CLUES FROM THE HISTORY AND NEUROLOGICAL EXAMINATION

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The neurologic examination

- Level of consciousness
- Speech fluency
- Pupillary size, reactivity
- Cranial nerve examination
- Muscle strength testing
- Cerebellar testing
- Deep tendon reflexes
- Babinski sign
- Sensation

Onset

- RAPID, LOCALIZED

  Seizure, Migraine, Vascular lesion
- GRADUAL AND LESS LOCALIZED

  Transient Ischemic Attack
- STUTTERING ONSET

  Ischemia

Onset

- GRADUAL OVER HOURS

  Toxic, Metabolic, Infectious, Inflammatory
- RELAPSING, DIFFERENT LEVELS

  Multiple Sclerosis
- SLOW PROGRESSION

  Neurodegenerative, Neoplasm, Chronic inflammation

Site involved

- CEREBRUM

  Gray matter (neuronal)

  Cognitive impairment; movement disorder; seizure

  White matter

  “Long Tract” disorders of motor, sensory, visual, or cerebellar pathways
- BRAIN STEM

  Cranial nerve lesion; crossed weakness

Site involved

- SPINAL CORD

  Mixed upper and lower motor neuron changes; weakness sparing head

- PROGRESSIVE, SYMMETRIC, NOT CIRCUMSCRIBED

  Metabolic; degenerative

Developmental milestones

Age	Development	Social
0-3 months	Rooting reflex	Orients to voice
3 months	Moro reflex lost; holds head up	Social smile
4-5 months	Rolls front to back; sits propped up	Recognizes people
7-9 months	Sits alone; crawls	Anxious with strangers
12-14 months	Upgoing Babinski disappears	
15 months	Walks; speaks few words	Separation anxiety
12-24 months	Climbs stairs; stacks 3 blocks	
18-24 months	Stacks 6 blocks	
24-48 months		Rapprochment; parallel play; gender identity
30-36 months	Stacks 9 blocks	Toilet training

Developmental milestones

AGE	DEVELOPMENT	SOCIAL
3 years	Rides tricycle; copies line and circle	900 word vocabulary; speaks in complete sentences
4 years	Draw stick figure; copy a cross; hop on one foot	Cooperative play; grooms self
6 years	Copy triangle	Reads; understands concept of death;
6-11 years	Rides bicycle	Same sex identification; conscience develops
11 years (girls); 13 years (boys)		Abstract reasoning; forms personality
	These milestones now occur at later ages than in earlier decades.	

Mental retardation

- Mental retardation and developmental deficits present in early childhood. One third genetic.
- Retarded are impaired in more than two of the following areas:
- Communication
- Self-care
- Home living
- Social and interpersonal skills
- Self-direction
- Academics
- Work (health and safety)
- Utilization of community resources

Headache

- Multifocal headaches and headaches described as sharp and stabbing have a benign cause.
- Site of extracranial origin is often palpable. (Temporal arteritis headache is unilateral, pounding.)
- Posterior fossa lesions present with occipito- nuchal headaches. Supratentorial lesions present with fronto- temporal headaches. Prostrating. Invariably unilateral.
- A headache of sudden onset (“thunderclap”) must be considered a subarachnoid hemorrhage . Aneurysms <1 cm size rarely cause headache or rupture. A “thunderclap” headache can only be diagnosed once subarachnoid hemorrhage is excluded.

Headache

- Meningeal irritation presents with nuchal rigidity.
- Glaucoma may present with severe eye pain. The pupil may be dilated. The eye is red.
- “Ice pick pains." are very brief, sharp, severe pains located in the scalp outside of the trigeminal distribution. They may be single or repetitive or occur in clusters, either at a single point or scattered over the scalp. Are severe enough to cause involuntary flinching.
- They are more common in migraine and cluster patients.

Migraine headache

- Migraines have slow buildup and last for up to 72 hours.
- Migraine headaches may wake the patient.
- Usually present with visual disturbances. Stars or flashes predominate; zigzags and scotomata are also common.
- Visual auras usually involve one portion of the visual field but may spread or migrate. Usually last less than 30 minutes. Precede onset of unilateral headache.
- Light and sound intolerance. Nausea and vomiting, rapid pulse and sweating. May have vertigo.

Migraine headache

- If the headache is pulsatile, lasts between 4-72 hours without medications, is unilateral, with nausea, and is disabling, the positive likelihood ratio (LR+) for a migraine headache is 24.
- Migraines can occur without aura. The headache must have at least two of the following qualities: unilateral, pulsating, aggravated by physical activity, limit physical activity; AND have either nausea (or vomiting) or photophobia (or phonophobia).
- Migraine aura without headache can also occur.
- Migraine may be precipitated by chocolate or cheese. Chocolate craving may be prodromal sign.

Aura

- Prolonged auras are seen in coagulopathy, central vein thrombosis, vessel dissection, and mitochondrial disorders
- Short auras are seen in seizure disorders, arteriovenous malformation

Migraine headache

- Hemicranial pain is most common pattern. Pain can also be holocephalic, bifrontal, or unilateral frontal in distribution. Localization to the occiput or the vertex is less common.
- NSAIDs may abort attack. Responds to Triptans. Ergotamine use associated with retroperitoneal fibrosis.
- β-blockers may be effective prophylaxis.

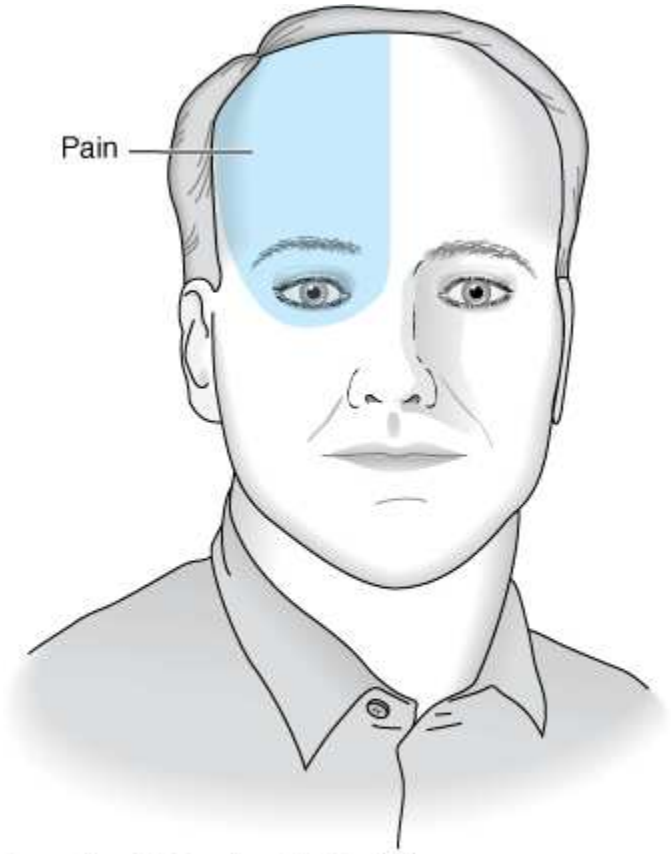

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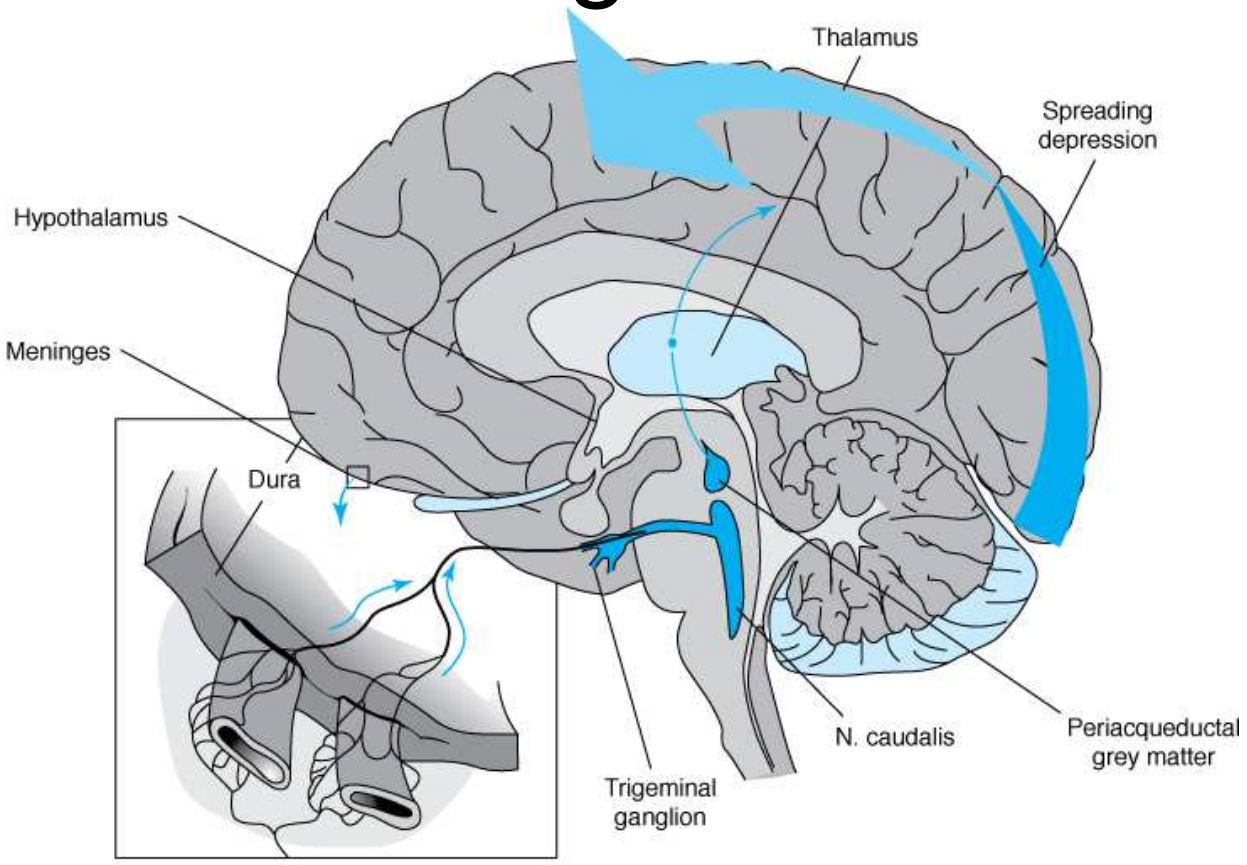
Fig. 2-13 Accessed 07/01/2010

Migraine

- Migraine may be initiated by the trigemino-vascular sensory system input from meningeal vessels, which projects through the nucleus caudalis to the periaqueductal gray, sensory thalamus, and sensory cortex. Pteropalatine ganglion return
- Increased retinal cortical blood flow
- Increased extracellular K+; intracellular Na+ and Cl- increase, with concomitant neuronal swelling (and neurotransmitter release)
- Glutamate as a trigger

Migraine



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Direction of spread (arrows) and maximal extent (colored region) of depressed cerebral blood flow in migraine with and without aura.

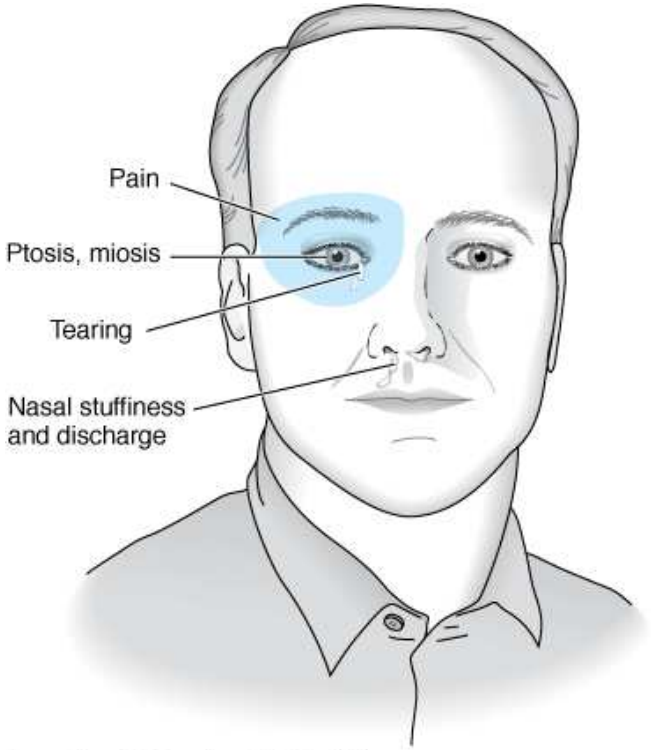
Fig. 2-10 Accessed 07/01/2010

Cluster headache

- A cluster headache has sudden onset, peaks in 3-5 minutes, and rapidly dissipates. Occur in cycles.
- Episodic, daily headache for at least 3 weeks. May have several daily. Cycle terminates spontaneously. Cycle can recur years later.
- Unilateral periorbital pain with an ipsilateral red, watery eye. Nasal discharge and nasal congestion may be present. (Possibly ipsilateral Horner's syndrome).
- Responds to 100% Oxygen.

Headache

Cluster



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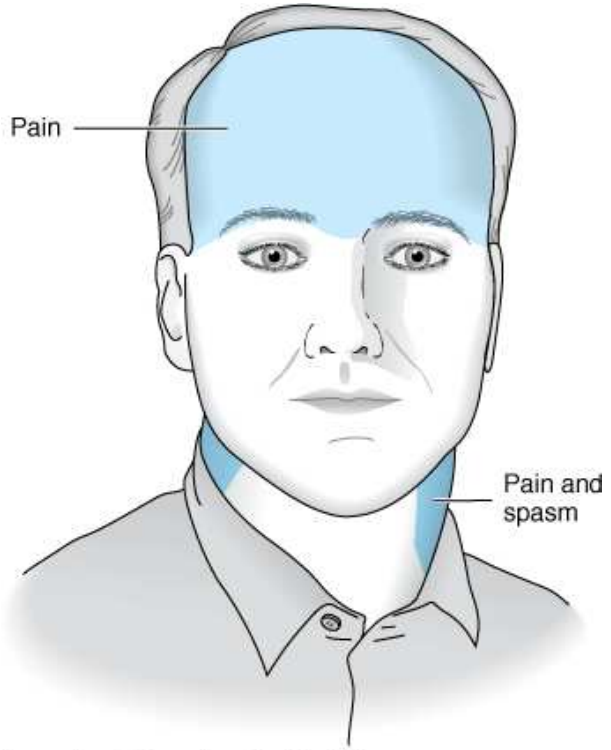
Tension



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Figs. 2-14 and 2-16 Accessed 07/01/2010

Tension headache

- Non-pulsatile, not aggravated by routine activity, bilateral, and are not associated with nausea or vomiting.
- Pericranial muscle spasm precipitating event.
- Respond to NSAIDs.
- Chronic tension headache may be managed with tricyclic antidepressant.

Temporal arteritis

- Jaw claudication (pain near the TMJ that develops only after a brief period of chewing) suggests temporal arteritis (positive likelihood ratio, LR+ 5.2).
- Often visual disturbances are present.
- Severe, throbbing headache (occasionally dull or burning) of acute onset and (in half the cases) bitemporal suggests temporal arteritis. This may be scalp tenderness.

Temporal arteritis

- The combination of headache, scalp tenderness, and jaw claudication in an older patient increases the likelihood of temporal arteritis as does finding a tender artery or finding diminished pulsations. (LR+, 18)
- An ESR <50mm/hr makes the diagnosis unlikely (LR+, 0.35).
- Corticosteroid therapy is begun once the disease is suspected.
- Biopsy is diagnostic

Intracerebral bleeds

- Bleeding is usually derived from arterioles and small arteries. Bleeding is localized and spreads along white matter pathways. Most common causes are hypertension, trauma, bleeding disorder, amyloid angiopathy, cocaine or metamphetamines, vascular malformations.
- Neurologic symptoms increase gradually over minutes or a few hours.
- Subarachnoid hemorrhage develops in an instant. Focal brain dysfunction is uncommon and suggests bleeding from an arterio-venous malformation.

Subarachnoid hemorrhage

- Prevalence of aneurysms in general population is 4%
- Aneurysms < 1cm diameter rarely cause symptoms and rarely rupture.
- Aneurysms >1 cm diameter rupture at a rate of 0.5% per year.
- 85% of subarachnoid hemorrhages are due to a rupture of a saccular aneurysm in circle of Willis. Common sites involve junction of anterior communicating artery with anterior cerebral artery, junction of posterior communicating artery with the internal carotid artery, and bifurcation of the middle cerebral artery.
- Present with headache; often, stiff neck, change in mental status

Subarachnoid hemorrhage

- Non-contrast CT highly sensitive within first 12 hours
- Angiography is performed to assist in surgical plan
- Lumbar puncture deferred for 6-12 hours after the onset of a headache in a patient with a suspicious headache and a negative CT scan; then, xanthochromia in the cerebrospinal fluid is diagnostic

Stroke criteria

- In patients >45 years old, with no history of seizures, who is not wheelchair bound or bedridden, (and whose blood glucose is between 60-400 mg/dl), the sudden (<24 hours) presence (and persistence for more than minutes) of any one of the following is diagnostic of an acute stroke:

  Facial asymmetry (show teeth or smile)

  Arm drift (patient closes eyes and extends both arms straight out for 10 seconds with the palms up; one arm drifts down)

  Weak grip (unilateral) or alteration in speech

Stroke

- Any of the three symptoms, facial paresis, arm drift, or abnormal speech has an positive likelihood ratio (LR+) of 5.2 for stroke in emergency patients without trauma or not in coma. The probability of a stroke is >10%.
- If all three are present, the LR+ is 14. If the neurologic deficits persist, the LR+ is 40.
- If none are present though onset is acute, the LR+ is 0.14.
- Deficits that last less than 4 hours are classified as a TIA. However, many are silent infarcts.
- The lower the NIHSS score, the greater the probability of excellent recovery.

Stroke

- Thrombotic stroke may originate in large vessels. Carotid stenosis is a significant factor for large artery thrombotic stroke. Atherothrombosis is the most common pathologic process. Defects may wax and wane over days.
- Small vessel disease refers specifically to penetrating arteries. They may thrombose due to atheroma formation at their origin or in the parent larger artery, or due to lipohyalinosis (hypertensive change). Can result in small deep (lacunar) infarcts. Evolve over hours.

Stroke

- Embolic fragments originate in the heart or aorta. Defects are of sudden onset, may resolve.
- Systemic hypoperfusion may lead to ischemia. 85% of strokes are ischemic in origin.
- Those of sub-Saharan origin, Asians, and women have a lower incidence of occlusive disease of the extracranial carotid and vertebral arteries than do men of European origin.
- Hypertensive intracerebral hemorrhage is more common among those of sub-Saharan origin or Asian origin. 15% of strokes. 66% involve basal ganglia.

Stroke

- Headache is typically a feature of hemorrhagic strokes.
- Vomiting is common in patients with intracerebral hemorrhage, subarachnoid hemorrhage, and vertebrobasilar artery ischemia.
- Reduced alertness favors hemorrhage. Accompanying neurologic signs favor intracerebral hemorrhage. Loss of consciousness, vertebrobasilar artery ischemia.

Oxford classification of subtypes of cerebral infarction

- **Total anterior circulation infarction syndrome**:
- A combination of (1) higher cerebral dysfunction such as dysphasia, dyscalculia, visuospatial disorder; with (2) homonymous visual field defect; and (3) ipsilateral motor or sensory deficit of at least two areas (face, arm, leg)
- Reflects proximal occlusion of the internal carotid or trunk of the middle cerebral artery

Oxford classification of subtypes of cerebral infarction

- **Partial anterior circulation infarction syndrome**:
- If face deficit with contralateral lower extremity involvement, anterior cerebral artery involved
- If aphasia with contralteral hemiparesis, middle cerebral artery involved
- **Lacunar infarction syndrome**:
- Pure motor stroke (internal capsule)
- Pure sensory stroke (thalamus)
- Sensorimotor stroke, or cerebellar dysfunction with ipsilateral long-tract deficit (ataxic hemiparesis) reflects infarct in pons

Oxford classification of subtypes of cerebral infarction

- **Posterior circulation infarction syndrome**:
- Any ipsilateral cranial nerve palsy with contralateral motor or sensory deficit; or bilateral motor or sensory deficit; or disorder of conjugate eye movement; or cerebellar dysfunction without ipsilateral long-tract deficit; or isolated homonomynous visual field defect
- Reflects verterbrobasilar distribution stroke

Pupil signs in stroke

- Pinpoint pupils (pons)
- Poorly reactive pupils (thalamus)
- Dilated pupils (putamen)

Arousal and awareness

- Patients in coma cannot be aroused
- Patients who are asleep can be aroused
- Arousal in a patient with a pontine lesion is demonstrated through directed eye movements
- Patients who are awake but unresponsive to stimuli are unaware
- Patients in a vegetative state can be aroused but are unaware
- Inattention to verbal stimuli may be due to hearing loss

Syncope or seizure

- Incontinence or even some automatisms (lip smacking, for example) DO NOT distinguish syncope from seizure.
- A seizure usually has an aura associated with its onset. Sweating, nausea, pallor are not described. Following the seizure the patient may have myalgias. Mental confusion may persist for minutes.
- Syncope has no aura. Sweating, nausea, pallor are generally described. Following the syncopal episode, mental confusion may last for seconds. Myalgias are uncommon.

Syncope or seizure

- Syncope occurs generally in the daytime. It does not awaken from sleep. Seizures may occur at any time and may awaken from sleep.
- Syncopal episodes are brief (seconds); seizures last minutes.
- Usual causes of syncope are neuro-cardiogenic (20%); orthostatic (11%); arrhythmia (14%); TIA, seizure, migraine (7%)

Seizures

- Initiated by two concurrent events:
- **High frequency bursts of action potentials**. Caused by relatively long depolarization of the neuronal membrane leading to influx of extracellular Calcium ion, which leads to the opening of voltage dependent Sodium ion channels, allowing Sodium ion influx, and generation of repetitive action potentials.
- This is followed by a hyperpolarizing after-potential mediated by GABA receptors or Potassium ion channels depending upon cell type.

Seizures

- **Hypersynchronization**. Repetitive discharges lead to an increase in extracellular Potassium ion , which blunts hyper-polarization and depolarizes neighboring neurons.
- Calcium ion accumulates in pre-synaptic terminals, leading to enhanced neurotransmitter release.
- Depolarization induced activation of the NMDA subtype of the excitatory glutamate receptor causes Calcium ion influx and neuronal activation.

- Neuronal recruitment and propagation follow.

Seizure

Focal site	Type	Signs
Frontal lobe	**Simple or complex partial seizures**	**Abrupt onset. May occur several times daily. Adversive head movements or complex movements. Mood change. Autonomic dysfunction. Speech arrest. Limited post-ictal confusion.**
Temporal lobe	**Complex partial seizures**	**Nausea, heat sensation. Visual or auditory hallucinations. Compulsive thoughts; déjà vu; automatisms. Dyspnea, palpitations, urinary urgency. Post-ictal confusion.**
Parietal lobe	**Simple seizures**	**Sensory and major motor (Jacksonian) seizures. Post-ictal confusion.**
Occipital lobe	**Simple seizures**	**Unformed visual hallucinations.**

Partial seizures	Signs
Simple	No loss of consciousness; seizures involve only sensory or motor systems
Complex	Impaired consciousness; onset localized, but spreads; involves limbic system; usually involves temporal lobe; may involve automatisms

Generalized seizures	Signs
Tonic-clonic (Grand mal)	Loss of consciousness; associated with generalized tonic muscle contractions (flexion: arms; extension: legs) followed by rhythmic contractions of limbs. Post-ictal phase can involve altered consciousness. 10-12Hz spikes on EEG .
Tonic	Loss of consciousness; rigid, violent muscle contractions; limbs fixed in abnormal positions
Clonic	Loss of consciousness; rhythmic muscle jerks; involves all parts of the body
Absence (Petit mal)	Brief loss of consciousness; may be frequent . 3Hz bilateral discharges on EEG.
Atonic	Sudden loss of muscle tone (fall)

Seizures

- Neonates

  ischemia, hypoxia, trauma, metabolic (glucose, Calcium, Magnesium, pyridoxine), developmental, genetic
- Infants (1 month – 12 years of age)

  infection, trauma, genetic
- Adolescents (12-18 years of age)

  trauma, infection, genetic, tumor, drugs

Seizures

- Young Adults (18 – 35 years of age)

  trauma, alcohol or drugs, tumor

- Adults (over 35 years of age)

  CVA, tumor, alcohol or drugs, metabolic, degenerative

  A first seizure in someone older than 40 should raise the possibility of bacterial endocarditis.

Concussion

- Transient loss of consciousness due to trauma to reticular formation in brainstem.
- Boxing knockout blow deflects head up and posteriorly; causes torque on brainstem leading to functional paralysis.
- Blow to the temporal skull may cause fracture but not loss of consciousness as lateral movement is inhibeted by the Falx.

Contusion

- Traumatic bruise of the brain caused by acceleration of the head in an anterior-posterior direction, or, in the case of a fall, downward direction.
- Crests are most susceptible to injury.
- Contusion at site of impact is called a coup injury.
- Contusion at site opposite impact is called a contrecoup injury.

Coma

- Drug overdose, trauma, cardiac arrest are three most common causes of coma.
- 2-33% of those resuscitated outside of the hospital survive to reach the hospital. 80% of those are comatose. 10-30% have meaningful recovery.
- Cerebral O_2 stores lost within 20 seconds of the onset of cardiac arrest; glucose and ATP stores are lost by 5 minutes.

Delirium

- A diagnosis of delirium requires a cognitive deficit.
- This criterion includes disorientation but may alternatively be fulfilled by memory impairment or disorganized thinking.
- May fluctuate throughout the day
- 3D Confusion Assessment Method is a 3-minute nursing assessment recommended for use throughout day
- delirium does not always resolve
- May persist for months after resolution of primary cause, particularly in older, frail patients.
- Increased duration associated with increased morbidity and mortality

Delirium

- Lasting and even accelerated progression of long-term cognitive impairment after an episode of delirium
- Confusion is not part of aging
- Neurocognitive decline is the major risk for delirium in the elderly
- Use of opioids for paint control as well as inadequate treatment of pain is associated with confusion in the elderly_
- Advancing age, functional limitations, visual impairment, and alcohol misuse are contributory

Delirium

- Precipitating factors believed to cause delirium directly include polypharmacy (in particular, anticholinergic agents), a wide range of electrolyte and metabolic disturbances, infections, restraint, catheter use, and surgery
- Late-life onset of psychosis is rare
- Psychotic symptoms such as delusions or hallucinations presenting in middle age and beyond suggest a medical and/or substance-induced cause

Delirium

- Non-auditory hallucinations, especially visual and tactile, should immediately raise suspicion for delirium.
- The more vivid the visual content the more likely there is a medical cause.
- Audio-visual hallucinations occurring at the boundary of sleep and wake (hypnagogic or hypnopompic hallucinations) are typically non-pathological.
- Similarly, new-onset delusions, bizarre beliefs, or significant misinterpretation of environment or interpersonal cues should raise suspicion for delirium

Delirium

- There is a bidirectional risk of dementia and delirium
- Delirium in patients with baseline cognitive impairment predicts a more precipitous cognitive decline than is seen in patients who do not develop delirium
- Every point increase in dementia severity on the Global Deterioration Scale (dementia) is associated with a 50% increase in delirium risk.
- Post-operative mortality doubles at 1 year in patients with dementia who develop delirium

Delirium

- There is no good evidence for use of antipsychotics in the treatment of delirium
- The Practice Guideline on the Use of Antipsychotics to Treat Agitation or Psychosis in Patients With Dementia states:
- “Nonemergency antipsychotic medication should be used only for the treatment of agitation or psychosis in patients … when symptoms are severe, are dangerous, and/or cause significant distress to the patient”
- Target disturbance (e.g., removing endotracheal tube) and stop medication when behavior resolves

Delirium

- More than 50% of delirium patients present with hypoactivity
- May be confused with depression
- Withdrawn patients require proactive intervention
- Refusal of food, physical therapy, intravenous fluids, or medications
- Sleepiness upon awakening is common; inability to stay awake throughout the day

Features of delirium

Headache	**Head trauma, meningitis, subarachnoid hemorrhage**
Vital signs	
Fever	**Infectious meningitis, anticholinergic intoxication, withdrawal from ethanol or sedative drugs, sepsis**
Hypothermia	**Intoxication with ethanol or sedative drugs, hepatic encephalopathy, hypoglycemia, hypothyroidism, sepsis**
Hypertension	**Anticholinergic intoxication, withdrawal from ethanol or sedative drugs, hypertensive encephalopathy, subarachnoid hemorrhage, sympathomimetic intoxication**
Tachycardia	**Anticholinergic intoxication, withdrawal from ethanol or sedative drugs, thyrotoxicosis, sepsis**
Bradycardia	**Hypothyroidism**
Hyperventilation	**Hepatic encephalopathy, hyperglycemia, sepsis**

Features of delirium

General examination	
Meningismus	**Meningitis, subarachnoid hemorrhage**
Skin rash	**Meningococcal meningitis**
Tetany	**Hypocalcemia**
Cranial nerves	
Papilledema	**Hypertensive encephalopathy, intracranial mass**
Dilated pupils	**Head trauma, anticholinergic intoxication, withdrawal from ethanol or sedative drugs, sympathomimetic intoxication**
Constricted pupils	**Opioid intoxication**
Nystagmus/ ophthalmoplegia	**Intoxication with ethanol, sedative drugs, or phencyclidine, vertebrobasilar ischemia, Wernicke encephalopathy**

Features of delirium

Motor	
Tremor	**Withdrawal from ethanol or sedative drugs, sympathomimetic intoxication, thyrotoxicosis**
Asterixis	**Metabolic encephalopathy**
Hemiparesis	**Cerebral infarction, head trauma, hyperglycemia, hypoglycemia**
Other	
Seizures	**Withdrawal from ethanol or sedative drugs, head trauma, hyperglycemia, hypoglycemia**
Ataxia	**Intoxication with ethanol or sedative drugs, Wernicke encephalopathy**

Drugs of abuse

Drug	Pupils	Reflexes	Other signs	Psyche
Cocaine	**Dilated**	**Hyperactive**	**Chorea, tremor, dystonia, myoclonus**	**Anxiety, agitation, hypervigliance.**
Amphetamines	**Dilated**	**Hyperactive**	**Chorea, tremor, muscle spasms**	**Euphoria or dysphoria, hyperactivity, hypervigilance, hallucinations**
MDMA	**Dilated**	**Hyperactive**	**Tremor, rigidity**	**Anxiety, hyperactivity, psychosis**
PCP	**Dilated, nystagmus**	**Hyperactive**	**Ataxia, tremor, increased muscle tone**	**Euphoria or dysphoria, psychosis, aggression**

Drugs of abuse

Drug	Pupils	Reflexes	Other signs	Psyche
Opiates	**Pinpoint**	**Depressed**	**Hypokinesia**	**Euphoria, somnolence**
LSD	**Dilated, sluggish**	**Hyperactive**	**Tremor**	**Euphoria, hallucinations, panic**

Alcohol withdrawal signs

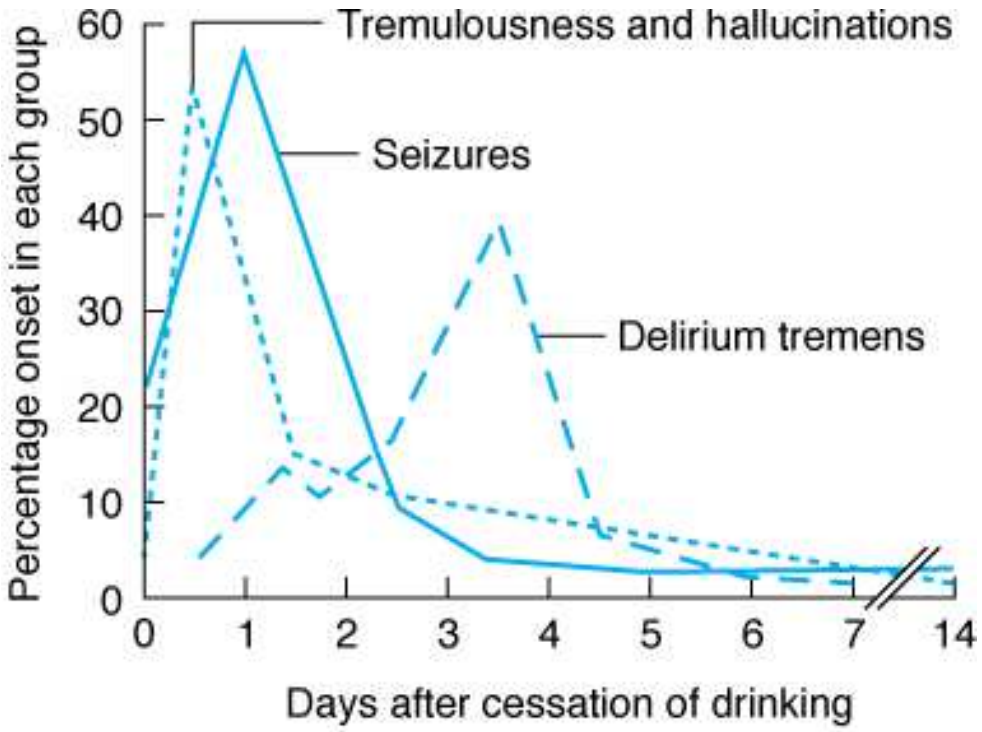

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Fig. 1-3 Accessed 07/01/2010

Administration of glucose without prior administration of thiamine may precipitate Wernicke's encephalopathy

Features of dementia

History	
Unprotected sexual intercourse, intravenous drug abuse, hemophilia, or blood transfusions	HIV-associated dementia
Family history	Huntington disease, Wilson disease
Headache	Brain tumor, chronic subdural hematoma
Vital signs	
Hypothermia	Hypothyroidism
Hypertension	Vascular dementia
Hypotension	Hypothyroidism

Features of dementia

General examination	
Meningismus	**Chronic meningitis**
Jaundice	**Acquired hepatocerebral degeneration**
Cranial nerves	
Papilledema	**Brain tumor, chronic subdural hematoma**
Argyll Robertson pupils	**Neurosyphilis**
Ophthalmoplegia	**Progressive supranuclear palsy**
Pseudobulbar palsy	**Vascular dementia, progressive supranuclear palsy**

Features of dementia

Motor	
Tremor	**Dementia with Lewy bodies, corticobasal ganglionic degeneration, acquired hepatocerebral degeneration, Wilson disease, HIV-associated dementia**
Asterixis	**Acquired hepatocerebral degeneration**
Myoclonus	**Creutzfeldt-Jakob disease, HIV-associated dementia**
Rigidity	**Dementia with Lewy bodies, corticobasal ganglionic degeneration, acquired hepatocerebral degeneration, Creutzfeldt-Jakob disease, progressive supranuclear palsy, Wilson disease**
Chorea	**Huntington disease, Wilson disease**
Other	
Gait apraxia	**Normal pressure hydrocephalus**
Polyneuropathy with hyporeflexia	**Neurosyphilis, vitamin B_{12} deficiency, HIV-associated dementia**

Dementia or depression

Dementia	Depression
Indifferent to memory impairment. Semantic paraphasia.	Can describe memory impairment precisely and in detail
Cognitive deficit	No or minimal cognitive deficit
Signs of depression secondary	Brooding, anxiety, sleep disturbance, loss of appetite, self doubt at presentation
Rarely past history of depression	Usually past history of depression

Amnesia

Manifestations	Lesion
Acute onset. **Can perform usual activities.** **Anxious but not confused.** **Anterograde or retrograde amnesia.**	**If transient ischemic episode, there may be full recovery.** **Migraine.** **Partial or complex seizure.** **Post-traumatic stress.** **If persistent, anterior cerebral artery infarction (hippocampus, thalamus).** **Orbito-frontal or medio-basal skull trauma may produce direct injury.** **Hypoxia (Ammon's horn).**
Subacute onset. **Initially confused.** **Anterograde or retrograde amnesia.**	**Wernicke-Korsakoff syndrome** **Herpes simplex encephalitis, HIV** **Basilar meningitis (fungus, TB)** **Sarcoid**
Progressive **Evolving retrograde amnesia**	**Tumor of temporal lobe or 3rd ventricle** **Paraneoplastic syndrome (lung cancer)** **Alzheimer**

Glasgow-Pittsburgh cerebral performance categories

- **Good cerebral performance**: Conscious. Alert. available to lead a normal life.
- (May have mild dysphasia, nonincapacitating hemiparesis, or minor cranial nerve abnormalities.)
- **Moderate cerebral disability**: Conscious. Sufficient cerebral function for work in sheltered environment or for independent activities of daily life (dressing, traveling by public transportation, preparing food).
- May have hemiplegia, seizures, ataxia, dysarthria, dysphasia, or permanent disability or mental damage.

Glasgow-Pittsburgh cerebral performance categories

- **Severe cerebral disability**: Conscious. Dependent upon others for daily support because of impaired brain function. At least limited cognition.
- May be ambulatory with severe memory disturbance or dementia. May be paralyzed and able to communicate only with eyes.
- **Coma or vegetative state**: Not conscious. Unaware of surroundings. No cognition. No verbal or psychological interactions with environment.

Coma outcomes

- There are no clinical findings that strongly predict a good clinical outcome.
- The findings at 24 and, then, 72 hours are more important than those immediately post-resuscitation.
- At 24 hours, a poor clinical outcome is predicted by the absence of a pupillary response (positive likelihood ratio of 10; negative, 0.8) and the absence of a corneal reflex, (13; 0.6).
- At 72 hours, a poor clinical outcome is predicted by the absence of motor response (positive likelihood ratio of 9.2; negative, 0.7). Seizures at 72 hours have minimal prognostic value.

Speech

- Aphasia implies cortical dysfunction
- How did the problem begin?
- Global aphasia: “Uh..well..umm..like”
- Expressive aphasia: “1..2..days..bed”
- Receptive aphasia (phonemic paraphrasia): “When whence beside bespoke”
- Conductive aphasia: “Started..well..started”
- Broca aphasia results in deficits of speech and writing
- Dysarthria implies motor dysfunction

Speech

- Peripheral lesions are associated with slurred speech, dyspnea, hoarseness, or whispering. May “speak through nose”.
- Cerebellar lesions associated with clipped, scanning speech.
- Basal ganglia lesions are associated with monotonous, soft, slurred speech. May see explosive, loud, uncoordinated, clipped speech, however.
- Slurred, effortful, slow speech is seen with metabolic disturbances.
- Monotonous, slow, hoarse, pressured speech is seen with white matter lesions. Deep, variable pitch.

Motor cortex lesions

- Irritative lesions may cause seizures that begin as focal twitching and spread (in a somatotopic manner, reflecting the organization of the homunculus) to involve large muscle groups (Jacksonian epilepsy). As abnormal electrical discharge spreads across the motor cortex, the seizure "marches" along the body. There may also be modification of consciousness and post-convulsive weakness or paralysis.
- Destructive lesions of the motor cortex (area 4) produce contra-lateral flaccid paresis, or paralysis, of affected muscle groups. Spasticity is more apt to occur if area 6 is also ablated.

Sensory cortex lesions

- Irritative lesions produce paresthesias (eg, numbness, abnormal sensations of tingling, electric shock, or pins and needles) on the opposite side of the body.
- Destructive lesions produce subjective and objective impairments in sensibility, such as an impaired ability to localize or measure the intensity of painful stimuli and impaired perception of various forms of cutaneous sensation. Complete anesthesia on a cortical basis is rare.

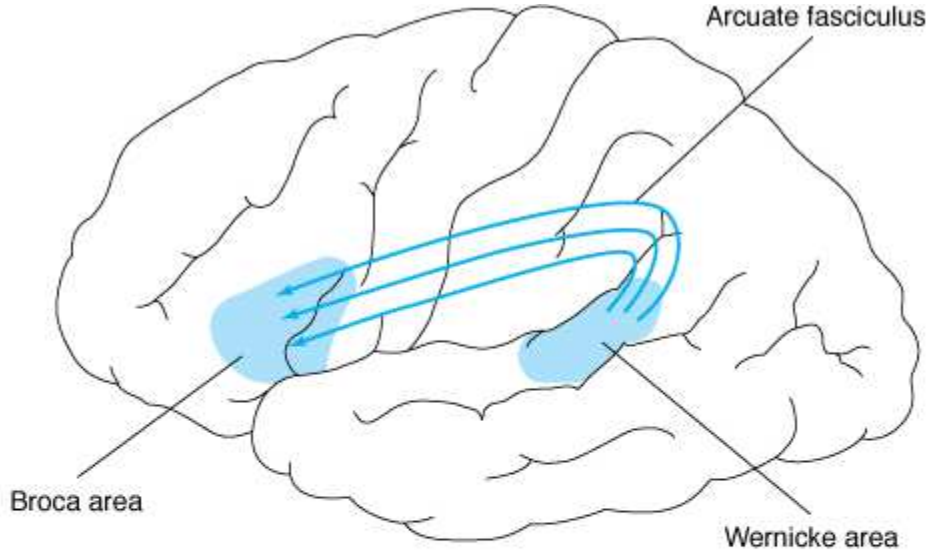
Visual cortex lesions

- Irritative lesions of area 17 can produce such visual hallucinations as flashes of light, rainbows, brilliant stars, or bright lines.
- Destructive lesions can cause contralateral homonymous defects of the visual fields. This can occur without destruction of macular vision, a phenomenon called "macular sparing." Injury to areas 18 and 19 can produce visual disorganization with defective spatial orientation in the homonymous halves of the visual field.

Auditory cortex lesions

- Irritation of the region in or near the primary auditory receptive area in humans causes buzzing and roaring sensations. A unilateral lesion in this area may cause only mild hearing loss, but bilateral lesions can result in deafness.
- Damage to area 22 in the dominant hemisphere produces a syndrome of pure word deafness (in which words cannot be understood although hearing is not impaired), also called Wernicke's aphasia.

Aphasia



Pathologic Site	Type of Aphasia	Language Functions Preserved		
		Comprehension	Repetition	Fluency
Wernicke area	Receptive	–	–	+
Arcuate fasciculus	Conductive	+	–	+
Broca area	Expressive	+	–	–

Source: Simon RP, Greenberg DA, Aminoff MJ:
Clinical Neurology, 7th Edition: http://www.accessmedicine.com

Fig. 1-2 Accessed 07/01/2010

Examples of aphasia

- How did the problem begin?
- Global aphasia: “Uh..well..umm..like”
- Expressive aphasia: “1..2..days..bed”
- Receptive aphasia (phonemic paraphrasia): “When whence beside bespoke”
- Conductive aphasia: “Started..well..started”

Testing of higher cortical functions

- These functions are tested in detail if the patient's history or behavior during the general examination has provided a reason to suspect some defect.
- Psychiatric aspects incorporate affect, mood, and normality of thought processes and content.
- Neurologic aspects include the level of consciousness, awareness (attention), language, memory, and visual-spatial abilities.

Testing of higher cortical functions

- Questions are first directed toward determining the patient's orientation in time and place and insight into his current medical problem.
- Attention, speed of response, ability to give relevant answers to simple questions, and the capacity for sustained and coherent mental effort all lend themselves to straightforward observation.

Testing of higher cortical functions

- The patient's account of his recent illness, dates of hospitalization, and his day-to-day recollection of recent incidents are excellent tests of memory;
- the narration of the illness and the patient's choice of words (vocabulary) provide information about his language ability and coherence of thinking.

Testing of higher cortical functions

- Useful bedside tests of attention, concentration, memory, and clarity of thinking include:
- the repetition of a series of digits in forward and reverse order;
- serial subtraction of 3s from 20 or 7s from 100;
- and recall of three items of information or a short story after an interval of 3 min.

Testing of higher cortical functions

- If there is any suggestion of a speech or language disorder, the nature of the patient's spontaneous speech should be noted.
- In addition, the accuracy of reading, writing, and spelling,
- executing spoken commands,
- repeating words and phrases spoken by the examiner,
- naming objects and parts of objects,
- and solving simple arithmetical problems should be assessed.

Testing of higher cortical functions

- The ability to carry out tasks (praxis) has great importance in the evaluation of several aspects of cortical function.
- Bisecting a line, drawing a clock or the floor plan of one's home or a map of one's country, and copying figures are useful tests of visual-spatial perception and are indicated in cases of suspected cerebral disease.

Testing frontal lobe functions

- Digit span (“Repeat the sequence 1, 2, 3, 7, 5, 9 forwards and backwards.”)
- Verbal Trails ("I'm going to tell you a sequence of letters and numbers, and I want you to pick up the pattern and keep going: 1, A, 2, B, 3....”)
- Spell "WORLD" backwards
- Serial 7s OR serial 3s (Begin subtracting 7 from 100 and continue subtracting 7 for five sequences; or begin subtracting 3 from 20 and continue subtracting for five sequences)
- Luria sequence (have the patient learn and imitate a sequence of hand movements: fist, palm, edge). Useful with the deaf.

Testing frontal lobe functions

- Abstraction:

  Analogies (“How are an apple and an orange alike?, a poem and a statue?, a tree and a dog?”)

  Proverbs (familiar--"what does 'people who live in glass houses shouldn't throw stones?' mean?"; unfamiliar--"what does 'when elephants fight, the grass gets trampled' mean?“)
- Phonemic fluency (“In one minute name as many words as you can that start with the letter S (or A or F), no proper nouns allowed.)
- Semantic fluency (“In one minute name as many 4-legged animals as you can.”)

Testing frontal lobe functions

- Contrasting programs (“When I hold up 2 fingers, you hold up 1 finger; when I hold up 1 finger, you hold up 2 fingers” and repeat this ten times.)
- Go-no go (a set-shift from contrasting programs: "Now, when I hold up 1 finger, you still hold up 2 fingers; but when I hold up 2 fingers, you don't hold up any" and repeat this ten times.)
- Crossed-response inhibition ("Close your eyes and rest your hands on your knees; when I touch your right hand, I want you to lift your left hand; and when I touch your left hand, I want you to lift your right hand" and repeat this ten times.)

Testing frontal lobe functions

- Echopraxia present?
- 3-step command ("I'm going to give you some instructions, and I want you to do them in the exact order I say, without talking while you do them. Don't start till I say ok. I want you to touch your left hand to your right ear, close your mouth, and open your eyes. Ok, you can start“.)

Testing dominant parietal-temporal lobe functions

- Language fluency (observe)
- Language comprehension (Observe. Can patient follow commands?)
- Repetition ("No ifs, ands, or buts"; "Methodist Episcopal")
- Reading
- Writing
- Naming objects, parts of objects, and fingers
- Simple calculations

Testing dominant parietal-temporal lobe functions

- Ideomotor praxis ("I want you to show me how you'd use a hammer/ a comb/ a screwdriver. Pretend you're holding the hammer and show me how you'd use it". If patient tries to use their body part as the tool, say "No, not that your finger is a screwdriver, pretend you're holding a screwdriver“.)
- Left-Right confusion present?

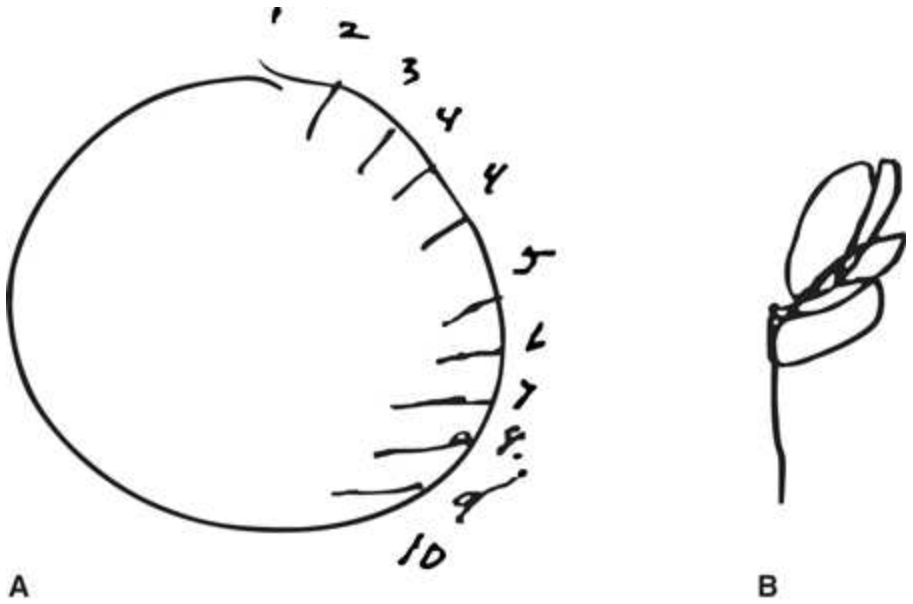
Testing non-dominant parietal-temporal lobe functions

- Obvious neglect present?
- Line cancellation (Give the patient a sheet with a bunch of short diagonal lines on it. Tell them to cross out all the lines and turn them into X's. See if the patient misses any. This is a test for neglect.)
- Line bisection (Draw a straight horizontal line, have the patient draw an X in the middle. See if the patient puts the mark prominently towards or on the end of the line. This is another test for neglect.)
- Figure copying (I use intersecting pentagons)

Testing non-dominant parietal-temporal lobe functions

- Prosody ("I'm going to tell you a sentence, and I want you to tell me if my voice sounds happy or sad. 'I just won the lottery' (said in a sad voice); "Someone stole my car" (said in a happy voice)).
- Clock drawing ("Draw a big circle. Now pretend it's a clock. Put the hands and numbers on it, and set the time at 10 past 11. Draw it so a child could read it.")

Disturbances of orientation



Source: Waxman SG: *Clinical Neuroanatomy, 26th Edition*:
http://www.accessmedicine.com

Testing non-dominant parietal-temporal lobe functions

- Extinction ("Put your hands on your knees and close your eyes. I'm going to touch your hands, and you tell me it it's your right hand or left hand I'm touching". Touch the patient's right hand, their left hand, their right hand again, and then both hands. If the patient says you're just touching the right hand when you touch both, they are showing extinction to the left-sided stimulus, a sign of neglect).

Testing bilateral parietal-temporal lobe functions

- Graphesthesia ("Close your eyes and hold out your hand. I'm going to write a number on your palm, and you tell me what it is". I like to use a pen with the ballpoint retracted. Make sure you and the patient are facing the same way so you'd both "read" the number in the same direction).
- Stereognosis ("Close your eyes and hold out your hand. I'm going to put something in it and I want you to tell me what it is". Use keys or a coin or a paperclip.)

Memory

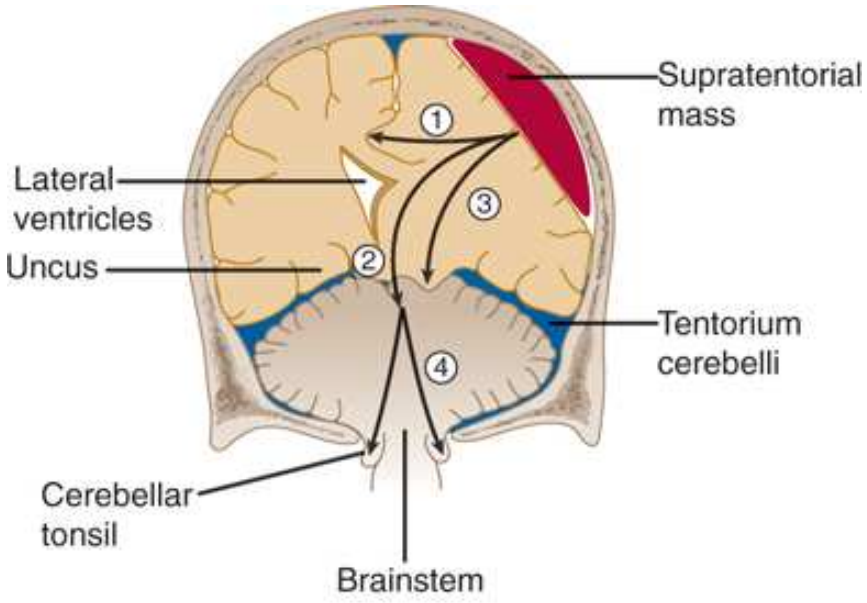
- Immediate recall ("I'm going to tell you 3 words, and I want you to say them back to me. Rose, umbrella, fear. Ok, now say them back to me. Now remember those words".)
- Delayed recall (assess after 5 minutes):

  “What were the words I asked you to remember?” If the response is not spontaneous, cue by categories ("One of the words was a flower/something you'd use outside/an emotion“) or cue by multiple choice ("Was it a lily, a rose, or a tulip?/An umbrella, a raincoat, or sunglasses?/Anger, sadness, or fear?“)

Memory

- Presidents/current events (Tailor to patient's interest. Someone not interested in politics might only know the name of the President. However, if they are sports fans, ask them for Super Bowl winners, World Series winners, etc; If they are gardeners, ask about their plants and some characteristics. Or their pets. If you, the examiner, do not know the answers to those, write down the patients' responses and look them up later.)

Herniation



(Reproduced with permission from Aminoll ML, Greenberg DA, Simon RP. Clinical News 1094, 6th edn, McGraw-Hill, 2005.)
Fig. 11-8 Accessed 07/01/2010

An expanding supra-tentorial mass lesion may cause brain tissue to be displaced into an adjacent intracranial compartment, resulting in (1) cingulate herniation under the falx, (2) downward transtentorial (central) herniation, (3) uncal herniation over the edge of the tentorium, or (4) cerebellar tonsillar herniation into the foramen magnum. Coma and ultimately death result when (2), (3), or (4) produces brainstem compression.

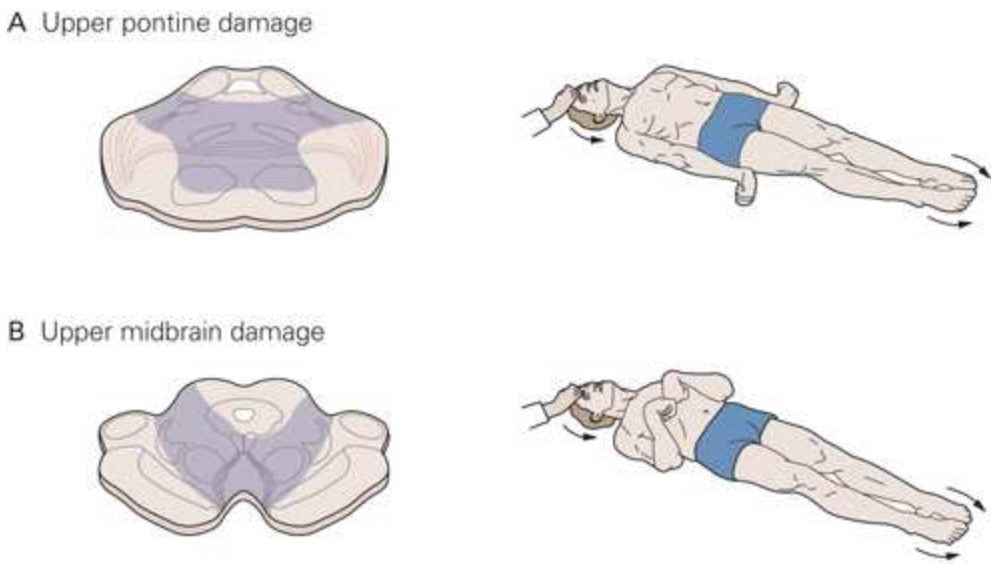
Posture

- Central monoparesis is distinguished from a peripheral paresis in that contraction of antagonist muscles is not impaired. For example, the hand may drop but grasp is unimpaired in a central lesion.
- Lesion of the internal capsule leads to contra-lateral hemiparesis.
- Lesion of the brainstem leads to ipsilateral cranial nerve injury and contra-lateral hemiparesis.

Posture

- Crossed paresis occurs at the level of decussation in the pyramids. For example, paresis in the right arm with paresis in the left leg.
- Spastic paraparesis occurs because of a lesion in the central spinal cord.
- Decerebrate posture is characterized by limb extension. Decorticate posture is characterized by flexion of the upper limbs with extension of the lower limbs.

Posture



Decerebrate

Decorticate

Source: Barrett KE, Barman SM, Boitano S, Brooks H: *Ganong's Review of Medical Physiology, 23rd Edition*: http://www.accessmedicine.com

(Modified from Kandel ER, Schwartz JH, Jessell TM (editors): *Principles of Neural Science,* 4th ed. McGraw-Hill, 2000.)
Fig. 16-8 Accessed 07/01/2010

Sudden fall without loss of consciousness

- **Drop attack**
- TIA (basilar artery) With dizziness, diplopia, ataxia, or paresthesia
- TIA (anterior cerebral artery)
- Posterior fossa tumor. Follows flexion of neck.
- Colloid cyst of 3rd ventricle. Position dependent headache
- **Catalepsy**
- Loss of muscle tone triggered by emotional stimuli. May occur with narcolepsy.

Disequilibrium or vertigo

- Disequilibrium (loss of balance) suggests a central process.
- Vertigo (spinning) as a peripheral process is associated with short course, precipitating factor, autonomic symptoms. Facial sensory changes and tinnitus may be described. Nystagmus is always present; unidirectional, never vertical.
- Vertigo as a central process may have no obvious precipitating factor. Autonomic symptoms are not prominent. Ataxia, sensory and motor symptoms. Nystagmus may be absent; if present, is uni- or bidirectional or may be may be vertical.

Vertigo

- No hearing loss and episodic vertigo that lasts seconds; positional; recurrent: benign positional vertigo (positive likelihood ratio, LR+, 11; LR-, 0.1)
- 10% dizzy patients have benign positional vertigo
- No hearing loss and persistent vertigo: vestibular neuronitis
- Hearing loss and persistent vertigo: labyrinthitis
- Hearing loss and episodic vertigo that lasts minutes to hours; tinnitus or aural fullness: Ménière's disease
- Panic attack common; lightheadedness as result of hyperventilation

Ataxia

- Cerebellar lesions are ipsilateral.
- Cerebellar ataxia. Unable to stand with feet together. Lurch. May see to and fro movement of trunk. “Drunken” gait. This is a lesion of the anterior lobe of Cerebellum. Lesions of the vermis involve gait disturbance alone.
- Cerebellar hemispheric lesions are associated with limb ataxia and nystagmus. The shoulder is lower on the affected side. The patient falls to the side of the lesion.
- Lesions of the 4th Ventricle are associated with truncal ataxia. There is no motor incoordination but the inability to stand upright without support.

Disequilbrium

	Vestibular	Cerebellar	Sensory
Vertigo	Present	May be present	Absent
Nystagmus	Present	Often present	Absent
Dysarthria	Absent	May be present	Absent
Limb ataxia	Absent	Usually present (one limb, unilateral, legs only, or all limbs)	Present (typically legs)
Stance	May be able to stand with feet together; typically worse with eyes closed	Unable to stand with feet together and eyes either open or closed	Often able to stand with feet together and eyes open but not with eyes closed (Romberg sign)
Vibratory and position sense	Normal	Normal	Impaired
Ankle reflexes	Normal	Normal	Depressed or absent

Disequilibrium

Site	Signs	Causes
Midline	Nystagmus, head and trunk titubation, gait ataxia	Tumor, multiple sclerosis
Superior vermis	Gait ataxia	Wernicke encephalopathy, alcoholic cerebellar degeneration, tumor, multiple sclerosis
Cerebellar hemisphere	Nystagmus, ipsilateral gaze paresis, dysarthria (especially left hemisphere lesion), ipsilateral hypotonia, ipsilateral limb ataxia, gait ataxia, falling to side of lesion	Infarction, hemorrhage, tumor, multiple sclerosis
Pancerebellar	Nystagmus, bilateral gaze paresis, dysarthria, bilateral hypotonia, bilateral limb ataxia, gait ataxia	Drug intoxications, hypothyroidism, hereditary cerebellar degeneration, paraneoplastic cerebellar degeneration, Wilson disease, infectious and parainfectious encephalomyelitis, Creutzfeldt-Jakob disease, multiple sclerosis

Gait disturbance

- Hemiparetic. Upper motor neuron disease. Spastic: affected arm flexed and affected leg extended; difficulty in flexing hip and knee as well as dorsiflexing ankle; paretic leg swings out.
- Paraperetic. Thoracic level lesion. Spastic: slow, stiff movements; hips adducted, legs extended. May cross one leg over the other when walking (“scissor” gait).
- Parkinsonian. Substantia nigra lesion. Difficulty initiating movement. General flexion of body. Rigid, with tremor. Short intitiating steps (“shuffle”) with rapid movement at end of sequence (“fenestration”). May see retropulsive steps if mild force exerted on patient.

Gait disturbance

- Proprioceptive difficulty. Sway with eyes closed. Wide based stance. Slap ground with feet (“tabetic”gait).
- Incoordination of voluntary movements is associated with lesions of the dentate nucleus of the cerebellum.

Testing of gait and stance

- Observe the patient stand and walk.
- An abnormality of stance or gait may be the most prominent or only neurologic abnormality, as in certain cases of cerebellar or frontal lobe disorder;
- An impairment of posture and highly automatic adaptive movements in walking may provide the most definite diagnostic clues in the early stages of diseases such as Parkinson disease.

Testing of gait and stance

- The cerebellum is required for precise control and coordinated movements. Functions to modify acts initiated by the motor cortex and basal nuclei.
- Vestibulo-cerebellar and Spino-cerebellar output affect the descending motor system at the brainstem level (red nucleus, CN motor nuclei and vestibular nuclei); they affect the motor act while it is in progress.
- Cerebro-cerebellar inputs information about a command about to be executed so it can modulate motor command information before execution of the command.

Testing of gait and stance

- Having the patient walk tandem or on the sides of the soles may bring out a lack of balance or dystonic postures in the hands and trunk.
- Hopping or standing on one foot may also betray a lack of balance or weakness,
- Standing with feet together and eyes closed will bring out a disequilibrium that is due to deep sensory loss (Romberg test).

Testing of gait and stance

- Walking on heels and toes not possible if cerebellar lesion. May see broad based gait.
- Lack of coordination (ataxia) is usually due to ipsilateral cerebellar damage
- Inability to stand with feet together and with eyes open indicates cerebellar damage. May see to and fro movement of trunk. “Drunken” gait.
- Inability to stand with feet together with eyes closed indicates decreased proprioceptive sense.
- Inability to stand upright without support but without motor incoordination indicates a lesion of the 4th ventricle.

Testing of gait and stance

- Problems with initiating or slowing of movement are a result of damage to upper motor neurons or the basal ganglia
- Hemiparetic gait. Upper motor neuron disease. Spastic: affected arm flexed and affected leg extended; difficulty in flexing hip and knee as well as dorsiflexing ankle; paretic leg swings out.
- Paraperetic gait. Thoracic level lesion. Spastic: slow, stiff movements; hips adducted, legs extended. May cross one leg over the other when walking (“scissor” gait).

Testing of gait and stance

- Parkinsonian gait. Substantia nigra lesion. Difficulty initiating movement. General flexion of body. Rigid, with tremor. Short intitiating steps (“shuffle”) with rapid movement at end of sequence (“fenestration”). May see retropulsive steps if mild force exerted on patient.
- Proprioceptive difficulty. Sway with eyes closed. Wide based stance. Slap ground with feet (“tabetic”gait).

Tremor

- Physiologic tremor is discrete, asymptomatic. May occur when holding heavy object (isometric). May be exaggerated with stress.
- Essential tremor is familial. 60% autosomal dominant. Involves hands, head, voice, trunk (in decreasing frequency). May be postural. Improved by alcohol.
- Intention tremor reflects cerebellar dysfunction. Postural tremor and head/neck tremor may be seen when patient standing.

Tremor

- Resting tremor seen in nigrostriatal or cerebello-spinothalamic tract lesions (e.g., Parkinson's). Usually proximal.
- Neuropathic tremor may be proximal or distal.

Nystagmus

- Loss of tonic vestibular discharge on side leads to imbalance (nystagmus and vertigo).
- Peripheral lesions (nerve or receptor destruction) result in no brainstem input. Fast component beats away from diseased ear.
- Central lesions produce vertical nystagmus.

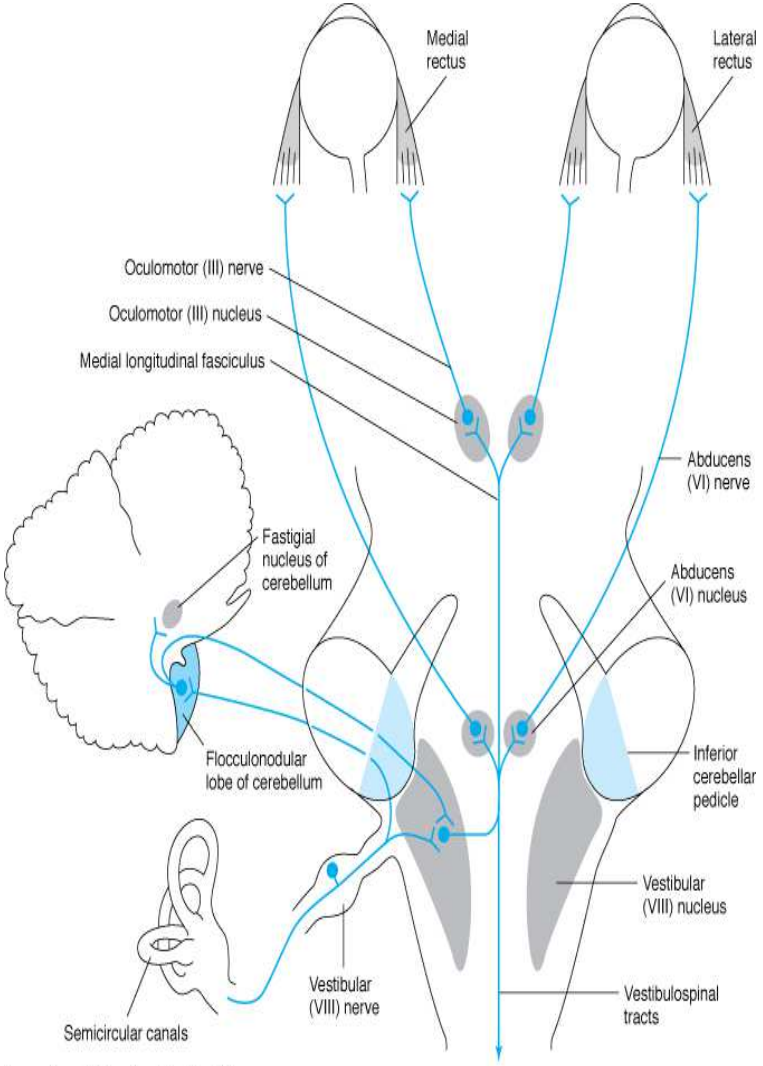


Fig. 3-1 Accessed 07/01/2010

Central lesions

- Horizontal plane: Tendency to fall to ipsilateral side. Diminished caloric sensation on inpsilateral side. Nystagmus to ipsilateral side.
- Saggital plane: Tendency to fall forward/ backward. Elevator sensation. Nystagmus is vertical.
- Frontal plane: Tendency to fall sideways (latero-pulsion). Nystagmus is vertical and to side of lesion.
- Retraction nystagmus is associated with bilateral dorsal midbrain lesion. Vertical up and downbeat nystgmaus is present in brain stem lesion.

Spino-cerebellar and cerebro-cerebellar injury

- Affect the most precise movements of the extremities
- Asynergy
- Dysdiadochkinesia
- Dysmetria
- Decomposition
- Hypotonia and pendular patellar tendon reflex

- It is difficult to separate vestibulo-cerebellar injury from injury to the nuclei.

Cranial nerve lesions

- Complete oculomotor palsy is associated with loss of conjugate gaze. Best seen when staring straight ahead. The unaffected eye is straight; the affected eye is pulled laterally.
- Trochlear palsy will show the affected eye up and medially while the unaffected eye is straight.
- Abducens palsy will show the affected eye pulled medially while the unaffected eye is straight.

Cranial nerve lesions

- Intranuclear ophthalmoplegia involves a lesion of the medial longitudinal fasciculus. Manifest on lateral gaze (contra-lateral eye does not respond).
- Upward gaze lesions may be pontine or supra-tentorial.
- Facial nerve lesions are associated with paresis of frontalis muscle, paresis at the corner of the mouth, paresis of the platysma, lagging lid closure.

Long tract signs

- In the brain stem, long tract signs are contralateral to the lesion. Cranial nerve signs are ipsilateral to the lesion.
- Therefore, a brain stem lesion may be localized to side of the cranial nerve. (Ipsilateral cranial nerve and contralateral long tract signs.)
- At spinal cord level, loss of pain and temperature is on side opposite of lesion. Long tract signs are not all on one side.

Motor system injury

- UPPER MOTOR NEURONS:
- Babinski positive
- Spasticity
- With time, hyperactive reflexes, leading to clonus
- Absent cremaster, abdominal reflexes
- LOWER MOTOR NEURONS:
- Flaccidity
- Absent reflexes, muscle atrophy
- Fasciculations and fibrillation

Other clues

- L'Hermitte sign: ectopic impulse generation with cervical flexion (demyelination of cervical cord)
- Symptoms worsen with heat or exercise (conduction block in demyelinated nerve)
- Recurrent dysphagia or dysarthria with exercise or fatigue (Myasthenia Gravis)
- Slowly advancing visual defects with luminous edges (Migraine)
- Constant pain in the ear without evidence of middle ear disease suggests cancer of the pharynx.

Testing of cranial nerves

- The function of the cranial nerves must generally be investigated more fully in patients who have neurologic symptoms than in those who do not.
- If one suspects a lesion in the anterior fossa, the sense of smell should be tested in each nostril; then it should be determined whether odors can be discriminated.
- Weak, familiar odors are used for testing CN I.
- (The distinct odor emanating from an alcohol wipe pad can be distinguished at 10cm.)
- Strong odors are irritants and detected by CN V.

Testing of cranial nerves

- Blindness in one eye: damage to eye or to CN II
- Visual fields should be outlined by confrontation testing, in some cases by testing each eye separately.
- (The examiner is 8-12 inches away from the patient, eye to eye.)
- If any abnormality is suspected, it should be checked on a perimeter and scotomata sought by computerized perimetry.

Testing of cranial nerves

- Bilateral hemianopsia: medial damage at chiasm
- Unilateral loss of nasal visual fields: lateral damage at chiasm
- Homonoymous hemianopsia: damage to optic tract approaching lateral geniculate ganglion, damage to the lateral geniculate ganglion, damage to optic raditation, or damage to visual cortex

Testing of cranial nerves

- Superior quadrant hemianopsia: temporal lobe damage (lateral optic radiation and Meyer loop)
- Inferior quadrant hemianopsia: parietal lobe damage
- Visual loss with macular sparing: primary visual cortex
- Visual loss with superior quadrant anopsia: below calcarine sulcus
- Visual loss with inferior quadrant anopsia: above calcarine sulcus

Testing of cranial nerves

- Pupil size and reactivity to light while in a dim ambient light (direct, consensual, and during convergence), the position of the eyelids (CN III), and the range of ocular movements (CN III, IV, VI) should next be observed.
- Asymmetrical pupil: CNIII or sympathetic dysfunction (Horner's syndrome due to lateral medullary damage)
- Pupils of unequal size may be congenital. Acute appearance may be medical emergency (Horner's syndrome due to dissecting carotid aneurysm or medial temporal lobe herniation)

Testing of cranial nerves

- Afferent pupillary defect: both pupils constrict if healthy eye is stimulated with light, but no pupillary constriction either direct or consensual if damaged eye stimulated by light (CN II)
- Unilateral, large, unreactive pupil: undamaged eye constricts with light stimulation of undamaged as well as damaged eye, but no pupillary constriction either direct or consensual in damaged eye (CN II)
- If a pupil does not constrict as a response to light but does constrict with focus on near object: Argyll-Robertson pupil (syphilis)

Testing of cranial nerves

- If neither eye responds to light, midbrain lesion
- If pupils fixed, dilated: midbrain lesion
- If pupils pinpoint: pontine damage or narcotic use
- Papilledema indicates increased intracranial pressure (manifest on CN II)

Testing of cranial nerves

- Drooping eyelid (ptosis): CN III or sympathetic dysfunction (Horner's syndrome)
- Convergence relies on intact medial rectus bilaterally (CN III)
- Accomodation relies on thickening of the lens
- Increasing depth of focus a result of miosis (pupillary constriction)

Testing of cranial nerves

- Double vision: CN III, IV, VI, or medial longitudinal fasciculus
- Eye moves down and out, pupil dilates, eyelid droops (CN III)
- Eye unable to direct gaze down and in (CN IV)
- Eye unable to abduct (CN VI)

Testing of cranial nerves

- Sensation over the face is tested with a pin and wisp of cotton. There is no overlap in sensory distribution of trigeminal nerve branches
- The presence or absence of the corneal reflexes (direct and consensually) may be determined. (CN V_1 provides sensation to the cornea; CN VII controls lid closure). It cannot be faked.

Testing of cranial nerves

- Facial movements should be observed as the patient speaks and smiles, for a slight weakness may be more evident in these circumstances than on movements to command.
- Paralysis of upper and lower face: ipsilateral lower motor neuron lesion (CN VII) or peripheral nerve lesion (Bell's palsy)
- Unilateral weakness of bite: lower motor neuron lesion; an upper motor neuron lesion produces minimal effect on bite due to bilateral cortical control
- If lower facial weakness only, contralateral upper motor neuron lesion

Testing of cranial nerves

- If facial weakness with loss of hearing and balance disturbance, lesion is peripheral and at the internal acoustic meatus
- If facial weakness with paralysis of ipsilateral lateral gaze, there is a dorsomedial pontine lesion
- Loss of hearing (CN VIII auditory branch).
- The auditory meati and tympanic membranes should be inspected with an otoscope.

Testing of cranial nerves

- If there is a concern as to hearing deficit, a vibrating high-frequency (512 Hz) tuning fork held at the vertex of the skull may be used to lateralize hearing loss (Weber). Then it is held next to the ear and on the mastoid to distinguish middle-ear (conductive) from neural deafness (Rinné).
- Audiograms and other special tests of auditory and vestibular function are needed if there is any suspicion of disease of the eighth nerve or the cochlear and labyrinthine end organs.

Nystagmus

- Loss of tonic vestibular discharge on side leads to imbalance (nystagmus and vertigo).
- Peripheral lesions (nerve or receptor destruction) result in no brainstem input. Fast component beats away from diseased ear.
- Central lesions produce vertical nystagmus.

Nystagmus

- Horizontal plane: Tendency to fall to ipsilateral side. Diminished caloric sensation on inpsilateral side. Nystagmus to ipsilateral side.
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- Retraction nystagmus is associated with bilateral dorsal midbrain lesion. Vertical up and downbeat nystgmaus is present in brain stem lesion.

Testing of cranial nerves

- Dysarthria (or dysphagia) implies motor dysfunction. It may be upper or motor lower neuron damage of CN IX, CN X, or represent cerebellar involvement
- The jaw jerk and the snout, buccal, and sucking reflexes should be sought, particularly if there is a question of dysphagia, dysarthria, or dysphonia.
- Gag reflex tests CN IX (sensory) and CN X (motor)
- Voluntary pharyngeal elevation and elicited reflexes are meaningful if there is a difference on the two sides; bilateral absence of the gag reflex is seldom significant.

Testing of cranial nerves

- The vocal cords must be inspected in cases of suspected medullary or vagus nerve disease, especially when there is hoarseness.
- Have the patient shrug shoulders against resistance as well as turn the head against resistance.
- Difficulty turning head to contralateral side with concomitant ipsilateral shoulder loop (CN XI)
- Tongue deviation (CN XII) is to the weak side. If it is an upper motor neuron lesion, the deviation is contralateral; if a lower motor neuron lesion, ipsilateral

Testing of cranial nerves

- Inspection of the tongue, both protruded and at rest, is helpful; atrophy and fasciculations may be seen and weakness detected. Slight deviation of the protruded tongue as a solitary finding can usually be disregarded, but a major deviation represents a deficit in action of the hypoglossal nerve and muscle on that side.
- The pronunciation of words should be noted.

Cortico-nulcear tract assymetries

- Innervation to the cranial nerve motor nuclei are mainly bilateral with these exceptions:
- Facial nucleus – the part that innervates the lower part of the face gets only contralateral input
- Hypoglossal nucleus – the neurons that innervate the genioglossus muscle only get contralateral input
- Nucleus Ambiguous – neurons that innervate the soft palate and uvula get only contralateral input
- Spinal Accessory – mainly ipsilateral input

Testing of motor function

- As a general rule, muscles essential in maintaining posture have bilateral innervation and are not useful for determining lateralized weakness.
- Principally uncrossed innervation is noted of dorsiflexors of the wrist, extensors of the elbow, dorsiflexors of the great toe, and flexors of the lower leg at the knee.
- Observations of the speed and strength of movements and of muscle bulk, tone, and coordination are considered in the context of the state of tendon reflexes.

Testing of motor function

- Flaccid muscle tone associated with lower motor neuron lesion (may be an acute upper motor neuron lesion)
- Lower motor neuron damage involves damage to the motor neuron innervating muscle. May see atrophy.
- Damage is ipsilateral to affected side of body

Testing of motor function

- Upper motor neuron damage involves the corticospinal tracts (as well as rubrospinal, vestibulospinal, reticulospinal tracts) or cortical control of these tracts
- Distal extremity weakness with loss of fine control and hyperreflexia is well as spastic paralysis are seen with upper motor neuron lesions.
- Increased tone that fades at the end of rigidity (as in opening a clasp knife) indicates an upper motor neuron lesion
- Ankle clonus as well as a Babinski reflex are also associated with upper motor neuron lesions.

Testing of motor function

- In the brain stem, long tract signs are contralateral to the lesion. Cranial nerve signs are ipsilateral to the lesion.
- Therefore, a brain stem lesion may be localized to side of the cranial nerve. (Ipsilateral cranial nerve and contralateral long tract signs.)
- At spinal cord level, loss of pain and temperature is on side opposite of lesion. Long tract signs are not all on one side.

Testing of motor function

- Increased tone throughout movement (as if a lead pipe) indicates basal ganglia disease
- Cog wheel rigidity produces ratchet-like catches and releases and is seen in Parkinson's disease
- Hypotonia with pendular reflexes indicates cerebellar darmage

Testing of motor function

- The strength of the legs can be similarly tested with the patient prone and the legs flexed at hips and knees, and observing downward drift of the weakened leg. In the supine position at rest, weakness due to an upper motor neuron lesion causes external rotation of the hip.
- It is essential to have the limbs exposed and to inspect them for atrophy and fasciculations.

Testing of motor function

- Fasciculations are visible twitches of individual motor units and indicate damage to lower motor neuron cell bodies or motor nerve roots. Fibrillations are not visible, refer to individual fibers
- Resting tremor indicates contralateral basal ganglia damage
- Intention tremor (most apparent at end of movement) is a sign of ipsilateral cerebellar damage.
- Athetosis or choreiform movements indicate basal ganglia dysfunction
- Ballismus indicates contralateral basal ganglia damage (subthalamic nucleus)

Testing of motor function

- Abnormalities of movement and posture as well as tremors may be exposed by observing the limbs at rest and in motion.
- This is accomplished by watching the patient maintain the arms outstretched in the prone and supine positions;
- accomplish simple tasks such as buttoning clothes, opening a safety pin, or handling common tools;

Testing of motor function

- perform simple tasks such as alternately touching his nose and the examiner's finger; make rapid alternating movements that necessitate sudden acceleration and deceleration and changes in direction, such as tapping one hand on the other while alternating pronation and supination of the forearm; rapidly touch the thumb to each fingertip.

Testing of motor function

- Estimates of the strength of leg muscles with the patient in bed are often unreliable; there may seem to be little or no weakness even though the patient cannot arise from a chair or from a kneeling position without help.
- Running the heel down the front of the shin; alternately touching the examiner's finger with the toe and the opposite knee with the heel; and rhythmically tapping the heel on the shin are the only tests of coordination that need be carried out in bed.

Anatomical landmarks

Cord Segments	Vertebral Bodies	Spinous Processes
C8	Lower C6 and upper C7	C6
T6	Lower T3 and upper T4	T3
T12	T9	T8
L5	T11	T10
S	T12 and L1	T12 and L1

Motor function

Muscle	Main Root	Peripheral Nerve	Main Action
Diaphragm	C3, C4	Phrenic	Respiration
Supraspinatus	C5	Suprascapular	Abduction of arm
Infraspinatus	C5	Suprascapular	External rotation of arm at shoulder
Deltoid	C5	Axillary	Abduction of arm
Biceps	C5, C6	Musculocutaneous	Flexion of forearm
Brachioradialis	C5, C6	Radial	Flexion of forearm
Extensor carpi radialis longus	C6, C7	Radial	Wrist extension
Flexor carpi radialis	C6, C7	Median	Wrist flexion
Extensor carpi ulnaris	C7	Radial	Wrist extension
Extensor digitorum	C7	Radial	Finger extension
Triceps	C8	Radial	Extension of forearm

Motor function

Muscle	Main Root	Peripheral Nerve	Main Action
Flexor carpi ulnaris	C8	Ulnar	Wrist extension
Abductor pollicis brevis	T1	Median	Abduction of thumb
Opponens pollicis	T1	Median	Opposition of thumb
First dorsal interosseous	T1	Ulnar	Abduction of index finger
Abductor digiti minimi	T1	Ulnar	Abduction of little finger
Iliopsoas	L2, L3	Femoral	Hip flexion
Quadriceps femoris	L3, L4	Femoral	Knee extension
Adductors	L2, L3, L4	Obturator	Adduction of thigh
Gluteus maximus	L5, S1, S2	Inferior gluteal	Hip extension
Gluteus medius and minimus, tensor fasciae latae	L4, L5, S1	Superior gluteal	Hip abduction

Motor function

Muscle	Main Root	Peripheral Nerve	Main Action
Hamstrings	L5, S1	Sciatic	Knee flexion
Tibialis anterior	L4, L5	Peroneal	Dorsiflexion of ankle
Extensor hallucis longus	L5	Peroneal	Dorsiflexion of great toe
Extensor digitorum longus	L5, S1	Peroneal	Dorsiflexion of toes
Extensor digitorum brevis	S1	Peroneal	Dorsiflexion of toes
Peronei	L5, S1	Peroneal	Eversion of foot
Tibialis posterior	L4	Tibial	Inversion of foot
Gastrocnemius	S1, S2	Tibial	Plantar flexion of ankle
Soleus	S1, S2	Tibial	Plantar flexion of ankle

Testing of motor function

- The inability to maintain a supinated arm against gravity is a sensitive indicator of a focal lesion. The weak arm, tiring first, soon begins to sag.
- Drfiting down of extended arm with pronation of the hand and flexion of the forearm (with hypertonia) is seen with an upper motor neuron lesion.
- Drifiting down of extended arm without pronation of the hand (with hypotonia) is associated with cerebellar damage
- If extended arm drifts up, there is a lack of proprioceptive information (sensory damage)

Testing of reflexes

- Deep tendon reflexes are designed to test sensory limb, motor limb, and upper motor neuron modulation of muscle stretch reflex
- Testing of the biceps, triceps, supinator-brachioradialis, patellar, Achilles, and plantar reflexes permits an adequate sampling of reflex activity of the spinal cord.
- Elicitation of tendon reflexes requires that the involved muscles be relaxed; underactive or barely elicitable reflexes can be facilitated by voluntary contraction of other muscles (Jendrassik maneuver).

Testing of reflexes

- Dorsiflexion of the large toe and fanning of the other toes is the well-known Babinski sign .
- The plantar response poses some difficulty because several different reflex responses can be evoked by stimulating the sole of the foot along its outer border from heel to toes.
- (1) the normal quick, high-level avoidance response that causes the foot and leg to withdraw;
- (2) the pathologic slower, spinal flexor protective reflex (flexion of knee and hip and dorsiflexion of toes and foot, "triple flexion”).

Testing of reflexes

- (3) plantar grasp reflexes;
- (4) support reactions in infants.
- Avoidance and withdrawal responses interfere with the interpretation of the Babinski sign and can sometimes be overcome by utilizing the several alternative stimuli (e.g., squeezing the calf or Achilles tendon, flicking the fourth toe, downward scraping of the shin, lifting the straight leg, and others) or by having the patient scrape his own sole.

Testing of reflexes

- An absence of the superficial cutaneous reflexes (abdominal, cremasteric) are useful ancillary tests for detecting corticospinal lesions particularly when unilateral.

Testing of sensory function

- Subjective. Requires patient's cooperation.
- Reserved for the end of the examination.
- If the findings are to be reliable, the tests should not be prolonged for more than a few minutes.
- Each test should be explained briefly; too much discussion of these tests with a meticulous, introspective patient might encourage the reporting of meaningless minor variations of stimulus intensity.

Testing of sensory function

- It is not necessary to examine all areas of the skin surface. A quick survey of the face, neck, arms, trunk, and legs with a pin takes only a few seconds.
- Usually one is seeking differences between the two sides of the body. It is better to ask whether stimuli on opposite sides of the body feel the same than to ask if they feel different.
- Moving the stimulus from an area of diminished sensation into a normal area is recommended because it enhances the perception of a difference.

Testing of sensory function

- A level below which sensation is lost, or a zone of relative or absolute analgesia (loss of pain sensibility) or anesthesia (loss of touch sensibility) is an important finding.
- Regions of sensory deficit can then be tested more carefully and mapped out.
- Dermatomal sensory loss indicates peripheral, segmental damage
- “Stocking and glove” distribution of sensory loss suggests metabolic peripheral neuropathy

Testing of sensory function

- Inability to locate point of stimulus with eyes closed or inability to identify common items by touch and manipulation or decreased two point discrimination suggests parietal lobe damage

Testing of vibratory function

- The sense of vibration may be tested by comparing the thresholds at which the patient and examiner lose perception at comparable bony prominences.
- Record the number of seconds for which the examiner appreciates vibration at the malleolus, toe, or finger after the patient reports that the fork has stopped buzzing.
- The finding of a zone of heightened sensation ("hyperesthesia“) calls attention to a disturbance of superficial sensation.
- Variations over several examinations may be unimportant.

Spinal cord lesions

- A small central lesion in the cord can affect the decussating fibers of the spinothalamic tract from both sides without affecting other ascending or descending tracts. As a result, these lesions can produce dissociated sensory abnormalities with loss of pain and temperature sensibility in appropriate dermatomes but with preserved vibration and position sense (e.g., in syringomyelia).

Spinal cord lesions

- A large central lesion in the cord involves, in addition to the pain and temperature pathways, portions of adjacent tracts, adjacent gray matter, or both. Thus, there can be lower motor neuron weakness in the segments involved, together with upper-motor-neuron dysfunction and, in some cases, loss of vibratory and position sense at levels below the lesion.

Spinal cord lesions

- A dorsal column lesion affects the dorsal columns, leaving other parts of the spinal cord intact. Proprioceptive and vibratory sensation are involved, but other functions are normal (e.g., tabes dorsalis)
- An irregular peripheral lesion (e.g., penetrating wound or compression of the cord) involves long pathways and gray matter; functions below the level of the lesion are abolished.

Spinal cord lesions

- Complete hemisection of the cord produces a Brown–Séquard syndrome. Lesions outside the cord (extramedullary lesions) may affect the function of the cord itself as a result of direct mechanical injury or secondary ischemic injury resulting from the compromise of the vascular structures or vasospasm.

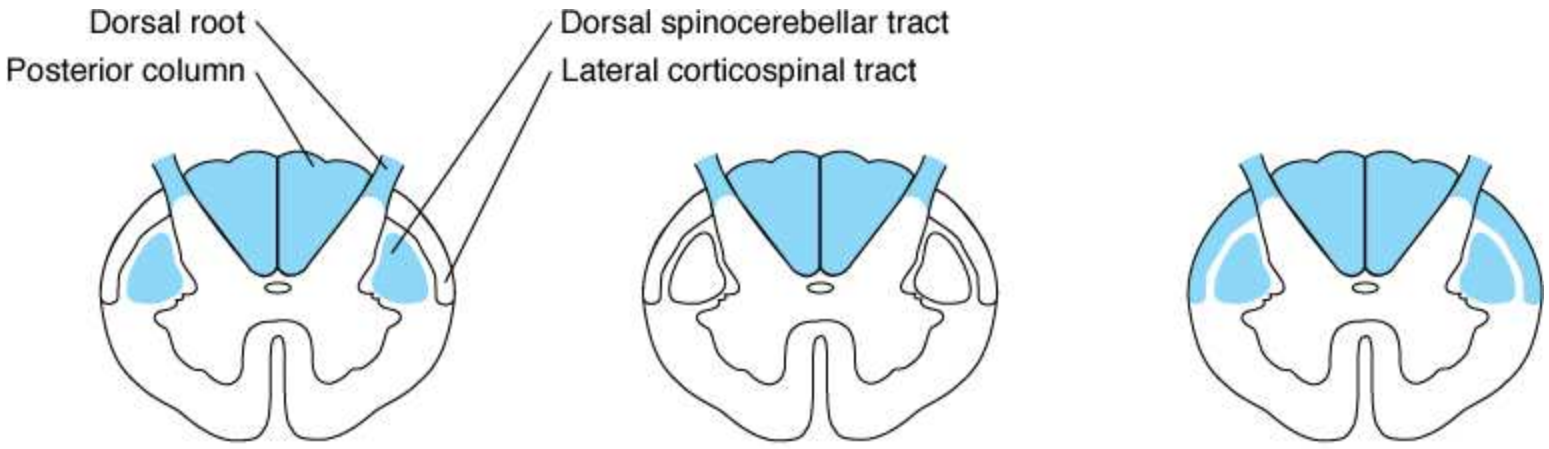
Spinal cord lesions

- A tumor of the dorsal root (such as a neurofibroma or schwannoma) involves the first-order sensory neurons of a segment and can produce pain as well as sensory loss. Deep tendon reflexes at the appropriate level may be lost because of damage to Ia fibers.

Spinal cord lesions

- A tumor of the meninges (primary or metastatic) or the bone (extramedullary masses) may compress the spinal cord against a vertebra, causing dysfunction of ascending and descending fiber systems.
- Herniated intervertebral disks can also compress the spinal cord.

Sensory ataxia



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