)
• Peripheral lesion – complete facial weakness
• Central lesion – forehead muscles usually spared
Hearing (CN VIII)

Classification of hearing loss:

• **Sensorineural** (inner ear, cochlea or auditory nerve)
• **Conductive** (any cause that limits the amount of external sound from gaining access to the inner ear – cerumen impaction, middle ear fluid, ossicular chain fixation)
• **Mixed loss** (combination of the above)
<table>
<thead>
<tr>
<th>Causes of hearing loss</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Conductive</strong></td>
</tr>
<tr>
<td>Outer-ear causes</td>
</tr>
<tr>
<td>Congenital microtia or atresia</td>
</tr>
<tr>
<td>External otitis</td>
</tr>
<tr>
<td>Trauma</td>
</tr>
<tr>
<td>Squamous cell carcinoma</td>
</tr>
<tr>
<td>Exostosis</td>
</tr>
<tr>
<td>Osteoma</td>
</tr>
<tr>
<td>Psoriasis</td>
</tr>
<tr>
<td>Cerumen</td>
</tr>
<tr>
<td>Middle-ear causes</td>
</tr>
<tr>
<td>Congenital atresia or ossicular chain malformation</td>
</tr>
<tr>
<td>Otis media</td>
</tr>
<tr>
<td>Cholesteatoma</td>
</tr>
<tr>
<td>Otosclerosis</td>
</tr>
<tr>
<td>Tympanic membrane perforation</td>
</tr>
<tr>
<td>Temporal bone trauma</td>
</tr>
<tr>
<td>Glomus tumors</td>
</tr>
<tr>
<td>Sensorineural</td>
</tr>
<tr>
<td>Inner-ear causes</td>
</tr>
<tr>
<td>Hereditary hearing loss</td>
</tr>
<tr>
<td>Congenital viral infections</td>
</tr>
<tr>
<td>Congenital malformations</td>
</tr>
<tr>
<td>Presbycusis</td>
</tr>
<tr>
<td>Meningitis</td>
</tr>
<tr>
<td>Thyrotoxicosis</td>
</tr>
<tr>
<td>Viral coxchleitis</td>
</tr>
<tr>
<td>Ototoxic drugs</td>
</tr>
<tr>
<td>Otopologic surgery</td>
</tr>
<tr>
<td>Meniere disease</td>
</tr>
<tr>
<td>Noise exposure</td>
</tr>
<tr>
<td>Barotrauma</td>
</tr>
<tr>
<td>Penetrating trauma</td>
</tr>
<tr>
<td>Acoustic neuroma</td>
</tr>
<tr>
<td>Meningioma</td>
</tr>
<tr>
<td>Autoimmune disease</td>
</tr>
<tr>
<td>Multiple sclerosis</td>
</tr>
<tr>
<td>Cerebrovascular ischemia</td>
</tr>
</tbody>
</table>
**Webber test**: Place the base of a struck tuning fork on the bridge of the forehead, nose, or teeth. In a normal test there is no lateralization of sound. With unilateral conductive loss, sound lateralizes towards affected ear. With unilateral sensorineural loss, sound lateralizes to the normal or better-hearing side.

**Rinne test**: Place the base of a struck tuning fork on the mastoid bone behind the ear. Have the patient indicate when sound is no longer heard. Move fork (held at base) beside ear and ask if now audible. In a normal test, AC > BC; patient can hear fork at ear. With conductive loss, BC > AC; patient will not hear fork at ear.

**AC**: air conduction; **BC**: bone conduction.
Interpreting Weber and Rinne tests: Conductive versus sensorineural hearing loss

<table>
<thead>
<tr>
<th></th>
<th>Weber lateralizes</th>
<th>Rinne test</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Conductive loss</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Good ear</td>
<td></td>
<td>AC &gt; BC</td>
</tr>
<tr>
<td>Bad ear</td>
<td>To bad ear</td>
<td>BC &gt; AC</td>
</tr>
<tr>
<td><strong>Sensorineural loss</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Good ear</td>
<td>To good ear</td>
<td>AC &gt; BC</td>
</tr>
<tr>
<td>Bad ear</td>
<td></td>
<td>AC &gt; BC</td>
</tr>
</tbody>
</table>

AC > BC: air conduction better than bone conduction (normal Rinne).
BC > AC: bone conduction better than air conduction (abnormal Rinne).
Vestibular function (CN VIII)

- Gait/balance observation
- Nystagmus (spontaneous and/or elicited)
Vertigo

• Symptom of illusory movement.
• It is a SYMPTOM, not a DIAGNOSIS.
## Causes of vertigo

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<th>Peripheral causes</th>
</tr>
</thead>
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<td>Benign paroxysmal positional vertigo</td>
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<tr>
<td>Vestibular neuritis</td>
</tr>
<tr>
<td>Herpes zoster oticus (Ramsay Hunt syndrome)</td>
</tr>
<tr>
<td>Meniere disease</td>
</tr>
<tr>
<td>Labyrinthine concussion</td>
</tr>
<tr>
<td>Perilymphatic fistula</td>
</tr>
<tr>
<td>Semicircular canal dehiscence syndrome</td>
</tr>
<tr>
<td>Cogan’s syndrome</td>
</tr>
<tr>
<td>Recurrent vestibulopathy</td>
</tr>
<tr>
<td>Acoustic neuroma</td>
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<tr>
<td>Aminoglycoside toxicity</td>
</tr>
<tr>
<td>Otitis media</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Central causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Migrainous vertigo</td>
</tr>
<tr>
<td>Brainstem ischemia</td>
</tr>
<tr>
<td>Cerebellar infarction and hemorrhage</td>
</tr>
<tr>
<td>Chiari malformation</td>
</tr>
<tr>
<td>Multiple sclerosis</td>
</tr>
<tr>
<td>Episodic ataxia type 2</td>
</tr>
<tr>
<td>Clinical features of common causes of vertigo*</td>
</tr>
<tr>
<td>---------------------------------------------</td>
</tr>
<tr>
<td>Benign paroxysmal positional vertigo</td>
</tr>
<tr>
<td>Vestibular neuritis</td>
</tr>
<tr>
<td>Meniere disease</td>
</tr>
<tr>
<td>Migrainous vertigo</td>
</tr>
<tr>
<td>Vertebrobasilar TIA</td>
</tr>
<tr>
<td>Brainstem infarction</td>
</tr>
<tr>
<td>Cerebellar infarctions or hemorrhage</td>
</tr>
</tbody>
</table>

* Other diagnoses described in text: "Pathophysiology and differential diagnosis of vertigo".
* Peripheral characteristics of nystagmus: horizontal or horizontal-torsional: suppresses with visual fixation, does not change direction with gaze. Central characteristics of nystagmus: may be horizontal, torsional, or vertical, does not suppress with visual fixation, may change direction with gaze.
<table>
<thead>
<tr>
<th>Clinical features of central versus peripheral vertigo</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<tr>
<td><strong>Nystagmus</strong></td>
</tr>
<tr>
<td>Direction</td>
</tr>
<tr>
<td>Type</td>
</tr>
<tr>
<td>Effect of visual fixation</td>
</tr>
<tr>
<td>Other neurologic signs</td>
</tr>
<tr>
<td>Postural instability</td>
</tr>
<tr>
<td>Deafness or tinnitus</td>
</tr>
</tbody>
</table>
Dix Hallpike maneuver

With the patient sitting, the neck is extended and turned to one side. The patient is then placed supine rapidly, so that the head hangs over the edge of the bed. The patient is kept in this position and observed for nystagmus for 30 seconds. In patients with benign paroxysmal positional vertigo, nystagmus usually appears with a latency of a few seconds and lasts less than 30 seconds. It has a typical trajectory, beating upward and torsionally, with the upper poles of the eyes beating toward the ground. After it stops and the patient sits up, the nystagmus will recur but in the opposite direction. Therefore, the patient is returned to upright and again observed for nystagmus for 30 seconds. If nystagmus is not provoked, the maneuver is repeated with the head turned to the other side. If nystagmus is provoked, the patient should have the maneuver repeated to the same (provoked) side; with each repetition, the intensity and duration of nystagmus will diminish.
Head thrust test

At rest, the patient is asked to fixate on a distant target. The patient's head is rotated rapidly by the examiner, first to the left (A to B), then to the right (C to D). In a normal response, the eyes remain on target (B). In an abnormal response, the eyes are dragged off target (D), followed by a saccade back to the target (E). This response implies a peripheral vestibular lesion on the right.
Palatal movement (CN IX and X)

• Ask the patient to say “aaah” or yawn while sitting
• Gag reflex – generally no need to test it – 20% of normal individuals don’t have it
Dysarthria (CN IX, X and XII)

• Dysarthria is an impairment of the motor functions necessary for speech production, it is **not a language disorder**.

• Listen for articulation errors, abnormalities of voice quality and irregularities of rate and rhythm.
Head rotation/shoulder elevation (CN XI)

• Ask the patient to turn the head all the way to the left, and test the right sternocleidomastoid muscle
• Shoulder elevation
• Peripheral lesion – ipsilateral weakness of SCM and trapezius
• Central lesion – ipsilateral SCM and contralateral trapezius.
Tongue movement (CN XII)

- Ask the patient to protrude the tongue and move it rapidly from side to side. Also, ask the patient to push the tongue against the cheek.
- CN XII receives descending cortical input from both hemispheres equally, except for genioglossus muscle (from contralateral hemisphere)
MOTOR EXAMINATION
Motor examination

- Gait
- Coordination
- Finger tapping
- Rapid alternating movements
- Finger-to-nose testing
- Heel-to-shin testing
- Pronator drift
Motor examination

Involuntary movements

• Tremor
• Myoclonus – rapid, shock-like muscle jerks
• Chorea – rapid, jerky twitches, similar to myoclonus but more random in location and more likely to blend into one another
• Athetosis – slow, writhing movements of the limbs
• Ballismus – large amplitude flinging limb movements
• Tics – abrupt, stereotyped, coordinated movements or vocalizations
• Dystonia – maintenance of an abnormal posture or repetitive twisting movements
Strength testing

GRADING STRENGTH (Medical Research Council scale)

- 0 = no contraction
- 1 = visible muscle twitch but no movement of the joint
- 2 = weak contraction insufficient to overcome gravity
- 3 = weak contraction able to overcome gravity but no additional resistance
- 4 = weak contraction able to overcome some resistance but not full resistance
- 5 = normal, able to overcome full resistance.
Terminology of weakness

- Monoparesis
- Hemiparesis
- Paraparesis
- Quadriparesis
Muscle bulk

• Inspected and palpated for evidence of atrophy
• Fasciculations
Muscle tone

It is the slight residual tension present in voluntary relaxed muscle

Hypertonia:
• Spasticity
• Rigidity
• Paratonia

Hypotonia

Upper versus lower motor neuron lesions
Nerve roots and peripheral nerves corresponding to the principal movements of the upper extremity

<table>
<thead>
<tr>
<th>Movement</th>
<th>Nerve root</th>
<th>Peripheral nerve</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Shoulder abduction</td>
<td>C5</td>
<td>Axillary</td>
</tr>
<tr>
<td>B. Elbow flexion</td>
<td>C5-6</td>
<td>Musculocutaneous</td>
</tr>
<tr>
<td>C. Elbow extension</td>
<td>C6-7</td>
<td>Radial</td>
</tr>
<tr>
<td>D. Wrist extension</td>
<td>C6-7</td>
<td>Radial</td>
</tr>
<tr>
<td>E. Wrist flexion</td>
<td>C7-8</td>
<td>Median</td>
</tr>
<tr>
<td>F. Finger flexion</td>
<td>C8</td>
<td>Median</td>
</tr>
<tr>
<td>G. Finger extension</td>
<td>C8</td>
<td>Radial</td>
</tr>
<tr>
<td>H. Finger abduction</td>
<td>T1</td>
<td>Ulnar</td>
</tr>
</tbody>
</table>

The letters labeling the movements form a spiral down the extremity. The nerve roots and peripheral nerves corresponding to each movement are listed below. Figure redrawn with permission from Gelb, DJ. The Neurologic Examination.

Nerve roots and peripheral nerves corresponding to the principal movements of the lower extremity

<table>
<thead>
<tr>
<th>Movement</th>
<th>Nerve roots</th>
<th>Peripheral nerve</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Hip flexion</td>
<td>L2-3</td>
<td>Femoral (<em>nerve to iliopectoas</em>)</td>
</tr>
<tr>
<td>B. Knee extension</td>
<td>L3-4</td>
<td>Femoral</td>
</tr>
<tr>
<td>C. Ankle dorsiflexion</td>
<td>L4-5</td>
<td>Peroneal</td>
</tr>
<tr>
<td>D. Hip extension</td>
<td>L4-5</td>
<td>Gluteal</td>
</tr>
<tr>
<td>E. Knee flexion</td>
<td>L5-S1</td>
<td>Sciatic</td>
</tr>
<tr>
<td>F. Ankle plantar flexion</td>
<td>S1-2</td>
<td>Tibial</td>
</tr>
</tbody>
</table>

The letters labeling the movements proceed in order from proximal to distal down the front of the limb, and then repeat from proximal to distal down the back of the limb. The nerve roots and peripheral nerves corresponding to each movement are listed below.

REFLEX EXAMINATION
Tendon reflexes

- Biceps
- Triceps
- Brachioradialis
- Knee
- Ankle

Jendrassik maneuver
Grading reflexes

- 0 = absent
- 1 = reduced (hypoactive)
- 2 = normal
- 3 = increased (hyperactive)
- 4 = clonus
Additional reflexes

• Plantar response – **ONLY EXTENSOR** response is called **BABINSKI SIGN**. Flexor or extensor.

• Superficial reflexes: abdominal, cremasteric

• Primitive reflexes: the grasp, root, snout and palmomental reflexes (**FRONTAL RELEASE SIGN**)
SENSORY EXAMINATION
Sensory examination

• First test the primary sensory modalities (light touch, pain and temperature, vibration, joint position sense), and then the discriminatory sensory functions (stereognosis, graphesthesia, point localizations, two point discrimination and extinction)

• Light touch
• Pain/temperature
• Joint position sense
• Vibration
• Graphesthesia
• Stereognosis
• Two-point discrimination
• Localization of sensory deficits
Schematic representation of the cervical and T1 dermatomes. There is no C1 dermatome. Patients with nerve root syndromes may have pain, paresthesias, and diminished sensation in the dermatome of the nerve that is involved.
Schematic representation of the lumbosacral dermatomes. Patients with sciatica may have pain, paresthesias, and diminished sensation in the dermatome of the nerve root that is involved.