2
Clinical Consequences of Stroke

Robert Teasell MD, Norhayati Hussein MBBS

Last updated November 2013

Abstract

Cerebrovascular disorders represent the third leading cause of mortality and the second major cause of long-term disability in North America (Delaney and Potter 1993). The impairments associated with a stroke exhibit a wide diversity of clinical signs and symptoms. Disability, which is multifactorial in its determination, varies according to the degree of neurological recovery, the site of the lesion, the patient's premorbid status and the environmental support systems. Clinical evidence is reviewed as it pertains to stroke lesion location (cerebral, right & left hemispheres; lacunar and brain stem), related disorders (emotional, visual spatial perceptual, communication, fatigue, etc.) and artery(s) affected.

Table of Contents

Abstract 1

Table of Contents 2

2. Clinical Consequences of Stroke 3

2.1 Localization of the Stroke 3
2.2 Cerebral Hemispheres (Carotid/Anterior Circulation) 4
2.2.1 Anterior Cerebral Artery (ACA) 5
2.2.2 Middle Cerebral Artery (MCA) 6
2.2.3 Right vs. Left Hemispheric Lesions 8
2.3 Right Hemisphere Disorders 8
2.3.1 Visual Spatial Perceptual Disorders 8
2.3.2 Emotional Disorders 10
2.3.3 Communication Problems 10
2.4 Left Hemisphere Disorders 11
2.4.1 Aphasia 11
2.4.2 Apraxias 14
2.4.3 Emotional Disorders 15
2.5 Brain Stem (Vertebral Basilar/Posterior Circulation) Strokes 15
2.5.1 Clinical Syndromes 15
2.5.2 Posterior Inferior Cerebellar Artery (PICA) 18
2.5.3 Basilar Artery 20
2.5.4 Posterior Cerebral Artery (PCA) 20
2.6 Dysphagia and Aspiration Post Stroke 22
2.7 Lacunar Infarcts 24
2.8 Cognitive Impairments Post Stroke 25
2.9 Fatigue Post Stroke 26
References 27

Dr. Robert Teasell
801 Commissioners Road East, London, Ontario, Canada, N6C 5J1
Phone: 519.685.4000 ● Web: www.ebrsr.com ● Email: Robert.Teasell@sjhc.london.on.ca

2. Clinical Consequences of Stroke

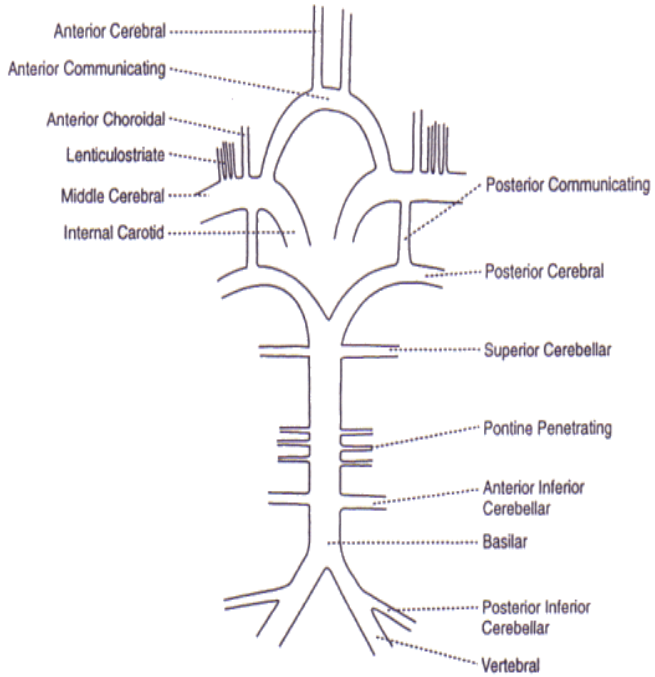
Cerebrovascular disorders represent the third leading cause of mortality and the second major cause of long-term disability in North America (Delaney and Potter 1993). The impairments associated with a stroke exhibit a wide diversity of clinical signs and symptoms. Disability, which is multifactorial in its determination, varies according to the degree of neurological recovery, the site of the lesion, the patient's premorbid status and the environmental support systems.

2.1 Localization of the Stroke

One of the first tasks in the neurologic diagnosis of stroke is localization of the lesion. Certain types of strokes tend to occur in specific areas; for instance, lacunar infarcts occur most often in subcortical regions (Dombovy et al. 1991). The most common presentation of a stroke patient requiring rehabilitation is contralateral hemiparesis or hemiplegia. Other neurological manifestations will vary depending upon the side of the stroke lesion and whether the stroke occurs in the cerebral hemispheres or the brainstem. The arterial territory affected will determine the clinical manifestations; hence, localization of a stroke is often described in such terms.

The clinical consequences of stroke are best classified based upon the anatomical regions(s) of the brain affected. This is best understood by dividing the brain into the cerebral hemispheres, where all but the posterior hemispheres are supplied by the (carotid/anterior circulation), left and right side, and the brain stem and posterior hemispheres (vertebral basilar/posterior circulation) (Figure 2.1).

Figure 2.1 Arterial Blood Supply to the Brain. (Circle of Willis) This can be divided into the carotid/anterior circulation and the posterior/vertebral-basilar circulation

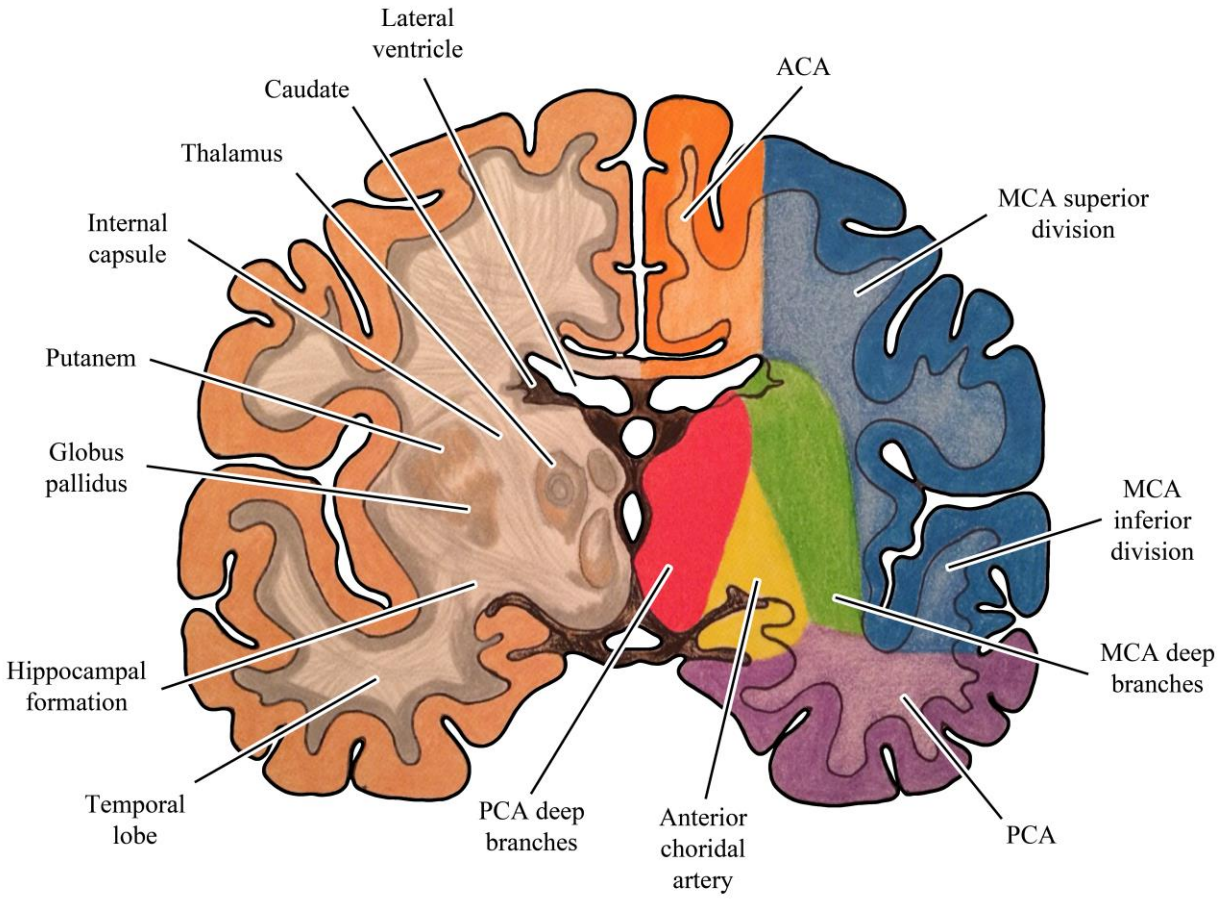


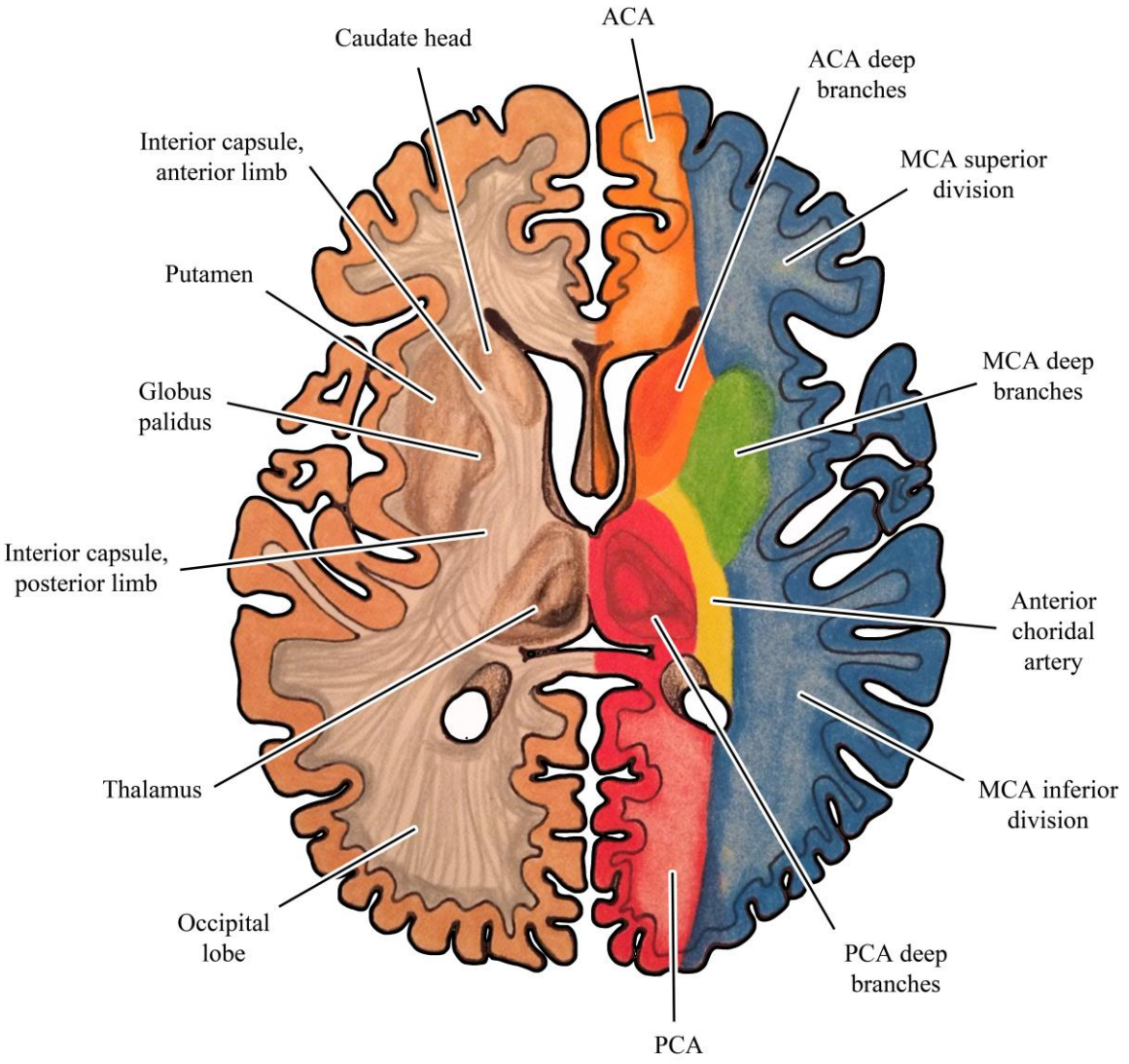
There is a large degree of specialization within the brain with different neurologic functions divided amongst the two hemispheres and the brainstem. The clinical picture of a stroke depends upon which specialized centers have been damaged with subsequent loss of the specialized neurological function they control. However, this schematic view of the brain is in many ways too simplistic. Brain functioning occurs in an integrated fashion. Even a simple activity, such as bending over to pick up an object, requires the integrated function of the entire central nervous system. When damage occurs in one region of the brain, not only are those specialized centers associated with the impaired region affected, but also the entire brain suffers from loss of input from the injured part.

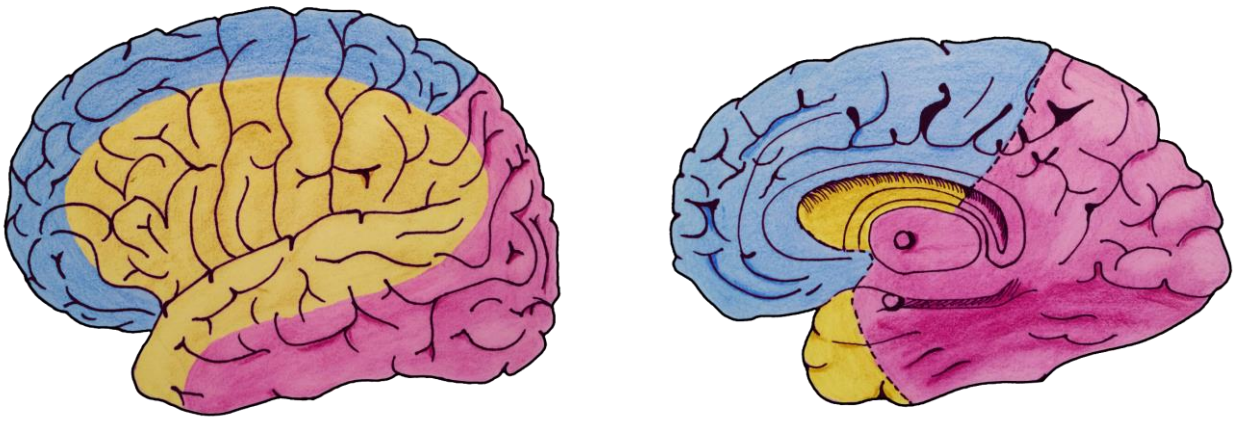
2.2 Cerebral Hemispheres (Carotid/Anterior Circulation)

A stroke in this vascular distribution often results in contralateral paralysis or weakness (hemiparesis/hemiplegia), sensory loss and visual field loss (homonymous hemianopsia) (Adams et al. 1997). Middle cerebral artery involvement is very common while anterior cerebral artery strokes are less common (Teasell 1998). The middle cerebral artery covers two-thirds of the medial surface of the cerebral hemisphere (Kiernan 1998, Scremin 2004). This vascular territory includes the medial aspect of the frontal and parietal lobes, the anterior half of the internal capsule, the anterior inferior head of the caudate, and the anterior four fifths of the corpus callosum. The territory also includes the supplementary motor area and the primary motor and sensory areas for the contralateral lower extremity (see Figure 2.2).

Figure 2.2 Vascular territories of anterior, middle and posterior cerebral arteries.




ACA
Caudate head
ACA deep branches
Interior capsule, anterior limb
MCA superior division
Putamen
MCA deep branches
Globus palidus
Interior capsule, posterior limb
Anterior choridal artery
Thalamus
MCA inferior division
Occipital lobe
PCA deep branches
PCA



2.2.1 Anterior Cerebral Artery (ACA)

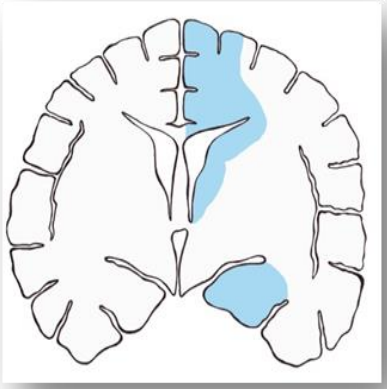
The ACA supplies the anterior two Infarctions involving the ACA territory account for less than 3% of all strokes (Bogousslavsky and Regli 1990, Gacs et al. 1983, Kazui et al. 1993, Kumral et al. 2002). The Circle of Willis generally compensates for lesions proximal to the anterior communicating arteries.

Infarctions of the ACA may present with the following clinical features:

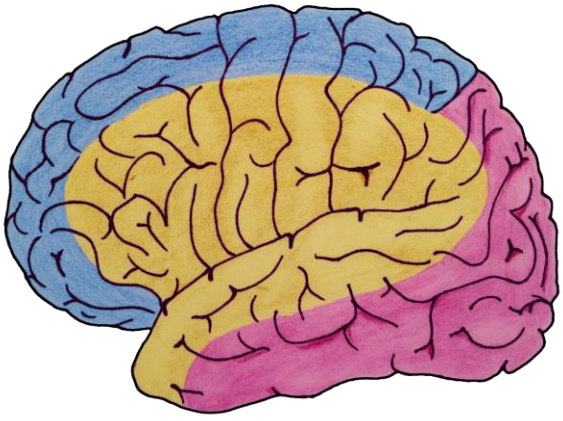
- Contralateral weakness/sensory loss, affecting distal contralateral leg more then upper extremity
- Mutism (Abulia)
- Urinary incontinence
- Contralateral grasp reflex and paratonic rigidity
- Transcortical motor aphasia (on left)
- Gait apraxia

Table 2.1 Occlusions of the ACA

Distal occlusions	Weakness of the opposite leg and a contralateral cortical sensory deficit, most marked in the leg.
Bilateral lesions	Incontinence, abulia or slow mentation and the appearance of primitive reflexes.
Proximal occlusion	All of the above signs plus facial and proximal arm weakness and frontal apraxia, with left side involvement.
Interruption of Commissural Fibers (between frontal lobes)	Sympathetic apraxia of the left arm, right motor paresis.



2.2.2 Middle Cerebral Artery (MCA)



Cortical branches of the MCA supply 2/3 of the lateral surface of the hemisphere as well as the temporal pole (Kiernan 1998, Scremin 2004). Important areas of neurological specialization within the MCA territory include the primary motor and sensory areas for the face and upper extremity as well as Broca's and Wernicke's language areas in the dominant hemisphere (see Figures 2.3 and 2.4). An infarction in the MCA territory is the most common site of cerebral ischemia (Adams et al. 1997). In North America, the etiology of this infarction is generally embolic rather than atherothrombotic (Adams et al. 1997). However, an atherothrombotic infarction of the internal carotid artery invariably presents with symptoms predominantly in the MCA territory. The clinical consequences are similar to those with involvement of the anterior/carotid circulation (see above). Unlike strokes involving the ACA, there is greater facial and

upper extremity involvement (Adams et al. 1997). Additional clinical signs and symptoms occur depending on whether the right or left hemisphere is involved.

Infarctions of the ACA may present with the following clinical features:

- Contralateral hemiparesis/hemiplegia
- Contralateral sensory loss
- Contralateral homonymous hemianopsia
- Left hemispheric: Aphasia
- Right hemispheric: Visual perceptual deficits including left neglect

Middle Cerebral Artery is divided into 2 main divisions – superior (M1) and inferior (M2)

Superior Division

- Contralateral hemiparesis/ hemiplegia
- Contralateral sensory loss
- Left hemispheric: Expressive aphasia
- Right hemispheric: Visual perceptual disorders

Inferior Division

- Superior quantrantonopsia or homonymous hemianopsia
- Left hemispheric: Wernicke's aphasia
- Right hemispheric: Left visual neglect

Figure 2.3 Areas of the cerebral cortex associated with specific functions

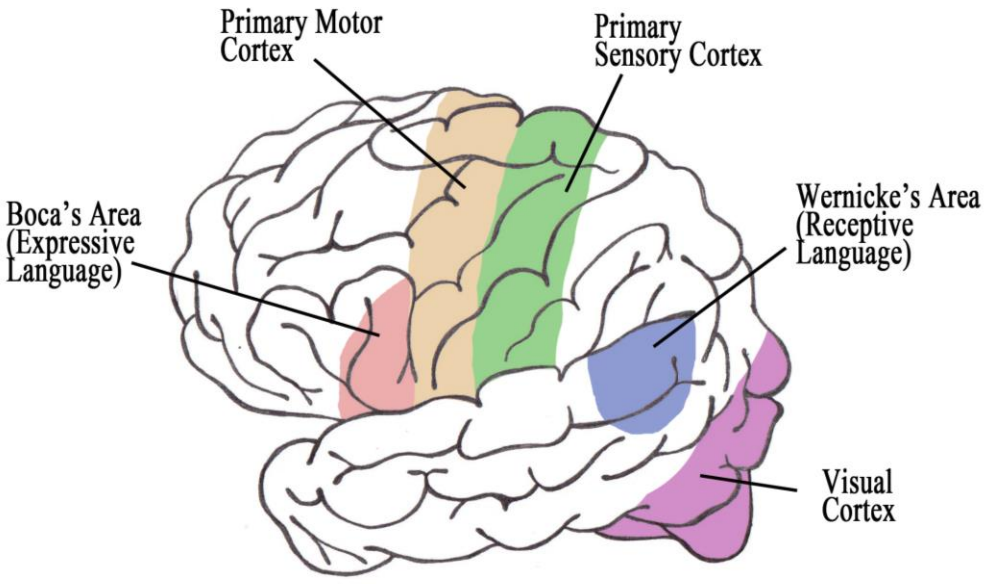
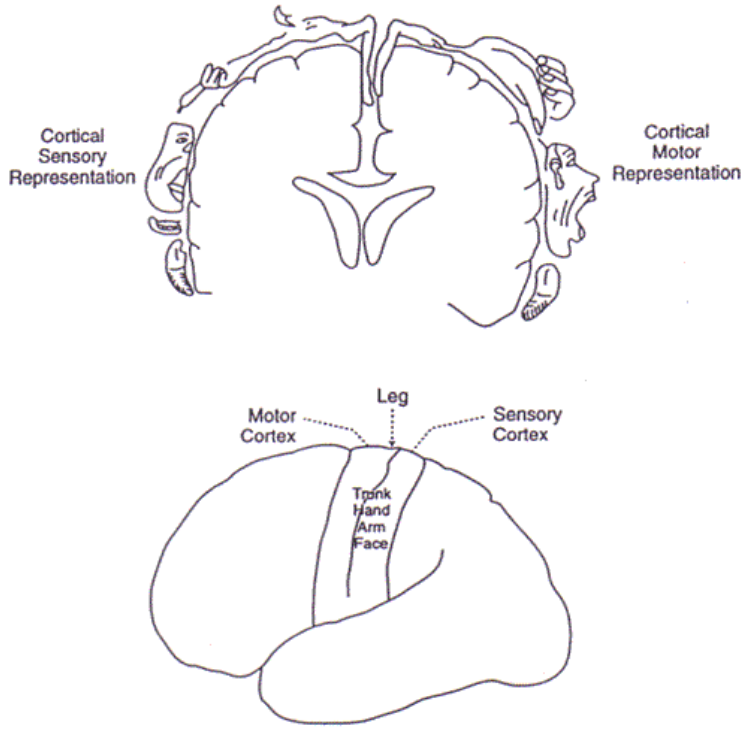


Figure 2.4 Representation of the body on the primary motor and sensory cortex. This explains greater arm involvement in a middle cerebral artery occlusion and greater leg involvement in an anterior cerebral artery occlusion.



2.2.3 Right vs. Left Hemispheric Lesions

Each hemisphere is responsible for initiating motor activity and receiving sensory information from the opposite side of the body. However, as mentioned previously, each hemisphere has a large degree of specialization. Despite this specialization, normal thinking and carrying out of activities requires the integrated function of both hemispheres, neither of which is truly dominant over the other. Many stroke patients have diffuse cerebrovascular disease and other conditions resulting in impaired cerebral circulation. While there may be one major area of infarction, there may be other areas of ischemic damage located throughout the hemispheres that may complicate the clinical presentation.

2.3 Right Hemisphere Disorders

The right hemisphere mediates learned behaviors that require voluntary initiation, planning and spatial perceptual judgement. Clinical signs and symptoms include visual-spatial perceptual deficits, emotional disorders and subtle communication problems.

2.3.1 Visual Spatial Perceptual Disorders

The right hemisphere is dominant for visuospatial orientation, constructional praxis and judgement in over 90% of the population (Delaney and Potter 1993). Therefore, in a right hemisphere middle cerebral infarct, visual-spatial perceptual disorders include left-sided neglect, figure ground

disorientation, constructional apraxia and asterognosis (the later seen with left hemisphere disorders). The most commonly seen visual-perceptual spatial problem is the unilateral neglect syndrome.

Unilateral Spatial Neglect

Kwasnica (2002) has noted that the incidence of unilateral neglect in patients with acute right hemispheric stroke varies between 22%-46% (Pederson et al 1997, Hier et al. 1983a). Acutely following a large right MCA infarct, neglect is characterized by head and eye deviation to the left. Kwasnica (2002) noted that, *"they often do not orient to people approaching them from the contralateral side (Rafal 1994). Patient may be noted not to dress the contralateral side of the body, or shave the contralateral side of the face. Some may fail to eat food on the contralateral side of their plates, unaware of the food they have left (Mesulam 1985)."*

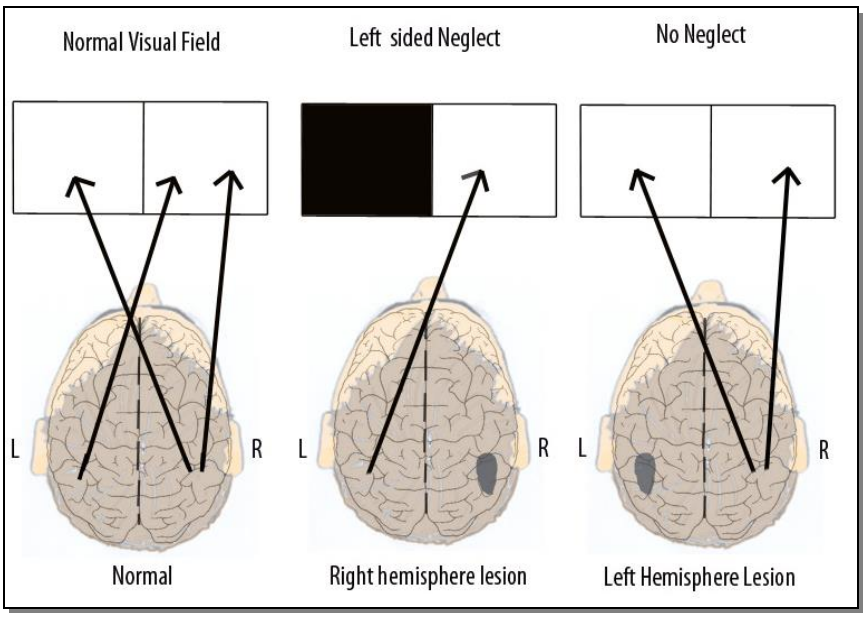
Chronically, it is rare to see significant unilateral neglect following stroke (Kwasnica 2000). Kwasnica (2000) noted that, *"Hier et al. (1983b) studied the recovery of behavioral abnormalities after right hemispheric stroke. He found that neglect, as measured by failure to spontaneously attend to stimuli on the left, had a median time to recovery of nine weeks; approximately 90% of the patients recovered by 20 weeks. He also measured unilateral spatial neglect, scored from a drawing task; 70% of patients recovered in 15 weeks. In chronic stroke patients unilateral neglect is usually subtle and may only be seen when competing stimuli are present, such as a busy therapy gym where patients may find it difficult to direct their attention to a therapist on their left side"* (Kwasnica 2000).

Unilateral Spatial Neglect:

- Defined as a failure to report, respond, or orient to sensory stimuli presented to the side contralateral to the stroke lesion
- More obvious forms of neglect involve colliding with environment on involved side, ignoring food on one side of plate, and attending to only one side of body
- More subtle forms are more common, more apparent during high levels of activity such as driving, work, or interacting with others
- Milder neglect involves various degrees of ignoring the affected side when faced with stimulation on the unaffected side (extinction).
- USN is found in about 23% of stroke patients
- More common in patients with right sided lesions (42%) than left sided lesions (8%) and is more persistent with right sided strokes
- Neuroanatomical studies found left hemisphere modulated arousal and attention for right visual field but right hemisphere controlled process for both right and left visual fields so intact right hemisphere is able to compensate
- Recovery of USN common; most recovery occurs in first 6 months and later recovery rare
- USN associated with negative prognosis for functional outcome, poorer mobility, longer LOS in rehab, and slower rates of improvement

Why is Left Sided Neglect More Common than Right Sided Neglect?

- The right hemisphere regulates attention more than the left hemisphere.
- The left hemisphere is responsible for modulating attention and arousal for the right visual field only, while the right hemisphere is responsible for controlling these processes in both the right and left hemispheres.
- Hence the right hemisphere is more able to compensate for the left hemisphere, when it suffers a stroke, while the left hemisphere is not able to compensate for the right hemisphere if it is injured in a stroke.



Anosognosia

- Refers to unawareness of loss of an important bodily function, primarily hemiplegia
- Involves primarily large right hemispheric strokes which involve the parietal region

Kwasnica (2002) notes that, *"anosognosia is another behavioral abnormality that occurs in patients with unilateral neglect. The term refers to a lack of knowledge or awareness of disability. These patients can fail to notice their contralesional limbs, whether or not they are hemiparetic. They also frequently deny their hemiplegia or minimize its impact on their functional status. In the extreme, they may deny ownership of the hemiparetic limb (Myer 1999). This exists in as much as 36% of patients with right hemisphere strokes (Hier et al. 1983a)".* Hier et al. (1983) found that after right hemispheric lesions, recovery from unilateral neglect and anosognosia was the most rapid. Recovery from constructional and dressing apraxia was intermediate while recovery was slowest for hemiparesis, hemianopsia and extinction.

A strong relationship has been established between visual, spatial, perceptual and motor dysfunction and the ADL performance of right hemispheric stroke patients (Campbell et al. 1991). Such perceptual impairments have been shown to adversely influence the rate of achieving independent sitting and stair climbing (Mayo et al. 1991).

2.3.2 Emotional Disorders

Patients with right hemispheric lesions may speak well so that their actual abilities are often overestimated. These patients tend to have a lack of insight into their own deficits. Difficulties generally labeled as emotionally related include indifference reaction or flat affect, impulsivity (often leading to multiple accidents) and emotional lability.

2.3.3 Communication Problems

Although aphasia is commonly noted to occur with left hemispheric strokes, it may occur rarely in right hemispheric strokes. Annett (1975) demonstrated aphasia occurred after right hemispheric strokes in 30% of left-handed people and 5% of right-handed people. Moreover, patients with nondominant hemispheric lesions often have associated communication difficulties, whereby they have difficulty in utilizing intact language skills effectively, that is, the pragmatics of conversation. The patient may not observe turn-taking rules of conversation, may have difficulty telling, or understanding, jokes (frequently missing the punchline), comprehending ironic comments and may be less likely to appropriately initiate conversation. This tends to result in social dysfunction that may negatively impact on family and social support systems (Delaney and Potter 1993).

2.4 Left Hemisphere Disorders

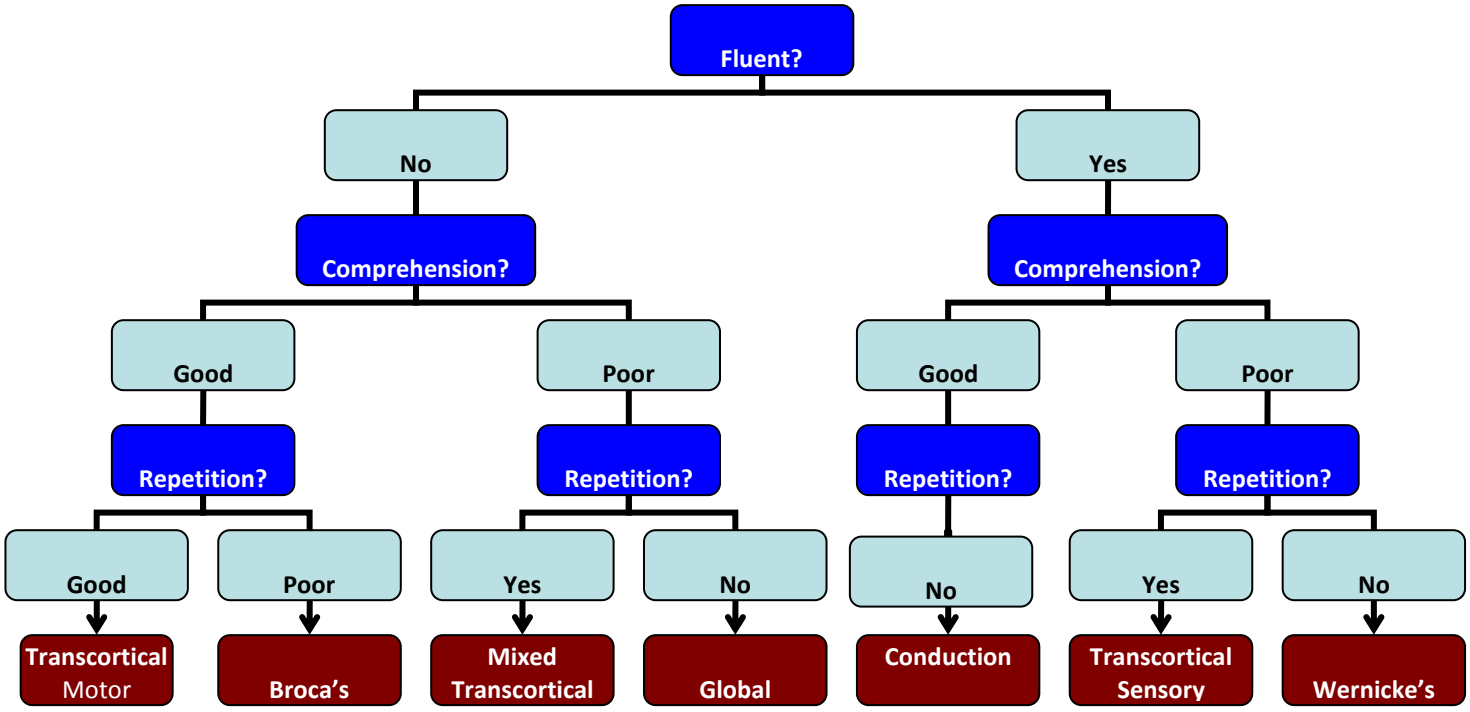
The left hemisphere is specialized for learning and using language symbols. Clinical signs and symptoms include aphasia, apraxia, and arguably emotional disorders.

2.4.1 Aphasia

Ninety-three percent of the population is right-handed, with the left hemisphere being dominant for language in 99% of right-handed individuals (Delaney and Potter 1993). In left-handed individuals, 70% have language control in the left hemisphere, 15% in the right hemisphere, and 15% in both hemispheres (O'Brien and Pallet 1978). Therefore 97% of the population has language control primarily in the left hemisphere. Language function is almost exclusively the domain of the left hemisphere, except for 35% of left handers (3% of population) who use the right hemisphere for language function. A disorder of language is referred to as aphasia with expressive (Broca's) aphasia the language disorder most commonly seen with left hemispheric MCA strokes. A classification of the aphasias is provided in Table 2.2.

Table 2.2 Characteristic Features of Aphasia

Type	Fluency	Comprehension	Repetition
Broca's	Nonfluent	Good	Poor
Transcortical motor	Nonfluent	Good	Good
Wernicke's	Fluent	Poor	Poor
Transcortical sensory	Fluent	Poor	Good
Global	Nonfluent	Poor	Poor
Conduction	Fluent	Good	Poor



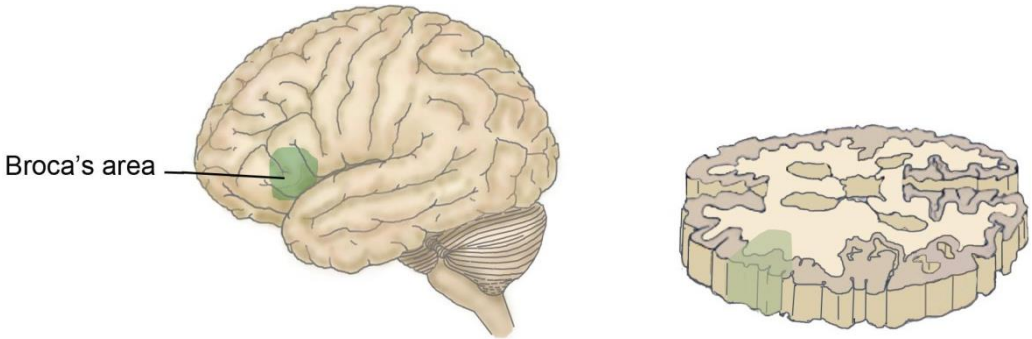
Paraphasias

Incorrect substitutions of words or parts of words. These can be:

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

Broca's Aphasia

- Motor aphasia
- Problems with output; understanding intact
- Nonfluent, hesitant, labored, and paraphasic speaking
- Vocabulary and confrontation naming is severely impaired
- Writing is similarly affected
- Posterior-inferior frontal lobe stroke characterized by nonfluent, effortful speech with preserved comprehension and poor repetition.
- Associated with marked paraphasias and articulatory errors and often described as telegraphic.



Anomic Aphasia

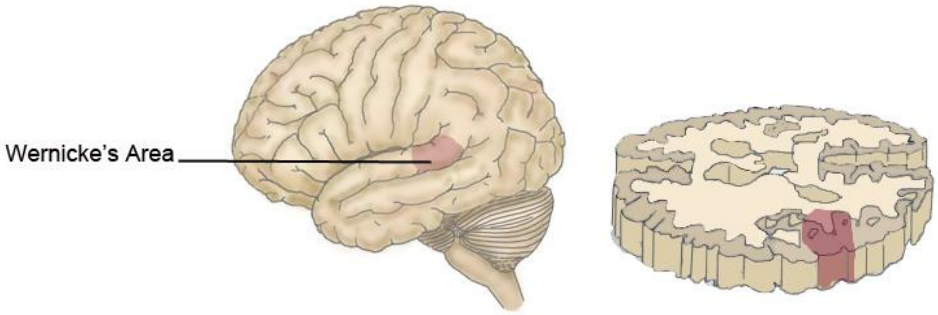
- Mild motor aphasia
- Problem with output; understanding intact
- Word-finding difficulties or mild articulatory errors (often called verbal apraxia)

Transcortical Motor Aphasia

- Stroke is located in the frontal lobe, anterior or superior to Broca's area or in the subcortical region deep to Broca's area.
- Characterized by nonfluent (reduced rate of speech and limited language output), good comprehension and good repetition.

Wernicke's Aphasia

- Sensory aphasia
- Problem with input
- Fluent speech with severe comprehension deficit, poor repetition and often unintelligible jargon; reading is similarly affected.
- Posterior part of superior (first) temporal gyrus stroke characterized by fluent speech but poor comprehension and poor repetition.
- Associated with marked paraphasias and neologisms.

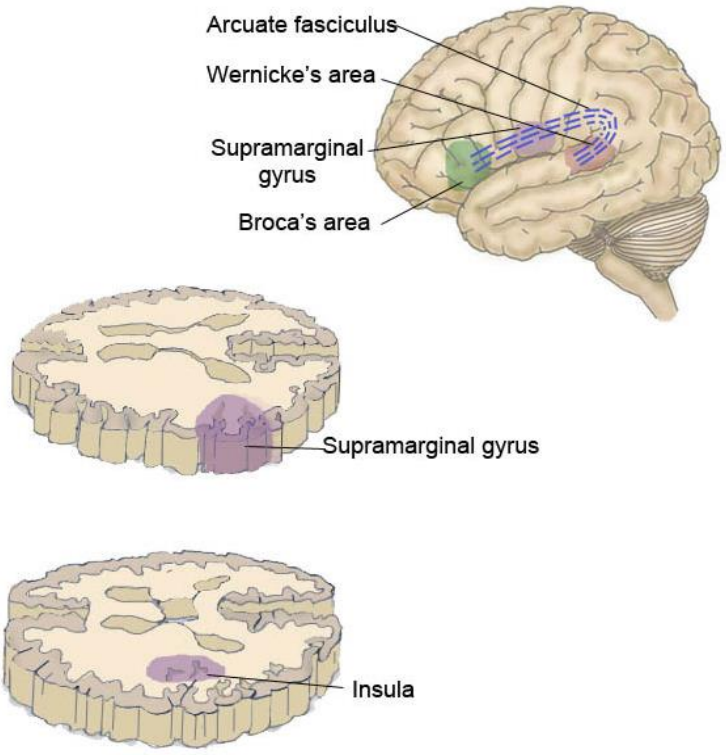


Transcortical Sensory Aphasia

- Watershed stroke isolating the perisylvian speech structures (Broca's and Wernicke's areas) from the posterior brain.
- Characterized by fluent speech (neologisms), poor comprehension and good repetition (possibly echolalia).

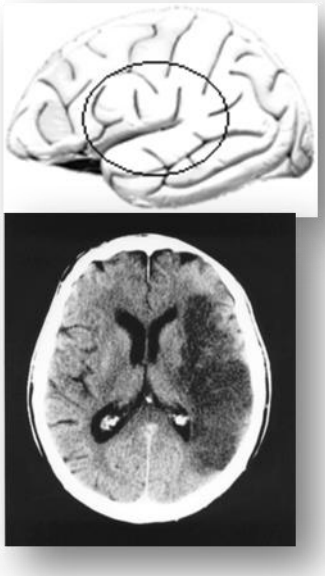
Conduction Aphasia

- Stroke of the parietal operculum (arcuate fasciculus) or insula or deep to the suramarginal gyrus characterized by disproportional impairment in repeating spoken languages.
- Literal paraphasias with "targeting" of words (until getting the right one).



Global Aphasia

- Motor and sensory aphasia
- Problem with input and output
- No communication even with gestures and no speech or only stereotypical repetitive utterances
- Reading and writing affected
- Not good rehabilitation candidates
- Generally involve the entire MCA region with moderate to severe impairment of language of all language function.



2.4.2 Apraxias

Apraxia is a disorder of voluntary movement wherein one cannot execute willed, purposeful activity despite the presence of adequate mobility, strength, sensation, co-ordination and comprehension (Adams et al. 1997). Left hemispheric stroke patients often demonstrate apraxias including general apraxias such as motor, ideomotor or ideational apraxias, as well as specific apraxias that include constructional apraxia, apraxia of speech (verbal apraxia), dressing apraxia and apraxia of gait (see Table 2.5).

Table 2.5 Classification of Apraxias

Type	Site of Lesion	Manifestation
Motor or Ideomotor	Often left hemisphere	Can automatically perform a movement but cannot carry it out on command.
Ideational	Often bilateral parietal	Can perform separate movements but cannot coordinate all steps into an integrated sequence.
Constructional	Either parietal lobe, right > left	Unable to synthesize individual spatial elements into a whole (eg, cannot draw a picture).
Verbal	Commonly associated with Broca's aphasia	Mispronunciation with letter substitution, effortful output and impaired melody of speech.
Dressing	Either hemisphere, right > left	Inability to dress oneself despite adequate motor ability.
Gait	Frontal lobes	Difficulty initiating and maintaining a normal walking pattern when sensory and motor functions seem otherwise unimpaired.

2.4.3 Emotional Disorders

Post stroke depression occurs in 50% of stroke patients (Robinson et al. 1984), more commonly in patients with frontal damage. Occasionally rage and frustration reactions are seen, especially in nonfluent and fluent aphasic patients.

2.5 Brain Stem (Vertebral Basilar/Posterior Circulation) Strokes

2.5.1 Clinical Syndromes

A stroke in this vascular distribution can produce very diverse manifestations because the vertebral basilar artery system provides the vascular supply to the occipital and medial temporal lobes, brainstem and cerebellum (Kiernan 1998, Scremin 2004). Clinical signs and symptoms are listed in Table 2.6. In contrast to the major cognitive or language disorders seen with hemispheric strokes, brainstem strokes in isolation spare cognitive and language functions.

Table 2.6 Clinical Features of Vertebrobasilar Artery Disorders

System	Signs and Symptoms
Cranial Nerves	Bilateral visual and cranial nerve problems Vertigo Dysarthria / Dysphagia Diplopia Facial numbness or paresthesia
Motor	Hemiparesis or quadriparesis Ataxia
Sensory	Hemi- or bilateral sensory loss
Other	Drop attacks

Intact cognitive abilities are important in later regaining functional abilities lost as a consequence of the stroke (Feigenson et al. 1977). Memory loss can be associated with medial temporal and thalamic damage and selective visual-perceptual disorders such as object or face agnosia (inability to recognize

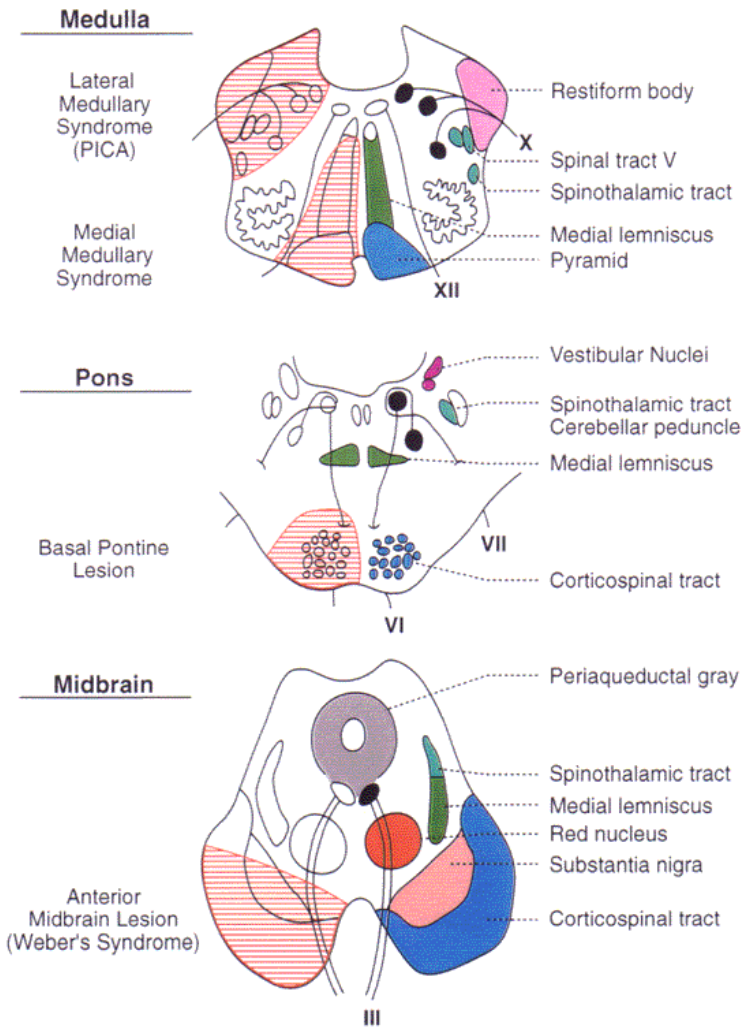
objects or faces) and pure alexia (reading difficulty with otherwise intact language) can be associated with medial occipital temporal damage.

Brainstem strokes are categorized into a variety of well-defined syndromes depending on the vascular territory involved. These syndromes are listed in Table 2.7. Specific impairments resulting from brainstem syndromes include the involvement of ipsilateral cranial nerves (diplopia, dysarthria and dysphagia), pyramidal tracts (hemiparesis), sensory tracts (hemisensory deficits) and cerebellar tracts (ipsilateral ataxia and incoordination). Dysarthria is characterized by unclear speech of various types including slurred, scanning, spastic, monotonous, lisping, nasal, or expulsive speech (Pryse-Philips and Murray 1978). Dysphagia is simply defined as difficulty with swallowing. The management of brainstem strokes with dysphagia often requires the use of prolonged feeding by an alternate route.

Table 2.7 Classic Brainstem Syndromes

Lesion Location	Syndrome	Clinical Picture
Lateral medulla (PICA and/or VA) Medial medulla	Wallenburg's	Vertigo Nausea and vomiting Sensory loss of ipsilateral face and contralateral limb Ipsilateral limbs ataxia Rotatory or horizontal gaze nystagmus Hoarseness and dysphonia Dysphagia and dysarthria Ipsilateral Horner's syndrome Contralateral limb paralysis (facial sparing) Contralateral decrease in position and vibration sense Ipsilateral tongue paralysis
Medulla	Jackson's	Hoarseness and dysphonia Weakness of trapezius and sternocleidomastoid muscles
Lower pons	Millard-Gubler	Alternating or crossed hemiparesis Unilateral UMN facial palsy Contralateral limb paralysis with no contralateral facial palsy
Lower pons	Fouille's	Crossed (alternating hemiparesis) Ipsilateral lateral gaze palsy
Lower pons	Raymond's	Abducens nerve palsy Contralateral hemiparesis
Superior colliculus	Parinaud's	Paralysis of upward conjugate convergence and frequently of downward gaze
Cerebellum	Cerebellum	Unilateral limb ataxia and truncal ataxia Vertigo Headache Occasionally patient may become comatose
Midbrain	Weber's	Contralateral hemiparesis Ipsilateral oculomotor paralysis with dilated pupil, lateral gaze only, ptosis
Midbrain	Benedict's or Claude's	Contralateral hemiparesis Tremor in paretic limbs on voluntary movement/limb ataxia Frequently contralateral sensory loss Ipsilateral oculomotor paralysis

Figure 2.5 Brainstem Anatomy and Involvement of Selected Stroke Syndromes



Pontine Infarct

Contralateral

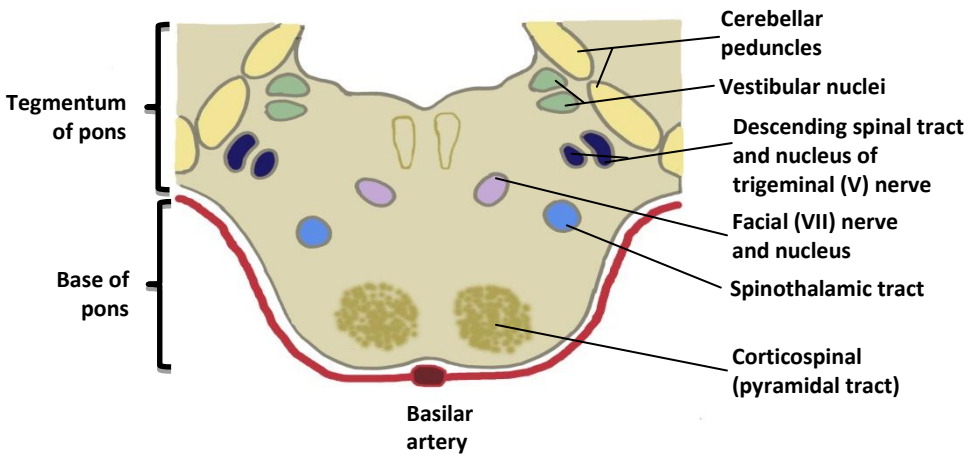
- Spastic hemiplegia (corticospinal tract)
- Hemisensory loss (limbs and trunk) (spinothalamic tract)

Ipsilateral

- Ataxia (cerebellar peduncles)
- Facial weakness (CN VII – facial nerve)
- Facial sensory loss (CN V – trigeminal nerve)
- Hearing loss (VN VIII – acoustic nerve)
- Vertigo
- Lateral gaze palsy

Others

- Dysphagia



2.5.2 Posterior Inferior Cerebellar Artery (PICA)

The PICAs originate from the vertebral arteries about 1 cm below the junction of the two vertebral arteries where they form the basilar artery. Each PICA courses around the lateral surface of the medulla and then loops back to supply portions of the cerebellum. It supplies a wedge of the lateral medulla and the inferior aspect of the cerebellum. Occlusion of the PICA results in a lateral medullary or Wallenburg's syndrome (see Table 2.7).

Lateral Medullary Syndrome

Associated with occlusion of vertebral arteries or posterior inferior cerebellar artery (PICA).

Clinical features of Lateral Medullary Syndrome (Wallenberg`s Syndrome) include:

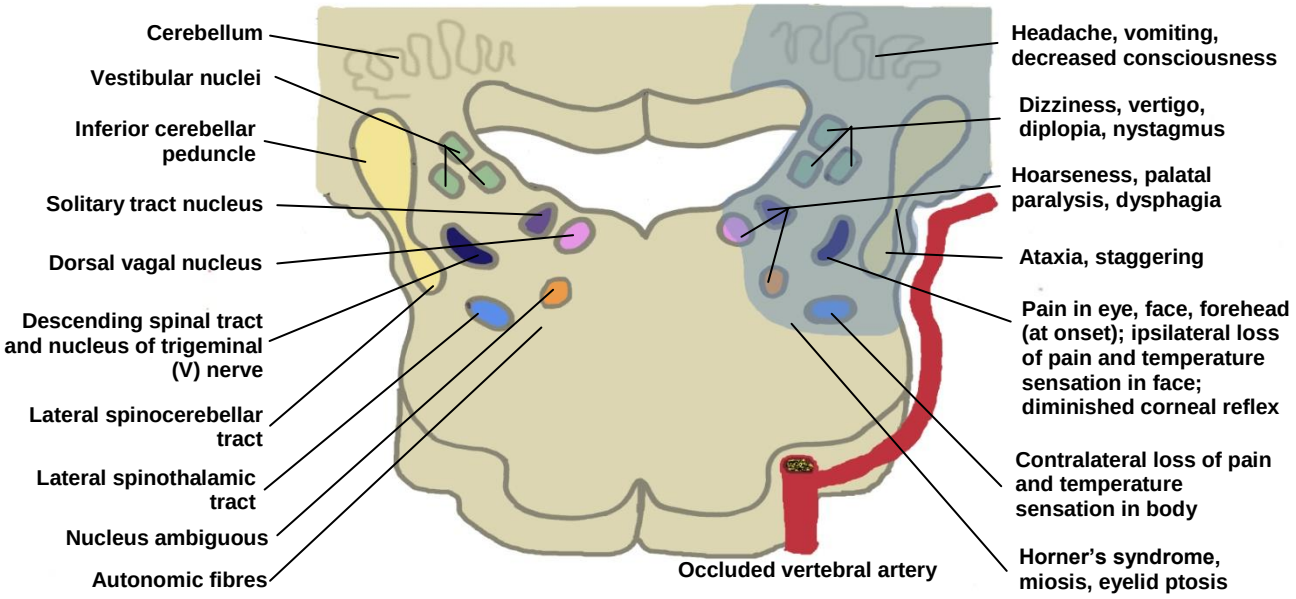
Ipsilateral

- Horner’s syndrome (ptosis, anhydrosis, and miosis)
- Decrease in pain and temperature ipsilateral face
- Cerebellar signs such as ataxia

Contralateral

- Decreased pain and temperature contralateral body
- Dysphagia, dysarthria, hoarseness and paralysis of vocal cord
- Vertigo, nausea and vomiting
- Hiccups
- Nystagmus, diplopia

Intracranial Occlusion of Vertebral Artery
Posterolateral medullary infarction (shaded area) and clinical manifestations



Medial Medullary Syndrome

Associated with occlusion of penetrating arteries.

Clinical features of Medial Medullary Syndrome include:

Ipsilateral

- Hypoglossal palsy (deviation toward the side of the lesion)

Contralateral

- Hemiparesis
- Lemniscal sensory loss (proprioception and position sense)

Cerebellar Stroke

Clinical features of a cerebellar stroke include:

- Ataxia
- Dyssynergia - impaired coordination of muscles involved in a single movement
- Dysmetria - impaired measure and extend as well as speed of intended movement
- Intention tremor
- Dysdiadokinesis – abrupt and jerky movements on alternating movements of agonist and antagonists
- Nystagmus
- Cerebellar (scanning and explosive) dysarthria

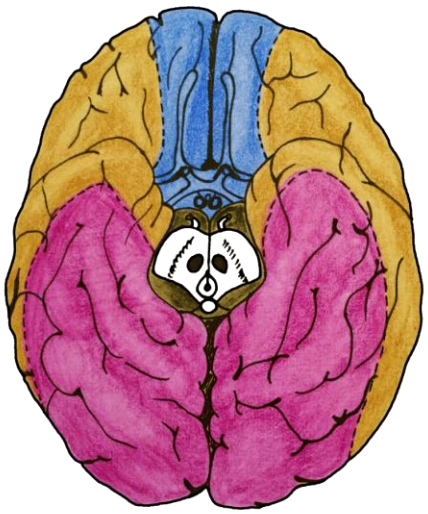
2.5.3 Basilar Artery

The basilar artery is formed at the junction of the medulla with the pons by the merger of the two vertebral arteries. There are 3 major branches of the basilar artery: the anterior inferior cerebellar artery, the superior cerebellar artery and the internal auditory or labyrinthine artery. These are known as the long circumferential arteries. There are also short circumferential arteries as well as small penetrating arteries that supply the pons and paramedian regions. Occlusion of these vessels may result in a variety of signs and symptoms (see Table 2.8).

Table 2.8 Clinical Features of Basilar Artery Ischemia

System	Signs and Symptoms
Sensorium	Alterations of consciousness
Cranial Nerves	Pupil abnormalities III, IV and VI with dysconjugate gaze Horner's syndrome V with ipsilateral facial hypoalgesia Nystagmus VII with unilateral LMN facial paralysis Caloric and oculocephalic reflexes Vertigo IX and X with dysphagia, dysarthria
Motor	Quadriplegia or contralateral hemiplegia
Sensory	Contralateral limb hypoalgesia
Cerebellum	Ipsilateral or bilateral cerebellar abnormalities
Respiratory	Respiratory irregularities
Cardiac	Cardiac arrhythmias and erratic blood pressure

2.5.4 Posterior Cerebral Artery (PCA)



Although the posterior cerebral arteries primarily supply the occipital cerebral hemispheres they usually arise from the posterior circulation. The posterior cerebral arteries arise as terminal branches of the basilar artery in 70% of individuals, from one basilar and the opposite carotid in 20-25% and directly from the carotid circulation in 5-10%. Both PCAs receive a posterior communicating vessel from the internal carotid artery and then arch posteriorly around the cerebral peduncles to the tentorial surface of the cerebrum. They supply the inferolateral and medial surfaces of the temporal lobe, the lateral and medial surfaces of the occipital lobe and the upper brainstem. Included in this area is the midbrain, visual cortex, cerebral peduncles, thalamus and splenium of the corpus callosum (see Figure 2.2). Occlusion of the PCA or any of its branches may produce a wide

variety of syndromes (see Table 2.9).

Patients with PCA infarctions present with:

- Homonymous hemianopsia
- Memory loss
- Hemisensory loss
- Alexia without agraphia

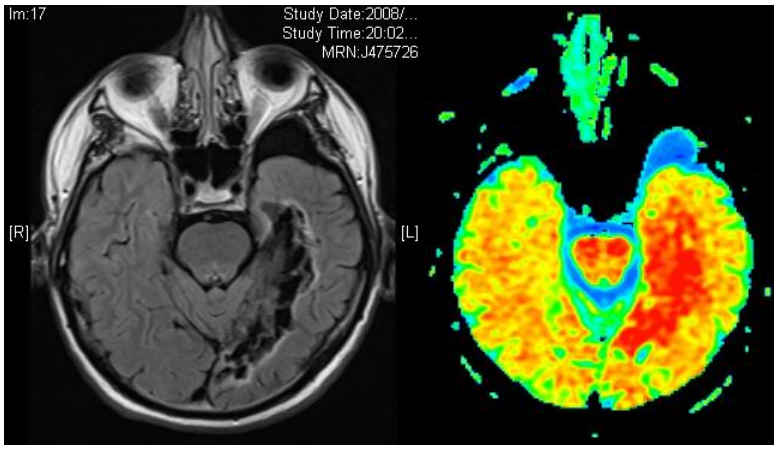
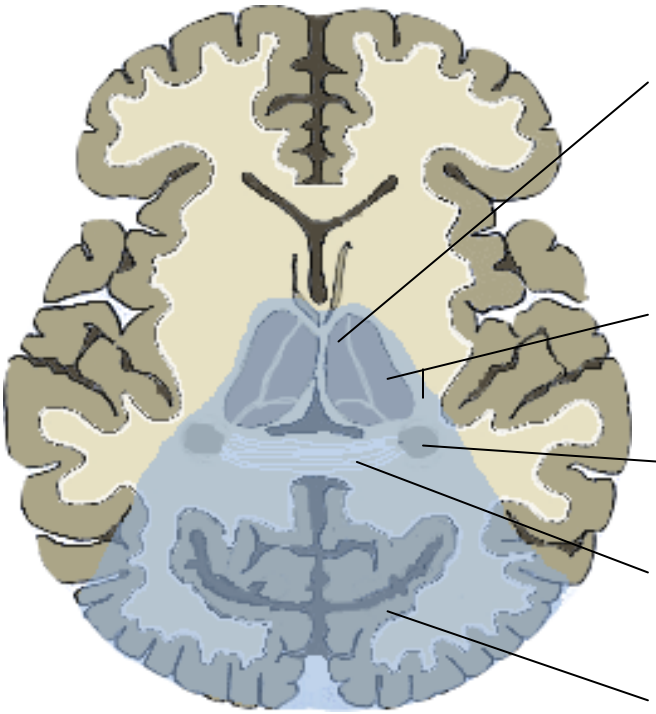


Table 2.9 Syndromes of PCA Occlusions

Thalamoperforate Branch Occlusion	Involuntary movement disorders Hemiataxia Intention tremor Weber's syndrome: Ipsilateral oculomotor palsy with contralateral hemiparesis Claude's or Benedict's syndrome: Ipsilateral oculomotor palsy with contralateral cerebellar ataxia
Thalamogeniculate Branch Occlusion (Thalamic Syndrome)	Contralateral sensory loss Transient contralateral hemiparesis Contralateral mild involuntary movements Intense, persistent, burning pain
Cortical Branch Occlusion	Contralateral homonymous hemianopsia Dominant hemisphere – alexia, memory impairment or anomia, especially for naming colors Non-dominant hemisphere –topographic disorientation (usually due to parietal damage) Prosopagnosia (failure to recognize faces)
Bilateral PCA Occlusions	Visual agnosia or cortical blindness (intact pupillary reflexes) Severe memory loss



Medial thalamus and midbrain
Hypersomnolence
Small, nonreactive pupils
Bilateral third cranial nerve palsy
Behavioural alterations
Hallucinations

Lateral thalamus and posterior limb of internal capsule
Hemisensory loss

Hippocampus and medial temporal lobes
Memory loss

Splenium of corpus callosum
Alexia without agraphia

Calcarine area
Hemanopsia (or bilateral blindness if both posterior arteries occluded)

2.6 Dysphagia and Aspiration Post Stroke

Dysphagia

- Very common
- Finestone et al. (1995) found 47% of rehab admissions had dysphagia, 49% were clinically malnourished
- Can lead to malnutrition and dehydration
- Malnutrition associated with worse functional outcomes
- Dysphagia is associated with aspiration

Signs and Symptoms of Dysphagia

- Choking on food
- Coughing during meals
- Drooling or loss of food from mouth
- Pocketing of food in cheeks
- Slow, effortful eating
- Difficulty swallowing pills
- Avoiding foods or fluids
- Complaining of:
- Food sticking in throat
- Problems swallowing

- Reflux or heartburn

Aspiration Post-Stroke

- Prospective studies of acute strokes (< 5 days) – 21%-42% aspirate
- 8%-15% at 3 months

Factors Associated with Aspiration Difficulties Post-Stroke

- Brainstem stroke
- Difficulty swallowing oral secretions
- Coughing/throat clearing, choking or wet gurgly voice quality after swallowing water
- Weak voice and cough
- Recurrent lower respiratory infections
- Aspiration or pharyngeal delay on VMBS
- Immunologically compromised or chronic lung disease
- Poor oral hygiene

Pneumonia Post-Stroke

- Varies with criteria used and population group studied
- 7% - 32% incidence in acute stroke population
- Up to 20% of individuals with stroke-related dysphagia die during the first year post-stroke of aspiration pneumonia

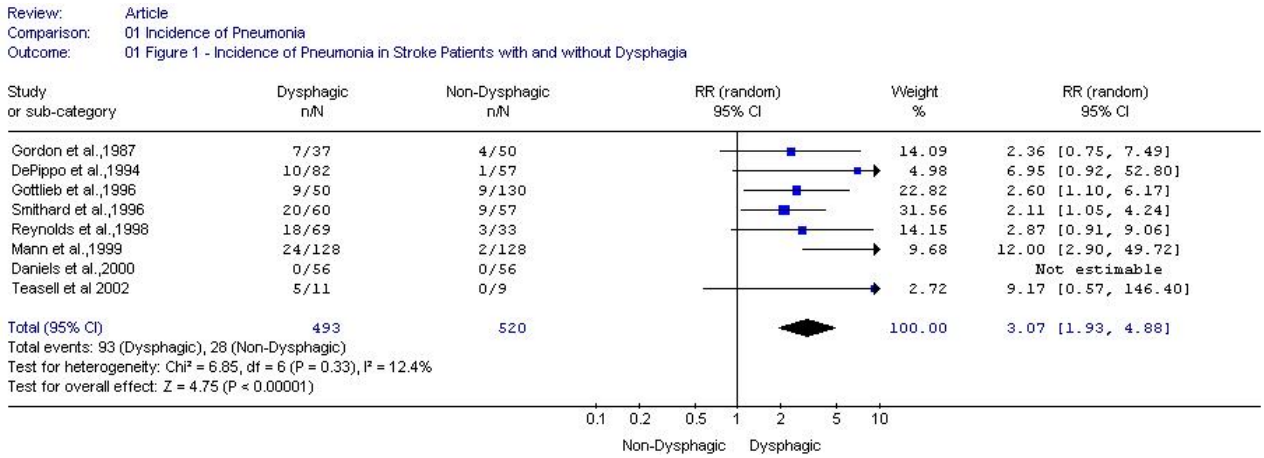
Association of Dysphagia or Aspiration and Pneumonia Post-Stroke

- Aspiration alone does not lead to pneumonia
- Aspiration of small amounts of saliva during sleep occurs in ½ of elderly
- Pneumonia is likely to occur when lung's natural defenses are overwhelmed by excessive or toxic aspirate

Table 2.10 Relationship Between Dysphagia and Pneumonia

Study	Incidence of Pneumonia Among Patients with and without Dysphagia	OR (95% CI, fixed effects model)
Gordon et al. 1987	7/37 vs. 4/50	2.63 (0.72 to 9.96)
De Pippo et al. 1994	10/82 vs. 1/57	7.78 (0.97 to 62.6)
Gottlieb et al. 1996	9/50 vs. 9/130	2.95 (1.10 to 7.94)
Smithard et al. 1996	20/60 vs. 9/57	2.67 (1.09 to 6.50)
Reynolds et al. 1998	18/69 vs. 3/33	3.53 (0.96 to 12.99)
Teasell et al. 2002	5/11 vs. 0/9	-
Falsetti et al. 2009	1/89 vs. 8/62	13.04 (1.44 to 286)
Combined estimate	70/398 vs. 34/398	2.28 (1.44 to 3.61)

Comparison of Pneumonia Frequency between Dysphagic and Non-Dysphagia Stroke Patients



Silent Aspiration Post-Stroke

- Penetration of food below level of true vocal cords without cough or outward sign of difficulty
- 8%-26% of aspirators acutely (< 5 days)
- Reliability of clinical assessment uncertain – increasing reliance on VMBS studies

2.7 Lacunar Infarcts

Short penetrating arteries that are end arteries with no anastomotic connections supply the medial and basal portions of the brain and brainstem. These small arteries arise directly from large arteries causing the gradation between arterial and capillary pressure to occur over a relatively short distance and exposing these small arteries to high arterial pressures. Occlusion of small penetrating arteries (50 to 500 u in diameter) may lead to small cerebral infarcts (usually < 10 12 mm) in the deep subcortical regions of the brain (Adams et al. 1997). They are associated with hypertension. Marked hypertrophy of the subintimal hyaline (lipohyalinosis) occurs with eventual obliteration of the vascular lumen. On healing after infarction a small cavity or "lacune" forms.

Most lacunar infarcts occur within the deep grey nuclei and some may involve multiple sites (see Table 2.10). The onset of a focal deficit may occur suddenly or progress over several hours. Similarly, both the time frame and extent of recovery in these patients is variable. Lacunar infarcts are often mistaken for a thromboembolic TIA. CT scan may show a small, deep infarct; however, many are too small to be seen without MRI. Smaller lacunar infarcts may be asymptomatic. Fischer (1982) has described 21 lacunar syndromes. The four most common lacunar syndromes are shown in Table 2.11.

Table 2.11 Sites of Lacunar Infarcts

Lacunar Lesions	%
Lenticular nuclei (especially putamen)	65
Pons	39
Thalamus	32
Internal capsule (posterior limb) and corona radiate	27
Caudate	24

Frontal white matter	17

Table 2.12 Common Lacunar Syndromes

Syndrome Manifestation	Lesion Site	Clinical
Pure motor hemiparesis	Posterior limb of internal capsule Lower pons (basis pontis)	Contralateral weakness of face, arm and leg No sensory involvement
Pure sensory stroke	Sensory nucleus of the thalamus	Sensory signs and/or symptoms involving contralateral half of the body
Dysarthria – clumsy hand	Upper pons (basis pontis)	Dysarthria and dysphagia Weakness of one side of the face and tongue Clumsiness and mild weakness of the hand
Ataxic hemiparesis	Upper pons (basis pontis)	Hemiparesis and limb ataxia on the same side

2.8 Cognitive Impairments Post Stroke

Defining Cognitive Impairments Post Stroke

Vascular dementia is defined as loss of cognitive function resulting from ischemic brain lesions due to cerebrovascular disease or cardiovascular pathology. Vascular cognitive impairment refers to the continuum of mild vascular cognitive impairment to vascular dementia.

Vascular Cognitive Impairment

Stroke survivors with vascular cognitive impairment but no dementia exhibit impairments of attention, executive function, and processing speed but have preservation of memory and orientation when compared to those who have Alzheimer's dementia

Prevalence of Dementia Post-Stroke

- As many as two-thirds of stroke pts go on to experience cognitive impairment or decline following stroke
- Approximately one-third develop dementia
- Risk of developing dementia may be 10x greater among individuals with stroke than those without

Diagnosis of Vascular Cognitive Impairment

At present there is no gold standard for the diagnosis and assessment of vascular cognitive impairment.

Vascular vs. Alzheimer's Dementia

Characteristic	Vascular Dementia	Alzheimer's Disease
Onset	Sudden or gradual	Gradual
Progression	Slow, stepwise fluctuation	Constant insidious decline

Neurological findings	Evidence of focal deficits	Subtle or absent
Memory	Mildly affected	Early and severe deficit
Executive function	Early and severe	Late
Dementia Type	Subcortical	Cortical
Neuroimaging	Infarcts or white matter lesions	Normal; hippocampal atrophy
Gait	Often disturbed early	Usually normal
Cardiovascular history	TIAs, strokes, vascular risk factors	Less common

2.9 Fatigue Post Stroke

According to de Groot et al. (2003), fatigue is a common complaint after stroke that occurs in 30% to 60% of stroke survivors (Staub and Bougousslavsky 2001a, 2001b, Michael 2002, Ingles et al. 1999, van der Werf et al. 2001, Glader et al. 2002). In a study by Ingles et al. (1999), 68% of 88 subjects who had strokes reported problems with fatigue at 3 and 13 months after stroke. Along the same lines, van der Werf et al. (2001) found that while 50% of the stroke group reported that fatigue was their main complaint, only 16% of the non-stroke group gave a similar response.

In discussing the effects of fatigue, Inges et al. (1999) found that stroke survivors who reported fatigue on a daily basis attributed more functional limitations to it in both physical and psychosocial (but not cognitive) domains than the controls. Similarly, Glader et al. (2002) reported that fatigue independently predicted decreased functional independence, institutionalization, and mortality, even after adjusting for age. The authors suggested that impairments after stroke likely contribute to fatigue which in turn contributes to impairment. Fatigue was also found to correlate significantly with measures of functional disability and neuropsychologic problems (van der Werf et al. 2001). Although there is limited information on the factors associated with post-stroke fatigue, some have reported an association with living alone or in an institution, impairment in ADLs, poor general health, anxiety, pain, depression, and a previous stroke (Glader et al. 2002, van der Werf et al. 2001). There is currently no evidence that fatigue is associated with time since stroke, severity of stroke, or side of lesion (Ingles et al. 1999, van der Werf et al. 2001, Staub et al. 2000, Glader et al. 2002). There is preliminary evidence that location of stroke may increase likelihood of fatigue (Staub et al. 2000).

References

Adams RD, Victor M, Ropper AH. Cerebrovascular Disease. In: Adams RD, Victor M, Ropper AH, editors. Principles of Neurology. New York: McGraw-Hill, Health Professions Division, 1997: 777-873.

Annet M. Hand preference and the laterality of cerebral speech. Cortex 1975; 11:305-328.

Bogousslavsky J, Regli F. Anterior cerebral artery territory infarction in the Lausanne Stroke Registry. Clinical and etiologic patterns. Arch Neurol 1990; 47(2):144-150.

Campbell A, Brown A, Schildroth C, et al. The relationship between neuropsychological measures and self-care skills in patients with cerebrovascular lesion. J Natl Med Assoc 1991; 83: 321-324.

Crossman AR, Neary D. Neuroanatomy: an illustrated colour text (2nd Ed). Churchill Livingston, Harcourt Publishers Limited, London, England 2000.

De Groot MH, Phillips SJ, Eskes GA. Fatigue associated with stroke and other neurologic conditions: Implications for stroke rehabilitation. Arch Phys Med Rehabil. 2003; 84(11):1714-20.

Delaney G, Potter P. Disability post stroke. In: Teasell RW (ed). Long-Term Consequences of Stroke. Physical Medicine and Rehabilitation: State of the Art Reviews, Hanley & Belfus Inc., Philadelphia; 7(20):27-42, 1993.

Dombovy ML. Stroke: Clinical course and neurophysiologic mechanisms of recovery. Critical Reviews in Physical and Rehabilitation Medicine 1991; 2(17):171-188.

Duncan PW, Lai SM. Stroke recovery. Topics Stroke Rehabil 1997; 4(17): 51-58.

Feigenson JS, McCarthy ML, Greenberg SD, et al. Factors influencing outcome and length of stay in a stroke rehabilitation unit: Part II. Comparison of 318 screened and 248 unscreened patients. Stroke 1977; 8:657.

Fisher CM. Lacunar stroke and infarcts- a review. Neurology 1982; 32: 871-876.

Gacs G, Fox AJ, Barnett HJ, Vinuela F. Occurrence and mechanisms of occlusion of the anterior cerebral artery. Stroke 1983; 14(6):952-959.

Glader EL, Stegmayr B, Asplund K. Poststroke fatigue: a 2-year follow-up study of stroke patients in Sweden. Stroke. 2002 May; 33(5):1327-33.

Hier DB, Mondlock J, Caplan LR. Behavioral abnormalities after right hemisphere stroke. Neurology. 1983; 33(3):337-344 (a).Hier DB, Mondlock J, Caplan LR. Recovery of behavioural abnormalities after right hemisphere stroke. Neurology 1983; 33:345-350 (b).

Ingles JL, Eskes GA, Phillips SJ. Fatigue after stroke. Arch Phys Med Rehabil. 1999 Feb; 80(2):173-8.

Kazui S, Sawada T, Naritomi H, Kuriyama Y, Yamaguchi T. Angiographic evaluation of brain infarction limited to the anterior cerebral artery territory. Stroke 1993; 24(4):549-553.

Kiernan JA. Blood Supply of the Central Nervous System. In: Kiernan JA, editor. Barr's The human nervous system: an anatomical viewpoint. Philadelphia: Lippincott-Raven, 1998: 439-455.

Kumral E, Bayulkem G, Evyapan D, Yunten N. Spectrum of anterior cerebral artery territory infarction: clinical and MRI findings. Eur J Neurol 2002; 9(6):615-624.

Kwasnica CM. Unilateral neglect syndrome after stroke:theories and management issues. Critical Reviews in Physical and Rehabilitation Medicine 2002; 14(1):25-40.

Mayo NE, Korner-Bitensky NA, Becker R. Recovery time of independent function post-stroke. Am J Phys Med Rehabil 1991; 70:5-12.

Mesulam M. Ateention, confusional states and neglect. In Mesulam M (ed.) Principles of Behavioral Neurology. FA Davis, Philadelphia,PA, 1985,pp125-127.

Michael K. Fatigue and stroke. Rehabil Nurs. 2002 May-Jun; 27(3):89-94, 103.

Myers PS. Right Hemispheric Damage-Disorders of Communication and Cognition. 1st edition, Singular Publishing Group Inc, San Diego,CA, 1999.

O'Brien MT, Pallet PJ. Total care of the stroke patient. Little Brown &Co., 1978.

Pedersen PM, Jorgensen HS, Nakayama H, Raaschou HO, Olsen TS.Hemineglect in acute stroke--incidence and prognostic implications. The Copenhagen Stroke Study. Am J Phys Med Rehabil 1997; 76(2):122-7.

Pryse-Phillips W, Murray TJ. Essential Neurology. Garden City, NY, Medical Examination Publishing Company; pp. 4 and 358-385, 1978.

Rafal RD. Neglect. Curr Opin Neurobiol 1994 Apr; 4(2):231-6.

Robinson RG, Starr LB, Lipsey JR, Rao K, Price TR. A two-year longitudinal study of post-stroke mood disorders: dynamic changes in associated variables over the first six months of follow-up. Stroke 1984; 15(3):510-517.

Scremin OU. Cerebral Vascular System. In: Paxinos G, Mai JK, editors. The Human Nervous System. San Diego: Elsevier Academic Press, 2004: 1325-1348.

Staub F, Bogousslavsky J. Fatigue after stroke: a major but neglected issue. Cerebrovasc Dis. 2001 Aug; 12(2):75-81. (a)

Staub F, Bogousslavsky J. Post-stroke depression or fatigue. Eur Neurol. 2001; 45(1):3-5. (b)

Staub F, Bogousslavsky J. Fatigue after stroke: a pilot study (abstract). Cerebrovasc Dis 2000; 19:62. (c)

Teasell RW. Stroke rehabilitation. Physical Medicine and Rehabilitation: State of the Art Reviews 1998; 12(3):355-592.

van der Werf SP, van den Broek HL, Anten HW, Bleijenberg G. Experience of severe fatigue long after stroke and its relation to depressive symptoms and disease characteristics. Eur Neurol. 2001; 45(1):28-33.