1

STROKE

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INTRODUCTION

🕮 DEFINITION OF STROKE

- Sudden focal (sometimes global) neurologic deficit secondary to occlusion or rupture of blood vessels supplying the brain
- Symptoms > 24 hours = stroke
- Symptoms < 24 hours = transient ischemic attack (TIA)
- Reversible ischemic neurologic deficit (RIND) = (this term is no longer used)

EPIDEMIOLOGY

- Stroke, after heart disease and cancer, is the third leading cause of death in the United States.
- The American Heart Association (AHA) estimates 600,000 strokes annually; 500,000 new cases, and 100,000 recurrent cases. (2000 AHA Heart and Stroke Statistical Update)
- Nearly four million stroke survivors in the United States
- 46% decline in cerebral infarcts and hemorrhages from 1950–1954 period to 1975–1979 period (Broderick, 1993)
  - Decline attributed to better management of blood pressure (BP), heart disease, decrease in cigarette smoking, etc.
- Incidence increases 17% from 1975–1979 period to 1980–1984 period (attributed to increased use of CT scan)
- There has been no change in the incidence of aneurysmal rupture
- Mortality from strokes has been steadily declining since 1950s
  - A sharp decline noted in the 1970s, possibly related to improved diagnosis (Dx) and treatment (Tx) of hypertension (HTN)
  - Improved Dx by modern diagnostic imaging tools (CT and MRI), may also have created a statistical decline in calculated mortality as smaller (less severe) strokes were identified (Sacco, 1995).

🕮 RISK FACTORS (Stewart, 1999)

Nonmodifiable:

- Age—single most important risk factor for stroke worldwide; after age 55, incidence increases for both males and females
- Risk more than doubles each decade after age 55
- Sex (male > female)
- Race (African Americans 2× > whites > Asians)
- Family history (Hx) of stroke

Modifiable (treatable) risk factors:

- Hypertension—probably the most important modifiable risk factor for both ischemic and hemorrhagic stroke; increases risk by sevenfold
- History of TIA/prior stroke (~ 5% of patients with TIA will develop a completed stroke within 1 month if untreated)
- Heart disease (Dz.)
  - Congestive heart failure (CHF) and coronary artery disease (CAD): increases risk by twofold
  - Valvular heart Dz. and arrhythmias atrial fibrillation (A. Fib.)—increases risk of embolic stroke
    - A. Fib.: fivefold increase risk (Ryder, 1999)
- Diabetes—twofold increase in risk; unfortunately, good blood sugar control has not been shown to alter the risk of stroke
- Cigarette smoking
- Carotid stenosis (and carotid bruit); risk of stroke decreases with carotid endarterectomy (CEA) on selected symptomatic patients (> 70% stenosis)
- ETOH abuse/cocaine use
- High-dose estrogens (birth control pills)—considerable increase in risk when linked to cigarette smoking
- Systemic diseases associated with *hypercoagulable states*
  - Elevated RBC count, hematocrit, fibrinogen
  - Protein S and C deficiency
  - Sickle-cell anemia
  - Cancer
- Hyperlipidemia—several clinical trials have shown a reduction in stroke with use of cholesterol reducing agents (~ 30% reduction risk of stroke with use of HMG-CoA reductase inhibitors)
- Migraine headaches
- Sleep apnea
- Patent Foramen Ovale

[Obesity/sedentary life style (no clear relationship with increased risk of stroke)]

BASIC NEUROANATOMICAL REVIEW OF THE MAJOR VESSELS INVOLVED IN STROKE

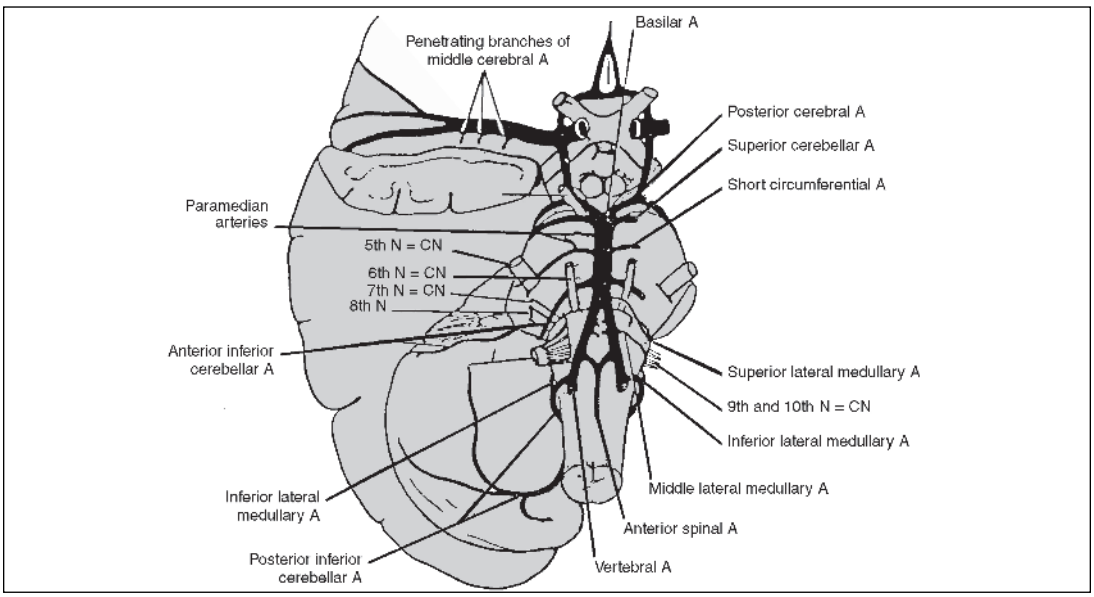


FIGURE 1–1 The principle vessels of the vertebrobasilar system in relation to the brainstem. A = artery; CN = cranial nerve

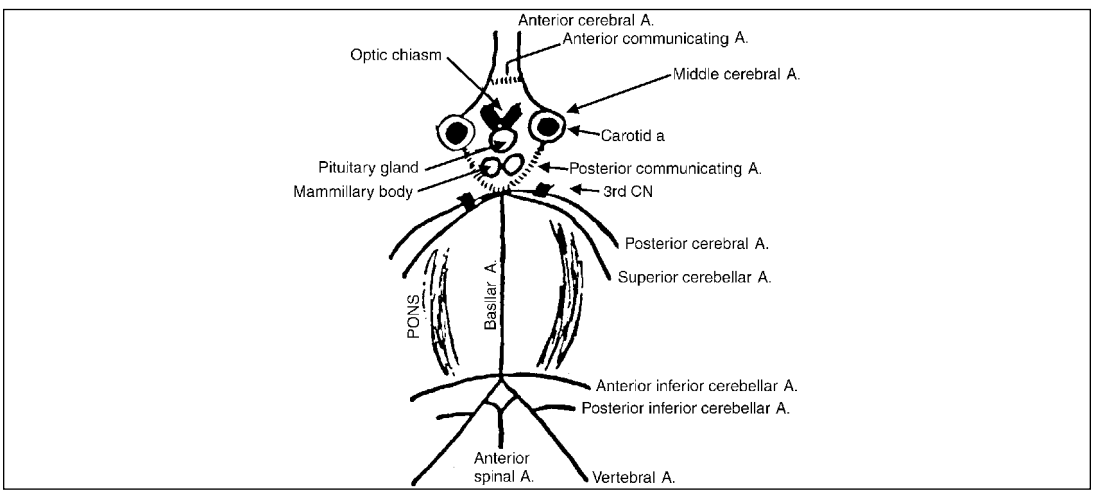


FIGURE 1–2 The Circle of Willis is a ferocious spider that lives in the brain. His name is Willis! Note that he has a nose, angry eyebrows, two suckers, eyes that look outward, a crew cut, antennae, a fuzzy beard, 8 legs, a belly that, according to your point of view, is either thin (basilar artery) or fat (the pons, which lies from one end of the basilar artery to the other), two feelers on his rear legs, and male genitalia. In Fig. 1–2 the brain is seen from below, so the carotid arteries are seen in cross section. (Reprinted with permission from Goldberg S. Clinical Neuroanatomy Made Ridiculously Simple. Miami: Medmaster Inc.; 1997.)

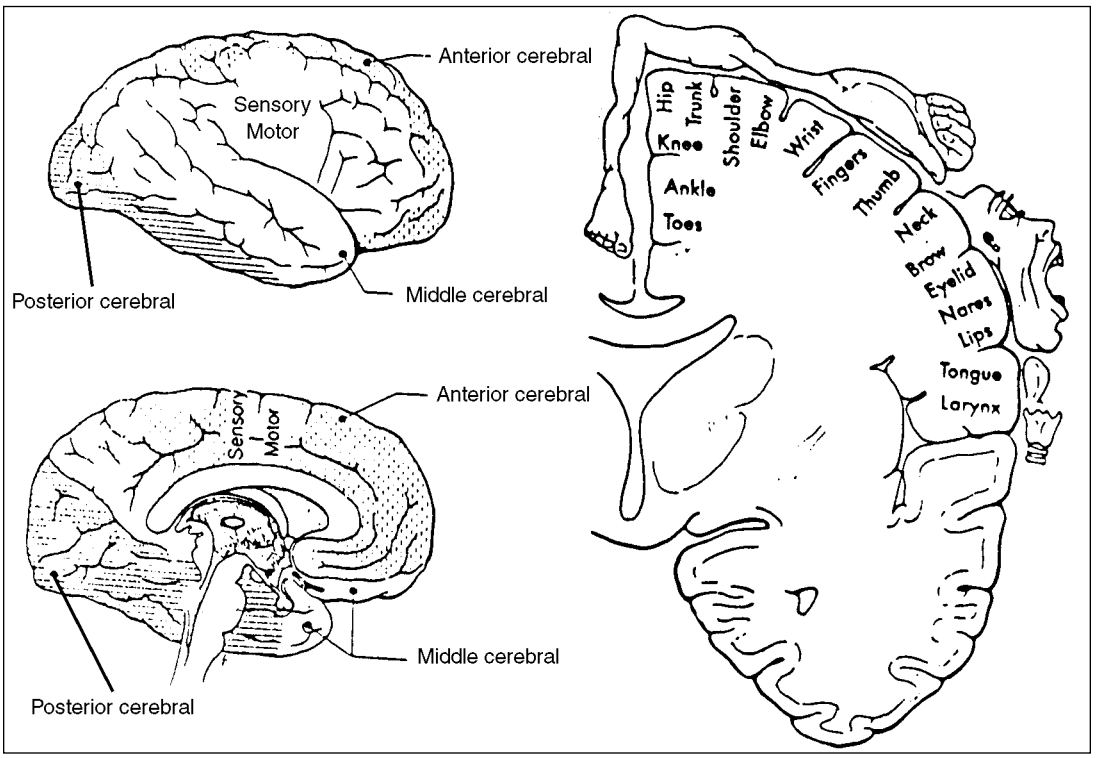


FIGURE 1–3 Major vascular supply to brain and functional diagram of motor strip. It is evident that lower-limb motor strip is in anterior cerebral artery distribution while upper-extremity motor strip is supplied by middle cerebral artery. (Reprinted with permission from Rosen P. Emergency Medicine–Stroke 3rd ed. St. Louis: Mosby; 1992.)

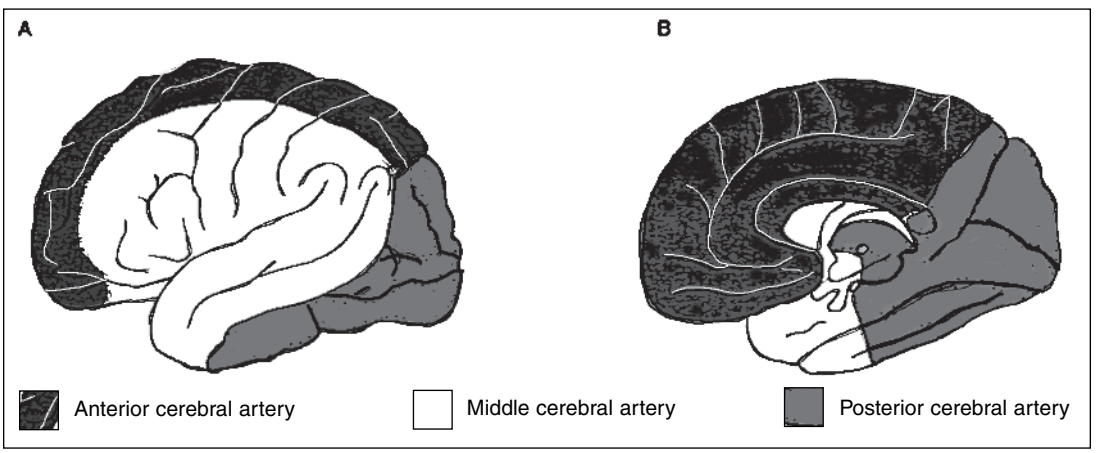


FIGURE 1–4 The three cerebral arteries' cortical territories. **A.** Lateral aspect of the hemisphere. **B.** Medial and inferior aspects of the hemisphere.

1. Most of the lateral aspect of the hemisphere is mainly supplied by the middle cerebral artery.
2. The anterior cerebral artery supplies the medial aspect of the hemisphere from the lamina terminalis to the cuneus.
3. The posterior cerebral artery supplies the posterior inferior surface of the temporal lobe and the visual cortex.

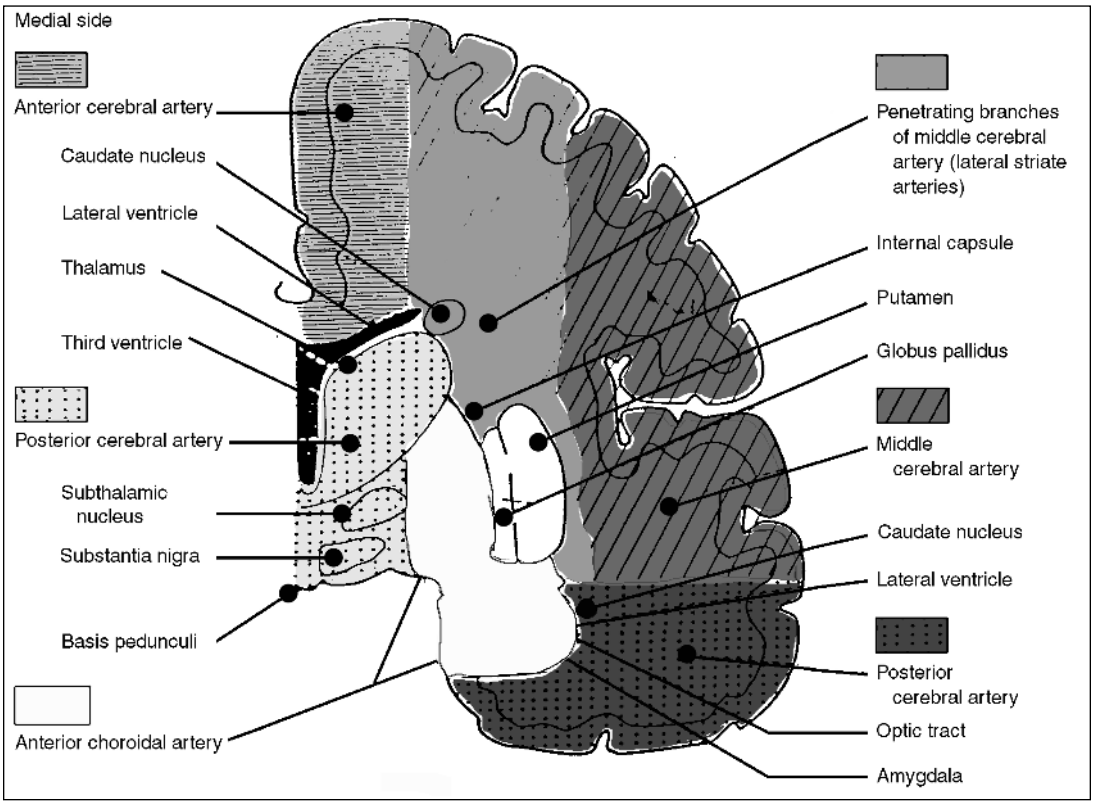


FIGURE 1–5 Major vascular territories are shown in this schematic drawing of a coronal section through the cerebral hemisphere at the level of the thalamus and the internal capsule.

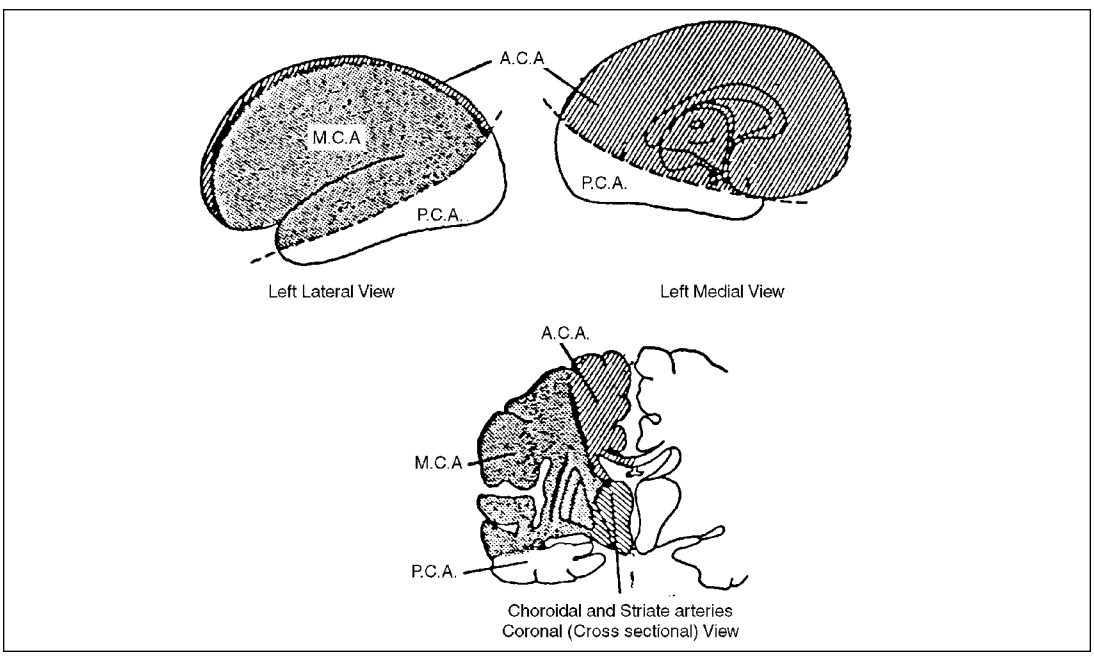


FIGURE 1–6 The cerebral blood circulation. MCA, middle cerebral artery; ACA, anterior cerebral artery; PCA, posterior cerebral artery. (Reprinted with permission from Goldberg S. Clinical Neuroanatomy Made Ridiculously Simple. Miami: Medmaster Inc.; 1997.)

🕮 TYPES OF STROKE

TABLE 1–1

	Ischemic 85%			Hemorrhagic 15%	
Type	Thrombotic	Embolic	Lacunar	Intracerebral (hypertensive) hemorrhage	Subarachnoid hemorrhage (ruptured aneurysms)
Frequency (%)	35	30	20	10	5
Factors associated with onset	Occurs during sleep	Occurs while awake		In 90% of cases occurs when the patient is calm and unstressed Blacks > whites	Occurs during activity (often strenuous activity)
Major causes/ etiology	Perfusion failure distal to site of severe stenosis or occlusion of major vessels	Due mainly to cardiac source	Small lesions seen mainly: –putamen –pons –thalamus –caudate –internal capsule/corona radiata	Hypertension	From ruptured aneurysms and vascular malformations
Presentation	Slowly (gradually) progressive deficit	Sudden, immediate deficit (seizures may occur)	Abrupt or gradual onset	Gradual onset (over minutes to days) or sudden onset of local neurologic deficits	Sudden onset
Link with TIA	50% with preceding TIA (50% occurring same vascular territory of preceding TIA)	TIA less common than in thrombotic 11% with preceding TIA	23% with preceding TIA	8% with preceding TIA	7% with preceding TIA

ISCHEMIC STROKES

Thrombotic (large artery thrombosis): 35% of all strokes

- Usually occurs during sleep (patient often awakens unaware of deficits)
- May have "stuttering," intermittent progression of neurologic deficits or be slowly progressive (over 24–48 hours)
- Profound loss of consciousness rare, except when area of infarction is large or when brainstem involved

- Neurologic deficit varies according to cerebral territory affected
- Perfusion failure distal to site of severe stenosis or occlusion of major vessels
- Emboli from incompletely thrombosed artery may precipitate an abrupt deficit. May have embolism from extracranial arteries affected by stenosis or ulcer

Embolic: 30% of all strokes

- Usually occurs during waking hours
- Deficit is immediate
- Seizures may occur at onset of stroke
- Cortical signs more frequent
- Most often embolus plugs one of the branches of the middle cerebral artery. An embolus may cause severe neurologic deficits that are temporary; symptoms resolve as the embolus fragments
- Presence of atrial fibrillation, history of recent myocardial infarction (MI) and occurrence of emboli to other regions of the body support Dx of cerebral embolism
- Suggested by history and by hemorrhagic infarction on CT (seen in 30% of patients with embolism) also by large low-density zone on CT encompassing entire territory of major cerebral artery or its main divisions
- Most commonly due to cardiac source: mural thrombi and platelet aggregates
- Chronic atrial fibrillation is the most common cause. Seen with myocardial infarction, cardiac aneurysm, cardiomyopathy, atrial myxoma, valvular heart disease (rheumatic, bacterial endocarditis, calcific aortic stenosis, mitral valve prolapse), sick sinus syndrome
- 75% of cardiogenic emboli go to brain

Lacunar infarction: 20% of all strokes

📖 Lacunes are small (less than 15 mm) infarcts seen in the putamen, pons, thalamus, caudate, and internal capsule
- Due to occlusive arteriolar or small artery disease (occlusion of deep penetrating branches of large vessels)
- Occlusion occurs in small arteries of 50—200 μm in diameter
- Strong correlation with hypertension (up to 81%); also associated with micro-atheroma, microembolism or rarely arteritis
- Onset may be abrupt or gradual; up to 30% develop slowly over or up to 36 hours
- CT shows lesion in about 2/3 of cases (MRI may be more sensitive)
- Relatively pure syndromes often (motor, sensory)—discussed below
- Absence of higher cortical function involvement (language, praxis, non-dominant hemisphere syndrome, vision)

Neuroanatomic Location of Ischemic Stroke (Adams, 1997)

1. Anterior Circulation

INTERNAL CAROTID ARTERY (ICA): (most variable syndrome): Occlusion occurs most frequently in the first part of the ICA immediately beyond the carotid bifurcation. ICA occlusions often asymptomatic (30–40% of cases) (Fig. 1–7)

- Ocular infarction: (embolic occlusion of either retinal branch or central retinal artery)
- Transient monocular blindness *(amaurosis fugax)*: the ICA nourishes the optic nerve and retina as well as the brain; transient monocular blindness occurs prior to onset of stroke in approximately 25% of cases of internal carotid occlusion. Central retinal artery ischemia is very rare because of collateral supply

- Cerebral infarction: Presentation of complete ICA occlusion variable, from no symptoms (if good collateral circulation exists) to severe, massive infarction on ACA and MCA distribution. Failure of distal perfusion of internal carotid artery may involve all or part of the middle cerebral territory and, when the anterior communicating artery is small, the ipsilateral anterior cerebral artery. Contralateral motor and/or sensory symptoms present.

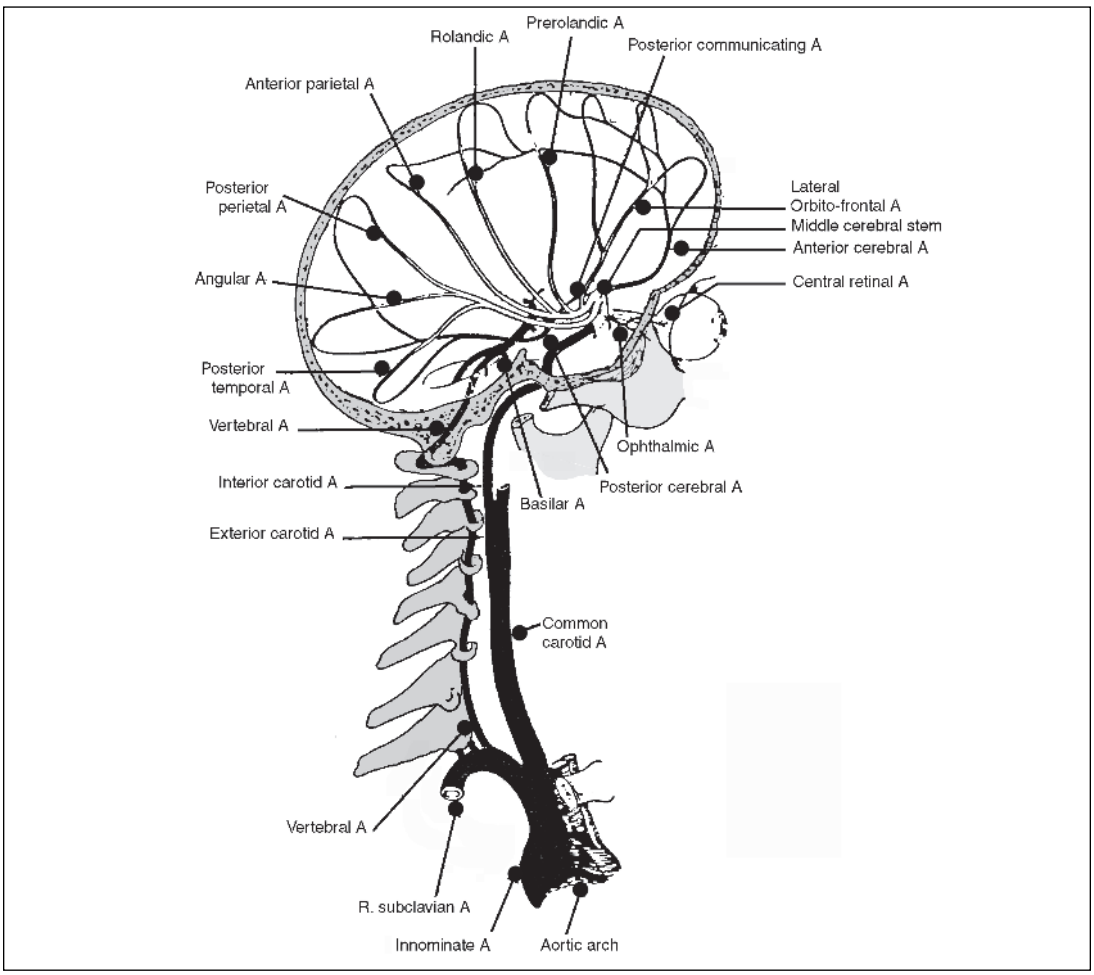


FIGURE 1–7 Arterial anatomy of major vessels on the right side carrying blood from the heart to the brain. Note location and course of the internal carotid artery.

MIDDLE CEREBRAL ARTERY (MCA): Occlusion occurs at stem of middle cerebral or at one of the two divisions of the artery in the sylvian sulcus. (Figure 1–8)

Superior Division

📖 Most common cause of occlusion of superior division of MCA is an embolus (superior division of MCA supplies rolandic and prerolandic areas)

Presentation:

- Sensory and motor deficits on contralateral face and arm > leg
- Head and eyes deviated toward side of infarct
- With left-side lesion (dominant hemisphere)—global aphasia initially, then turns into Broca's aphasia (motor speech disorder)

- Right side lesion (nondominant hemisphere)—deficits on spatial perception, hemineglect, constructional apraxia, dressing apraxia
- Muscle tone usually decreased initially and gradually increases over days or weeks to spasticity
- Transient loss of consciousness is uncommon

Inferior division (lateral temporal and inferior parietal lobes)

Presentation:
- With lesion on either side—superior quadrantanopia or homonymous hemianopsia
- Left side lesion—Wernicke's aphasia
- Right side lesion—left visual neglect

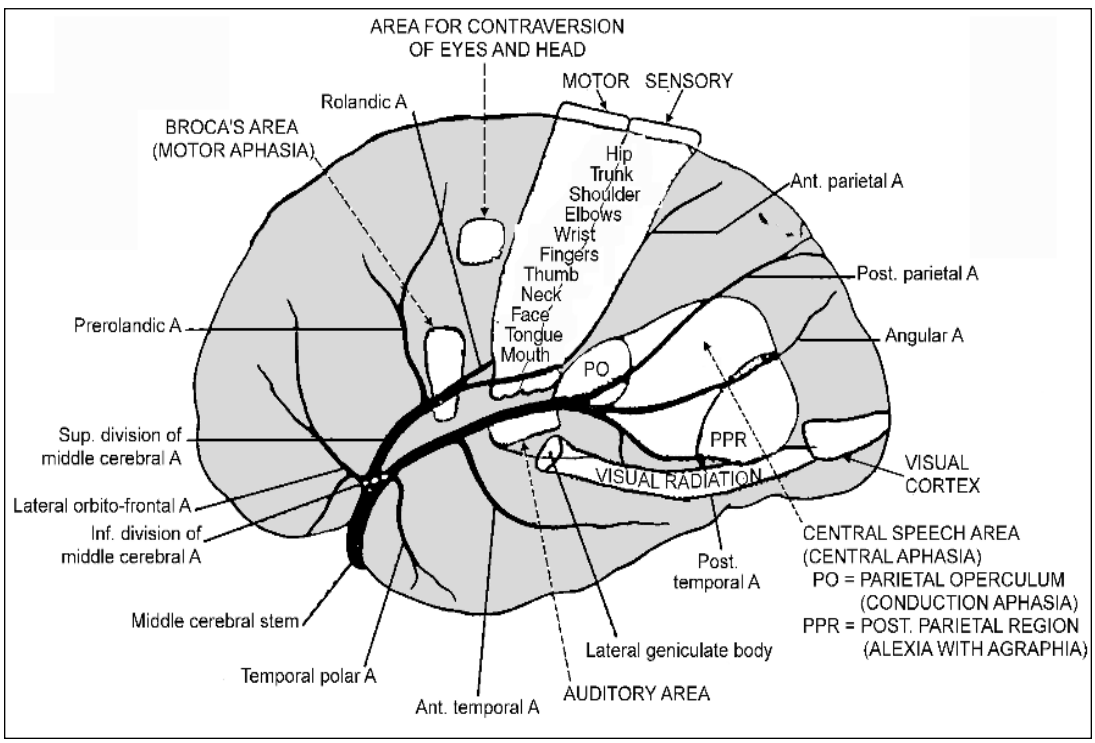


FIGURE 1–8 The distribution of the middle cerebral artery on the lateral aspect of the cerebral hemisphere. Principal regions of cerebral localization are noted.

ANTERIOR CEREBRAL ARTERY (ACA) (Figure 1–9):

- If occlusion is at the stem of the anterior cerebral artery proximal to its connection with the anterior communicating artery ⇒ it is usually well tolerated because adequate collateral circulation comes from the artery of the opposite side
- 🕮 If both anterior cerebral arteries arise from one stem ⇒ major disturbances occur with infarction occurring at the medial aspects of both cerebral hemispheres resulting in aphasia, paraplegia, incontinence and frontal lobe/personality dysfunction
- Occlusion of one anterior cerebral artery distal to anterior communicating artery results in:
  - 🕮 Contralateral weakness and sensory loss, affecting mainly distal contralateral leg (foot/leg more affected than thigh)
  - Mild or no involvement of upper extremity

– Head and eyes may be deviated toward side of lesion acutely
– Urinary incontinence with contralateral grasp reflex and paratonic rigidity may be present
– May produce transcortical motor aphasia if left side is affected
– 🕮 Disturbances in gait and stance = gait apraxia

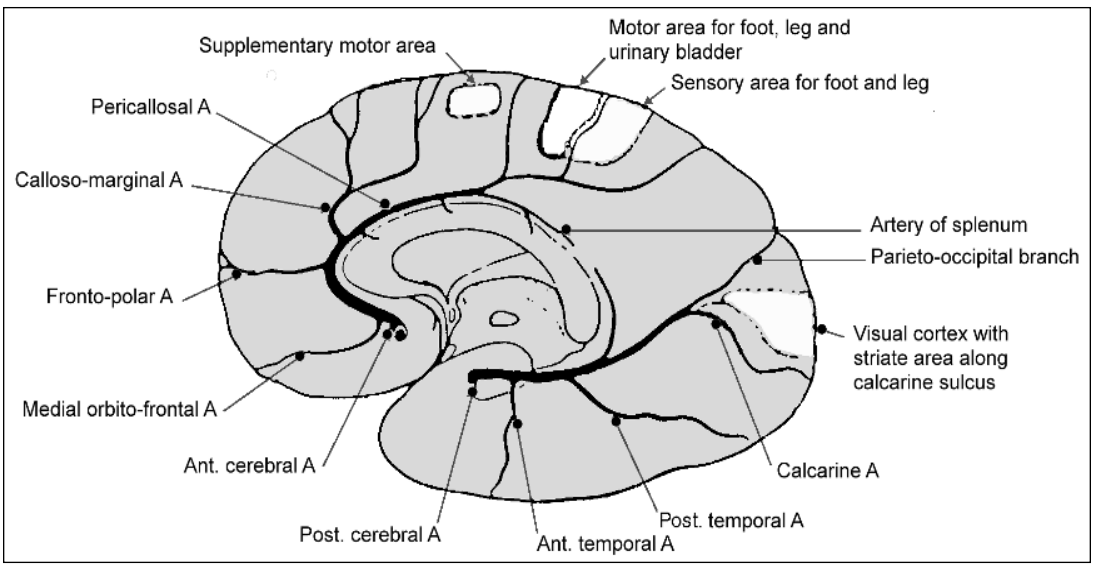


FIGURE 1–9 The distribution of the anterior cerebral artery on the medial aspect of the cerebral hemisphere, showing principal regions of cerebral localization.

2. Posterior Circulation: Vertebrobasilar Arteries & Posterior Cerebral Arteries

POSTERIOR CEREBRAL ARTERY (PCA):
Occlusion of PCA can produce a variety of clinical effects because both the upper brainstem and the inferior parts of the temporal lobe and the medial parts of the occipital lobe are supplied by it.

Particular area of occlusion varies for PCA because anatomy varies

- 70% of times both PCAs arise from basilar artery; connected to internal carotids through posterior communicating artery
- 20%–25%: one PCA comes from basilar; one PCA comes from ICA
- 5%–10%: both PCA arise from carotids

Clinical presentation includes:

- Visual field cuts (including cortical blindness when bilateral)
- May have prosopagnosia (can't read faces)
- palinopsia (abnormal recurring visual imagery)
- alexia (can't read)
- transcortical sensory aphasia (loss of power to comprehend written or spoken words; patient can repeat)
- 🕮 Structures supplied by the interpeduncular branches of the PCA include the oculomotor cranial nerve (CN 3) and trochlear (CN 4) nuclei and nerves

- 🕮 Clinical syndromes caused by the occlusion of these branches include oculomotor palsy with contralateral hemiplegia = Weber's syndrome (discussed below) and palsies of vertical gaze (trochlear nerve palsy)

VERTEBROBASILAR SYSTEM:

- Vertebral and basilar arteries: supply midbrain, pons, medulla, cerebellum, and posterior and ventral aspects of the cerebral hemispheres (through the PCAs.)
- Vertebral arteries: branches of the subclavian; are the main arteries of the medulla. At the pontomedullary junction, the two vertebral arteries join to form the basilar artery, which supplies branches to the pons and midbrain. Cerebellum is supplied by posterior-inferior cerebellar artery (PICA) from vertebral arteries, and by anterior-inferior cerebellar artery (AICA) and superior cerebellar artery, from basilar artery
- Vertebrobasilar system involvement may present any combination of the following signs/symptoms: vertigo, nystagmus, abnormalities of motor function often bilateral. usually ipsilateral cranial nerve dysfunction
- Crossed signs: motor or sensory deficit on ipsilateral side of face and opposite side of body; ataxia, dysphagia, dysarthria

🕮 **Important:** There is *absence* of *cortical signs* (such as aphasias or cognitive deficits) that are characteristic of anterior circulation involvement

Syndromes of the Vertebrobasilar System

I. 🕮 Lateral Medullary (Wallenberg's) Syndrome

This syndrome is one of the most striking in neurology. It occurs due to occlusion of the following:

1. vertebral arteries (involved in 8 out of 10 cases)
2. posterior inferior cerebellar artery (PICA)
3. superior lateral medullary artery
4. middle lateral medullary artery
5. inferior lateral medullary artery

- **Wallenberg's syndrome** also known as lateral medullary syndrome, PICA syndrome, and vertebral artery syndrome.
  Signs and symptoms include the following:
  - Ipsilateral side
    - Horner's syndrome (ptosis, anhydrosis, and miosis)
    - decrease in pain and temperature sensation on the ipsilateral face
    - cerebellar signs such as ataxia on ipsilateral extremities (patient falls to side of lesion)
  - Contralateral side
    - Decreased pain and temperature on contralateral body
  - Dysphagia, dysarthria, hoarseness, paralysis of vocal cord
  - Vertigo; nausea and vomiting
  - Hiccups
  - Nystagmus, diplopia
    Note: No facial or extremity muscle weakness seen in this syndrome

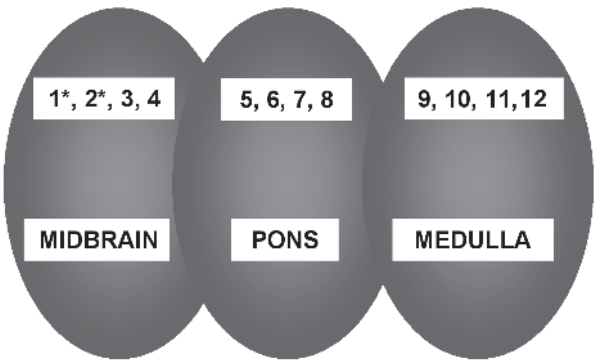
II. Benedikt's Syndrome (Red Nucleus/Tegmentum of Midbrain):

- Obstruction of interpeduncular branches of basilar or posterior cerebral artery or both
- Ipsilateral III nerve paralysis with mydriasis, contralateral hypesthesia (medial lemniscus), contralateral hyperkinesia (ataxia, tremor, chorea, athetosis) due to damage to red nucleus

III. Syndromes of the ParamedianArea (Medial Brainstem):

Paramedian area contains:
- Motor nuclei of CNs
- Cortico-spinal tract
- Medial lemniscus
- Cortico-bulbar tract

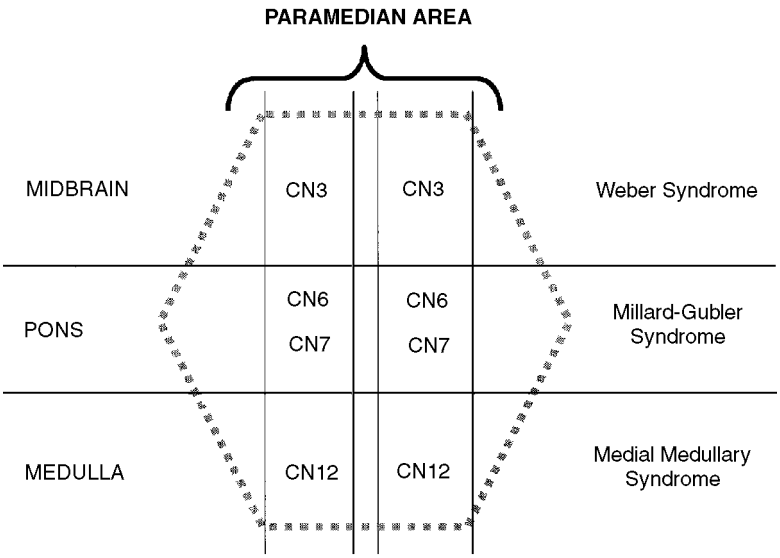
Signs/symptoms include:
- contralateral hemiparalysis
- ipsilateral CN paralysis



Location (grossly) of cranial nerve nuclei on brainstem
*📖 *NOTE: nucleus of CN 1 and CN 2 located in forebrain. Spinal division of CN 11 arises from ventral horn of cervical segments C1–C6.*

TABLE 1–2 Syndromes of the Paramedian Area (Medial Brainstem)

Weber Syndrome	Millard-Gubler Syndrome	Medial Medullary Syndrome "Another Lesion"
• Ipsilateral CN 3 palsy	• Ipsilateral CN 6 paralysis (often CN 7 also involved)	• Ipsilateral CN 12 palsy
• Contralateral hemiplegia	• Contralateral hemiplegia (extension into medial lemniscus is Foville's Syndrome with gaze palsy to side of lesion).	• Contralateral hemiplegia
	• Contralateral lemniscal (tactile sensation) sensory loss secondary to damage to medial lemniscus	• Contralateral lemniscal sensory loss



Gross depiction of the paramedian area of the brainstem and associated syndromes.

Weber Syndrome

(Base of midbrain): Obstruction of interpeduncular branches of posterior cerebral artery or posterior choroidal artery or both. Ipsilateral CN 3 cranial nerve paralysis, contralateral hemiplegia, contralateral Parkinson's signs, contralateral dystaxia (mild degree of ataxia).

Millard-Gubler Syndrome

(Base of pons): Obstruction of circumferential branches of basilar artery. Ipsilateral facial (CN 7) and abducens (CN 6) palsy, contralateral hemiplegia, analgesia, hypoesthesia.

- Extension to medial lemniscus = Raymond-Foville's Syndrome (with gaze palsy to side of lesion)

🕮 Medial Medullary Syndrome

Caused by an infarction of the medial medulla due to occlusion (usually atherothrombotic) of penetrating branches of the vertebral arteries (upper medulla) or anterior spinal artery (lower medulla and medullo-cervical junction).

- Rare; ratio of medial medullary infarct to lateral medullary infarct ~ 1–2 : 10
- Typical syndrome:
  - Ipsilateral hypoglossal palsy (with deviation toward the side of the lesion)
  - Contralateral hemiparesis
  - Contralateral lemniscal sensory loss (proprioception and position sense)

TABLE 1–3 Syndromes of the Brainstem

	Main Arteries	Medial Brain Stem Lesions (Paramedian area syndromes)	Lateral Brain Stem Lesions
Midbrain	PCA	Weber syndrome	
Pons	Basilar	Millard-Gubler syndrome	
Medulla	Vertebral (or anterior spinal artery)	Medial medullary syndrome	Wallenberg syndrome

IV. Basilar Artery Occlusion Syndrome

Occlusion may arise in several ways:

- atherosclerotic plaque in the basilar artery itself (usually lower third)
- occlusion of both vertebral arteries
- occlusion of one vertebral artery when it is the only one of adequate size

Note:

- Thrombosis usually only obstructs a branch of basilar artery rather than the trunk
- Emboli, if they get through the vertebral arteries, usually lodge in one of the posterior cerebral arteries or at the upper bifurcation of the basilar artery

May cause internuclear ophthalmoplegia, conjugate horizontal gaze palsy, ocular bobbing. Ptosis, nystagmus common but variable. May see palatal myoclonus, coma.

🕮 ***Locked-in syndrome:*** tetraparesis with patients only able to move eyes vertically or blink; patient remains fully conscious secondary to sparing of the reticular activating system; caused by bilateral lesions of the ventral pons (basilar artery occlusion). Some degree of paresis accompanies nearly all cases of basilar artery occlusion.

Neuroanatomic Location of Lacunar Infarction Syndromes

TABLE 1–4

Lacunar Syndrome	Anatomical Location
1. Pure motor hemiplegia –Weakness involves face, arm, and leg; no sensory deficits, aphasia or parietal signs	Posterior limb of internal capsule Corona radiata Pons
2. Pure sensory stroke	Thalamus (ventro-lateral) Parietal white matter Thalamocortical projections
3. 🕮 Dysarthria—clumsy hand syndrome	Basis pontis Internal capsule (anterior limb)
4. Sensorimotor stroke	Junction of internal capsule and thalamus
5. Ataxia and leg paralysis	Pons Midbrain Internal capsule Cerebellum Parietal white matter Coronal Radiata
6. Hemichorea-hemiballismus	Head of the caudate Thalamus Subthalamic nucleus

HEMORRHAGIC STROKES (see Table 1–1)

15% of all strokes may be secondary to hypertension, ruptured aneurysm, arteriovenous malformation (AVM), blood dyscrasias/bleeding disorders, anticoagulants, bleeding into tumors, angiopathies.

I. Hypertensive Intracerebral Hemorrhage

- Linked to chronic HTN (> one-third occur in normotensives)
- Sudden onset of headache, and/or loss of consciousness
- Vomiting at onset in 22%–44%
- Seizures occur in 10% of cases (first few days after onset)
- Nuchal rigidity common
- Sites: putamen, thalamus, pons, cerebellum; some from white matter
- Frequently extends to ventricular subarachnoid space
- Preceded by formation of "false" aneurysms (microaneurysms) of Charcot & Bouchard = arterial wall dilations 2° to HTN

Locations

1. **Putamen:** Most common; hemiplegia 2° to compression of adjacent internal capsule. Vomiting in ~ 50%; headache frequent but not invariable
   - Large hemorrhage: Stupor/coma + hemiplegia with deterioration in hours.
   - With smaller hemorrhages: Headache (HA) leading to aphasia, hemiplegia, eyes deviate away from paretic limbs
   - These symptoms, occurring over few minutes to one-half hour, are strongly suggestive of progressive intracerebral bleeding
2. **Thalamus:** Hemiplegia by compression of adjacent internal capsule; contralateral sensory deficits; aphasia present with lesions of the dominant side; contralateral hemineglect with involvement on the nondominant side. Ocular disturbances with extension of hemorrhage into subthalamus
3. **Pontine:** Deep coma results in a few minutes; total paralysis, small pupils (1 mm) that react to light; decerebrate rigidity → death occurs in few hours. Patient may survive if hemorrhage is small (smaller than 1 cm)
4. **Cerebellum:** Develops over several hours. Coma/loss of consciousness (LOC) unusual vomiting, occipital HA, vertigo, inability to sit, stand or walk, eyes deviate to opposite side (ipsilateral CN 6 palsy); dysarthria, dysphagia
5. **Lobar hemorrhage:** HA and vomiting. A study of 26 patients revealed:
   - 11 occipital: Dense homonymous hemianopsia and pain ipsilateral eye
   - 7 temporal: Partial hemianopsia/fluent aphasia/pain ear
   - 4 frontal: Contralateral hemiplegia (mainly the arm) and frontal headache
   - 3 parietal: Hemisensory deficit (contralateral)/anterior temporal HA

II. Subarachnoid Hemorrhage (SAH) (Ruptured Saccular Arterial Aneurysm)

- Saccular aneurysms = Berry aneurysms
- Arterial dilations found at bifurcations of larger arteries at base of brain (circle of Willis or major branches). (Fig. 1–10)
  📖 90%–95% of saccular aneurysms occur on the anterior part of the circle of Willis. Presumed to result from congenital medial and elastica defects vs hemodynamic forces causing focal destruction of internal elastic membrane at bifurcations. (Adams, 1997)
- Multiple in 20% of patients (either unilateral or bilateral)
- Other types of aneurysms: arteriosclerotic, mycotic, dissecting aneurysms, traumatic, neoplastic
- More likely to rupture if 10mm or larger (rupture may occur in smaller aneurysms)
- Rupture occurs usually when patient is active rather than during sleep (e.g., straining, coitus)
- Peak age for rupture = fifth and sixth decade

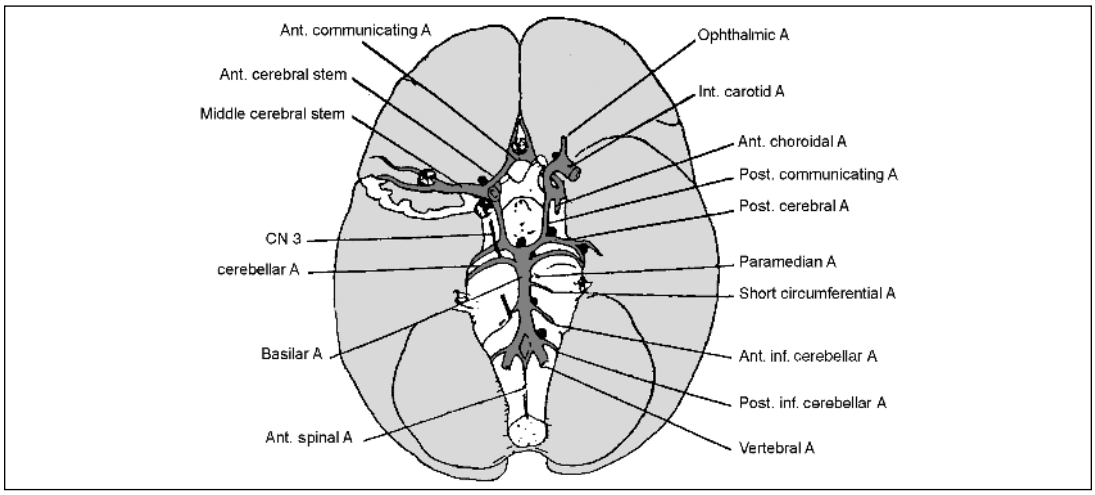


FIGURE 1–10 The principle sites of saccular aneurysms. The majority (approximately 90%) of aneurysms occur on the anterior half of the Circle of Willis.

Clinical Presentation of Saccular Aneurysms/SAH:
Symptoms due to aneurysms; presentation can be either:

1. None, usually asymptomatic prior to rupture. (intracranial aneurysms are common, found during 3%–5% of routine autopsies)
2. Compression of adjacent structures
   (e.g., Compression of oculomotor nerve (CN 3) with posterior communicating—internal carotid junction aneurysm or posterior communicating—posterior cerebral artery aneurysm)

   Signs of CN 3 involvement:
   - Deviation of ipsilateral eye to lateral side (lateral or divergent strabismus) because of unopposed action of lateral rectus muscle
   - Ptosis
   - Mydriasis (dilated pupil) and paralysis of accommodation due to interruption of parasympathetic fibers in the CN 3
3. Rupture of aneurysm producing subarachnoid hemorrhage with or without intracerebral hematoma
   - "Sentinel" HA prior to rupture in ~ 50% of patients
   - With subarachnoid hemorrhage, blood is irritating to the dura causing severe HA classically described as "worst headache of my life"
   - Sudden, transient loss of consciousness in 20%–45% at onset
   - May have CN 3 or CN 6 palsy (from direct pressure from the aneurysm vs. accumulation of an intracerebral hematoma vs early development of arterial spasm), hemiparesis, aphasia (dominant hemisphere), memory loss
   - Seizures: 4% at onset/25% overall
   - Mortality 25% during first 24 hours
   - Risk of rebleeding within one month 30%; 2.2% per year during first decade
   - Mortality from rebleeding in the early weeks after initial event: 50% to 60%
   - Vasospasm: common complication occurring in ~ 25% of cases; caused by the presence of blood breakdown products (vasoactive amines) on the subarachnoid space, acting in the adventitia of the arteries. Occurs 3–12 days after rupture (frequently ~ 7 days after rupture)

- Meds: nimodipine, a calcium channel blocker, is useful in the treatment of cerebral blood vessel spasm after subarachnoid hemorrhage (see Treatment section below)

III. Vascular Malformations/AVMs

- Consists of a tangle of dilated vessels that form an abnormal communication between the arterial and venous systems: an arteriovenous (AV) fistula
- Congenital lesions originating early in fetal life
- AVM composed of coiled mass of arteries and veins with displacement rather than invasion of normal brain tissue
- 🕮 AVMs are usually low-pressure systems; the larger the shunt, the lower the interior pressure. Thus, with these large dilated vessels there needs to be an occlusion distally to raise luminal pressures to cause hemorrhage
- Hemorrhage appears to be more common in smaller malformations, which is probably due to higher resistance and pressure within these lesions
- Patients are believed to have a 40%–50% risk of hemorrhage from AVM in life span
- Natural history of AVMs: bleeding rate per year = 2%–4%
- Rebleeding rate 6% first year post-hemorrhage
- Annual mortality rate: 1% (per year)
- First hemorrhage fatal in ~10% of these patients
- Bleeding commonly occurs between the ages of 20–40 years

Clinical Presentation of AVM Rupture:

- Hemorrhage: Majority of symptomatic patients present with hemorrhage. Cerebral hemorrhage first clinical manifestation in ~ 50% of cases; may be parenchymal (41%), subarachnoid (23%), or intraventricular (17%) (Brown et al., 1996)
- Seizures: presenting feature in 30% of cases
- Headaches: presenting complaint in 20% of cases; 10% (overall) with migraine-like headache

■ DIAGNOSTIC STUDIES

TABLE 1–5 Diagnostic Studies

	Infarct	**Hemorrhage**
CT	Focally decreases density (hypodense) = darker than normal **BLACK** Not seen immediately (unless there is a mass effect) May be seen after 24 hrs. (due to increase in edema); seen best within 3 to 4 days	Blood Hyperdense (radio-opaque) **WHITE** Seen immediately
MRI	Edema Fluid: high density **WHITE** Can be seen immediately as bright area on T_2	Blood Low signal density **BLACK** (on either T1 or T2)

1. CT Scan:

Major role in evaluating presence of blood (cerebral hemorrhage or hemorrhagic infarction), especially when anticoagulation is under consideration.

If intracranial (IC) hemorrhage suspected, *CT scan without contrast* is the study of choice
Why?: To avoid confusing blood with contrast, as both appear white on CT scan.

Cerebral Infarction:

- Regardless of stroke location or size, CT is often normal during the first few hours after brain infarction
- Infarcted area appears as hypodense (black) lesion usually after 24–48 hours after the stroke (occasionally positive scans at 3–6 hours ↔ subtle CT changes may be seen early with large infarcts, such as obscuration of gray-white matter junction, sulcal effacement, or early hypodensity)
- Hypodensity initially mild and poorly defined; edema better seen third or fourth day as a well-defined hypodense area
- CT with contrast: IV contrast provides no brain enhancement in day 1 or 2, as it must await enough damage to blood brain barrier; more evident in 1–2 weeks. Changes disappear in 2 to 3 months
- Some studies suggest worse prognosis for patients receiving IV contrast early
- Hemorrhage can occur within an infarcted area, where it will appear as a hyperdense mass within the hypodense edema of the infarct

Hemorrhagic Infarct:

- High density (white) lesion seen immediately in ~100% cases. Proved totally reliable in hemorrhages 1.0 cm or more. Demonstration of clot rupture into the ventricular system (32% in one series) not as ominous as once thought

Subarachnoid Hemorrhage:

- Positive scan in 90% when CT performed within 4–5 days (may be demonstrated for only 8–10 days). SAH can really be visualized only in the acute stage, when blood is denser (whiter) than the cerebrospinal fluid (CSF)
- Appears as hyperdense (or isodense) area on CT scan—look for blood in the basal cisterns or increase density in the region around the brainstem. May sometimes localize aneurysm based upon hematoma or uneven distribution of blood in cisterns.
- Once diagnosis of SAH has been established, angiography is generally performed to localize and define the anatomic details of the aneurysm and to determine if others aneurysms exist

2. MRI Scan:

More sensitive than CT scan in detecting small infarcts (including lacunar) and posterior cranial fossa infarcts (because images are not degraded by bone artifacts); ischemic edema detected earlier than with CT—within a few hours of onset of infarct.

Cerebral Infarction:

- Early, increased (white) signal intensity on T2 weighted images, more pronounced at 24 hours to 7 days (Tl may show mildly decreased signal)
- Chronically (21 days or more), decreased Tl and T2 weighted signals

Intracerebral Hemorrhage:

- Acute hemorrhage: decreased (black) signal or isointense on Tl and T2 weighted images
- Edema surrounding hemorrhage appears as decreased intensity on Tl weighted image; increased (white) signal on T2 images
- As hemorrhage ages, it develops increased signal on Tl and T2 images

Subarachnoid or Intraventricular Hemorrhage:

- Acutely, low signal (black) on Tl and T2 images

MRI/MRA:
Detects most aneurysms on the basal vessels; insufficient sensitivity to replace conventional angiography

Lacunes:
CT scan documents most supratentorial lacunar infarctions, and MRI successfully documents both supratentorial and infratentorial infarctions when lacunes are 0.5 cm or greater

3. **Carotid Ultrasound:**
   Real time B-mode imaging; direct doppler examination. Screening test for carotid stenosis; identification of ulcerative plaques less certain. Useful in following patients for progression of stenosis.

4. **Angiography:**
   Conventional angiography, intravenous digital subtraction angiography (DSA), and intra-arterial digital subtraction angiography
   - DSA studies: safer, may be performed as outpatient
   - Evaluation of extracranial and intracranial circulation
   - Valuable tool for diagnosis of aneurysms, vascular malformations, arterial dissections, narrowed or occluded vessels, and angiitis
   - Complications: occur in 2% to 12%; complications include aortic or carotid artery dissection, embolic stroke, vascular spasm, and occlusion
   - Morbidity associated with procedure: 2.5%
   - Carotid and vertebral angiography—only certain means of demonstrating an aneurysm—positive in 85% of patients with "clinical" SAH

MRA:
Can reliably detect extracranial carotid artery stenosis; may be useful in evaluating patency of large cervical and basal vessels

5. **Lumbar Puncture:**
   Used to detect blood in CSF; primarily in subarachnoid hemorrhage when CT not available or, occasionally, when CT is negative and there is high clinical suspicion

6. **Transesophageal Echo:**
   Transesophageal echocardiographic findings can be helpful for detecting potential cardiac sources of embolism in patients with clinical risks for cardioembolism or unexplained stroke.

■ TREATMENT

IMMEDIATE MANAGEMENT (Ferri, 1998; Rosen, 1992; Stewart, 1999)

- **Respiratory support**/ABCs of critical care
- **Airway obstruction** can occur with paralysis of throat, tongue, or mouth muscles and pooling of saliva. Stroke patients with recurrent seizures are at increased risk of airway obstruction. Aspiration of vomiting is a concern in hemorrhagic strokes (increased association of vomiting at onset). Breathing abnormalities (central) occasionally seen in patients with severe strokes
- **Control of blood pressure** (see following)

- **Indications for emergent CT scan**
  - Because the clinical picture of hemorrhagic and ischemic stroke may overlap, CT scan without contrast is needed in most cases to definitively differentiate between the two
  - Determine if patient is a candidate for emergent thrombolytic therapy
  - Impaired level of consciousness/coma: If there is acute deterioration of level of consciousness, evaluate for hematoma/acute hydrocephalus; treatment: emergency surgery
  - Coagulopathy present (i.e., rule out (R/O) hemorrhage)
  - Fever and concern regarding brain ulcers or meningitis
- **Seizure management (see below)**
- **Obtain blood sugar levels** immediately
  - Hypoglycemia → bolus 50% dextrose
  - Hyperglycemia: shown to potentiate severity of brain ischemia in animal studies.
  - Insulin if blood sugar > 300 mg/dl
- **Control of Intracranial Pressure (ICP) (see below)**
- **Fever**: potentially damaging to the ischemic brain.
  - Antipyretics (acetaminophen) should be given early while the source of fever is being ascertained
- **Intravenous Fluid**: Normal Saline Solution (NSS) or Ringer's lactate; avoid hypotonic solutions or excessive loading because they may worsen brain edema
- **Keep patient NPO** if at risk of aspiration

Blood Pressure Management:

Management of blood pressure after acute ischemic and hemorrhagic stroke is controversial. Many patients have HTN after ischemic or hemorrhagic strokes but few require emergency treatment. Elevations in blood pressure usually resolve without antihypertensive medications during the first few days after stroke. (Biller and Bruno, 1997)

Antihypertensive treatment can lower cerebral perfusion and lead to worsening of stroke. The response of stroke patients to antihypertensive medications can be exaggerated.

Current treatment recommendations are based on the type of stroke, ischemic vs. hemorrhagic

Ischemic Stroke:

TABLE 1–6 American Heart Association Recommendations for HTN Management in ***Ischemic Stroke***

Nonthrombolytic candidates	Treat if :	SBP >220
		DBP >120
		or MAP >120
Thrombolytic candidates	Treat if :	SBP >185
(before thrombolytic treatment give)		DBP >110

- IV labetalol and enalapril are favored antihypertensive agents.

Hemorrhagic Strokes:

Treatment of increased BP during hemorrhagic strokes is controversial. Usual recommendation is to treat at lower levels of blood pressure than for ischemic strokes because of concerns of rebleeding and extension of bleeding.

- Frequent practice is to treat BP if: SBP > 180, DBP > 105
- Agent of choice: IV labetalol (labetalol does not cause cerebral vasodilation, which could worsen increased ICP)

Seizure Management:

- Recurrent seizures: potentially life-threatening complication of stroke (see Stroke Rehabilitation)
- Seizures can substantially worsen elevated ICP
- Benzodiazepines = first-line agents for treating seizures
- IV lorazepam or diazepam
- If seizures don't respond to IV benzodiazepines, treat with long acting anticonvulsants:
  Phenytoin – 18 mg/kg
  Also fosphenytoin – 17 mg/kg
  Phenobarbital – 1000 mg or 20 mg/kg

Intracranial Pressure Management:

- Increased ICP reduces cerebral blood perfusion
- Cerebral perfusion calculated by subtracting ICP from mean arterial pressure (MAP). It should remain > 60 mm Hg to ensure cerebral blood flow
- Fever, hyperglycemia, hyponatremia, and seizures can worsen cerebral edema by increasing ICP

Keep ICP <20 mmHg

Management of ICP:

- **Correction of factors exacerbating increased ICP**
  - Hypercarbia
  - Hypoxia
  - Hyperthermia
  - Acidosis
  - Hypotension
  - Hypovolemia
- **Positional**
  - Avoid flat, supine position; elevate head of bed 30°
  - Avoid head and neck positions compressing jugular veins
- **Medical Therapy**
  - Intubation and hyperventilation: reduction of $PaCO_2$ through hyperventilation is the most rapid means of lowering ICP. Keep ICP < 20 mmHg
  - Hyperventilation should be used with caution because it reduces brain tissue PO_2 ($PbrO_2$); hypoxia may lead to ischemia of brain tissue, causing further damage in the CNS after stroke
  - Optimal $PaCO_2$ ~ 25–30 mmHg
  - Hyperosmolar therapy with mannitol improves ischemic brain swelling (by diuresis and intravascular fluid shifts)
  - Furosemide/acetazolamide may also be used
  - High doses of barbiturates (e.g., thiopental) rapidly lower ICP and suppress electrical brain activity
- **Fluid Restriction**
  - Avoid glucose solutions; use normal saline; maintain euvolemia
  - Replace urinary losses with normal saline in patients receiving mannitol
- **Surgical Therapy**
  - Neurosurgical decompression

THROMBOLYTIC THERAPY

IV tissue – plasminogen activator (t-PA)

First FDA approved Tx for ischemic strokes in selected patients
- In National Institute of Neurologic Disorders (1995) trial, patients given t-PA within three hours of onset of stroke were 30% more likely to have minimal or no disability at three months compared to patrents treated with placebo
- There was a tenfold increase in hemorrhage (overall) with t-PA compared to placebo (6.4% vs. 0.6%) and in fatal ICH (3% vs. 0.3%)
- However, mortality was higher in placebo group than in t-PA groups; overall mortality: 17% t-PA (including hemorrhage cases) vs. 21% placebo

Inclusion criteria

- Age 18 yrs or older
- Time of onset of symptoms well established to be < three hours before treatment would begin
- Patients with measurable neurologic deficits (moderate to severe stroke symptoms)
- CT negative for blood

Exclusion criteria

- Minor stroke symptoms/TIA (symptoms rapidly improving)
- CT positive for blood
- Blood Pressure > 185/100 despite moderate Tx
- Increased PT/PTT
- Decreased PLTs
- Blood Sugar < 50, > 400
- Hx stroke past 3 month
- Hx of ICH, AVM or aneurysm
- Seizure at onset of stroke

Streptokinase

Three recent large randomized trials of streptokinase in stroke suspended because of increase in hemorrhage and mortality in treatment group

ANTICOAGULANT THERAPY

Heparin
- Frequently used in patients with acute ischemic stroke, but its value is unproven
- There is no unanimity on when heparin should be started, desired level of anticoagulation or if bolus dose should be given or not

Low molecular-weight heparin
- has more selective antithrombotic action than heparin (may be safer)
- Kay et al. (1995) study reporting improvement in survival and decrease in eventual rate of dependency (rated by Barthel Index) in patients treated with low molecular-weight heparin (LMWH) within 48 hours of onset of stroke compared to placebo

Aspirin, warfarin, ticlopidine (Ticlid®), clopidogrel, Plavix® (Creager, 1998)
- All have been shown to decrease the risk of subsequent stroke in patients with TIA.
- Usefulness in Tx of acute stroke unknown
- Anticoagulant therapy with warfarin: Stroke incidence and mortality in patients at high risk reduced; might be the best option for patients with a history of atrial fibrillation, prior stroke (or TIA), HTN, diabetes and CHF (Ryder, 1999)

Indications for Anticoagulation (Controversial)

- **Stroke in evolution:**

Neurologic deficit developing in stepwise progression (over 18 to 24 hours in carotid circulation; up to 72 hours in vertebrobasilar circulation). IV heparin efficacy unproven as previously mentioned. Generally, IV heparin given for at least several days to increase PTT to 1.5 to 2.5 times control. Coumadin® may be used for longer period (e.g, 6-month trial)

- **Cardiac emboli** (best reason to anticoagulate):
  - Primarily from nonvalvular atrial fibrillation and mural thrombus from myocardial infarction (MI)
  - Anticoagulation reported to reduce incidence of cerebral emboli in patients with MI by 75%
  - Timing of anticoagulation in patients with cardiac emboli controversial; probable risk of inducing cerebral hemorrhage or hemorrhagic infarction within large infarcts if anticoagulated in first 24–32 hours
  - If neurologic deficit is mild (and CT shows no hemorrhage) may begin anticoagulation immediately
  - If deficit severe (clinically and/or CT), wait 3–5 days before starting anticoagulation
  - 15% of cardiogenic emboli lodge in the brain. The most common cause is chronic atrial fibrillation

- **Transient Ischemic Attacks:**
  - Some studies suggest that a cluster of recent, frequent ("crescendo") TIAs is an indication for anticoagulation therapy. Use of anticoagulants (heparin, Coumadin®) in TIA is empirical
  - May consider use of Coumadin® when antiplatelet drugs fail to reduce attacks

- **Completed Stroke:**
  - Anticoagulation not considered beneficial after major infarction and usually not of great value once stroke is fully developed
  - Empirically, some will utilize anticoagulation (initially with IV heparin) in setting of mild infarct to theoretically prevent further progression in same vascular territory Coumadin® may be continued for several weeks to 3 to 6 months
  - Anticoagulation generally not employed for lacunar infarction

CORTICOSTEROIDS:

- No value in ischemic strokes
- Some studies suggest worsening in prognosis of stroke patients due to hyperglycemia

CAROTID ENDARTERECTOMY (CEA)

Symptomatic carotid stenosis

CEA for symptomatic lesions with > 70% stenosis (70%–99%) is effective in reducing the incidence of ipsilateral hemisphere stroke. (North American Symptomatic Carotid Endarterectomy Trial Collaborators, 1991), (Endarterectomy for moderate symptomatic carotid stenosis: Interim results from the MRC European Carotid Surgery Trial, 1996) (Executive Committee for Asymptomatic Carotid Artherosclerosis Study, 1995)

American Heart Association (AHA) guidelines for CEA (Moore, 1995)

1. CEA is proven beneficial in:
   - Symptomatic patients with one or more TIAs (or mild stroke) within the past 6 months and carotid stenosis ≥ 70%

2. CEA is "Acceptable but not proven":
   - TIAs or mild and moderate strokes within the last 6 months and stenosis 50% to 69%
   - Progressive stroke and stenosis ≥ 70%

CEA for Asymptomatic Carotid Stenosis

- Indications—Controversial
- AHA guidelines (Moore, 1995)

"Acceptable but not proven": in stenosis > 75% by linear diameter (asymptomatic cases)

Note: recent studies present opposing views on indications for surgery in asymptomatic carotid stenosis

Asymptomatic Carotid Atherosclerosis Study (ACAS) (Executive Committee for the Asymptomatic Carotid Artherosclerosis Study, 1995) (Young et al., 1996)

- Study showed a significant reduction (53%) in risk of ipsilateral stroke in a five-year period in asymptomatic patients with > 60% carotid stenosis (and < 3% rate perioperative morbidity/mortality); risk was 5.1% on patients treated surgically vs. 11.0% in patients treated medically.
- ACAS study not evaluated for AHA guidelines on 1995

ECST (Endarterectomy for moderate symptomatic carotid stenosis: Interim results from the MRC European Carotid Surgery Trial, 1996). This 3 year study showed:

- In patients with asymptomatic carotid stenosis < 70%, risk of stroke is low, 2%. In patients with stenosis > 70%, risk also is low, 5.7%
- Conclusion of study was that CEA is not justified in asymptomatic carotid stenosis.

TREATMENT OF SUBARACHNOID HEMORRHAGE (see also Tx of ICP)

- Bed rest in a quiet, dark room with cardiac monitoring (cardiac arrhythmias are common)
- Control of headaches with acetaminophen and codeine
- Mannitol to reduce cerebral edema
- Control of blood pressure—have the patient avoid all forms of straining (give stool softeners and mild laxatives)
- Early surgery (with clipping of aneurysm) better; reduces risk of rebleeding; does not prevent vasospasm or cerebral ischemia
- Nimodipine (calcium channel blocker) shown to improve outcome after SAH (decreased vasospasms). It is useful in the treatment of cerebral blood vessel spasm after SAH. It decreases the incidence of permanent neurologic damage and death. Therapy should be initiated within 96 hours of the onset of hemorrhage

TREATMENT OF INTRACRANIAL HEMORRHAGE

- Management of increased ICP and blood pressure (see previous)
- Large intracranial or cerebellar hematomas often require surgical intervention

TREATMENT OF ARTERIOVENOUS MALFORMATION (AVM) (Hamilton and Septzler, 1994; Schaller, Scramm, and Haun, 1998)

- Treatment advised in both *symptomatic* and *asymptomatic* AVMs
- Surgical excision if size and location feasible (and depending on perioperative risk)
- Embolization
- Proton Beam Therapy (via stereotaxic procedure)
- Small asymptomatic AVMs: radiosurgery/microsurgical resection recommended

STROKE REHABILITATION

INTRODUCTION

The primary goal of stroke rehabilitation is functional enhancement by maximizing the independence, life style, and dignity of the patient.

This approach implies rehabilitative efforts from a physical, behavioral, cognitive, social, vocational, adaptive, and re-educational point of view. The multidimensional nature of stroke and its consequences make coordinated and combined interdisciplinary team care the most appropriate strategy to treat the stroke patient.

Recovery from impairments

Hemiparesis and motor recovery have been the most studied of all stroke impairments. Up to 88% of acute stroke patients have hemiparesis

The process of recovery from stroke usually follows a relatively predictable, stereotyped series of events in patients with stroke-induced hemiplegia. These sequence of events have been systematically described by several clinical researchers.

Twitchell (1951) published a highly detailed report describing the pattern of motor recovery following a stroke (pattern most consistent in patients with cerebral infarction in the MCA distribution)

- His sample included 121 patients, all except three having suffered either thrombosis or embolism of one of the cerebral vessels
- Immediately following onset of hemiplegia there is total loss of voluntary movement and loss or decrease of the tendon reflexes
- This is followed (within 48 hours) by increased deep tendon reflexes on the involved side, and then (within a short time) by increased resistance to passive movement (tone returns → spasticity), especially in flexors and adductors in the upper extremity (UE) and extensors and adductors in the lower extremity (LE)
- As spasticity increased, clonus (in ankle plantar flexors) appeared in 1–38 days post- onset of hemiplegia
- Recovery of movement:
  - 6 to 33 days after the onset of hemiplegia, the first "intentional" movements (shoulder flexion) appears
  - In the UE, a flexor synergy pattern develops (with shoulder, elbow, wrist and finger flexion) followed by development of an extensor synergy pattern. Voluntary movement in the lower limb also begins with flexor synergy (also proximal—hip) followed by extensor synergy pattern
- With increase of voluntary movement, there is a decrease in the spasticity of the muscles involved
- Tendon reflexes remain increased despite complete recovery of movement
- At onset of hemiplegia, the arm is more involved than the leg, and eventual motor recovery in the leg occurs earlier, and is more complete, than in the arm
- Most recovery takes place in the first three months and only minor additional recovery occurs after six months post onset

Predictors of motor recovery:

- Severity of arm weakness at onset:
  - With complete arm paralysis at onset, there is a poor prognosis of recovery of useful hand function (only 9% gain good recovery of hand function)

- Timing of return of hand movement:
  - If the patient shows some motor recovery of the hand by four weeks, there is up to 70% chance of making a full or good recovery
  - Poor prognosis with no measurable grasp strength by four weeks
- Poor prognosis associated also with:
  - Severe proximal spasticity
  - Prolonged "flaccidity" period
  - Late return of proprioceptive facilitation (tapping) response > nine days
  - Late return of proximal traction response (shoulder flexors/adductors) > 13 days

Brunnstrom (1966, 1970) and Sawner (1992) also described the process of recovery following stroke-induced hemiplegia. The process was divided into a number of stages:

1. Flaccidity (immediately after the onset)
   No "voluntary" movements on the affected side can be initiated
2. Spasticity appears
   Basic synergy patterns appear
   Minimal voluntary movements may be present
3. Patient gains voluntary control over synergies
   Increase in spasticity
4. Some movement patterns out of synergy are mastered (synergy patterns still predominate)
   Decrease in spasticity
5. If progress continues, more complex movement combinations are learned as the basic synergies lose their dominance over motor acts
   Further decrease in spasticity
6. Disappearance of spasticity
   Individual joint movements become possible and coordination approaches normal
7. Normal function is restored

REHABILITATION METHODS FOR MOTOR DEFICITS: Major theories of rehabilitation training

Traditional Therapy:

Traditional therapeutic exercise program consists of positioning, ROM exercises, strengthening, mobilization, compensatory techniques, endurance training (e.g., aerobics). Traditional approaches for improving motor control and coordination: emphasize need of repetition of specific movements for learning, the importance of sensation to the control of movement, and the need to develop basic movements and postures. (Kirsteins, Black, Schaffer, and Harvey, 1999)

Proprioceptive (or peripheral) Neuromuscular Facilitation (PNF) (Knott and Voss, 1968)

- *Uses spiral and diagonal components of movement rather than the traditional movements in cardinal planes of motion with the goal of facilitating movement patterns that will have more functional relevance than the traditional technique of strengthening individual group muscles*
- Theory of spiral and diagonal movement patterns arose from observation that the body will use muscle groups synergistically related (e.g., extensors vs. flexors) when performing a maximal physical activity

- Stimulation of nerve/muscle/sensory receptors to evoke responses through manual stimuli to increase ease of movement-promotion function
- It uses resistance during the spiral and diagonal movement patterns with the goal of facilitating "irradiation" of impulses to other parts of the body associated with the primary movement (through increased membrane potentials of surrounding alpha motoneurons, rendering them more excitable to additional stimuli and thus affecting the weaker components of a given part)
- Mass-movement patterns keep Beevor's axiom: Brain knows nothing of individual muscle action but only movement

Bobath approach / neurodevelopmental technique (NDT) (Bobath, 1978)
- *The goal of NDT is to normalize tone, to inhibit primitive patterns of movement, and to facilitate automatic, voluntary reactions and subsequent normal movement patterns.*
- Based on the concept that pathologic movement patterns (limb synergies and primitive reflexes) must not be used for training because continuous use of the pathologic pathways may make it too readily available to use at expense of the normal pathways
- Probably the most commonly used approach
- Suppress abnormal muscle patterns before normal patterns introduced
- Mass synergies avoided, although they may strengthen weak, unresponsive muscles, because these reinforce abnormally increased tonic reflexes, spasticity
- Abnormal patterns modified at *proximal* key points of control (e.g., shoulder and pelvic girdle)
- *Opposite to Brunnstrom approach* (which encourages the use of abnormal movements); see the following

Brunstrom approach/Movement therapy (Brunnstrom, 1970)
- *Uses primitive synergistic patterns in training in attempting to improve motor control through central facilitation*
- Based on concept that damaged CNS regressed to phylogenetically older patterns of movements (limb synergies and primitive reflexes); thus, synergies, primitive reflexes, and other abnormal movements are considered normal processes of recovery before normal patterns of movements are attained
- Patients are taught to use and voluntarily control the motor patterns available to them at a particular point during their recovery process (e.g., limb synergies)
- Enhances specific synergies through use of cutaneous/proprioceptive stimuli, central facilitation using Twitchell's recovery
- *Opposite to Bobath* (which inhibits abnormal patterns of movement)

Sensorimotor approach/Rood approach (Noll, Bender, and Nelson, 1996)
- Modification of muscle tone and voluntary motor activity using cutaneous sensorimotor stimulation
- Facilitatory or inhibitory inputs through the use of sensorimotor stimuli, including, quick stretch, icing, fast brushing, slow stroking, tendon tapping, vibration, and joint compression to promote contraction of proximal muscles

Motor relearning program/Carr and Shepard approach (Carr et al., 1985)
- Based on cognitive motor relearning theory and influenced by Bobath's approach
- Goal is for the patient to relearn how to move functionally and how to problem solve during attempts at new tasks
- Instead of emphasizing repetitive performance of a specific movement for improving skill, it teaches general strategies for solving motor problems.
- Emphasizes functional training of specific tasks, such as standing and walking, and carryover of those tasks

Behavioral approaches (Noll, Bender, and Nelson, 1996) include:
- Kinesthetic or positional biofeedback and forced-use exercises
- Electromyographic biofeedback EMGBF: makes patient aware of muscle activity or lack of it by using external representation (e.g., auditory or visual cues) of internal activity as a way to assist in the modification of voluntary control
  - In addition to trying to modify autonomic function, EMGBF also attempts to modify pain and motor disturbances by using volitional control and auditory, visual, and sensory clues
  - Electrodes placed over agonists/antagonists for facilitation/inhibition
  - Accurate sensory information reaches brain through systems unaffected by brain → via visual and auditory for proprioception

UPPER EXTREMITY MANAGEMENT (Black-Shaffer, Kirsteins, and Harvey, 1999)

- Shoulder pain: 70%–84% of stroke patients with hemiplegia have shoulder pain with varying degrees of severity
- Of the patients with shoulder pain, the majority (85%) will develop it during the spastic phase of recovery
- It is generally accepted that the most common causes of hemiplegic shoulder pain are the shoulder-hand syndrome/reflex sympathetic dystrophy (RSD) and soft-tissue lesions (including plexus lesions)

Complex Regional Pain Syndrome Type I (CRPS, Type I)/Reflex Sympathetic Dystrophy (RSD)

- Disorder characterized by sympathetic-maintained pain and related sensory abnormalities, abnormal blood flow, abnormalities in the motor system, and changes in both superficial and deep structures with trophic changes
- Has been reported in 12% to 25% of hemiplegic stroke patients
- CRPS Type I = RSD
  (CRPS type II = causalgia—pain limited to a peripheral nerve distribution)
- Most common subtype of RSD in stroke is shoulder-hand syndrome

Stages:
- **Stage 1—acute:** burning pain, diffuse swelling/edema, exquisite tenderness, vasomotor changes in hand/fingers (with increased nail and hair growth, hyperthermia or hypothermia, sweating). Lasts three to six months
- **Stage 2—dystrophic:** pain becomes more intense and spreads proximally, skin/muscle atrophy, brawny edema, cold insensitivity, brittle nails/nail atrophy, decrease ROM, mottled skin, early atrophy and osteopenia (late). Lasts three to six months
- **Stage 3—atrophic:** pain decreases, trophic changes, hand skin pale and cyanotic, with a smooth, shiny appearance and feels cool and dry, bone demineralization progresses with muscular weakness/atrophy, contractures/flexion deformities of shoulder/hand, tapering digits, no vasomotor changes

Pathogenesis
- Multiple theories postulated including:
  - Abnormal adrenergic sensitivity develops in injured nociceptors, and circulating or locally secreted sympathetic neurotransmitters trigger the painful afferent activity
  - Cutaneous injury activates nociceptor fibers → central pain-signaling system → pain
  - Central sensitization of pain-signaling system
  - Low-threshold mechanoreceptor input develops capacity to evoke pain
  - With time, efferent sympathetic fibers develop capacity to activate nociceptor fibers

Diagnosis

- **X rays**—in initial stages, X rays normal; periarticular osteopenia may be seen in later stages; use is questionable, given that bone mineral density starts to decrease in the paralytic arm one month after stroke
  - Need 30%–50% demineralization for detection

- **Bone Scan—**30 stroke survivors < 3 months onset, evaluated for CRPS Type I using triple phase bone scan (Kozin, 1981; Simon and Carlson, 1980; Habert, Eckelman, and Neuman, 1996)
  - Sensitivity ~ 92%
  - Specificity ~ 56%
  - Positive predictive value (PPV) ~ 58%
  - Negative predictive value (NPV) ~ 91% (Holder and Mackinnon, 1984)
  - Diffusely increased juxta-articular tracer activity on delayed images is the most sensitive indicator for RSD (sensitivity 96%, specificity 97%, and PPV 88%)

- **EMG—**as predictor for RSD (Cheng and Hong, 1995)
  - Association between spontaneous activity and eventual development of RSD (vs no spontaneous activity on EMG)

- **Clinical** (Wang et al., 1998)
  - Clinical diagnosis difficult, presentation fairly incomplete
  - Most consistent early diagnostic signs: shoulder pain with ROM (flexion/abduction/external rotation), absence of pain in elbow and with forearm pronation/supination; wrist dorsiflexion pain with dorsal edema; pain MCP/PIP flexion with fusiform PIP edema
  - Pain out of proportion to injury and clinical findings
  - Shoulder/hand pain preceded by rapid ROM loss
  - Tepperman et al. (1984) Greyson and Tepperman (1984)
  - Studied 85 consecutive post-CVA hemiplegic patients
  - 25% had radionuclide evidence for RSD: positive diagnosis was evident when delayed image showed increased uptake in wrist, MCP and IP joints
  - In this study, the most valuable clinical sign was MCP tenderness to compression with 100% predictive value, sensitivity 85%, and specificity 100%

- **Stellate ganglion block**
  - Alleviation of pain following sympathetic blockade of the stellate ganglion using local anesthetic: is the gold standard Dx of sympathetically mediated CRPS Type I

Treatment (Arlet and Mazieres, 1997)

- ROM exercises involved joint-pain free within three weeks, most < four to six days with passive stretch of involved joints
- Corticosteroids (systemic): a large majority of patients respond to systemic steroids instituted in the acute phase of the disease. Usually prednisone in doses up to 100–200 mg/day or 1 mg/kg, and tapered over two weeks
  - More effective in RSD confirmed by bone scan than on "clinical" RSD with negative bone scan. Bone scan may be useful not only in establishing the Dx of RSD, but also in identifying patients likely to respond to oral steroid therapy. In a study, 90% of the patients. with positive bone scan findings for RSD treated with corticosteroids had good or excellent response, whereas 64% of the patients, without bone scan abnormalities had poor or fair response.

  - In recent study, 31/34 MCA stroke patients with RSD became pain free by 14 days after starting methylprednisolone 8 mg PO QID (patients treated for two weeks, followed by two-week taper)
- Intra-articular injections with corticosteroids
- Analgesics (NSAIDs)
- Tricyclic antidepressants
- Diphosphonates
- Calcitonin
- Anticonvulsants (as Neurontin® or Tegretol®)
- Alpha-adrenergic blockers (clonidine, prazosin)
- Beta-blockers (propranolol and pindolol)
- Calcium channel blockers (nifedipine)
- Topical capsaicin
- TENS
- Contrast baths
- Edema control measures
- Desensitization techniques
- Ultrasound (U/S)
- Sympathetic ganglion blocks (i.e., stellate ganglion) may be diagnostic as well as therapeutic
- Local injections (procaine, corticosteroid)
- Sympathectomy

Shoulder Subluxation

Characterized by the presence of a palpable gap between the acromion and the humeral head

Etiology is unknown, but may be due to changes in the mechanical integrity of the glenohumeral joint

Pathogenesis: factors that are thought to be related to shoulder subluxation include: angulation of the glenoid fossa, the influence of the supraspinatus muscle on the humeral head sitting, the support of the scapula on the rib cage, the contraction of the deltoid and rotator cuff muscles on the abducted humerus

- A number of recent studies have failed to show any correlation between shoulder subluxation and pain
- There might be a correlation with between-shoulder pain and decrease in arm external rotation
- *Basmajian Principle:* Decreased trapezius tone—the scapula rotates and humeral head subluxates from glenoid fossa

Treatment

Shoulder slings: use is controversial

Routine use of sling for the subluxed shoulder (or for shoulder pain) is not indicated

- 🕮 **Friedland—**sling does not prevent/correct subluxation, not necessary to support pain-free shoulder (Friedland, 1975)
- 🕮 **Hurd—**no appreciable difference in shoulder ROM, subluxation, or shoulder pain in patients wearing slings or not (Hurd et al., 1974)

Pros: May be used when patient ambulates to support extremity (may prevent upper extremity trauma, which in turn may cause increase pain or predispose to development of RSD)

Cons: May encourage contractures in shoulder adduction/internal rotation, elbow flexion (flexor synergy pattern)

Other widely used treatments for shoulder subluxation:
- Functional Electrical Stimulation (FES)
- Armboard, arm trough, lapboard—used in poor upper-extremity recovery, primary wheelchair users
  - Arm board may overcorrect subluxation
- Overhead slings—prevents hand edema (may use foam wedge on armboard)

Prevention:
- Subluxation may be prevented by combining the early reactivation of shoulder musculature (specifically supraspinatus and post- and mid-deltoid) with the provision of FES or a passive support of the soft-tissue structures of the glenohumeral joint (e.g., arm trough)

🕮 Brachial Plexus/Peripheral Nerve Injury

Etiology: "Traction" neuropathy

Diagnosis:
- Clinical: atypical functional return, segmental muscle atrophy, finger extensor contracture, delayed onset of spasticity
- Electrodiagnostic studies (EMG)—lower motor neuron findings

Treatment:
- Proper bed positioning to prevent patient from rolling onto his paretic arm, trapping it behind his back or through the bed rails and place a traction stress on it.
- ROM to prevent contracture while traction avoided
- 45-degree shoulder-abduction sling for nighttime positioning
- Sling for ambulation to prevent traction by gravity
- Armrest in wheelchair as needed

Prognosis—may require 8 to 12 months for reinnervation

Bicipital Tendinitis

- Chronic pain anterolateral shoulder, pain in abduction/external rotation, painful over bicipital groove
- Positive Yergason test: with arm on side and elbow flexed, external rotation of the arm is exerted by the examiner (while pulling downward on the elbow) as the patient resists the movement. If the biceps tendon is unstable on the bicipital groove, it will pop-out and the patient will experience pain
- Greatest excursion of long head biceps from flexion/internal rotation, to elevation/abduction, depression/external rotation/extension
- May progress to adhesive capsulitis (frozen shoulder)

Diagnosis may be confirmed with decreased pain after injection of tendon sheath with lidocaine; bicipital tendinitis may respond to steroid injection of the tendon sheath.

Rotator Cuff Tear, Impingement Syndrome, Adhesive Capsulitis (frozen shoulder):

All are causes of poststroke shoulder pain—see Table 1.7; see Musculoskeletal chapter

TABLE 1–7 Post-Stroke Shoulder Pain

	Inferior Subluxation	**Rotator Cuff Tear**	**CRPS Type 1 (RSD)**	**Frozen Shoulder**	**Impingement Syndrome**	**Biceps Tendinitis**
EXAM	• Acromio-humeral separation • Flaccid	• Positive abduction test • Positive drop arm test • Flaccid or spastic	• MCP compression test • Skin changes color • Flaccid or spastic	• External rotation less than 15° • Early decrease in scapular motion • Spastic	• Pain with abduction of 70°–90° • End-range pain with forward flexion • Usually spastic	• Positive Yergason's test • Flaccid or spastic
DIGNOSTIC TEST	• X ray in standing position • Scapular plane view	• X ray • Arthrogram • Subacromial injection of lidocaine • MRI	• Triple phase bone scan • Stellate ganglion block	• Arthrogram	• Subacromial injection of lidocaine	• Tendon sheath injection of lidocaine
THERAPY	• Sling when upright • FES	• Steroid injection • PT/ROM • Possible surgical repair • Reduction of internal rotator cuff tone	• Oral corticosteroids • Stellate ganglion block	• PT/ROM • Debridement manipulation • Subacromial steroids • Intra-articular steroids • Reduction of internal rotator cuff tone	• PT/ROM • Scapular mobilization • Subacromial steroids • Reduction of internal rotator cuff tone	• Tendon sheath injection of steroids

Abbreviations: CRPS1: Complex/regional Pain Syndrome type 1
FES: Functional electrical stimulation
MCP: metacarpophalangeal
PT/ROM: Physical Therapy/Range of Motion
MRI: magnetic resonance imaging
RSD: Reflex Sympathetic Dystrophy
(Black-Schaffer, 1999)

Heterotopic Ossification

- Infrequent (in stroke), but may be seen in elbow, shoulder
- Occurs only on extensor side of elbow
- No problems in pronation/supination since proximal radioulnar joint not involved
- Treatment: joint mobilization/ROM, etidronate disodium

Dependent Edema

May be treated with use of compression glove, foam wedge, pneumatic compression, retrograde massage, arm elevation

OTHER ASPECTS OF STROKE REHABILITATION

Spasticity Management

For a detailed discussion of spasticity, see the Spasticity chapter

Spasticity in stroke:
- Usually seen days to weeks after ischemic strokes
- Usually follows classic upper-extremity flexor and lower-extremity extensor patterns
- Clinical features include velocity-dependent resistance to passive movement of affected muscles at rest, and posturing in the patterns previously mentioned during ambulation and with irritative/noxious stimuli

Treatment:
- Noninvasive Tx: stretching program, splints/orthosis, serial casting, electrical stimulation, local application of cold
- Medications:
  - The use of benzodiazepines, baclofen, dantrolene, and the alpha agonists clonidine and tizanidine, in stroke patients, remains controversial
  - These drugs have modest effects on the hypertonicity and posturing associated with stroke and their side-effects limit their usefulness
- Injection of chemical agents:
  - Botulinum toxin: may be particularly useful in control of increased tone in smaller muscles of the forearm and leg (e.g., brachioradialis, finger, wrist, and thumb flexors, in the upper extremity, and long and short toe flexors, extensor hallucis injury (EHL), and ankle invertors in the lower extremity)
  - Phenol: may remain the agent of choice for injection of large muscle groups (e.g., hip adductors and extensors, the pectorals, lats, and biceps)
- Intrathecal baclofen: limited experience of its use in stroke patients; usefulness remains to be determined in this population
- Surgical procedures:
  - Uncommonly used in stroke, probably because of expected decrease in survival and increase in rate of medical co-morbidities
  - May be useful in selected cases when specific goals are pursued (e.g., increase in function, improve hygiene, decrease in pain)

Deep Vein Thrombosis (DVT)

- Common medical complication after stroke; occurring in 20%–75% of untreated stroke survivors (60%–75% in affected extremity, 25% proximal DVT; PE, 1%–2%)
- **Diagnosis**: Usually can be made using noninvasive techniques:
  - Ultrasonography
  - Impedance plethysmography
  - Contrast venography reserved for cases with inconclusive results.
  - D-dimer assays (a cross-linked fibrin degradation product): may be useful as screening tool for DVT in stroke patients
- **Prophylaxis**:
  Currently, recommended prophylaxis regimens include:
  - Low dose subcutaneous (SQ) heparin/low molecular weight heparin
  - Intermittent pneumatic compression (IPC) of the lower extremity (LE) (for patients with a contraindication to heparin)
  - Gradient compression stockings in combination with SQ heparin or IPC

Bladder Dysfunction

- Incidence of urinary incontinence is 50%–70% during the first month after stroke and 15% after 6 months (similar to general population—incidence ≈ 17%.)
- Incontinence may be caused by CNS damage itself, UTI, impaired ability to transfer to toilet or impaired mobility, confusion, communication disorder/aphasia, and cognitive-perceptual deficits that result in lack of awareness of bladder fullness
- Types of voiding disorders: areflexia, uninhibited spastic bladder (with complete/incomplete emptying), outlet obstruction
- Treatment: implementation of timed bladder-emptying program
  - Treat possible underlying causes (e.g., UTI)
  - Regulation of fluid intake
  - Transfer and dressing-skill training
  - Patient and family education
  - Medications (if no improvement with conservative measures)
- Remove indwelling catheter—perform postvoid residuals, intermittent catheterization—perform urodynamics evaluation

Bowel Dysfunction

Patient unable to inhibit urge to defecate → incontinence
- Incidence of bowel incontinence in stroke patients 31%
- Incontinence usually resolves within the first two weeks; persistence may reflect severe brain damage
- Decrease in bowel continence may be associated with infection resulting in diarrhea, inability to transfer to toilet or to manage clothing, and communication impairment/inability to express toileting needs
- Tx: treat underlying causes (e.g., bowel infection, diarrhea), timed-toileting schedule, training in toilet transfers and communication skills

Impairment of intestinal peristalsis—constipation
- Management: adequate fluid intake/hydration, modify diet (e.g., increase in dietary fiber), bowel management (stool softeners, stool stimulants, suppositories), allow commode/bathroom privileges

Dysphagia

Dysphagia (difficulty swallowing), in stroke, has an incidence of 30% to 45% (overall)
- 67% of brainstem strokes
- 28% of all left hemispheric strokes
- 21% of all right hemispheric strokes
- More common in bilateral hemisphere lesions than in unilateral hemisphere lesions
- More common in large-vessel than in small-vessel strokes

Predictors on bed-side swallowing exam of aspiration include:
- Abnormal cough
- Cough after swallow
- Dysphonia
- Dysarthria
- Abnormal gag reflex
- Voice change after swallow (wet voice) (Aronson, 1990)

SWALLOWING

Three phases:
1. Oral
2. Pharyngeal
3. Esophageal

TABLE 1–8 Oral Phase

Voluntary vs. reflex	• Voluntary
Phase duration	• Variable; voluntary phase with duration influenced by consistency of material ingested, number of times person chews, etc.
Hallmarks of this phase	• Preparation of bolus • Tongue elevates and occludes the anterior oral cavity and compresses the bolus toward the oropharynx • Contraction of the palatopharyngeal folds • Elevation of the soft palate
Phase requires:	• Intact lip closure • Mobile tongue • Functional muscles of mastication
Problems seen in this phase:	• Drooling, pocketing, head tilt

TABLE 1–9 Pharyngeal Phase

Voluntary vs. reflex	• Reflex
Phase duration	• Lasts ~ 0.6 to 1 sec
Hallmarks of this phase	• Bolus propelled from mouth to esophagus • Aspiration most likely to occur during this phase • With initiation of pharyngeal phase, inhibition of breathing occurs to prevent aspiration.
🕮 **Phase requires:**	• Tongue elevation • Soft palate elevation (also seen in the oral phase) and velopharyngeal port closure—to close off the nasal cavity and prevent regurgitation into the nasopharynx • Laryngeal elevation, with folding of epiglottis and vocal cord adduction to prevent aspiration • Coordinated pharyngeal constriction and cricopharyngeal (upper esophageal sphincter) relaxation—to facilitate bolus transport into the esophagus
Problems seen in this phase:	• Food sticking, choking and coughing, aspiration, wet/gurgling voice, nasal regurgitation

TABLE 1–10 Esophageal Phase

Voluntary vs. reflex	• Reflex
Phase duration	• Longest phase—lasts 6–10 sec
Hallmarks of this phase	• Bolus passed from pharynx → esophagus → stomach • Esophageal clearance is assisted by gravity but requires relaxation of the gastroesophageal sphincter
Phase requires:	• Cricopharyngeal muscle contraction • Coordinated peristalsis and LES relaxation
Problems seen in this phase:	• Heartburn, food sticking

Important Definitions

- 🕮 **Chin tuck**—compensatory technique to provide airway protection by preventing entry of liquid into the larynx (probably by facilitating forward motion of the larynx). Also, chin tuck decreases the space between the base of the tongue and the posterior pharyngeal wall, and so creates increased pharyngeal pressure to move the bolus through the pharyngeal region
- 🕮 **Aspiration**
  - Aspiration, by definition, is the penetration of a substance into the laryngeal vestibule and below the vocal folds (true vocal cords) into the trachea
  - Aspiration is missed on bedside swallowing evaluations in 40% to 60% of patients (i.e., silent aspiration)
  - It can be reliably diagnosed on videofluoroscopic swallowing study (penetration of contrast material below the true vocal cords)
  - Using videofluoroscopic swallowing study, aspiration has been found to occur in 40% to 70% of stroke patients.
  - Predictors of aspiration on videofluoroscopic swallowing study include:
    - Delayed initiation of the swallow reflex
    - Decreased pharyngeal peristalsis

- **Aspiration pneumonia**
  Risk factors for development of pneumonia secondary to aspiration include:
  - Decreased level of consciousness
  - Tracheostomy
  - Emesis
  - Reflux
  - Nasogastric tube (NGT) feeding
  - Dysphagia
  - Prolonged pharyngeal transit time

As dysphagia is a frequent and potentially serious (because of aspiration) complication of stroke, careful bedside swallowing evaluation should be performed in all patients before oral feeding is started. If a patient is believed to be at high risk of recurrent aspiration after bedside and/or videofluoroscopic evaluation, he/she should be kept NPO and enterally fed, initially via NGT, and then via G-tube or J-tube if long-term enteral feeding is required.

- **Non-oral feeding:**
  - Clear contraindication for oral feeding is pulmonary pathology due to aspiration in the presence of documented airway contamination
  - NPO also indicated in patients at high risk of aspiration because of reduced alertness, reduced responsiveness to stimulation, absent swallow, absent protective cough, and difficulty handling secretions, or when there is significant reduction of oral pharyngeal and laryngeal movements
  - NPO is disadvantageous in treating dysphagia because swallowing itself is the best treatment

Treatment of dysphagia/prevention of aspiration:
- Changes in posture and head position
- Elevation of the head of the bed
- Feeding in the upright position
- Chin tuck
- Turning the head to the paretic side
- Diet modifications (e.g., thickened fluids, pureed or soft foods in smaller boluses)

Inconclusive evidence of long-term efficacy in dysphagia:
- Thermal stimulation (to sensitize the swallowing reflex)
- Oral/motor exercises (to improve tongue and lip strength, ROM, velocity, and precision, and vocal-fold adduction)

Other complications of dysphagia include dehydration and malnutrition:
- Malnutrition found in 49% of patients admitted to rehabilitation in recent study and was associated with a prolonged length of stay and slower rate of functional gains
- Malnourished patients—higher stress reaction, frequency of infection and decubiti

Recovery of dysphagia in stroke:
Few studies available on recovery of dysphagia in stroke:
- Gresham (1990) reports his findings regarding 53 patients in a swallowing program post-stroke
  - 85% (45/53) on full oral nutrition at discharge
  - 17% (9/53) could not drink thin liquids safely
  - 8% (4/53) could not adequately maintain cohesive bolus of varied texture
- Gordon (1987)
  - 41 of 91 (45%) stroke patients + dysphagia
  - 90% hemispheric lesions (17% bilateral)

  - Swallowing function regained within 14 days in 86% (of patients who survived unilateral stroke)
- 🕮 Logemann (1991)
  - Recovery of swallowing function in most brainstem strokes occurs in the first three weeks poststroke

🕮 **Nasal speech**: hypernasality caused by partial or complete failure of soft palate to close-off the nasal cavity from the oral cavity or by incomplete closure of the hard palate. Uplifting the soft palate prevents nasal speech (speech abnormally resonated in the nasal cavities).

APHASIA

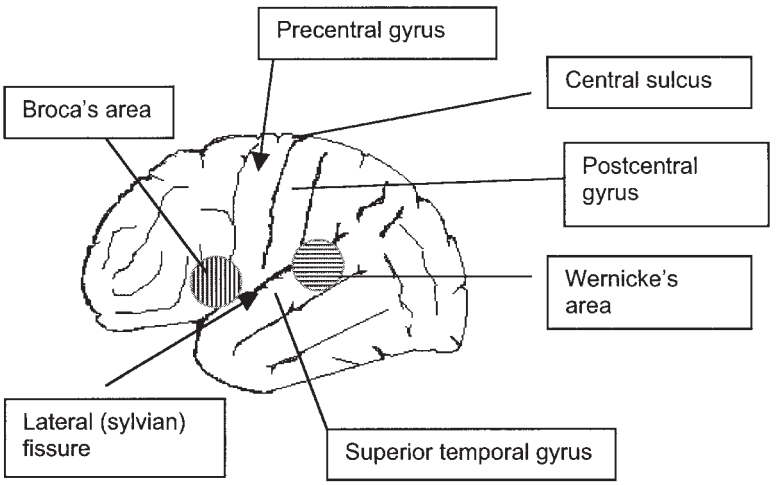
- Aphasia is an impairment of the ability to utilize language due to brain damage. Characterized by paraphasias, word-finding difficulties, and impaired comprehension. Also common, but not obligatory, features are disturbances in reading and writing, nonverbal constructional and problem-solving difficulty and impairment of gesture

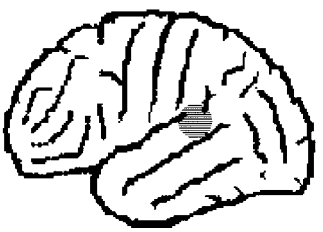
TABLE 1–11 Aphasias

Fluent				**Nonfluent**			
+ COMPREHENSION		– COMPREHENSION		+ COMPREHENSION		– COMPREHENSION	
REPETITION ⇩		*REPETITION* ⇩		*REPETITION* ⇩		*REPETITION* ⇩	
+	–	+	–	+	–	+	–
NAMING	conduction	Transcortical sensory	Wernicke's	Transcortical motor	Broca's	Mixed transcortical	Global
+	–						
normal	anomia						

Fluent	**Non-fluent**
Wernicke's	Broca's
Transcortical sensory	Transcortical motor
Conduction	Global
Anomia	Mixed transcortical

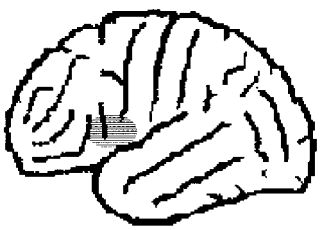
ANATOMIC LOCATION OF MAJOR SPEECH AREAS





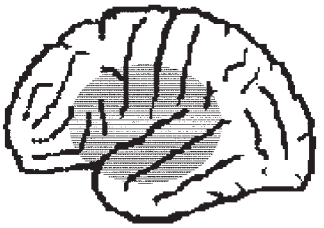
Wernicke's aphasia

📖 Location: posterior part of superior (first) temporal gyrus of the dominant (usually left) hemisphere
Characteristics:
- Fluent speech (normal rate/speed)
- Impaired comprehension
- Word deafness, difficulty in reading (alexia) and writing (agraphia)
- Marked paraphasias & neologisms



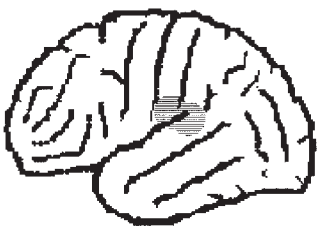
Broca's aphasia (remember "broken" speech)

Location: posterior-inferior frontal lobe (third frontal convolution) of dominant (usually left) hemisphere → anterior to motor cortex areas that supply the tongue, lips and larynx
Characteristics:
- Nonfluent speech (telegraphic)
- Impaired repetition
- Preserved comprehension
- Paraphasias & articulatory errors or struggle



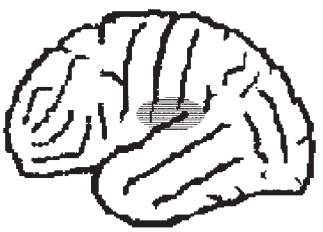
Global aphasia

Location: vary in size and location but usually involve distribution of the left MCA (entire perisylvian region)
Characteristics:
- Ranges from mutism (non-fluent) to total repetitive jargon or neologistic output (fluent but incomprehensible speech)
- Poor comprehension and repetition



Anomic aphasia

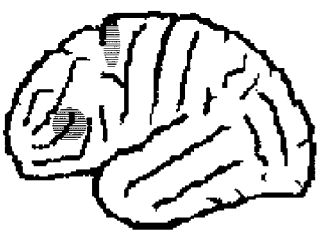
Location: temporo-parietal injury, angular gyrus
Characteristics:
- Fluent, essentially good comprehension and repetition
- Decreased output of nouns
- Word-finding difficulties
- Alexia and agraphia may be present



Conduction aphasia

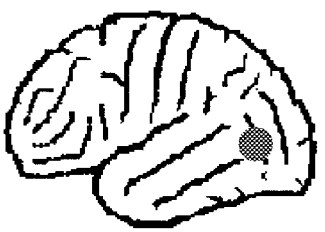
Location: lesion of the parietal operculum (arcuate fasciculus) or insula or deep to the supramarginal gyrus (usually left side)
Characteristics:
- Normal rate of speech (fluent)
- Preserved comprehension
- Impaired repetition
- Literal paraphasias with "targeting" of words (until getting the right one)

Note: **arcuate fasciculus** is a band of white matter running deep to the supramarginal gyrus and insula that joins Broca's and Wernicke's areas



Transcortical motor aphasia

Location: frontal lobe, anterior or superior to Broca's area or in the subcortical region deep to Broca's area
Characteristics:
- Reduced rate of speech, limited language output (with some fluent utterances)
- Reduced initiation & organization of speech good comprehension
- Preserved repetition



Transcortical sensory aphasia

Location: watershed lesion isolating perisylvian speech structures (Broca's and Wernicke's areas) from the posterior brain; angular gyrus or posterio-inferior temporal lobe
Characteristics:
- Poor comprehension
- Fluent speech (neologisms)
- Preserved repetition (possibly echolalia)

Transcortical mixed aphasia: Lesions in border zone of frontal, parietal, and temporal areas

Characteristics:
- Poor comprehension
- Nonfluent (decrease rate and initiation of speech)
- Preserved repetition (echolalia)

🕮 Note: Language areas are anatomically clustered around the sylvian fissure of the dominant hemisphere—left hemisphere in 95% of people.

Paraphasias: Incorrect substitutions of words or part of words

- Literal or phonemic paraphasias: similar sounds (e.g., "sound" for "found")
- Verbal or semantic paraphasias: word substituted for another from same semantic class (e.g., "fork" for "spoon")

🕮 Recovery Language Deficits/Aphasia Post Stroke:

The greatest amount of improvement in patients with aphasia occurs in the first two to three months after the onset; after six months, there is a significant drop in the rate of recovery.

In the majority of cases of patients with aphasia spontaneous recovery does not seem to occur after a year. However, there are reports of improvements many years after their stroke in patients undergoing therapy.

MEDICAL MANAGEMENT PROBLEMS

Poststroke Depression

- **Etiology:**
  - Organic: May be related to catecholamine depletion through lesion-induced damage to the frontal nonadrenergic, dopaminergic and serotonergic projections (Heilman and Valenstein, 1993)
  - Reactive: Grief/psychological responses for physical and personal losses associated with stroke, loss of control that often accompany severe disability, etc.
- Prevalence of depression in stroke patients reported ≈ 40% (25% to 79%); occur in similar proportion in their caregivers. (Flick, 1999)
- Most prevalent six months to two years
- A psychiatric evaluation for DSM-IV criteria and vegetative signs may be a clinically useful diagnostic tool in stroke patients
- There may be higher risk for major depression in left frontal lesions (relationship still controversial)
- Risk factors: prior psychiatric Hx, significant impairment in ADLs, high severity of deficits, female gender, nonfluent aphasia, cognitive impairment, and lack of social supports
- Persistent depression correlates with delayed recovery and poorer outcome
- Treatment: Active Tx should be considered for all patients with significant clinical depression
- Psychosocial interventional program: psychotherapy
- Medications: SSRIs preferred because of fewer side effects (compared to TCAs); methylphenidate has also been shown to be effective in poststroke depression
- SSRIs and TCAs also been shown to be effective in poststroke emotional lability

Sexual Dysfunction

- Well documented that the majority of elderly people continue to enjoy active and satisfying sexual relationships
- No significant changes in sexual interest or desire, but marked decline in behavior in both sexes (after stroke)
- There is a marked decline in sexual activity poststroke
- Fugl-Meyer (1980)—67 patients sexually active prestroke (Fugl-Meyer and Jaaski, 1980)
  - 36% remained active poststroke
  - 33% men resumed unaltered intercourse
  - 43% women resumed unaltered intercourse
  - Decreased frequency due to altered sensation, custodial attitudes taken by spouse

Other factors related to decrease in sexual activity poststroke:
Emotional factors—fear, anxiety and guilt; low self esteem; and fear of rejection by partner
Treatment: supportive psychotherapy, counseling.

Seizures

- Can be classified as occurring:
  - At stroke onset
  - Early after stroke (1–2 weeks)
  - Late after stroke (> 2 weeks)
- In prospective study after first time stroke, 27 of 1099 (2.5%) of patients had seizures within 48 hours postictus.
- Seizures associated with older age, confusion, and large parietal or temporal hemorrhages
- Majority of seizures were generalized tonic-clonic
- In-hospital mortality higher in patients with seizures
- Early seizures tend not to recur; these are associated with acute metabolic derangement associated with ischemia or hemorrhage.
- Stroke patients requiring inpatient rehabilitation have higher probability of developing seizures than the general stroke population
- Seizures occurring > 2 weeks after stroke have higher probability of recurrence
- In study with 77 ischemic stroke victims followed two to four years
  - 6%–9% develop seizures
  - 6/23 (26%) patients with cortical lesions develop seizures
  - 1/54 (2%) patients with subcortical lesions develop seizures
- Risk Factors: Cortical lesions, persistent paresis (6/12 = 50%)
- Treatment: choice of anticonvulsant drugs for patients with cerebral injury discussed in the TBI chapter.

FACTORS THAT PREDICT MORTALITY AND FUNCTIONAL RECOVERY IN STROKE PATIENTS

Mortality Factors

- Mortality of ischemic strokes in the first 30 days ranges from 17%–34%
- Hemorrhagic strokes are more likely to present as severe strokes and with mortality rate reported to be up to 48%

- Mortality in the first year after stroke 25% to 40%
- The risk of another stroke within the first year 12% to 25%

RISK FACTORS FOR ACUTE STROKE MORTALITY — 30 DAY MORTALITY
• Stroke severity • Low level of consciousness • Diabetes mellitus • Cardiac disease • Electrocardiograph abnormalities • Old age • Delay in medical care • Elevated blood sugar in non-diabetic • Brainstem involvement • Hemorrhagic stroke • Admission from nursing home

Functional Recovery and Disability Factors

- As stroke mortality has declined in the last few decades, the number of stroke survivors with impairments and disabilities has increased
- There are 300,000 to 400,000 stroke survivors annually
- 78% to 85% of stroke patients regain ability to walk (with or without assistive device)
- 48% to 58% regain independence with their self-care skills
- 10% to 29% are admitted to nursing homes

RISK FACTORS FOR DISABILITY AFTER STROKE
• Severe stroke (minimal motor recovery at 4 weeks) • Low level of consciousness • Diabetes mellitus • Cardiac disease • Electrocardiograph abnormalities • Old age • Delay in medical care • Delay in rehabilitation • Bilateral lesions • Previous stroke • Previous functional disability • Poor sitting balance • Global aphasia • Severe neglect • Sensory and visual deficits • Impaired cognition • Incontinence (>1–2 weeks)

Negative Factors of Return to Work (Black-Shaffer and Osberg, 1990)
- Low score on Barthel Index at time of rehabilitation discharge
- Prolonged rehabilitation length of stay
- Aphasia
- Prior alcohol abuse

(Barthel Index is a functional assessment tool that measures independence in ADLs on 0–100 scale)

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2

TRAUMATIC BRAIN INJURY

Elie Elovic, M.D., Edgardo Baerga, M.D., and Sara Cuccurullo, M.D.

INTRODUCTION

EPIDEMIOLOGY

- Trauma is the leading cause of death in ages 1–44 and more than half of these deaths are due to head trauma. Traumatic brain injury (TBI) is arguably the primary cause of neurologic mortality and morbidity in the United States
- About 500,000 per year traumatic brain injuries (requiring hospitalization) in the United States
- National Health Interview Survey (1985) provided the only national estimate of incidence of nonfatal TBI (hospitalized and non-hospitalized cases) for 1985–1987:
  - 1.975 million head injuries per year
  - Estimate probably includes head injuries in which no brain injury occurred
  - Fife (1987) concluded that only 16% of these injuries resulted in admission to a hospital
  - As a result of TBI Act, CDC is now sponsoring State Registries
- Overall incidence of traumatic brain injury 200 per 100,000 (150,000–235,000) per year (TBI cases requiring hospitalization)
  - Incidence is higher in males than in females (148–270 per 100,000 vs. 70–116 per 100,000)
- There is some evidence that TBI incidence is decreasing in the United States, comparing numbers from the 1970s and 1980s with more recent data (1990s) (Sosin, 1991)
- Peak ages 15–18 to 25 years
  - Age distribution is bimodal, with second peak in the elderly (ages 65–75); this group has a higher mortality rate
- Male to female ratio $\Rightarrow$ 2.5 : 1
  - Mortality in males is 3–4 times higher than in females
- Motor vehicle accident (MVA) is the most common cause (overall) of head injury in adolescents and adults (~ *50%* of cases)

  🕮 The single most common cause of death and injury in automobile accidents is ejection of the occupant from the vehicle. (Spitz, 1991)
- Violence/assault is the second most common cause of TBI in young adults
- ETOH use clearly related to TBI
  - Alcohol detected in blood in up to 86% of TBI patients
  - ETOH blood levels 0.10% or higher in 51% to 72% of patients at the time of the injury (Gordon, Mann, a nd Wilier, 1993)

TBI Model Systems Program from 1989 to 1998

Data collected from the 17 model system centers in the United States between March 1989 and Sept 1998, on 1,086 TBI cases: (U.S. Dept. of Ed., 1999)

- 58% MVA-related injuries
- 26% violence-related injuries
- 53% positive blood alcohol level at injury

Mortality in TBI

- There has been a change in trends from 1980s to 1990s in TBI mortality with success in decreasing deaths secondary to MVA, but failure to prevent injuries (and deaths) due to firearms/violence
- Study in TBI deaths from 1979 to 1992 (Sosin, Snizek, and Waxweiler, 1995)
  - Average 52,000 deaths per year in the United States secondary to TBI
  - There was a decline in overall TBI-related deaths of 22% from 1979 to 1992 (reasons not known, but may include more vehicles with air bags, increase in use of seat belts and other improvements in vehicle safety features, roadway safety improvements, etc.)
  - 25% decline in MVA-related deaths
  - 13% increase in firearm-related deaths
  - Firearms surpassed MVA as the largest single cause of death in TBI in 1990
- Mortality rate TBI : 14–30 per 100,000 per year
- Gunshot wound (GSW) to head—mortality risk 75%–80%
- The majority of GSW-related TBI are self-inflicted

Geriatrics

- The risk of TBI increases sharply after age 65
- TBI among the elderly are more frequently due to falls
- Severity of TBI among the elderly tends to be higher than that observed in other age groups; mortality is also higher in the elderly compared to other groups
- Predominant sex: none; male = female (grossly) (1.2 : 1 ratio)
  (National Institute on Disability and Rehabilitation Research, Traumatic Brain Injury Model Systems Program, 1999)

Pediatrics

- As mentioned above, TBI is the leading cause of death in children > 1 year of age
- 10 in every 100,000 children die each year secondary to head injuries
- Annual incidence of TBI in children—185 per 100,000
- Causes:
  - Transportation related (39%)
  - Falls (28%)
  - Sports and recreational activities (17%)
  - Assault (7%)

■

MECHANISM AND RECOVERY OF HEAD INJURY

MECHANISMS OF INJURY

Primary

Occurs at the moment of the impact and as a direct result of trauma

A. Contusions and lacerations of the brain surfaces—(Figure 2–1)
B. Diffuse axonal injury (DAI)—(Figure 2–2)
C. Diffuse vascular injury/multiple petechial hemorrhages
D. Cranial nerve injury

Secondary

Damage that occurs after the initial trauma and as a result of the injuring event. Most secondary injury occurs during the first 12 to 24 hours after trauma, but may occur up to 5 to 10 days postinjury in very severe brain injury. Because of the delayed presentation, secondary injury may be preventable.

A. Intracranial hemorrhage (epidural, subdural, subarachnoid and intracerebral hematoma)
B. Brain swelling/brain edema (see below)
C. Elevated Intracranial Pressure (ICP)
   – ↑ ICP ⇒ ↓ perfusion ⇒ ischemic brain damage
D. Brain damage secondary to hypoxia
E. Intracranial infection
F. Hydrocephalus
G. ↑ release of excitatory neurotransmitters secondary to diffuse axonal injury (DAI) = excitotoxicity
   – This will increase the activity of certain brain areas and overall metabolic demand in the already injured brain
H. Production of free-radical molecules

Other secondary causes of brain injury include:

- Hypotension
- Electrolyte imbalances
- Anemia
- Hyperthermia
- Hyperglycemia
- Hypercarbia
- Hypoglycemia
- Hyperemia
- Hyponatremia
- Infection
- Carotid dissection
- Epilepsy/seizures
- Vasospasm/ischemia

Primary Head Injury

Contusion—bruising of cerebral (cortical) tissue

- Occurs on the undersurface of the frontal lobe (inferior frontal or orbitofrontal area) and anterior temporal lobe, regardless of the site of impact (Figure 2–1)
- May produce focal, cognitive, and sensory-motor deficits
- Is not directly responsible for loss of consciousness following trauma
- May occur from relatively low velocity impact, such as blows and falls

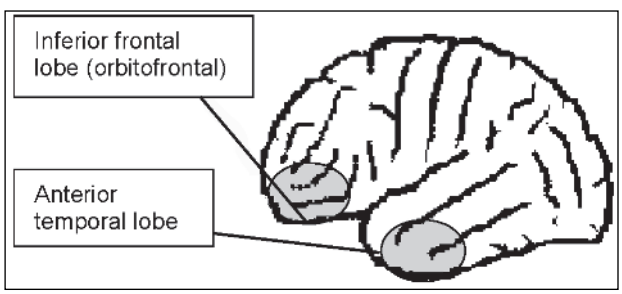

FIGURE 2–1. Location of Contusions

Diffuse axonal injury (DAI):

- DAI is seen exclusively in TBI
- Damage seen most often in the corpus callosum and other midline structures involving the parasagittal white matter, the interventricular septum, the walls of the third ventricle and the brain stem (midbrain and pons) (Figure 2–2)

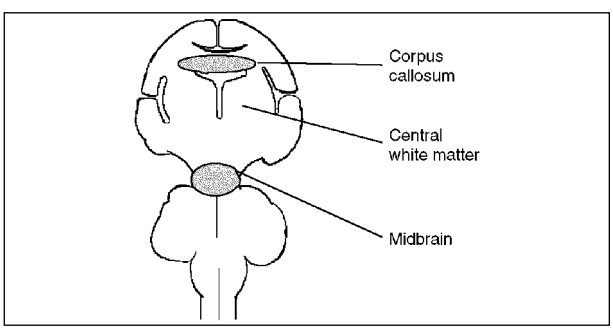

FIGURE 2–2. Locations of Diffuse Axonal Injury

- Responsible for the initial loss of consciousness seen in acute TBI
- Results from acceleration-deceleration and rotational forces associated with high-velocity impact (MVAs)
- The axonal injury seen in severe TBI is thought to be secondary to damage to the axoplasmic transport in axons (with ↑ Ca^{++} influx) leading to axonal swelling and detachment

Secondary Head Injury

Brain Swelling

- Occurs after acute head injury within 24 hours.
- Identified in CT as collapse of ventricular system and loss of cerebral spinal fluid (CSF) cisterns around the midbrain
- Is due to an increase in cerebral blood volume (intravascular blood)

Brain Edema

- Occurs later after head injury (in comparison to brain swelling)
- Is due to an increase in brain volume secondary to ↑ brain water content ⇒ extravascular fluid
- Two types:
  1. **Vasogenic** edema:
     - Due to outpouring of protein rich fluid through damaged vessels
     - *Extracellular* edema
     - Related to cerebral contusion
  2. **Cytogenic** edema:
     - Found in relation to hypoxic and ischemic brain damage
     - Due to failing of the cells' energy supply system ⇒ ↑ cell-wall pumping system ⇒ *intracellular* edema in the dying cells

PENETRATING HEAD INJURIES

Missile/Fragments

- Deficits are focal corresponding to location of lesions caused by bullet/fragment
- If the brain is penetrated at the lower levels of the brain stem, death is instantaneous from respiratory and cardiac arrest. 80% of patients with through-and-through injuries die at once or within a few minutes
- The mortality rate of patients who are initially comatose from gunshot wounds to head is 88%; this is more than twice the mortality from closed-head injury (CHI)
- Focal or focal and generalized seizures occur in the early phase of the injury in 15% to 20% of cases. Risk of long-term posttraumatic epilepsy is higher in penetrating head injuries compared to nonpenetrating injuries

RECOVERY MECHANISMS

🕮 Plasticity

- Brain plasticity is when the damaged brain has the capabilities to repair itself by means of morphologic and physiologic responses
- Plasticity is influenced by the environment, complexity of stimulation, repetition of tasks, and motivation

It occurs via 2 mechanisms:

1) Neuronal regeneration/neuronal (collateral) sprouting
2) Unmasking neural reorganization

Neuronal Regeneration

Intact axons establish synaptic connections through dendritic and axonal sprouting in areas where damage has occurred

- May enhance recovery of function, may contribute to unwanted symptoms, or may be neutral (with no increase or decrease of function)
- Thought to occur weeks to months post-injury

Functional Reorganization/Unmasking

Healthy neural structures not formerly used for a given purpose are developed (or reassigned) to do functions formerly subserved by the lesioned area.

> Brain plasticity—remember "PUN"
> Plasticity = Unmasking + Neuronal sprouting

OTHER RELATED PHENOMENA ASSOCIATED WITH HEAD INJURY RECOVERY

Synaptic Alterations

Includes diaschisis and increased sensitivity to neurotransmitter levels

📖 **Diaschisis**: Mechanism to explain spontaneous return of function (Figure 2–3)

1. Lesions/damage to one central nervous system (CNS) region can produce altered function in other areas of the brain (at a distance from the original site of injury) that were not severed if there is connection between the two sites (through fiber tracts). Function is lost in both injured and in morphologically intact brain tissue.
2. There is some initial loss of function secondary to depression of areas of the brain connected to the primary injury site, and resolution of this functional deafferenation parallels recovery of the focal lesion (Feeney, 1991).

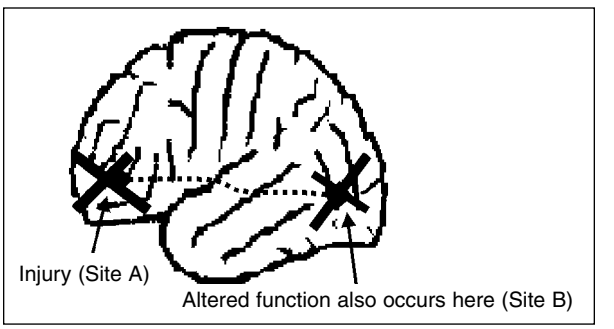

FIGURE 2–3. Example of Diaschisis: Injury to site A will produce inhibition on function at site B, which was not severed by the initial injury and is distant from the original site of injury (site A). Recovery of functions controlled by site B will parallel recovery of site A

Functional Substitution/Behavioral Substitution

Techniques/new strategies learned to compensate for deficits and achieve a particular task

Other Theories of Recovery Include

- **Redundancy**: Recovery of function based on activity of uninjured brain areas (latent areas) that normally would contribute to that function (and are capable of subserving that function)
- **Vicariation**: Functions taken over by brain areas not originally managing that function. These areas alter their properties in order to subserve that function.

DISORDERS OF CONSCIOUSNESS

LOCATION OF CONTROL OF CONSCIOUSNESS

🕮 Consciousness

Consciousness is a function of ascending reticular activating system (RAS) and the cerebral cortex

RAS is composed of cell bodies in the central reticular core of the upper brain stem (mainly midbrain) and their projections to widespread areas of the cerebral cortex via both the thalamic and the extrathalamic pathways.

Lesions that interrupt the metabolic or structural integrity of the RAS or enough of the cortical neurons receiving RAS projections can cause coma.

DISORDERS OF CONSCIOUSNESS

Coma

- It is a state of unconsciousness from which the patient cannot be aroused; there is no evidence of self- or environmental-awareness
- Coma is essentially universal in severe TBI
- Up to 50% of patients in coma > 6 hours die without ever regaining consciousness. Survivors who remain unresponsive for > 2–4 weeks evolve into vegetative state
- Eyes remain continuously closed
- No sleep-wake cycles on electroencephalogram (EEG)
- There is ***no*** spontaneous purposeful movement (e.g., patient will not scratch himself, will not grab bedrail); there is inability to discretely localize noxious stimuli
- No evidence of language comprehension or expression
- It is believed that transition from coma to vegetative state signals the return of brain stem arousal mechanisms and that persistent unconsciousness reflects damage to the thalamus and/or global cortical and subcortical damage

Vegetative State (VS)

- **Definition:** Loss of capacity to interact with the environment despite the preserved potential for spontaneous or stimulus-induced arousal (due to absence of cortical activity)
- **Characteristics:**
  - Patient opens eyes (either spontaneously or with noxious stimuli)
  - VS is characterized by the presence of intermittent wakefulness evidenced by sleep-wake cycles (these may be demonstrated on EEG)
  - In VS there is no perceivable evidence of purposeful behavior
  - There is no evidence of intelligible verbal or gestural communication
  - This condition may persist for years following TBI, but this is very rare
  - Coma and VS are characterized by the absence of function in the cerebral cortex as judged behaviorally
- The term persistent vegetative state (redefined by The Multi-Society Task Force on PVS, 1994) is used for vegetative state that is present ≥ one month after a traumatic or nontraumatic brain injury

The Task Force also introduced the term *permanent vegetative state* to denote irreversibility after 3 months following nontraumatic brain injury and 12 months following traumatic brain injury (Howsepian, 1996).

Persistent VS	VS present ≥ 1 month after TBI or Nontraumatic brain injury
Permanent VS	VS present > 3 months after Nontraumatic brain injury or VS present > 12 months after TBI, in both children and adults

- American Congress of Rehabilitation Medicine (1995)—advocates to simply use the term vegetative state (VS) followed by the length of time it persists instead of the terms persistent and permanent. The Aspen Neurobehavioral Conference (1996), supported the ACRM recommendations to use the term VS + specify cause of injury + specify length of time since onset.
- Neuropathology of VS ⇒
  - Related to diffuse cortical injury
  - Bilateral thalamic lesions are prominent findings in VS
- Transition to VS, when preceded by coma, is signaled by re-emergence of eye opening and spontaneous control of autonomic functions.
  - Vegetative or autonomic functions include respiration, cardiovascular, thermoregulatory and neuroendocrine functions
- Visual tracking is considered a signal of transition out of VS

Minimally Conscious State (MCS)

- Severely altered consciousness with minimal but definite behavioral evidence of self- or environmental-awareness.
- Patients in MCS demonstrate inconsistent but definite reproducible behavioral evidence of self-awareness or awareness of the environment
- There is evidence that the following behaviors are reproducible (or sustained), but inconsistent:
  - Simple command following
  - Object manipulation
  - Intelligible verbalization
  - Gestural or verbal yes/no responses
- Patient may also show:
  - Visual fixation
  - Smooth pursuit tracking
  - Emotional or motor behaviors that are contingent upon the presence of specific eliciting stimuli [e.g., patient will cry or get agitated (and behavior is reproducible) only after hearing voices of family members but not with voices of hospital staff]
- Emergence from MCS is signaled by:
  - Consistent command following
  - Functional object use
  - Reliable use of a communication system
- Prognosis is better for MCS than for VS

Treatment

- There is no evidence to support that any kind of therapy-based program (e.g., coma stimulation/sensory-stimulation program) will induce or accelerate the cessation of coma or VS

- Nevertheless, an organized treatment approach to low-functioning patients permits a quantifiable assessment of responses to stimulation and early recognition of changes or improvements in response to therapeutic interventions or through spontaneous recovery

Management/Therapy Program for Patients with Disorders of Consciousness

- Neuromedical stabilization
- Preventive therapeutic interventions may be implemented:
  - Manage bowel and bladder (B/B) function
  - Maintain nutrition
  - Maintain skin integrity
  - Control spasticity
  - Prevent contractures
- Pharmacologic treatment/intervention
  A. Elimination of unnecessary medicines (e.g., histamine-2 blockers, metoclopramide, pain meds, etc.) and selection of agents with fewest adverse effects on cognitive and neurologic recovery
  B. Addition of agents to enhance specific cognitive and physical functions
     - In patients emerging out of coma or VS, the recovery process may be (theoretically) hastened through the use of pharmacotherapy
     - Agents frequently used include:
       - Methylphenidate
       - Dextroamphetamine
       - Dopamine agonists (levocarbidopa and carbidopa)
       - Amantadine
       - Bromocriptine
       - Antidepressants—tricyclic antidepressants (TCA's) & selective serotonin reuptake inhibitors (SSRIs)
     - The efficacy of pharmacologic therapy to enhance cognitive function has not been proven
- Sensory stimulation—widely used despite little evidence of efficacy as previously mentioned.
  - Sensory stimulation should include all five senses, addressed one at a time, in specific therapy sessions and/or in the environmental state and developed in the room
  - Avoid overstimulation (educate family)
  - Patient may have adverse responses due to overstimulation, as ↑ confusion or agitation, ↑ reflex responses or avoidance reactions, which may interfere with performance

POSTURING SECONDARY TO HEAD INJURY

🕮 DECEREBRATE POSTURING

- This postural pattern was first described by Sherrington, who produced it in cats and monkeys by transecting the brain stem
- There is extension of the upper and lower extremities (hallmark: elbows extended) (Figure 2–4 A)

- Seen with midbrain lesions/compression; also with cerebellar and posteria fossa lesions
- In its fully developed form it consists of opisthotonus, clenched jaws, and stiff, extended limbs with internal rotation of arms and ankle plantar flexion (Feldman, 1971)

DECORTICATE POSTURING

- Posturing due to lesions at a higher level (than in decerebrate posture)—seen in cerebral hemisphere/white matter, internal capsule and thalamic lesions
- Flexion of the upper limbs (elbows bent) and extension of the lower limbs (Figure 2–4 B)

> **Hint:**
> Remember, de***COR***ticate → "**COR**" = heart = ♥
> ⇒ Patient brings hands close to his heart by flexing the elbows

- Arms are in flexion and adduction and leg(s) extended

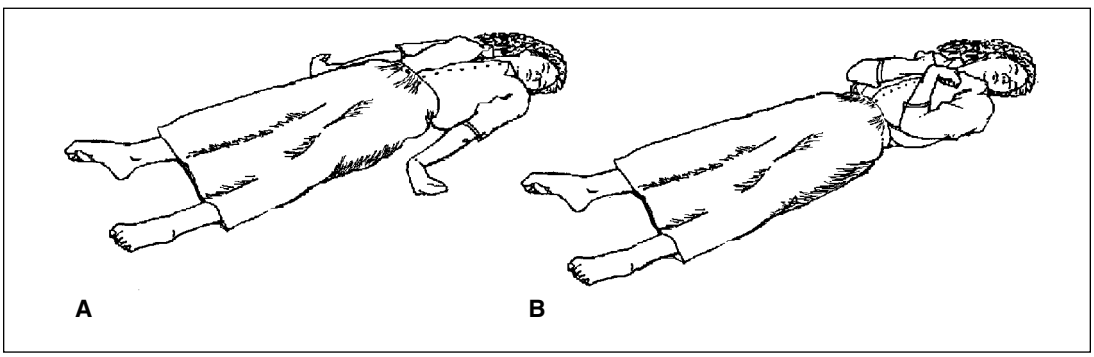

FIGURE 2–4. **A** Decerebrate Posture: There is extension of the upper and lower extremities. **B** Decorticate Posture: There is flexion of the upper extremities and extension of the lower limbs.

■ PREDICTORS OF OUTCOME AFTER TBI

🕮 WIDELY USED INDICATORS OF SEVERITY IN ACUTE TBI

- The best Glasgow Coma Scale (GCS) score within 24 hours of injury
- Length of coma
- Duration of posttraumatic amnesia (PTA)
  - Note: The *initial* GCS and the *worst* GCS (within the first 24 hours) have also been proposed as acute indicators of severity in TBI

Glasgow Coma Scale

TABLE 2–1 🕮 Glasgow Coma Scale: (Teasdate and Jennett, 1974)

Score	Best Motor Response 6	Best Verbal Response 5	Eye Opening 4
1	None	None	None
2	Decerebrate posturing (extension) to pain	Mutters unintelligible sounds	Opens eyes to pain
3	Decorticate posturing (flexion) to pain	Says inappropriate words	Opens eyes to loud voice (verbal commands)
4	Withdraws limb from painful stimulus	Able to converse—confused	Opens eyes spontaneously
5	Localizes pain/pushes away noxious stimulus (examiner)	Able to converse—alert and oriented	
6	Obeys verbal commands		

- Total GCS score is obtained from adding the scores of all three categories.
- Highest score = 15
- Lowest score = 3
- With GCS score < 8: patient is said to be comatose
- The lower the score, the deeper the coma

🕮 The GCS is a simple scoring for assessing the depth of coma

- Highest GCS score within the first few hours after the injury preferred as this reduces the likelihood of using excessively low, very early scores (often before cardiopulmonary resuscitation (CPR)) and of confounding factors such as decreased arousal due to use of sedatives or paralytic agents
- Severity of injury:
  GCS score of 3 to 8 = severe injury
  9 to 12 = moderate injury
  13 to 15 = mild injury
- 🕮 Of the three items in GCS, *best motor response* is the best acute predictor of outcome
- Jennet (1979): Relationship between best GCS score (within the first 24 hours) and outcome:
  - GCS scores of 3–4 resulted in death or VS in 87% of patients
  - Scores 5–7 = death or VS in 53% and moderate or good recovery in 34%
  - Scores 8–10 = moderate or good recovery in 68%
  - Score of 11 = moderate or good recovery in 87%
- Glasgow-Liege scale: Born (1985) proposed the addition of the brain stem reflex scale to the Glasgow Coma Scale—Glasgow-Liege Scale

The Glasgow-Liege Scale has been tested for reliability and prognostic power and has been shown to amplify the information provided by the standard GCS in comatose patients

Brain stem reflexes included in this scale:

- Fronto-orbicular reflex (orbicularis oculi): Orbicularis oculi contraction on percussion of the glabella
- Vertical oculocephalic and horizontal oculocephalic or oculovestibular reflex: "Doll's eye" maneuver (horizontal—moving head forward from side to side; vertical—moving head up and down)
- The pupillary light reflex
- The oculocardiac reflex: Bradycardia induced by increasing pressure on the eyeball

Duration of Posttraumatic Amnesia (PTA)

- PTA is one of the most commonly used predictors of outcome
- PTA is the interval of permanently lost memory that occurs following the injury
- 🕮 Resolution of PTA clinically corresponds to the period when incorporation of ongoing daily events occurs in the working memory
- Duration of PTA has a proportional relationship to coma duration. Katz and Alexander (1994)—PTA correlates with Glasgow Outcome Scale (GOS) score at 6 and 12 months—predictor of outcome
- PTA correlates strongly with length of coma (and with GOS—see below) in patients with DAI but poorly in patients with primarily focal brain injuries (contusions)
- 🕮 Galveston Orientation and Amnesia Test (GOAT)—developed by Harvey Levin and colleagues, is a standard technique for assessing PTA. It is a brief, structured interview that quantifies the patient's orientation and recall of recent events
  - The GOAT includes assessment of orientation to person, place, and time, recall of the circumstances of the hospitalization, and the last preinjury and first postinjury memories
  - The GOAT score can range from 0 to 100, with a score of 75 or better defined as normal
  - The end of PTA can be defined as the date when the patient scores 75 or higher in the GOAT for two consecutive days. The period of PTA is defined as the number of days beginning at the end of the coma to the time the patient attains the first of two successive GOAT scores ≥ 75 (Ellenberg, 1996)
- Categories of PTA: Duration of PTA is often used to categorize severity of injury according to the following criteria:

TABLE 2–2. Posttraumatic Amnesia

Duration of PTA	Severity of Injury Category
Less than 5 minutes	Very mild
5–60 minutes	Mild
1–24 hours	Moderate
1–7 days	Severe
1–4 weeks	Very severe
Greater than 4 weeks	Extremely severe

TABLE 2–3. Classification of Posttraumatic Amnesia

Length of PTA	Likely Outcome
1 day or less	Expect quick and full recovery with appropriate management (a few may show persisting disability)
More than 1 day, less than 1 week	Recovery period more prolonged—now a matter of weeks or months. Full recovery possible, for most of these cases, with good management.
1–2 weeks	Recovery a matter of many months. Many patients are left with residual problems even after the recovery process has ended, but one can be reasonably optimistic about functional recovery with good management.
2–4 weeks	Process of recovery is very prolonged—1 year or longer is not unusual. Permanent deficits are likely. There must be increasing pessimism about functional recovery when PTA reaches these lengths.
More than 4 weeks	Permanent deficits, indeed significant disability, now certain. It is not just a matter of recovery but of long-term retraining and management.

From Brooks DN and McKinlay WW, Evidence and Quantification in Head Injury: Seminar notes. Unpublished material, 1989, with permission.

Duration of Coma

- Katz and Alexander (1994): defined as the date when the patient shows the first unequivocal sign of responsiveness. In this study, the sign of responsiveness used was evidence of the patient following commands

Other Indicators of Outcome after TBI Include:

- **Age**
  - Children and young adults tend to have a generally more positive prognosis than older adults. However, young children (< 5 yrs) and older adults (> 65 yrs) have greater mortality
  - Katz and Alexander (1994): Age ≥ 40 correlates with worse functional outcome when compared with patients < 40
- **Rate of early recovery** reflected in serial disability rating scales (DRS): found to be predictive of final outcome
- **Pupillary reaction** to light:
  - 50% of patients with reactive pupils after TBI achieve moderate disability to good recovery (in DRS scale) vs 4% with nonreactive pupils
- **Time**
  - Most recovery usually occurs within the first 6 months postinjury
- **Postcoma use of phenytoin:**
  - Long-term use of phenytoin has been reported to have adverse cognitive effects (neurobehavioral effects in severe TBI patients compared to placebo group)

HEAD INJURY PREDICTOR SCALES AND TESTING

🕮 Prognosis in Severe Head Injury

TABLE 2–4

Predicative Indicator	Poorer	Better
Glasgow Coma Scale score	< 7	> 7
CT scan	Large blood clot; massive bihemispheric swelling	Normal
Age	Old age	Youth
Pupillary light reflex	Pupils remain dilated	Pupil contracts
Doll's eye sign	Impaired	Intact
Caloric testing with ice water	Eyes do not deviate	Eyes deviate to irrigated side
Motor response to noxious stimuli	Decerebrate rigidity	Localizes defensive gestures
Somatosensory evoked potentials	Deficient	Normal
Posttraumatic amnesia length	> 2 wks	< 2 wks

(Reprinted with permission from Braddom, RL. Physical Medicine and Rehabilitation. Philadelphia: W.B. Saunders Company; 1996: p. 1033, table 49-4.)

🕮 Glasgow Outcome Scale (GOS)

TABLE 2–5

	Category	Description
1	Death	Self-evident criteria
2	Persistent vegetative state	Prolonged unconsciousness with no verbalization, no following of commands. Absent awareness of self and environment; patient may open eyes; absence of cortical function as judged behaviorally; characterized by the presence of sleep-wake cycles
3	Severe disability	Patient unable to be independent for any 24-hour period by reason of residual mental and/or physical disability
4	Moderate disability	Patient with residual deficits that do not prevent independent daily life; patient can travel by public transport and work in a sheltered environment
5	Good recovery	Return to normal life; there may be minor or no residual deficits

- Widely used scale; documented correlation between acute predictors of outcome and GOS score at 6 months and 12 months
- Cons:
  - In the GOS, categories are broad; scale not sensitive enough
  - Not real indicator of functional abilities

(*Continued*)

🕮 Disability Rating Scale (DRS)

TABLE 2–6

1. Eye Opening	2. Communication	3. Motor Response
0 Spontaneous	0 Oriented	0 Obeying
1 To Speech	1 Confused	1 Localizing
2 To Pain	2 Inappropriate	2 Withdrawing
3 None	3 Incomprehensible	3 Flexing
	4 None	4 Extending
		5 None

4. Feeding	5. Toileting	6. Grooming
0.0 Complete	0.0 Complete	0.0 Complete
0.5	0.5	0.5
1.0 Partial	1.0 Partial	1.0 Partial
1.5	1.5	1.5
2.0 Minimal	2.0 Minimal	2.0 Minimal
2.5	2.5	2.5
3.0 None	3.0 None	3.0 None

7. Level of functioning (physical and cognitive disability)	8. "Employability" (as full-time worker, homemaker, or student)
0.0 Completely independent	0.0 Not restricted
0.5	0.5
1.0 Dependent in special environment	1.0 Selected jobs, competitive
1.5	1.5
2.0 Mildly dependent—limited assistance (nonresident helper)	2.0 Sheltered workshop, noncompetitive
2.5	2.5
3.0 Moderately dependent—moderate assistance (person in home)	3.0 Not employable
3.5	
4.0 Markedly dependent—assist all major activities, all times	
4.5	
5.0 Totally dependent—24-hr nursing care	

(Rappaport et al., 1982)

This is a 30-point scale covering the following eight dimensions:

1. Eye opening
2. Verbalization/communication
3. Motor responsiveness
4. Feeding*
5. Toileting*
6. Grooming*
7. Overall level of functioning/dependence
8. Employability

*Note: measuring cognitive skills only in these categories.

- The DRS was developed specifically for brain injury
- It provides a quantitative index of disability
- It is more sensitive to clinical changes than GOS

Rancho Los Amigos Levels of Cognitive Function Scale (LCFS)

TABLE 2–7

Level	Description
I	No response
II	Generalized response to stimulation
III	Localized response to stimuli
IV	Confused and agitated behavior
V	Confused with inappropriate behavior (nonagitated)
VI	Confused but appropriate behavior
VII	Automatic and appropriate behavior
VIII	Purposeful and appropriate behavior

(Reprinted with Permission from Rancho Los Amigos National Rehabilitation Center.)

- Eight-level global scale that focuses on cognitive recovery and behavior after TBI
- (Gouvier et al., 1987)—LCFS has lower validity and reliability than the Disability Rating Scale (DRS)

Functional Independence Measure (FIM)

- Ordinal scale with 18 items and 7 levels to assess physical and cognitive function
- Documented validity and reliability

TABLE 2–8 FIM

LEVELS		
	7 Complete Independence (Timely, Safely) 6 Modified Independence (Device)	NO HELPER
	Modified Dependence 5 Supervision 4 Minimal Assist (Subject = 75% +) 3 Moderate Assist (Subject = 50% +) Complete Dependence 2 Maximal Assist (Subject = 25% +) 1 Total Assist (Subject = 0% +)	HELPER

	ADMIT	DISCHG	FOL-UP
Self-Care			
A. Eating	☐	☐	☐
B. Grooming	☐	☐	☐
C. Bathing	☐	☐	☐
D. Dressing - Upper Body	☐	☐	☐
E. Dressing - Lower Body	☐	☐	☐
F. Toileting	☐	☐	☐
Sphincter Control			
G. Bladder Management	☐	☐	☐
H. Bowel Management	☐	☐	☐
Transfers			
I. Bed, Chair, Wheelchair	☐	☐	☐
J. Toilet	☐	☐	☐
K. Tub, Shower	☐	☐	☐
Locomotion			
L. Walk/Wheelchair	☐ Walk / Wheelchair / Both	☐ Walk / Wheelchair / Both	☐ Walk / Wheelchair / Both
M. Stairs	☐	☐	☐
Motor Subtotal Score	☐	☐	☐
Communication			
N. Comprehension	☐ Auditory / Visual / Both	☐ Auditory / Visual / Both	☐ Auditory / Visual / Both
O. Expression	☐ Vocal / Non-vocal / Both	☐ Vocal / Non-vocal / Both	☐ Vocal / Non-vocal / Both
Social Cognition			
P. Social Interaction	☐	☐	☐
Q. Problem Solving	☐	☐	☐
R. Memory	☐	☐	☐
Cognitive Subtotal Score	☐	☐	☐
Total FIM	☐	☐	☐

NOTE: Leave no blanks; enter 1 if patient not testable due to risk

(FIM™ Guide for Uniform Data Set for Medical Rehabilitation, SUNY-Buffalo, 1996.)

Coma Recovery Scale (CRS)

- Theorizes hierarchical responses (from generalized to cognitively mediated) for 25 items in 6 areas: auditory, visual, motor, oromotor/verbal, communication, and arousal
- Giacino et al. (1991) found that changes in CRS scores showed stronger correlations with outcome (as measured by the Disability Rating Scale) than initial, one-time scores. The change as a percentage of total score was greater for the CRS than for the GCS or DRS (Horn and Zasler 1996)

🕮 Neuropsychological Testing

Prior to the development of the CT Scan, neuropsychological assessment was targeted at determining whether a brain lesion was or was not present, and, if present, discerning its location and type

This diagnostic approach supported the development of the *Halstead-Reitan Neuropsychological Battery (HRNB).* This battery was initially designed to assess frontal-lobe disorders by W.C. Halstead (1947) and subsequently used by Reitan (1970 1974), who added some tests and recommended its use as a diagnostic test for all kinds of brain damage. Most examiners administer this battery in conjunction with the WAIS-R (Wechsler Adult Intelligence Scale—Revised) and WMS (Wechsler Memory Scale) or the Minnesota Multiphasic Personality Inventory (MMPI)

- *Wechsler Adult Intelligence Scale—Revised (WAIS-R):* eleven subtests (6 determine verbal IQ and 5 determine performance IQ), WAIS-R is the most frequently used measure of general intellectual ability.
- *Minnesota Multiphasic Personality Inventory (MMPI)* consists of 550 true/false questions that yield information about aspects of personality. It is the most widely and thoroughly researched objective measure of personality.

■ MEDICAL COMPLICATIONS AFTER TBI

Posttraumatic Hydrocephalus (PTH)

- Ventriculomegaly (ventricular dilation) is common after TBI , reported in 40%–72% of patients after severe TBI. However, true hydrocephalus is relatively rare; incidence is 3.9 to 8%
- Ventriculomegaly is usually due to cerebral atrophy and focal infarction of brain tissue (*ex vacuo* changes)
- Hydrocephalus in TBI is most often of the communicating or normal-pressure type
- Unfortunately, the classic triad of incontinence, ataxia/gait disturbance and dementia is of little help in severe TBI cases
- Radiographic evaluation (CT Scan) and further work-up (to rule out hydrocephalus) should be considered if there is failure to improve or deterioration of cognitive or behavioral function
- CT-Scan—periventricular lucency, lack of sulci, and uniformity in ventricular dilation favors PTH
- Initial manifestations of hydrocephalus can be intermittent HA, vomiting, confusion, and drowsiness
- Tx: Lumbar puncture, shunt placement

Elevated Intracranial Pressure (ICP)

- In a normal adult, reclining with the head and the trunk elevated to 45°, the ICP is between 2 to 5 mmHg
- ICP levels up to 15 mmHg are considered harmless
- Raised ICP: defined as ICP > 20 mmHg for more than 5 minutes
- Common after severe TBI (53% reported in a recent series)
- When ICP > 40 mmHg, there is neurologic dysfunction and impairment of the brain's electrical activity
- An ICP > 60 mmHg is invariably fatal; pressures in 20–40 mmHg area associated with increased morbidity
- 75% of the patients post severe TBI die due to deformation of tissue, shift, the development of internal hernias and secondary damage to the CNS
- If unchecked an increased ICP may cause death mainly because of deformation of tissue, brain shifts, herniation, and cerebral ischemia
- A unilateral mass lesion causes distortion of the brain, a reduction of the CSF volume, and, in the closed skull, the formation of internal hernias (including tentorial/uncal herniation—see below)
- Increased ICP reduces cerebral blood perfusion
- It is more important to maintain an adequate cerebral perfusion pressure (CPP) than controlling only the ICP
- Cerebral perfusion is calculated by subtracting ICP from mean arterial pressure (MAP). It should remain > 60 mmHg to ensure cerebral blood flow
- CPP = MAP—ICP
- Fever, hyperglycemia, hyponatremia, and seizures can worsen cerebral edema by ↑ ICP

Indications for Continuous Monitoring of Intracranial Pressure and for Artificial Ventilation
1. Patient in coma (GCS < 8) and with CT findings of ↑ ICP (absence of third ventricle and CSF cisterns) 2. Deep coma (GCS < 6) without hematoma 3. Severe chest and facial injuries and moderate/severe head injury (GCS < 12) 4. After evacuation of IC hemorrhage if patient is in coma (GCS < 8) beforehand

Factors that May Increase ICP

- Turning head, especially to left side if patient is completely horizontal or head down
- Loud noise
- Vigorous physical therapy
- Chest PT
- Suctioning
- Elevated blood pressure

Methods Used to Monitor ICP

- Papilledema: papilledema is rare in the acute stage after brain injury, despite the fact that ↑ ICP is frequent
  - Usually occurs bilaterally
  - May indicate presence of intracranial mass lesion
  - Develops within 12 to 24 hours in cases of brain trauma and hemorrhage, but, if pronounced, it usually signifies brain tumor or abscess, i.e., a lesion of longer duration
- CT Scan (see earlier)—if CT-Scan equivocal, cysternography may be done

- Lumbar puncture (LP) if no papilledema (must rule out mass lesion first)
  - LP carries a certain risk of causing fatal shift of brain tissue (i.e., herniation)

Management of ICP

- Elevate head of bed 30°
- Intubation and hyperventilation: reduction of $PaCO_2$ through hyperventilation is the most rapid mean of lowering ICP. However, it may negatively impact outcome
  - Hyperventilation should be used with caution as it reduces brain tissue PO_2 this may cause brain tissue hypoxia ⇒ this may lead to ischemia ⇒ ischemia may cause further damage in the CNS tissue of the head injury (HI) patient
  - Optimal $PaCO_2$ ~ 30 mmHg
- Osmotic agents (e.g., mannitol)—improves ischemic brain swelling (by diuresis and intravascular fluid shifts)
- Furosemide/acetazolamide may also be used
- Avoid HTN: can increase brain blood volume and increase ICP
- High doses of barbiturates (e.g., thiopental) rapidly lower ICP and suppress electrical brain activity
- Neurosurgical decompression
- Hypothermia may be used to ↓ ICP and it may protect brain tissue by lowering cerebral metabolism. Marion (1997)—treatment with hypothermia for 24 hours in severe TBI patients (GCS 5–7) associated with improved outcome
- Steroids—not proven to be beneficial management of ICP

Temporal Lobe—Tentorial (UNCAL) Herniation

- Uncal herniation results when the medial part of one temporal lobe (uncus and parahippocampal gyrus) is displaced over the edge of the ipsilateral tentorium so as to compress the third cranial nerve, midbrain, cerebral cortex, and subthalamus
- Occurs as a result of increased supratentorial pressure. It is commonly associated with hematoma (subdural or epidural) secondary to trauma or to a brain tumor
- Uncal herniation of the medial temporal lobe produces:
  1. Stretching of the third cranial nerve (oculomotor nerve) causes ipsilateral pupillary dilation; this may lead to complete ipsilateral third nerve palsy (with fixed pupil dilation, ptosis, and later, ophthalmoplegia)
  2. Ipsilateral hemiparesis results due to pressure on the corticospinal tract located in the contralateral crus cerebri
  3. Contralateral hemiparesis may result due to pressure (from edema or mass effect) on the precentral motor cortex or the internal capsule
- In uncal herniation, reduced consciousness and bilateral motor signs appear relatively late. Central hyperventilation may also occur late in uncal herniation

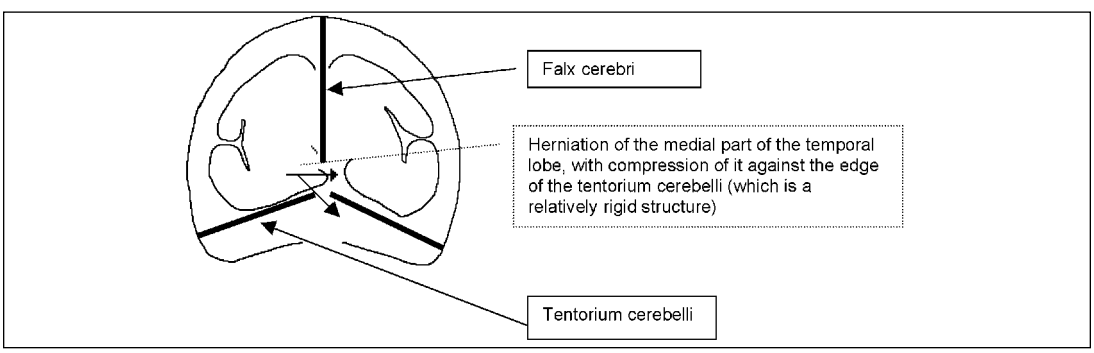

FIGURE 2–5. Temporal Lobe—Tentorial (Uncal) Herniation

Heterotopic Ossification (HO)

- HO is the formation of mature lamellar bone in soft tissue
- Common in TBI, with an incidence of 11%–76% (incidence of clinically significant cases is 10%–20%)

🕮 **Risk factors:**
- Prolonged coma (> 2 weeks)
  - Immobility
  - Limb spasticity/↑ tone (in the involved extremity)
  - Associated long-bone fracture
  - Pressure ulcers
  - Edema
- Period of greater risk to develop HO: 3 to 4 months post injury

Signs/Symptoms
- Most common: pain and ↓ range of motion (ROM)
- Also: local swelling, erythema, warmth joint, muscle guarding, low-grade fever
- In addition to pain and ↓ ROM, complications of HO include bony ankylosis, peripheral nerve compression, vascular compression, and lymphedema
- Joints most commonly involved:
  1. Hips (most common)
  2. Elbows/shoulders
  3. Knees

Differential Dx: DVT, tumor, septic joint, hematoma, cellulitis, and fracture

Diagnostic Tests/Labs

Serum Alkaline Phosphatase (SAP)
- SAP elevation may be the earliest and least expensive method of detection of HO
- It has poor specificity (may be elevated for multiple reasons, such as fractures, hepatic dysfunction, etc.)

Bone Scan
- Is a sensitive method for early detection of HO
- HO can be seen within the first 2–4 weeks after injury in Phase I (blood-flow phase) and Phase II (blood-pool phase) of a triple phase bone scan, and in Phase III (static phase/delayed images) in 4–8 weeks with normalization by 7 to 12 months

Plain X-rays
- Require 3 weeks to 2 months post injury to reveal HO. Useful to confirm maturity of HO

Prophylaxis
- ROM exercises
- Control of muscle tone
- Non Steroidal Anti-inflammatory Drugs (NSAIDs)
- Radiation—used perioperatively to inhibit HO in total hip replacement patients; concerns about ↑ risk of neoplasia limit its use in younger patient populations (e.g., TBI patients). Also, as radiation is used prophylactically to prevent HO formation of a particular joint, to use it in TBI patients would require essentially irradiation of the whole body (as HO can develop practically at any joint), which is not practical

Treatment
- Diphosphonates and NSAIDs (particularly indomethacin) have been used on patients to arrest early HO and to prevent postop recurrence, but their efficacy has not been clearly proven (TBI population)
- ROM exercises—used prophylactically to prevent HO and also used as a treatment for developing HO (to prevent ankylosis)
- Surgery—surgical removal of HO indicated only if ↑ in function is a goal (to ↑ hygiene, sitting, etc.)
- Surgical resection usually postponed 12 to 18 months to allow maturation of HO

Hypertension (HTN)

- Frequently observed post-TBI
- Estimated incidence 11%–25% post head injury
- Posttraumatic hypertension usually resolves spontaneously—long-term use of antihypertensive agents is rarely necessary
- Post TBI hypertension related to sympathetic hyperactivity usually seen in severe TBI—demonstrated by ↑ plasma and urine catecholamines levels
- Cases of HTN have been reported secondary to hydrocephalus several years after TBI
- If medication needed, propanolol recommended because:
  - ↓ plasma catecholamines levels
  - ↓ cardiac index
  - ↓ myocardial oxygen demand
  - ↓ heart rate
  - Improves pulmonary ventilation-perfusion inequality

Venous Thromboembolic Disease

- Venous thromboembolic disease (VTE), including deep vein thrombosis (DVT) and pulmonary embolus (PE), are among the most significant complications of TBI as they are related to ↑ mortality in the rehabilitation setting
- The incidence of DVT in TBI rehabilitation admissions is approximately 10%–18% (Cifu, 1996)
- VTE/DVT often clinically silent in the TBI population, with sudden death from PE being the first clinical sign in 70%–80%
- DVT occurs most commonly in the lower limbs and is traditionally associated with immobility, paresis, fracture, soft-tissue injuries, and ages > 40
- Remember Virchow's triad: venous stasis, vessel-wall damage, and hypercoagulable state

Prophylactic Regimens for DVT
- Low-dose unfractionated heparin (5000 U q 8 to 12 hours) and low-molecular-weight heparin—adequate anticoagulation generally achieved with these treatments
- Intermittent pneumatic compression—provide effective DVT prophylaxis in patients at risk of bleeding complications
- Warfarin (Coumadin®)
- Inferior vena cava (IVC) filter

Diagnostic tests
Doppler ultrasonography, impedance plethysmography (IPG), ^{125}I-fibrinogen scanning and contrast venography

TABLE 2–9

Diagnostic Test	Pros	Cons
Doppler ultrasonography	• 95% sensitivity and 99% specificity for symptomatic proximal thrombi	• Limited ability to detect calf thrombi
Impedance plethysmography	• 90%–93% sensitivity and 94% specificity for proximal thrombi	• Limited ability to detect calf thrombi
^{125}I-fibrinogen scanning	• 60%–80% sensitive in proximal thrombi	• Invasive • Involves injection of radioactive agent
Contrast venography	• Remains the gold standard for diagnosis of clinically suspected DVT	• Invasive • Contrast-induced thrombosis • Contrast allergy

Treatment of DVT

- Anticoagulation is first initiated with IV heparin or adjusted-dose subcutaneous heparin followed by oral anticoagulation; anticoagulation continued for 3–6 months. IVC filter used when anticoagulation is contraindicated

Posttraumatic Epilepsy/Posttraumatic Seizures (PTS)

Posttraumatic epilepsy is classified as:

1. Generalized (grand mal and tonic-clonic)
2. Partial (*simple*, if consciousness is maintained, or *complex*, if not)

The majority of PTS are of the partial type

Posttraumatic seizures are further classified as:

- Immediate PTS—occur within the first 24 hours post injury
- Early PTS—occur within the first week (24 hours to 7 days)
- Late PTS—occur after the first week
  - Immediate PTS has better prognosis than early epilepsy; early PTS associated with increased risk of late PTS

Incidence

Varies greatly according to the severity of the injury, the time since the injury, and the presence of risk factors (see below)

- 5% of hospitalized TBI patients (overall, closed-head injury) have late *PTS*
- 4%–5% of hospitalized TBI patients have one or more seizures in the first week after the injury (early PTS) (Rosenthal et al., 1990)

Study to evaluate association between different characteristics (severity) of TBI and development of seizures post injury. A group of 4541 patients with TBI {characterized by loss of consciousness (LOC), posttraumatic amnesia (PTA), SDH or skull fracture}, were divided into three categories:

Mild TBI—LOC or amnesia < 30 minutes
Moderate TBI—LOC for 30 minutes to 24 hours or skull fractures
Severe TBI—LOC or amnesia > 24 hours, SDH or brain contusion

Incidence of seizures in the different categories:

Mild TBI—1.5%
Moderate TBI -2.9%
Severe TBI -17%
Overall incidence (all patients)—3.1%

(Annegers et al., 1998)

🕮 Risk Factors Associated with Late Posttraumatic Seizures:
- Penetrating head injury—33%–50%
- Intracranial hematoma—25%–30%
- Early seizure (> 24 hours to 7 days)—25%
- Depressed skull fracture—3%–70%
- Prolonged coma or posttraumatic amnesia (> 24 hours)—35%

Other Risk Factors
- Dural tearing
- Presence of foreign bodies
- Focal signs such as aphasia and hemiplegia
- Age
- Alcohol abuse
- Use of tricyclic anti-depressants (TCAs)

Risk Factors Associated with Late Posttraumatic Seizures

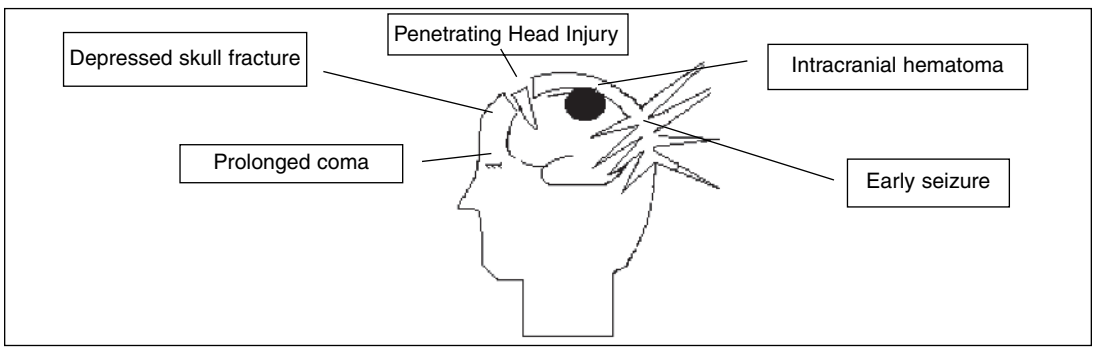

FIGURE 2–6.

- 50%–66% of PTS occur within one year; 75%–80% occur within two years; most PTS occur 1–3 months after injury
- 50% of patients with PTS will have only one seizure, and 25% have no more than three episodes

Diagnosis
- Clinical
- EEG
  - Standard
  - Sleep-deprived
  - 24 hour
- Prolactin level: ↑ prolactin level confirms true seizure activity (but normal prolactin levels will not exclude seizure activity)

Prophylactic Anticonvulsants
- Greater risk of development of PTS: within the first 2 years post injury
- Prophylactic use of anticonvulsants has not been proven effective in prospective, randomized, controlled studies
- Phenytoin—proven to be effective only during the first week post injury (with no benefit thereafter) at preventing early PTS. There is no proof of change in outcome with prophylactic use of phenytoin (Temkin et al., 1990)

Treatment

It has been suggested that carbamazepine and valproic acid are the drugs of choice (DOC) for the treatment of partial and generalized PTS, respectively.

TABLE 2–10 Anticonvulsant Medications: Uses and Adverse Reactions

Medication	Uses	Adverse Reactions
Carbamazepine	• Partial seizures • Tonic-clonic; generalized seizures • Stabilization of agitation and psychotic behavior • Bipolar affective disorder • Neuralgia	• Acute: stupor or coma, hyperirritability, convulsions, respiratory depression • Chronic: drowsiness, vertigo, ataxia, diplopia, blurred vision, nausea, vomiting, aplastic anemia, agranulocytosis, hypersensitivity reactions (dermatitis, eosinophilia, splenomegaly, lymphadenopathy), transient mild leukopenia, transient thrombocytopenia, water retention with decreased serum osmolality and sodium, transient elevation of hepatic enzymes
Gabapentin	• Partial seizures	• Somnolence, dizziness, ataxia, fatigue
Lamotrigine	• Partial seizures • Tonic-clonic; generalized seizures	• Dizziness, ataxia, blurred or double vision, nausea, vomiting, rash, Stevens-Johnson syndrome, disseminated intravascular coagulation
Phenobarbital	• Partial seizures • Tonic-clonic; generalized seizures	• Sedation, irritability, and hyperactivity in children, agitation, confusion, rash, exfoliative dermatitis, hypothrombinemia with hemorrhage in newborns whose mothers took phenobarbital, megaloblastic anemia, osteomalacia • Nystagmus and ataxia at toxic doses
Phenytoin	• Partial seizures • Tonic-clonic; generalized seizures • Neuralgia	• Intravenous administration: cardiac arrhythmias, hypotension, CNS depression • Oral administration: disorders of the cerebellar and vestibular systems (such as nystagmus, ataxia, and vertigo), cerebellar atrophy, blurred vision, mydriasis, diplopia, ophthalmoplegia, behavioral changes (such as hyperactivity, confusion, dullness, drowsiness, and hallucination), increased seizure frequency, gastrointestinal symptoms, gingival hyperplasia, osteomalacia, megaloblastic anemia, hirsutism, transient liver enzyme elevation, decreased antidiuretic hormone secretion leading to hypernatremia, hyperglycemia, glycosuria, hypocalcemia, Stevens-Johnson syndrome, systemic lupus erythematosus, neutropenia, leukopenia, red cell aplasia, agranulocytosis, thrombocytopenia, lymphadenopathy, hypothrombinemia in newborns whose mothers received phenytoin, reactions indicative of drug allergy (skin, bone marrow, liver function)
Valproic Acid	• Partial seizures • Tonic-clonic; generalized seizures • Myoclonic seizures • Absence seizures • Stabilization of agitation and psychotic behavior	• Transient gastrointestinal symptoms such as anorexia, nausea, and vomiting; increased appetite; sedation; ataxia; tremor; rash; alopecia; hepatic enzyme elevation, fulminant hepatitis (rare, but fatal); acute pancreatitis; hyperammoniemia

From Rosenthal M, Griffith ER, Kreutzer JS, Pentland B. Rehabilitation of the Adult and Child with Traumatic Brain Injury 3rd ed. Philadelphia: F.A. Davis; 1999, p. 67, table 4–2, with permission

- Anticonvulsant medications are usually started once *late* seizures occur
- 🕮 In the TBI population, carbamazepine (for partial seizures) and valproic acid (for generalized seizures) are often preferred to medications that are more sedating or associated with cognitive impairment (such as phenobarbital and phenytoin). Their superiority over phenytoin has been debated (differences among these three agents are probably minimal); carbamazepine may be as sedating as phenytoin. (Brain Injury Special Interest Group of the American Academy of Physical Medicine and Rehabilitation, 1998)
- Important to remember that all anticonvulsants may cause some degree of sedation and cognitive deficits (usually psychomotor slowing)
- Phenobarbital is clearly associated with greater cognitive impairment; this medication should not be used as first choice of anticonvulsant therapy in the TBI patient
- Long-term use of phenytoin has been associated with adverse cognitive effects. Animal and clinical (extrapolated from strokes) studies suggest that phenytoin may impede recovery from brain injury (Dikmen, 1991)
- Second generation anticonvulsants, such as gabapentin and lamotrigine, may also be used for treatment of PTS as adjuvant agents (not approved yet for monotherapy). These agents appear to have fewer cognitive side effects, but are still under investigation in the TBI population.

🕮 *Drug Interactions*

TABLE 2–11. Anticonvulsant Medications: Common Drug Interactions

Medication	Drug Interaction
Carbamazepine	• Increased metabolism of carbamazepine (decreased levels) with phenobarbital, phenytoin, and valproic acid • Enhances metabolism of phenobarbital • Enhances metabolism of primidone into phenobarbital • Reduces concentration and effectiveness of haloperidol • Carbamazepine metabolism inhibited by propoxyphene and erythromycin
Lamotrigine	• When used concurrently with carbamazepine, may increase levels of 10,11 –epoxide (an active metabolic of carbamazepine) • Half-life of lamotrigine is reduced to 15 hours when used concurrently with carbamazepine, phenobarbital, or primidone • Reduces valproic acid concentration
Phenobarbital	• Increased levels (as much as 40%) of phenobarbital when valproic acid administered concurrently • Phenobarbital levels may be increased when concurrently administering phenytoin • Phenobarbital has a variable reaction with phenytoin levels
Phenytoin	• Phenytoin levels may increase with concurrent use of chloramphenicol, cimetidine, dicumarol, disulfiram, isoniazid, and sulfonamides • Free phenytoin levels may increase with concurrent use of valproic acid and phenylbutazone • Decreased total levels of phenytoin may occur with sulfisoxazole, salicylates, and tolbutamide • Decreased phenytoin levels with concurrent use of carbamazepine • Decreased carbamazepine levels with concurrent use of phenytoin • Increased or decreased levels of phenytoin when concurrently administered with phenobarbital • When concurrently used with theophylline, phenytoin levels may be lowered and theophylline metabolized more rapidly • May decrease effectiveness of oral contraceptives • Enhances metabolism of corticosteroids
Valproic Acid	• Increases level of phenobarbital • Inhibits metabolism of phenytoin • Rare development of absence status epilepticus associated with concurrent use of clonazepam

(Reprinted with permission from Rosenthal et al., 1999, p. 68, table 4–3)

Withdrawal of Anticonvulsants (For Patients with Posttraumatic Seizures):

- 🕮 No clear indications. It has been suggested to withdraw anticonvulsant medications after a seizure-free interval of 3 months to 6 months up to 1–2 years. (One to two year seizure-free interval is used more often as time frame for withdrawal of anticonvulsant therapy.)
- Spontaneous resolution of PTS can occur

Posttraumatic Agitation

- Posttraumatic agitation is usually a self-limiting problem lasting 1–4 weeks
- Reported to occur in 33%–50% of patients with TBI in the acute care setting
- Posttraumatic agitation has been described as
  - A subtype of delirium unique to TBI survivors, in which the survivor is in a state of posttraumatic amnesia, and there are excesses of behavior, including a combination of aggression, disinhibition and/or emotional lability
    - Delirium is related to, but not sufficient for, a diagnosis of agitation.
- Often no pharmacologic intervention is required

First-line Intervention

- Patient should be maintained in a safe, structured, low-stimulus environment, which is frequently adequate to manage short-term behavior problems. Agitation may be controlled with alterations in environment and staff or family behavior
- Floor beds can eliminate need for restraints
- Use physical restraints only if patient is a danger to self or others; should be applied only to minimal degree and not as a substitute for floor bed or one-to-one or other environmental interventions

TABLE 2–12. Environmental Management of Agitation

1. Reduce the level of stimulation in the environment
Place patient in quiet private room
Remove noxious stimuli if possible, tubes, catheters, restraints, traction
Limit unnecessary sounds, TV, radio, background conversations
Limit number of visitors
Staff to behave in a calm and reassuring manner
Limit number and length of therapy sessions
Provide therapies in patient room
2. Protect patient from harming self or others
Place patient in a floor bed with padded side panels (Craig bed)
Assign 1:1 or 1:2 sitter to observe patient and ensure safety
Avoid taking patient off unit
Place patient in locked ward
3. Reduce patient's cognitive confusion
One person speaking to patient at a time
Maintain staff to work with patient
Minimize contact with unfamiliar staff
Communicate to patient briefly and simple, one idea at a time
4. Tolerate restlessness when possible
Allow patient to thrash about in floor bed
Allow patient to pace around unit with 1:1 supervision
Allow confused patient to be verbally inappropriate

(Reprinted with permission from Braddom RL. Physical Medicine and Rehabilitation, Philadelphia: W.B. saunders Company; 1996: Table 49-8.)

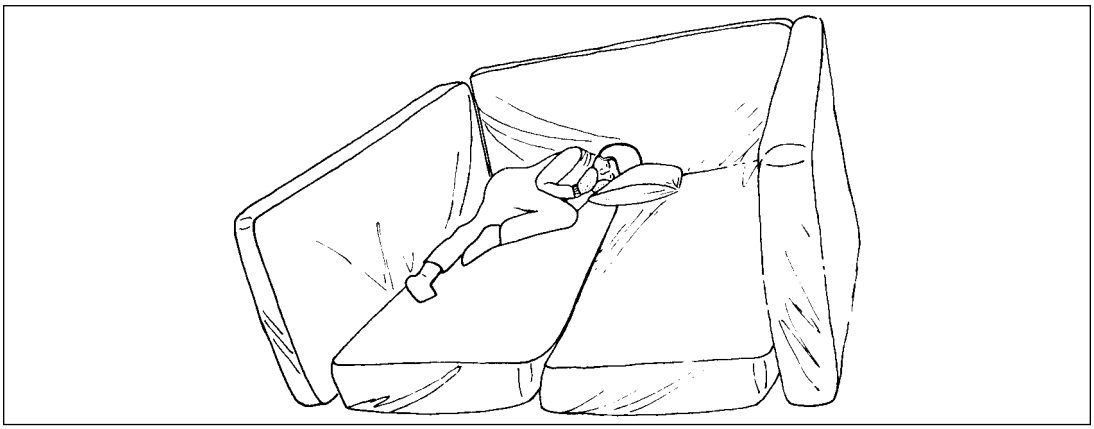

FIGURE 2–7 Agitated non-ambulatory patients often benefit from the use of a floor (Craig) bed. Mattresses can be laid on the floor and 3–4 ft. padded walls on four sides allow the patient to roll around. The use of a floor bed with one-to-one supervision and with the use of mitts and a helmet (if necessary) often eliminates the need for restraints. (Reprinted with permission from Braddom, R.L. Physical Medicine and Rehabilitation. Philadelphia: W.B. Saunders; 1996: p. 1038, figure 49-7.)

Medications for Treatment of Posttraumatic Agitation

- Pharmacologic treatment for agitation is controversial but it includes carbamazepine (Tegretol®) (most commonly used agent for posttraumatic agitation), TCAs, trazodone (Desyrel®), beta-blockers, SSRIs, valproic acid (Depakote®), lithium, amantadine (Symmetrel®), buspirone (BuSpar®)
- Avoid haloperidol (Haldol®), which is shown to decrease recovery in the injured brain tissue in animals (Feeny et al., 1982)

Urinary Dysfunction

- Neurogenic bladder with uninhibited detrusor reflex (contraction)
- TBI patients are frequently incontinent, usually presenting a disinhibited type of neurogenic bladder, in which the bladder volume is reduced but empties completely with normal postvoiding intravesicular volumes ⇒ Small voids with normal residuals
- 🕮 For this type of dysfunction, a time-void program is usually helpful, in which the patient is offered the urinal or commode at a regular scheduled interval
- Anticholinergic meds (to ↑ bladder capacity) may also be used
- (Note—For a more detailed description of bladder function, types of neurogenic bladder and treatments, see SCI section) (Rosenthal, 1999)

Cranial Neuropathies

Most frequently affected cranial nerves in blunt head trauma:

- Olfactory nerve (CN I)
- Facial nerve (CN VII)
- Audiovestibular/vestibulocochlear nerve (CN VIII)
- CN affected with intermediate frequency
  - optic nerve (CNII)
  - ocular motor nerves (CN IV > CN III > CN VI)
- Trigeminal nerve (CN V) and the lower cranial nerves are rarely involved

CN I (Olfactory)

- 🕮 Cranial nerve most often damaged by blunt head trauma
- Overall incidence ~ 7%, rising to 30% with severe head injuries or anterior fossa fractures

- Anosmia (loss of the ability to smell) is more common with occipital than with frontal blows and can result from trauma to any part of the head
- Anosmia and an apparent loss of taste result from CN I disruption thought to be secondary to a displacement of the brain with tearing of the olfactory nerve filaments in or near the cribriform plate through which they course
- Often associated with ↓ appetite/altered feeding behavior
- Associated with CSF rhinorrhea
- Recovery occurs in > one-third of cases, usually during the first 3 months

CN VII (Facial)
- Especially vulnerable to penetrating or blunt trauma to head because of its long, tortuous course through the temporal bone

CN VIII (Vestibulocochlear)
- Damage to the vestibulocochlear nerve results in loss of hearing or in postural vertigo and nystagmus coming on immediately after the trauma

CN II (Optic Nerve)
- Partial damage may result in scotomas and a troublesome blurring of vision or as homonymous hemianopsia. If nerve is completely involved or transected, patient will develop complete blindness (pupil dilated, unreactive to direct light but reactive to light stimulus to the opposite eye (consensual light reflex)

Endocrine Complications

Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH)
- Water retention resulting from excessive antidiuretic hormone (ADH) secretion from the neurohypophysis secondary to multiple causes including head trauma
- In SIADH, ADH excess considered to be inappropriate because it occurs in the presence of plasma hypo-osmolality
- In SIADH, Na^+ excretion in the urine is maintained by hypervolemia, suppression of the renin-angiotensin-aldosterone system, and ↑ in the plasma concentration atrial natriuretic peptide (usually > 20 mmol/L)

Common Causes of SIADH
- CNS Diseases
  - Thrombotic or hemorrhagic events
  - Infection
    - Meningitis
    - Encephalitis
    - Brain abscess
- Head Trauma
- Lung Disease
  - Pneumonia
  - Lung abscess
  - Positive pressure ventilation
- Malignancy
  - CA of the lung (especially small cell CA)
  - GI malignancy (e.g., pancreatic cancer)
  - Prostate CA
  - Thymoma
  - Lymphoma
- Drugs
  - 🕮 Carbamazepine
  - Vincristine
  - Clofibrate
  - Chlorpropamide
  - Phenotiazines
  - Amtriptyline
  - Morphine
  - Nicotine

Signs and Symptoms in SIADH:
- In mild SIADH (with Na^+ 130–135), or in gradually developing SIADH, symptoms may be absent or limited to anorexia and nausea/vomiting

- In severe SIADH (with significant hyponatremia) or in acute onset SIADH, there might be an increase in body weight and symptoms of cerebral edema—restlessness, irritability, confusion, coma, convulsions
- Edema (peripheral/soft tissue) almost always absent

Treatment
- Fluid restriction to ~ 1.0 L/day (800 ml to 1.2 L/day) (either alone or with a loop diuretic)
- Careful daily monitoring of weight changes and serum Na^+ until sodium level > 135 mmol/L
- Hypertonic saline (e.g., 5% NaCl solution), 200–300 ml, should be infused IV over 3–4 hours in patients with severe symptoms as confusion, convulsions, or coma
- It is important not to raise Na^+ concentration too rapidly to avoid development of serious neurological damage, pontine myelinolysis, or congestive heart failure (CHF); sodium may be corrected not more than 10 mEq/L over 24 hours until sodium reaches 125 mEq/L
- Chronic SIADH may be treated with demeclocycline, which normalizes serum Na^+ by inhibiting ADH action in the kidney; lithium carbonate acts similarly but is rarely used because it is more toxic

Cerebral Salt-Wasting (CSW) Syndrome
- CSW is another common cause of hyponatremia in TBI; may probably be a more common cause of hyponatremia in TBI patients than SIADH
- Hyponatremia in TBI is generally present in a hypotonic setting with either normal extracellular volume (isovolemia = SIADH) or reduced extracellular volume (hypovolemia = CSW).
- CSW is thought to occur because of direct neural effect on renal tubular function
- In CSW, hyponatremia is not dilutional (as in SIADH)—CSW patients are, in fact, volume depleted

- *Hallmark of CSW*
  - Decreased blood volume (↓ extracellular volume = hypovolemia) secondary to sodium loss (in urine) ⇒ this triggers ↑ in ADH secretion that is appropriate rather than inappropriate (differentiating this condition from SIADH)
  - Signs of dehydration

- *Treatment*
- Hydration/fluid replacement and electrolyte (Na+) correction
- It is important to differentiate CSW from SIADH and to recognize that there is water depletion in this condition, because treating it with fluid restriction (adequate Tx for SIADH) may further ↓ the extracellular fluid with disastrous results to the patient

Diabetes Insipidus (DI)
- Hallmark: Deficiency of ADH (vasopressin)
- May occur in severe head injuries; often associated with fractures of the skull
- 🕮 A fracture in or near the sella turcica may tear the stalk of the pituitary gland, with resulting DI (due to disruption of ADH secretion from the posterior lobe of the pituitary) in addition to other clinical syndromes depending on the extent of the lesion
- Spontaneous remissions of traumatic DI may occur even after 6 month, presumably because of regeneration of disrupted axons within the pituitary stalk

Clinical Manifestations
- Polyuria, excessive thirst and polydipsia
- Urinary concentration (osm < 290 mmol/kg, SG 1.010) is below that of the serum in severe cases but may be higher than that of serum (290–600 mmol/kg) in mild DI

- Normal function of the thirst center ensures that polydipsia closely matches polyuria, so dehydration is seldom detectable except by a mild elevation of serum Na^+
- However, when replenishment of excreted water is inadequate, dehydration may become severe, causing weakness, fever, psychic disturbances, prostration, and death
- These features are associated with a rising serum osmolality and serum Na^+ concentration, the latter is sometimes > 175 mmol/L

Treatment
- Hormone replacement
  - DDAVP® (desmopressin acetate)—analog of antidiuretic hormone (ADH) with prolonged antidiuretic effect and no significant pressor activity
  - May be given intranasally or intramuscular (IM)
- Chlorpropamide potentiates the effects of ADH on the renal tubules—used in partial ADH deficiency

TABLE 2–13. Comparison of SIADH, CSW and DI

	SIADH	DI	CSW syndrome
Serum ADH (rarely done as routine lab work)	↑ (inappropriately elevated)	↓	↑ (appropriately elevated)
Diagnostic Labs			
Serum Na^+	↓	↑	↓
Serum osmolality	↓	↑	↓
Extracellular volume	Normal (isovolemic)	Normal (isovolemic)	Reduced (hypovolemic)
Urine osmolality and SG	↑ (concentrated urine with osmolality usually > 300 mmol/kg)	↓	Normal

Spasticity

- Disorders of motor tone (e.g., spasticity, rigidity) are common after TBI
- Please refer to the Spasticity section for a full discussion on definition, clinical assessment/grading and treatment options for spasticity

MILD TRAUMATIC BRAIN INJURY AND POSTCONCUSSIVE SYNDROME

- Mild TBI constitutes 80% to 90% of TBI cases in the United States
- ~ 2.3 million cases in the United States
- Multiple terms, definitions, and diagnostic criteria available for mild or minor traumatic brain injury
- The American Congress of Rehabilitation (1995) has defined mild TBI as a traumatically induced physiologic disruption of brain function with at least one of four manifestations:
  - Any loss of consciousness (LOC)
  - Any loss of memory for events immediately before or after the injury
  - Any alteration in mental status at the time of the accident
  - Focal neurological deficits that may or may not be transient
- Usually, mild TBI has negative radiological findings (CT/MRI)

- The injury cannot exceed the following severity criteria:
  - LOC greater than 30 minutes
  - Posttraumatic amnesia (PTA) > 24 hours
  - Initial GCS ≤ 12 (13 to 15)
- Signs and symptoms after mild TBI include:
  - Headache (most common)
  - Dizziness
  - Tinnitus
  - Hearing loss
  - Blurred vision
  - Altered taste and smell
  - Sleep disturbances/insomnia
  - Fatigue
  - Sensory impairments
  - Attention and concentration deficits
  - Slowed mental processing (slowed reaction and information processing time)
  - Memory impairment (mostly recent memory)
  - Lability
  - Irritability
  - Depression
  - Anxiety
- Most mild TBI patients have a good recovery with symptoms clearing within the first few weeks or months postinjury (usually within 1 to 3 months)
- In some patients the symptoms (previously mentioned) persist and are associated with social and vocational difficulties that appear to be out of proportion to the severity of the neurologic insult. This condition has been termed postconcussive syndrome (PCS)
- In a recent study, 14 mild TBI patients with unusually persistent deficits evaluated with single photon emission computed tomography (SPECT) showed significant anterior mesial temporal (lobe) hypoperfusion and less striking dominant (left) orbitofrontal abnormalities
- Memory and learning deficits have been associated with lesions at the hippocampus and related structures in the medial temporal lobes or with injuries to structures that control attention, concentration, and information processing in the frontal and temporal lobe
- Pharmacologic intervention may be used including antidepressants and psychostimulants

Concussion/Sports Related Head Injuries

- Classification of concussion is controversial
- The most widely used grading systems for concussion/mild head injury are the Colorado and the Cantu guidelines

TABLE 2–14. Cantu and Colorado Head Injury Grading Systems

Grade	Cantu	Colorado
Grade I—mild	• No LOC • PTA < 30 min	• No LOC • Confusion w/o amnesia
Grade 2—moderate	• LOC < 5 min • PTA > 30 min	• No LOC • Confusion with amnesia
Grade 3—severe	•LOC > 5 min • PTA > 24 hrs	• LOC

LOC = loss of consciousness
PTA = posttraumatic amnesia
(Cantu, 1992) (Report of the Quality Standards Subcommittee, 1997)

Return to Play Guidelines

- Return to play criteria have been similarly controversial
- Colorado Medical Society and Cantu Guidelines are among the most widely used.

TABLE 2–15. Cantu's Guidelines for Return to Play after Concussion

Grade	First Concussion	Second Concussion	Third Concussion
Grade I—mild	May return to play if asymptomatic for 1 week	May return to play in 2 weeks if asymptomatic for 1 week	Terminate season, although patient may return to play next season if asymptomatic
Grade 2—moderate	May return to play after asymptomatic for 1 week	Minimum of 1 month out of competition, may return to play then if asymptomatic for 1 week and consider termination of season dependent on symptoms	Same as above
Grade 3—severe	Minimum of 1 month, may return to play if asymptomatic for 1 week	Terminate season, although may return to play next season if asymptomatic	

(Cantu , 1998)

The American Academy of Neurology endorsed the Colorado Medical Society Guidelines for classification and management of concussion in sports in its Report of the Quality Standards Subcommittee Practice Parameter published in Neurology, 1997.

TABLE 2–16. When to Return to Play—Colorado Medical Society Guidelines

Grade of Concussion:	Return to play only after being asymptomatic with normal neurologic assessment at rest with exercise:
Grade 1 concussion	15 minutes or less
Multiple Grade 1 concussions	1 week
Grade 2 concussion	1 week
Multiple Grade 2 concussions	2 weeks
Grade 3—brief loss of consciousness (seconds)	1 week
Grade 3—prolonged loss of consciousness (minutes)	2 weeks
Multiple Grade 3 concussions	1 month or longer, based on decision of evaluating physician

(Report of the Quality Standards Subcommittee, 1997)

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