Chapter 3
Stroke

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3.1 Introduction and Historical Background

Cerebrovascular disease or stroke refers to a group of disorders of the brain vasculature that can affect the vascular supply of the underlying tissues. Stroke, an important cause of prolonged disability, often makes the survivors unable to return to work or continue their duties as family members or citizens. Stroke has a considerable psychosocial and economic impact worldwide. The term stroke was often known as "apoplexy," a Greek word meaning "struck suddenly with violence." In the vast majority of cases, "stroke undoubtedly alters the history of the world for the survivor" as the loss of function is often instantaneous, totally unanticipated and impairments more or less permanent and devastating. The loss of function may include one or more features like sudden inability to move a limb, stand or walk, speak or understand spoken language, see, read, write, think, and feel.

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For many of the eminent leaders in medicine, science, and politics, stroke had prematurely cut short their productivity. Louis Pasteur, a biologist, microbiologist, and chemist; Russell DeJong, the first editor of the journal *Neurology*; Raymond Escourolle, the neuropathologist; and H. Houston Merritt, the writer of *Merritt's Neurology* were a few who got severely disabled by multiple strokes. Prominent political leaders, including Vladimir Lenin, Franklin D. Roosevelt, Winston Churchill, and Joseph Stalin, had severe cerebrovascular disease during the helm of their careers.

In 400 BC, perhaps Hippocrates, the Greek physician, was the first to write about the medical aspects of the stroke. He was mostly interested in the prognosis of the illness and was a keen observer. In his aphorisms on apoplexy, he stated that most subjects were between the ages of forty and sixty and mentioned that "attacks of numbness" and "pains in the head" were reflections of the "impending apoplexy." The Greeks recognized that interruption of the blood vessels to the brain was the cause for loss of consciousness, and they named those arteries carotid, meaning "deep sleep," from the Greek word *karos*. Roman physician and philosopher Galen, a few hundred years after Hippocrates, described the anatomy of the brain and its blood vessels following dissections of animals. Galen believed the disease to be a disequilibrium between the four body humors: the black bile, yellow bile, phlegm, and blood. Until the fourteenth century, his voluminous writings were blindly followed by physicians, and dissection, experimentation, and observations were discouraged and considered unscholarly.

Andreas Vesalius, a physician and an anatomist in the sixteenth century, challenged the Galenic tradition by dissecting humans and, based on his personal observations, wrote books on human anatomy known as *De humani corporis fabrica libri septem*, meaning on the fabric of the human body. Vesalius is considered the founder of modern human anatomy. In the latter half of the seventeenth century, two eminent physicians, Johann Jakob Wepfer and Thomas Willis, made further anatomical and clinical observations on apoplexy. Wepfer performed meticulous examinations of the brains of patients dying of apoplexy. It was Wepfer who described the carotid siphon and the course of the middle cerebral artery in the Sylvian fissure. He mentioned that apoplexy could be either due to obstruction of the carotid and vertebral arteries or due to a bleed into the brain. Thomas Willis, a physician and a neuroanatomist, best known for his "circulus arteriosus cerebri" or cerebral arterial circle, also recognized transient ischemic attacks and the consequence of embolism. In 1689, William Cole, an English physician, in a medical essay concerning apoplexy, first introduced the word "stroke" in the field of medicine.

In the eighteenth century, Giovanni Battista Morgagni, the father of modern anatomical pathology, focused his attention on the pathology and cause of apoplexy. He recognized that paralysis was on the side of the body opposite to the brain lesion and gave an elaborate description of the intracerebral hemorrhage. His work helped in shifting the emphasis from anatomy to etiology, pathology, and clinical manifestations of the diseases. In 1812, John Cheyne, a British physician and a surgeon, in his book, made a strong attempt to separate apoplexy from the phenomena of lethargy and coma. In the nineteenth century, John Abercrombie, a physician and a

philosopher, made a detailed clinical classification of apoplexy and published the book *Pathological and Practical Researches on Diseases of the Brain and the Spinal Cord*, a book regarded as the first textbook in neuropathology. Abercrombie also speculated the etiological mechanisms underlying the spasm of vessels, interruption of the circulation, and rupture of vessels causing hemorrhage. In the later part of the nineteenth century, Rudolf Virchow, a German physician and a pathologist, mentioned certain important experimental and pathological information about vascular disease, particularly relating to the thrombus and infarction. A French neurosurgeon, Henri Düret, made a detailed observation about the distribution of the arteries and veins in the cranium, including the arteries supplying the cranial nerve nuclei. During the same period, several clinicians gathered information on the clinical findings of stroke that involved various regions of the brain. However, minimal concern was given to the pathogenesis, laboratory confirmation, or management for the same. The medical textbooks and scholarly articles written by William Osler, William Richard Gowers, Samuel Alexander Kinnier Wilson, and Charles Foix made detailed clinical descriptions about many stroke syndromes. Charles Miller Fisher, a Canadian neurologist and a neuropathologist, mentioned "...fleeting attacks of paralysis, numbness, tingling, speechlessness, unilateral blindness or dizziness…" as the warning symptoms of carotid artery disease. During the mid- and later years of the twentieth century, Fisher made major pathological and clinical observations of carotid artery disease, intracerebral hemorrhage, lacunar stroke, and vascular lesions of the posterior circulation.

In the last few decades, there has been an explosive growth of interest in and knowledge about stroke. Recent advances in science and technology allowed better visualization of the normal, pathological anatomy, and functional aspects of the brain. Technology has also helped to pool up the databases and registries of large numbers of stroke studies to identify and quantify the most common clinical and laboratory findings in various stroke syndromes. Researches in the field of epidemiology have helped in accurately identifying the risk factors for stroke and plausible prevention strategies for the same. Even significant advances in surgical, medical, and radiological management have shown success in reducing the morbidity and mortality of stroke.

3.2 Normal Arterial Blood Supply of the Brain

The human brain, though weighing over a kilogram, consumes approximately 20% of the body's oxygen supply at rest and must continuously receive a voluminous amount of blood, about one liter per minute. Two pairs of arterial trunks, the right and the left internal carotid arteries, and the right and the left vertebral arteries supply blood to the brain. These four arteries lie within the subarachnoid space, and their branches anastomose at the base of the brain around the optic chiasma, the stalk of the pituitary gland, and the hypothalamus. The following section of this chapter focuses on arteries and their branches supplying the brain parenchyma.

3.2.1 *Internal Carotid Artery*

Both the right and the left internal carotid arteries are the branches of common carotid arteries. Typically, the right common carotid artery arises from the brachiocephalic artery and the left common carotid artery arises directly from the aortic arch (Fig. 3.1). Each common carotid artery, roughly near the C4 level or the upper level of the thyroid cartilage, bifurcates to form the respective internal and external carotid arteries. Each internal carotid artery consists of cervical, petrous, cavernous, and cerebral segments (Fig. 3.2). The cervical segment traversing through the neck enters the cranial cavity through the carotid canal of the petrous part of the temporal bone (petrous segment) and reaches the cavernous sinus from below (cavernous segment). Subsequently, the internal carotid artery pierces the roof of the cavernous sinus and enters the cranial cavity, flanked by the oculomotor and optic nerves. The cerebral segment of the internal carotid artery is quite short and extends upward and backward to give rise to all the major branches of the internal carotid artery. From the neck to the termination, the internal carotid artery makes several, almost 90° turns, and these turns reduce the pressure and the velocity of the blood it brings to the thin-walled arteries of the brain.

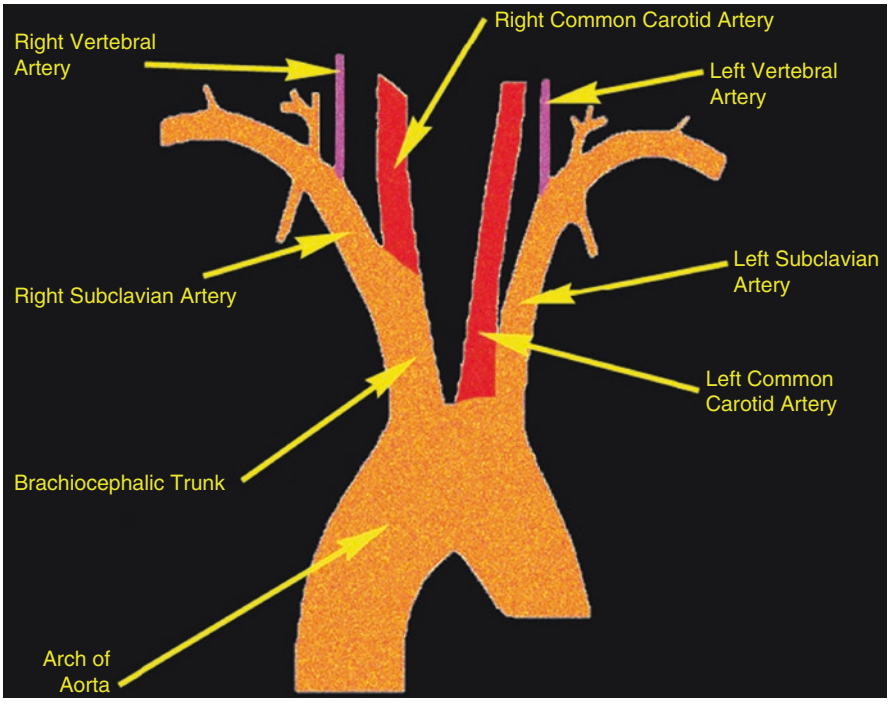

Fig. 3.1 An illustration of the right common carotid artery arising from the brachiocephalic artery and the left common carotid artery arising from the arch of the aorta

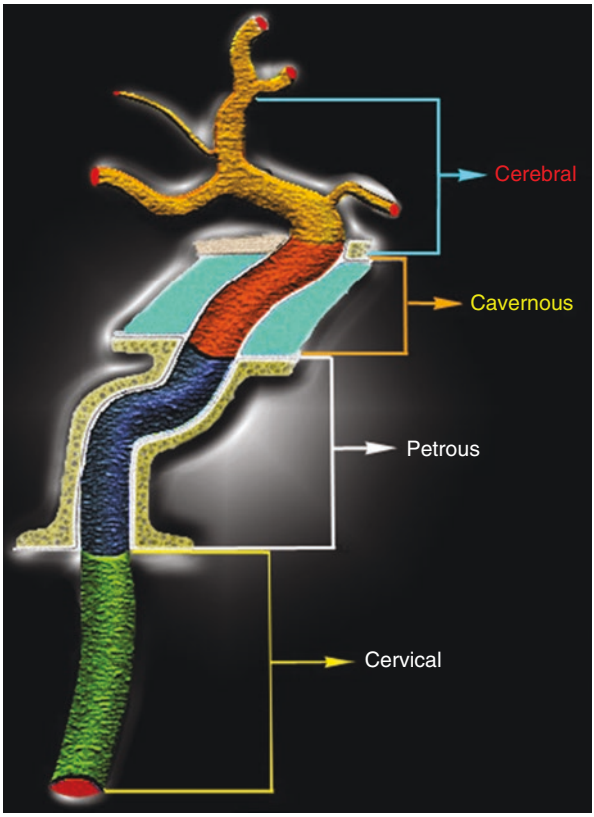

Fig. 3.2 The cervical, petrous, cavernous, and cerebral segments of the internal carotid artery

The cerebral segment of the internal carotid artery branches into ophthalmic, anterior choroidal, posterior communicating, anterior cerebral, and middle cerebral arteries (Fig. 3.3). The ophthalmic artery supplies all the structures in the orbit. The anterior choroidal artery, a small narrow artery arising from the posterior aspect of the internal carotid artery, while traversing along the path of the optic tract toward the lateral geniculate body, supplies the optic tract, lateral geniculate body, optic radiation, hippocampus, posterior limb of the internal capsule, tail of the caudate nucleus, and choroid plexus of the inferior horn of the lateral ventricle. The posterior communicating artery, a small vessel that originates from the cerebral segment of the internal carotid artery, close to its terminal bifurcation, runs posteriorly above the oculomotor nerve to join the posterior cerebral artery forming part of the cerebral arterial circle. Usually, the size of the right and left posterior communicating arteries is not identical; one can be frequently smaller than the other and at times entirely absent or doubled. The main function of these arteries is to ensure sustainable blood supply to the brain in case if the internal carotid or vertebral artery occludes.

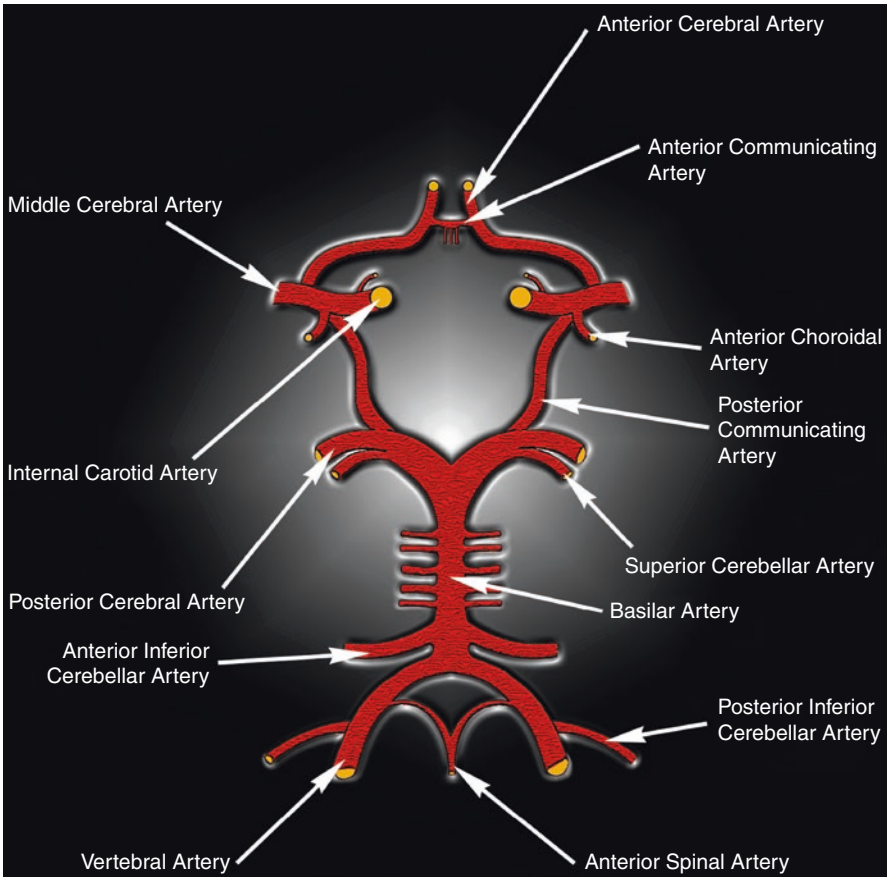

Fig. 3.3 An illustration of the formation of the "circle of Willis" by the anterior and posterior circulations

3.2.2 Anterior Cerebral Artery

The anterior cerebral artery, one of the two terminal branches of the internal carotid artery, runs forward and medially, superior to the optic nerve, to enter the great longitudinal fissure. Here, it connects with its counterpart of the contralateral side by a short branch named the anterior communicating artery. In the longitudinal fissure, the right and left anterior cerebral arteries lie in a close approximation of about 4–5 mm, and these arteries follow the genu and then the superior border of the corpus callosum until they anastomose with their corresponding posterior cerebral arteries. The anterior communicating, the central, and the cortical are three branches of each anterior cerebral artery. The anterior communicating artery is approximately 4 mm long and gives off numerous branches that supply the structures, including the

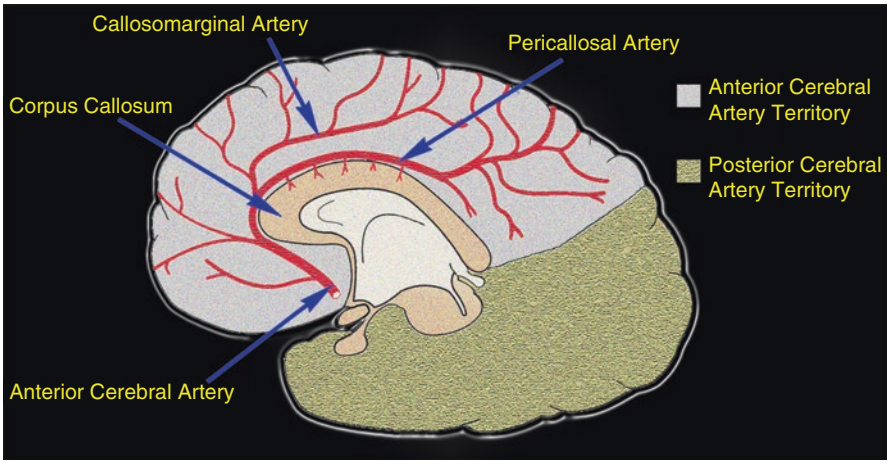

Fig. 3.4 The vascular territory of the anterior cerebral artery and its branches

optic chiasma, hypothalamus, para-olfactory areas, fornix, and cingulate gyrus. The central branches arising from the initial part of the anterior cerebral artery, just before or after the origin of the anterior communicating artery, supply the anterior part of the corpus callosum, head of the caudate nucleus, part of the putamen, globus pallidus, and anterior limb of the internal capsule. Among the central branches, the recurrent artery of Heubner (medial striate artery) is the largest perforating branch from the central branches and is the only one routinely seen on angiography.

The names of the cortical branches of the anterior cerebral artery according to their distribution are orbital, frontal, and parietal. The orbital branch supplies the orbital surface of the frontal lobe, olfactory lobe, medial orbital gyrus, and gyrus rectus. The frontal branch supplies the medial frontal gyrus, cingulate gyrus, paracentral lobule, superior frontal gyrus, middle frontal gyrus, and precentral gyrus. Whereas, the parietal branch supplies the superior parietal lobule (precuneus) and the adjoining lateral surface. To sum up, the cortical branches of the anterior cerebral artery supply the motor and somatosensory cortices representing the lower limb, i.e., the entire medial surface of the cerebral cortex up to the parieto-occipital sulcus (Fig. 3.4) including a small strip of cortex on the adjoining lateral surface (Fig. 3.5).

3.2.3 Middle Cerebral Artery

The middle cerebral artery, the larger terminal branch of the internal carotid artery, runs first in the lateral sulcus (also known as lateral or Sylvian fissure) then posterosuperiorly on the insula and divides into central and cortical branches. Like the anterior cerebral artery, the central branches supply blood to the deeper structures,

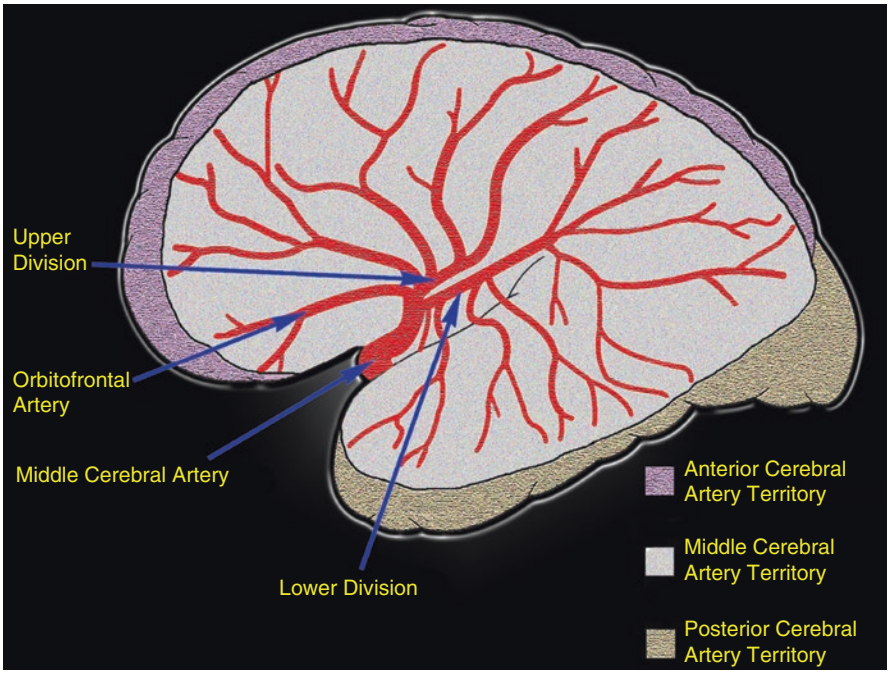

Fig. 3.5 The vascular territory of the middle cerebral artery and its branches

and cortical branches supply blood to the cortical regions. The central branches have 10–15 slender striate arteries (lenticulostriate arteries) that pierce the floor of the lateral sulcus to supply the deeper cerebral structures, including the corpus striatum, most of the caudate nucleus and the lenticular nucleus, and the external and the internal capsules.

The cortical branches, during their course along the lateral sulcus, are named according to their location and distribution as the orbital, frontal, parietal, and temporal branches. The orbital branches serve the inferior frontal gyrus and the lateral orbital surface of the frontal lobe. The frontal branches serve the middle frontal, the precentral, and a part of the inferior frontal gyri, and the parietal branches serve the postcentral gyrus, the whole inferior parietal lobule, the lower part of the superior parietal lobule, and the angular and supramarginal gyri. The temporal branches serve the lateral surface of the entire temporal lobe up to the occipital gyri. Therefore, the cortical branches of the middle cerebral artery supply the whole lateral surface of the cerebral cortex (Fig. 3.5) except the narrow strip supplied by the anterior cerebral artery and the occipital pole and the inferolateral surface of the cerebral cortex supplied by the posterior cerebral artery, i.e., the cortical branches supply the motor and somatosensory cortices representing the entire body, except the lower limb (Fig. 3.4).

3.2.4 *Vertebral Artery*

Each of the vertebral arteries, arising from the respective first part of the subclavian artery, ascends cephalically through the transverse processes of the upper six cervical vertebrae and pierces the atlanto-occipital membrane and the dura mater to enter the posterior cranial fossa through the foramen magnum. Close to the anterolateral aspect of the medulla, the right and the left vertebral arteries converge medially as they ascend, and approximately at the pontomedullary junction level, the right and the left vertebral arteries unite to form the basilar artery (Fig. 3.6). Together the intracranial branches of the vertebral and basilar arteries supply the spinal cord, brainstem, cerebellum, posterior parts of the diencephalon, and parts of occipital and temporal lobes of the cerebral cortex.

The anterior and posterior spinal arteries, the posterior inferior cerebellar artery, and the medullary divisions are the main branches of the vertebral artery supplying the brainstem and the spinal cord. The anterior spinal artery emerges near the end of the vertebral artery and descends anteriorly to anastomose with its counterpart from the opposite side at the mid-medullary level. The single trunk then descends on the anterior median fissure of the spinal cord. In the majority of the subjects, the posterior spinal arteries arise from the posterior inferior cerebellar artery or may emerge directly from the vertebral artery near the medulla. The right and left posterior

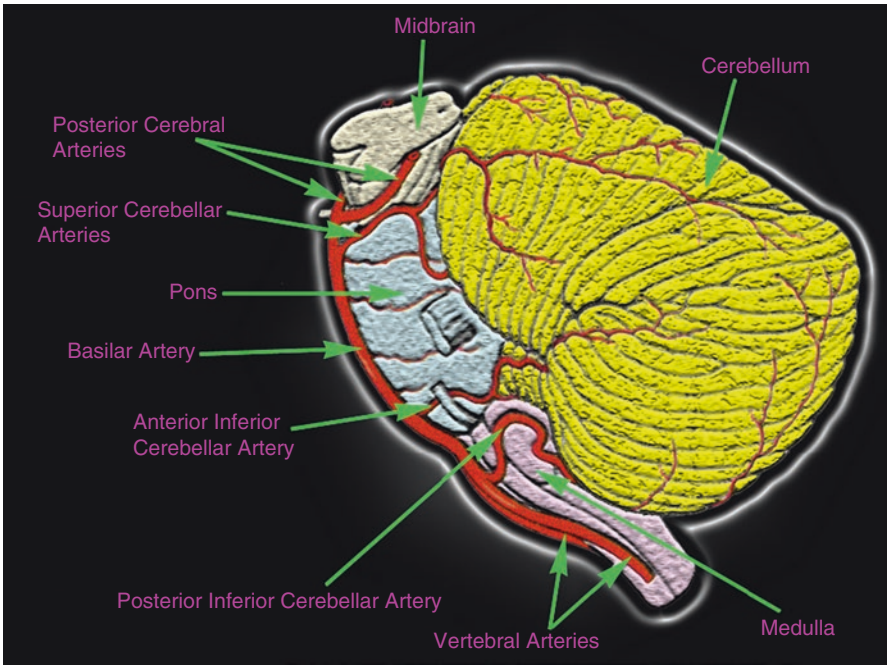

Fig. 3.6 Illustration of the posterior circulation of the brain

spinal arteries descend along the respective posterolateral surface of the spinal cord and supply the ipsilateral grey and white dorsal columns of the spinal cord. The anterior spinal artery and the right and left posterior spinal arteries are reinforced by radicular arteries that enter the vertebral canal through the intervertebral foramina, sequentially from the vertebral, ascending cervical, posterior intercostal, and first lumbar arteries.

The posterior inferior cerebellar artery, the largest branch of the vertebral artery, arises near the lower end of the olivary nucleus, curves posteriorly, and ascends behind the roots of the ninth and tenth cranial nerves. Near the inferior border of the pons, the artery divides, and the medial branch supplies the cerebellar hemisphere and inferior vermis. The lateral branch supplies the inferior cerebellar surface as far as its lateral border and anastomoses with the anterior inferior and superior cerebellar arteries of the basilar artery. The trunk of the posterior inferior cerebellar artery supplies the medulla oblongata dorsal to the olive, lateral aspect of the hypoglossal nucleus near its emerging roots, the dentate nucleus, and the choroid plexus of the fourth ventricle. The medullary divisions, small branches, emerging from the cranial portion of the vertebral artery, supply the medulla oblongata (also served by the posterior inferior cerebellar artery and/or the anterior and posterior spinal arteries).

3.2.5 Basilar Artery

The basilar artery is a large median vessel formed by the union of the vertebral arteries at the mid-medullary level. It lies in the shallow median groove on the ventral pontine surface and extends from the rostral end of the medullary pyramids to the rostral end of the pons. Near the interpeduncular cistern, the artery terminates by dividing into two posterior cerebral arteries (Fig. 3.6). The branches of the basilar artery are the pontine divisions, labyrinthine artery, anterior inferior cerebellar artery, and superior cerebellar artery. The small pontine divisions or branches arising at 90° to the basilar artery penetrate and serve the substance of the pons and the midbrain. The labyrinthine artery, usually a branch of the anterior inferior cerebellar artery and less frequently a branch of the basilar artery, is larger than the pontine branches, accompanies the facial and the vestibulocochlear nerves, and supplies the internal ear.

The anterior inferior cerebellar artery, just rostral to the medullary pyramids, emerges from the basilar artery and follows the sixth cranial nerve and loops around the seventh and eighth cranial nerves and reaches the cerebellopontine angle. The small pontine branches and the medial and lateral branches of the anterior inferior cerebellar artery serve the pons and midbrain and the anteromedial and anterolateral aspects of the cerebellum, respectively. The branches of the artery mentioned above anastomose with branches of the posterior inferior cerebellar and superior cerebellar arteries. The superior cerebellar artery emerges from the basilar artery, caudal to the third cranial nerve, and curves around the cerebral peduncle to supply the

superior surface of the cerebellum. The branches also vascularize the pineal body, the midbrain, and the choroid plexus of the third ventricle. The branches of the superior cerebellar artery anastomose with branches of the anterior inferior cerebellar artery.

3.2.6 Posterior Cerebral Artery

The posterior cerebral artery, larger than the superior cerebellar artery, a terminal branch of the basilar artery originates near the third cranial nerve, lateral to the midbrain. The right and left posterior cerebral arteries run laterally, parallel with the superior cerebellar arteries, and join with the corresponding posterior communicating arteries. Each artery then winds around the cerebral peduncle and reaches the superior surface of the tentorium cerebelli to serve the temporal and occipital lobes. In addition to the choroidal branch, like the anterior and middle cerebral arteries, the posterior cerebral artery also has cortical and central branches. Figure 3.7 illustrates the cortical vascular area supplied by the posterior cerebral artery. The temporal, the lateral and medial occipital, and the splenial are the cortical branches of the posterior cerebral artery. The temporal branch supplies the uncus, the parahippocampal, and the medial and lateral occipitotemporal gyri. The occipital branches supply the cuneus, precuneus, lingual gyrus, and posterolateral surface of the occipital lobe.

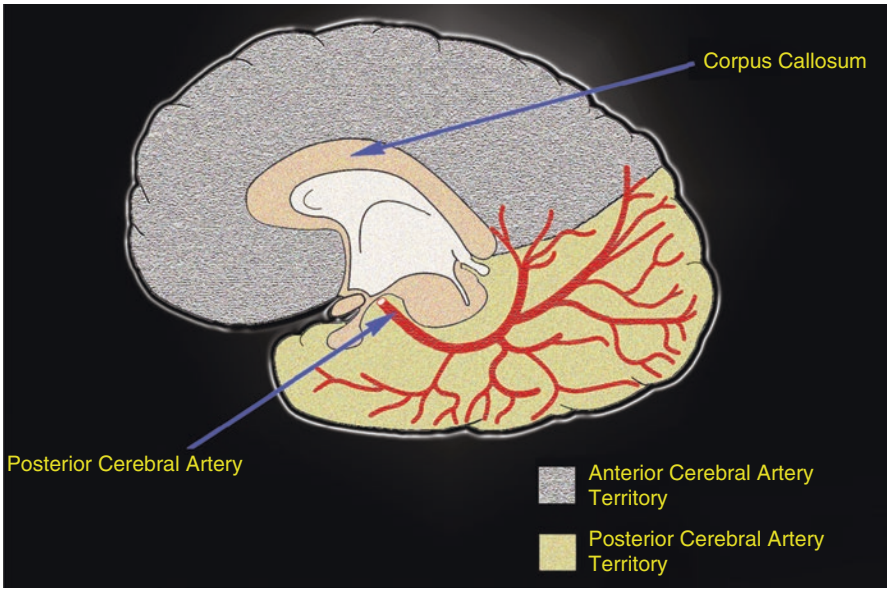

Fig. 3.7 The vascular territory of the posterior cerebral artery

The splenial artery supplies the splenium and posterior portion of the corpus callosum.

The central branches of the posterior cerebral artery vascularize the subcortical structures. Several central branches of the posterior cerebral artery, along branches from the posterior communicating artery, supply the optic tract, the mammillary bodies, and the thalamus. In addition to that, they supply the geniculate bodies, the cerebral peduncles and the interpeduncular area, the corticospinal tracts, the mesencephalic reticular formation, the substantia nigra, and the tegmentum of the midbrain. The choroidal branches supply the choroid plexus of the lateral and third ventricle, the medial and lateral geniculate bodies, the peduncle, the tegmentum, the colliculi, the pulvinar, the posterior and medial portions of the thalamus, and the pineal body.

3.2.7 Cerebral Arterial Circle

The cerebral arterial circle, also known as the circle of Willis or "circulus arteriosus," is a large arterial anastomosis at the base of the brain around the optic chiasma, infundibulum, and other structures of the interpeduncular fossa. This circle forms an anastomosis between proximal portions of the anterior and posterior cerebral arteries, the distal-most portion of the internal carotid artery, and the anterior and posterior communicating arteries (Fig. 3.8). Anteriorly, the anterior cerebral arteries are connected by the small anterior communicating artery, and, posteriorly, the two posterior cerebral arteries are merged to the corresponding side internal carotid artery by a posterior communicating artery. This arterial wreath equalizes the blood flow to various parts of the brain. In normal situations, due to equal blood pressure, only a minimal exchange of blood occurs between the right and left halves of the circle. Following the occlusion of one or more of the arteries contributing to the circle, alterations of blood flow in the arterial circle are bound to occur.

Variations in the caliber of the arteries forming the cerebral arterial circle are common. The hemodynamics of the arterial circle is influenced by variations in the caliber of communicating arteries and the segments of the anterior and posterior cerebral arteries that form a part of the circle. Among them, the greatest individual divergence seen is the presence or absence and the caliber of the posterior communicating arteries.

The circle is also a common site for saccular aneurysms. Congenital defects in the blood vessel wall and high turbulence of blood are a few key factors involved in the development of aneurysms. The most common sites for the development of saccular aneurysms are the origins of the anterior and posterior communicating artery, the bifurcation of the middle cerebral artery, the cavernous segment of the internal carotid artery, the bifurcation of the internal carotid artery, and various locations on the vertebrobasilar arteries. Rupture of these saccular cerebral aneurysms leads to subarachnoid hemorrhages and often results in serious neurological impairments.

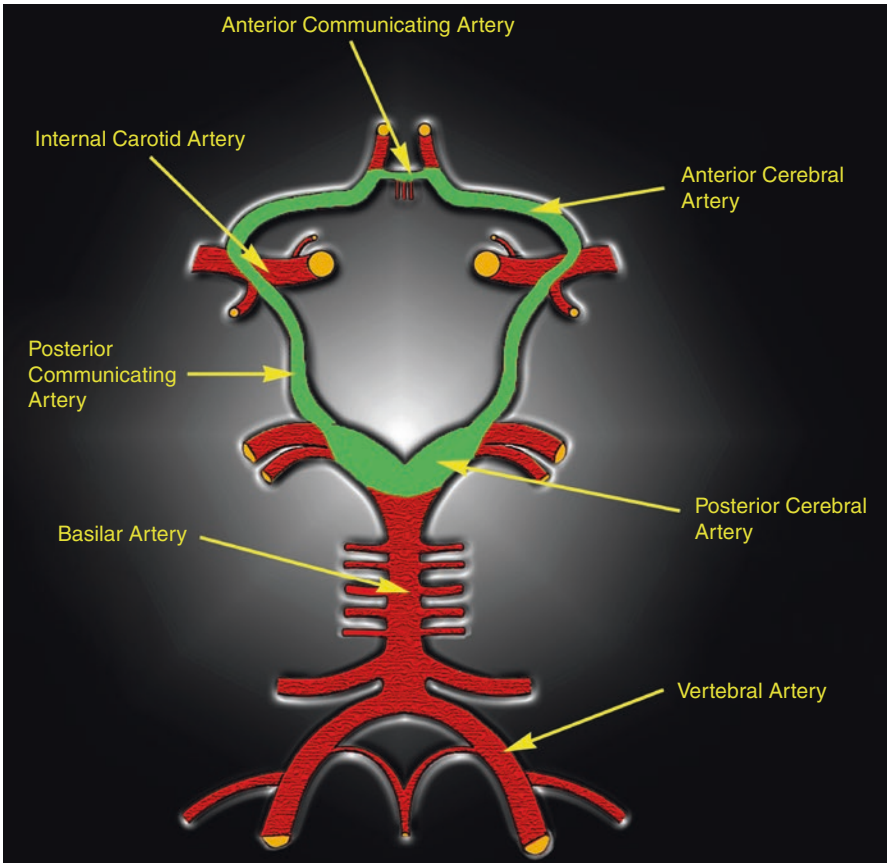

Fig. 3.8 The anastomosis of cerebral vessels forming the "circle of Willis"

3.3 Functional Anatomy of Cerebral Cortex and Internal Capsule

A longitudinal fissure separates the two cerebral hemispheres of the human brain. The outer layer, made up of gray matter, called the cerebral cortex (cerebral mantle), and the inner layer, called the white matter, constitute each cerebral hemisphere. The two cerebral hemispheres are joined beneath the cerebral cortex by the corpus callosum. The cerebral cortex consists of six lobes: the frontal, parietal, temporal, occipital, insular, and limbic lobes. The following section covers only the first four lobes of the cortex as the insular cortex and the limbic lobe connections with the rest of the brain and functions are complex and beyond the scope of this chapter.

3.3.1 *Frontal Lobe*

Regarding the lobes, the frontal lobe is considered the largest and accounts for approximately 40% of the cerebral cortex. The frontal lobe, the rostral region of the cerebral hemisphere and the youngest part of the brain based on the phylogeny, is located above the lateral fissure or Sylvian fissure and is limited posteriorly by the central sulcus. The primary motor cortex, premotor cortex, supplementary motor cortex, frontal eye field, and Broca's area of speech are the main regions of the motor cortex of the frontal lobe (Fig. 3.9). The dorsolateral, medial, and orbitofrontal regions constitute the prefrontal cortex region of the frontal lobe.

The primary motor cortex (Brodmann area 4), located within the precentral gyrus and anterior to the central sulcus, is involved in the control of voluntary movements through its projection of the cortical neurons to the brainstem and spinal cord. The primary motor cortex contains a topographically organized map known as the motor homunculus of the opposite half of the body, representing the head most laterally and the legs and feet medially on the hemisphere in the paracentral lobule. The representation of body parts is disproportionate to their physical size but proportionate to the ability to produce fine-controlled or fractionated movements. In addition to receiving input from the neighboring primary somatosensory area, premotor cortex, and ventral lateral nucleus of thalamus, the cortex area 4 has major thalamic connections and loops projected from the deep cerebellar nuclei and the basal ganglia. Inputs from these centers modulate the output of the primary motor cortex by promptly providing information about the position, timing, and coordination of voluntary movements.

The premotor cortex (Brodmann area 6), located immediately rostral to the primary motor cortex, assists in the integration of sensory and motor information for

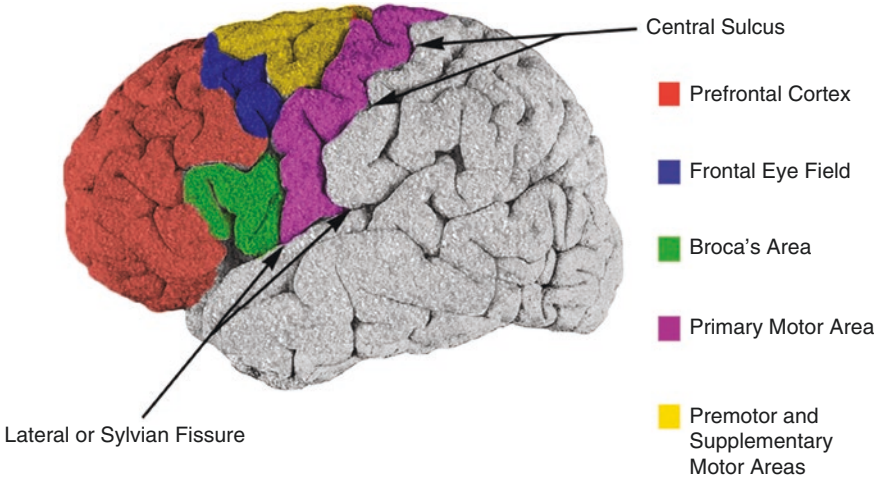

Fig. 3.9 An illustration of the frontal lobe

the performance of an action. The premotor area has neuronal projections from the secondary somatosensory area, the ventral anterior thalamic nucleus, and the premotor area of the contralateral side. The neurons in this cortex have extensive reciprocal connections with the primary motor cortex, which is in addition to those influencing motor behavior directly through the axons projecting to the lower motor neurons (corticobulbar and corticospinal pathways) of the brainstem and the spinal cord. Damage to the premotor cortex may result in apraxia (inability to perform purposeful skilled movements in the absence of paralysis, sensory loss, abnormal posture and tone, involuntary movement, incoordination, or inattentiveness), deficits in performing complex patterns of movements, and initiation and selection of movements.

The supplementary motor cortex lies medial to area 6, and this area has extensive reciprocal connections with the thalamus and the ipsilateral frontal lobe, including the primary motor cortex, the premotor area, the prefrontal area, and the frontal eye field. This cortex even has connections to the ipsilateral superior parietal area and the contralateral supplementary motor area and the motor cortices of the contralateral frontal lobe. Primarily, the role of the supplementary motor area is to control the movement involving complex tasks that require the temporal organization of sequential movements and retrieval of motor memory. The consequences of supplementary motor area damage strikingly resemble the effects of basal ganglia dysfunction, namely, akinesia and impairments in the performance of sequential and complex movements.

The pyramidal tract, the most important output from the motor cortex, consists of approximately 30% fibers from the primary motor cortex, 30% from the premotor and supplementary motor cortices, and the remaining from the somatosensory cortex of the parietal lobe and the cingulate gyrus. The projections from the primary motor cortex to the brainstem and spinal cord are facilitatory. The destruction of the pure pyramidal fibers arising from the primary motor cortex or the interruption of its pyramidal projections to the brain stem causes flaccid weakness. However, in most scenarios, cortical lesions are more extensive and involve premotor and supplementary motor areas. The latter two areas are normally inhibitory to the ventromedial bulbar reticular formation, and the inhibitory influences are conducted down to the spinal cord by the dorsal reticulospinal tract. Lesions involving the premotor and supplementary motor cortices or the projections to the brainstem and spinal cord will cause spastic weakness and hyperactive stretch reflex in upper motor neuron lesion conditions, including stroke.

The frontal eye field (Brodmann area 8) located rostral to the premotor cortex has extensive ipsilateral connections with several visual areas in the occipital, parietal, and temporal lobes. The eye field receives fibers from the prefrontal cortices and projects to the motor and premotor cortices. In addition to the above, there are prominent neuronal projections from the eye field to the superior colliculus, the pontine gaze center within the pontine reticular formation, and the cranial nuclei for extraocular muscles. The primary function of the frontal eye field is to control the voluntary movements of the eyes toward the contralateral visual field, and the eye

field damage will cause the inability to move the eyes toward the contralesional side voluntarily.

Broca's area (Brodmann areas 44 and 45), an essential area for language, which helps to put thoughts into words, lies in the inferior frontal gyrus of the dominant cerebral hemisphere. Broca's area is structurally and functionally heterogeneous, and this area has been implicated in diverse cognitive functions, extending well beyond articulation and comprehension. The area is connected to many regions of the brain, including the Wernicke's area (through a neuronal tract known as the arcuate fasciculus), prefrontal cortex, primary motor cortex, premotor cortex, supplementary motor area, parietal lobe (specifically the inferior parietal lobule), and visual cortices of the occipital lobe. The supplementary motor cortex of the frontal lobe is believed to participate in spontaneous and automatic speech and may play a part in the programming of speech by its connections with Broca's area. In addition to the above, the Broca's area also has projections to the basal ganglia. Broca's area functions in the assembly of phonemes into words and words into sentences and for forming the grammatical structure for sentences. The area is also involved in motor-related activities and sensorimotor learning and integration. The lesion in this area mainly leads to reduced fluency, improper or distorted articulation of phonemes, agrammatism, and impaired retrieval of words, resulting in non-fluent and effortful speech, with impaired repetition, word-finding difficulties with relatively preserved comprehension.

The prefrontal cortex (Brodmann areas 9 and 46) located on the lateral surface of the cerebral hemisphere receives major thalamic afferents and corticocortical connections within the frontal lobe, including the supplementary motor area, the premotor cortex, and the frontal eye field. All these thalamic and corticocortical connections are reciprocal. In addition to the above, the prefrontal area receives association fibers from the posterior and middle superior temporal gyrus, including the auditory association areas, the parietal lobe, and the limbic lobe. The prefrontal cortex has commissural connections with the homologous parts of the contralateral hemisphere and with the contralateral inferior parietal cortex. The prefrontal cortex is important for the spatial processing of afferent information, organization of working memory, the mnemonic processing of objects, understanding the value of time, normal expression of emotions, and the ability to predict the consequences of actions. The medial and orbitofrontal regions of the prefrontal lobes are closely linked with the limbic system, and damage to the same regions can cause disinhibition of affective behavior due to which the patient can be unduly jocular and unmindful about his or her intellectual disability. Impaired motivation has an association with lesions of the dorsolateral prefrontal cortex and anterior cingulate. Patients with prefrontal dysfunction show deficits in planning and decision-making, poor judgment, easy distractibility, inability to look ahead in time or generate hypotheses for future events, reduction in total verbal output, restricted range of sentence structures, echolalia (automatic imitative repetition of spoken words made by another person), and inability to appreciate verbal and nonverbal humor. Patients with prefrontal dysfunction will have emotional dysregulation and emotional blunting, behavioral rigidity, poor frustration tolerance, defective social and moral

reasoning, lack of self-awareness and thoughts and feelings for others, and increased susceptibility to psychiatric syndromes such as depression, mania, apathy, and obsessive-compulsive disorder. Uninhibited bladder may occur with bilateral brain lesions above the pontine micturition center, especially when the lesion is in the medial prefrontal area and anterior cingulate gyrus of the frontal lobe.

3.3.2 Parietal Lobe

The parietal lobe is located posterior to the central sulcus and superior to the lateral fissure (Fig. 3.10). The lobe is clearly demarcated from the occipital lobe by the parieto-occipital sulcus near the medial aspect of the cerebral hemisphere. However, on the posterolateral aspect of the cerebral hemisphere, the lobe has less distinct boundaries with the occipital lobe and the temporal lobe. The primary somatosensory cortex, the secondary somatosensory cortex, and the superior and inferior parietal lobules are the main regions of the parietal lobe. The primary somatosensory cortex of the postcentral gyrus is located between the central and postcentral sulci. Like the primary motor cortex, the primary somatosensory cortex (Brodmann areas 3, 1, and 2) also has its topographical mapping of the contralateral half of the body, with the face, tongue, and lips represented inferiorly, the trunk and upper limbs represented superolaterally, and the lower limbs medially. This area has complex internal connectivity and apparently has a stepwise hierarchical progression of information processing occurring from area 3 through areas 1 and 2. The primary sensory area has ipsilateral association connections with the motor cortices of the frontal lobe and superior parietal lobe. The area has reciprocal corticocortical

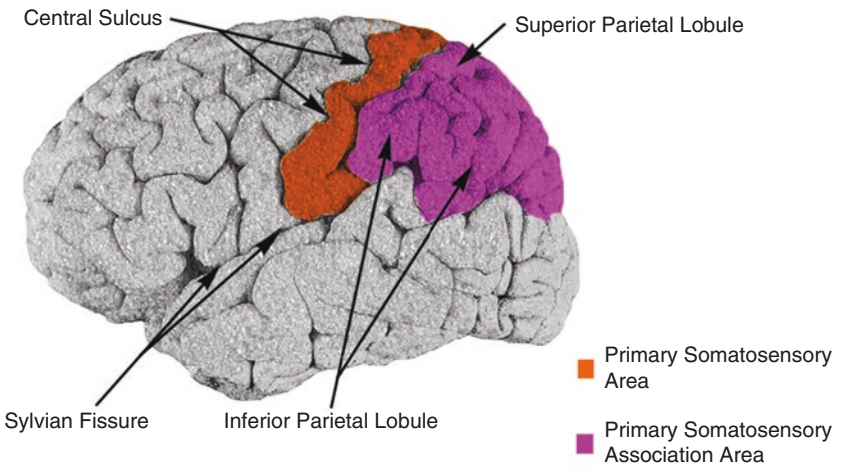

Fig. 3.10 An illustration of the parietal lobe

connections with the secondary somatosensory area, commissural connections with its contralateral homologue, and subcortical connections with the thalamus and basal ganglia. It also has projections to the brainstem via the corticopontine and corticotectal fibers.

The secondary somatosensory area located posterior to the central sulcus, along the upper bank of the lateral fissure, has reciprocal connections with the thalamus. The area has neuronal projections to the dorsal horn of the cervical and thoracic spinal cord, the posterior cingulate gyrus, the primary motor cortex, the superior parietal lobe, the principal trigeminal nucleus, and the periaqueductal gray matter of the midbrain. Within the parietal cortex, the secondary sensory area has reciprocal connections with the primary sensory area and commissural connections with the secondary sensory area of the contralateral hemisphere. Unilateral lesions of the secondary somatosensory area may degrade tactile discrimination abilities, impair some elements of sensory discrimination, and elevate pain thresholds without elimination of pain sensibility.

Posterior to the postcentral sulcus, a large area of the parietal lobe is divided by the intraparietal sulcus into the superior and inferior parietal lobules. The somatosensory association cortex (Brodmann areas 5 and 7) is located posterior to the primary sensory cortex in the parietal lobe. Similar to the primary somatosensory area, the superior parietal lobule has complex and extensive corticocortical, commissural, association fibers, and thalamic connections. The area also contributes to the corticospinal tract. Superior parietal lobule injury can lead to astereognosis (inability to recognize familiar solid objects by touch), a variety of body scheme or body image dysfunctions, hemineglect (neglect of the contralateral side of the world, usually seen in nondominant hemisphere lesion), asomatognosia (inability to recognize the affected limb as one's own), and a variety of syndromes, including dressing apraxia. The inferior parietal lobe (Brodmann areas 39 and 40) of the dominant hemisphere includes parts of Wernicke's speech area, and damage of the dominant hemisphere inferior parietal lobule can present with a tetrad of symptoms known as Gerstmann's syndrome. In adults, this syndrome can arise as a result of impaired blood flow to the inferior parietal lobe of the dominant hemisphere and is characterized by the inability to perform mathematical calculations (acalculia or dyscalculia), inability to write (agraphia or dysgraphia), inability to identify one's own finger (finger agnosia), and inability to make the distinction between the right and the left side of the body. In addition to the above, the patients may have difficulty expressing themselves through speech and difficulty in reading and spelling.

3.3.3 Temporal Lobe

The temporal lobe, the auditory language and speech comprehension area, is located inferior to the lateral fissure. The medial aspect of the temporal lobe consisting of the hippocampus and entorhinal cortex (an area in the medial temporal lobe essential for memory, navigation, and perception of time) and the areas of the neocortex adjacent to these limbic regions are grouped as medial temporal association cortex.

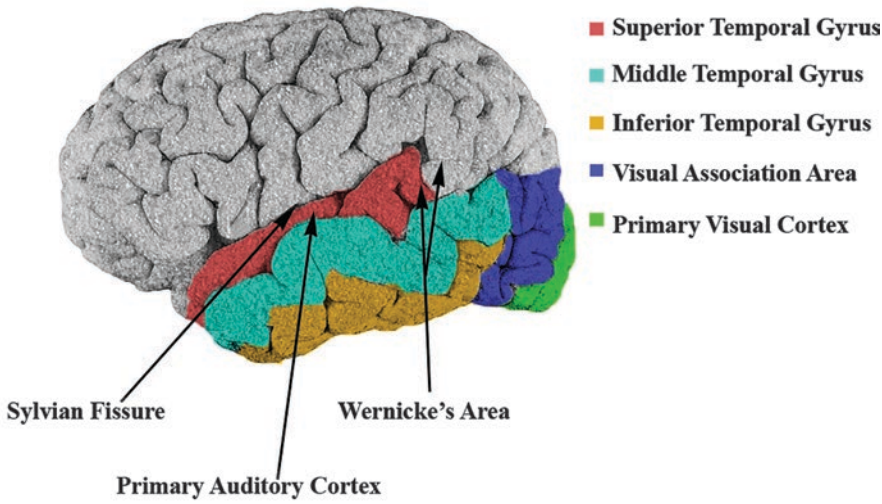

Fig. 3.11 An illustration of the temporal and occipital lobes

Their connections and functions are beyond the scope of this chapter. The superior and inferior temporal sulci located on the lateral surface of the temporal lobe divide the area into the superior, middle, and inferior temporal gyri (Fig. 3.11). The primary auditory cortex (Brodmann area 41 and 42) is located in the superior temporal gyrus, and the posterior area constitutes the Wernicke's area (auditory association area, Brodmann area 22). The auditory association cortex (Brodmann areas 22 and 42), located inferior and posterior to the primary auditory cortex (along the superior bank of the middle temporal gyrus), is the area concerned with memory and classification of sounds. The primary auditory cortex is reciprocally connected with the medial geniculate body and receives additional thalamocortical projections. Injury of the primary auditory cortex produces cortical deafness, verbal and non-verbal auditory agnosias. Both the primary and association areas, located in the auditory cortex of the superior temporal gyrus, have interconnections with the prefrontal cortex and the frontal eye field. It has contralateral connections with the adjacent and same regions of the other hemisphere. The connections of other sensory association pathways converge toward the superior temporal sulcus. Damage to the auditory association area in the superior temporal gyrus of the dominant hemisphere causes receptive fluent aphasia.

The middle temporal gyrus (Brodmann area 21) also is polysensory and has its connections with the auditory, somatosensory, and visual cortical association pathways. It has complex connections with the auditory association area of the superior temporal gyrus and has connections with the prefrontal cortex, the eye field, and the visual association area. In addition to the above, the middle temporal gyrus also has thalamic connections. The inferior temporal gyrus (Brodmann area 20), the higher visual association area, receives major ipsilateral corticocortical fibers from the visual association areas and has neuronal projections to the prefrontal cortex, the limbic areas, and the frontal eye field and reciprocal connections with the thalamus. Commissural

connections are between the corresponding areas and the adjacent visual association areas of the contralateral hemisphere. Due to its complexity, damage to the temporal lobe can lead to considerable disturbance of intellectual function, including visuospatial difficulties, prosopagnosia (also called face blindness, an inability to recognize familiar faces), and sensory aphasia, particularly when the dominant hemisphere is involved. Vascular lesions of frontal and/or temporal lobes can cause an acquired communication disorder named aphasia, characterized by impaired language comprehension, oral expression, and use of gestures or symbols to communicate ideas. Table 3.1 explains various types of aphasia and their associated features.

3.3.4 Occipital Lobe

The occipital lobe, located in the rearmost portion of the cerebral hemisphere, being the smallest of the four lobes, lies posterior to the parietal and temporal lobes (Fig. 3.11) and rests on the tentorium cerebelli. The lobe is primarily responsible for processing visual information and is comprised of Brodmann areas 17, 18, and 19. The primary visual cortex (Brodmann area 17) is located mostly on the medial aspect of the lobe and occupies the upper and lower lips and depths of the posterior part of the calcarine sulcus. The visual cortex of each cerebral hemisphere receives impulses from the two retinal halves (temporal half of one side and nasal half of the other side) that represent the contralateral half of the binocular visual field. The inferior half of the visual field represented by the superior retinal quadrants project to the visual cortex above the calcarine sulcus and the upper half of the visual field represented by the inferior retinal quadrants project to the visual cortex below the calcarine sulcus.

The main purpose of the primary visual cortex is to receive, segment, and integrate visual information. The processed information from the cortex is sent to the other regions of the brain to be analyzed and utilized to recognize objects and patterns quickly without a significant conscious effort. The corticocortical fibers from the primary visual cortex project to functional areas (Brodmann areas 18 and 19), the adjacent regions of the parietal and temporal cortices, the middle temporal area, and the medial superior temporal region. The efferent projections connect to the superior colliculus, pretectum, thalamus, and reticular formation of the pons. The commissural fibers reciprocally connect the primary visual cortex to the contralateral side and the geniculo-cortical projections are reciprocal. Injury to the primary visual cortex, being the initial processing center of all visual information, causes loss of vision in the contralesional visual field including homonymous hemianopia and upper (superior) or lower (inferior) quadrantanopia. Figure 3.12 depicts the visual field deficits due to lesions of the visual pathway.

The secondary visual cortex (Brodmann area 18) and the visual association area (Brodmann area 19), located near the primary visual cortex, project to several visual areas in the temporal and parietal association cortices and the frontal eye fields. The thalamocortical connections to these visual cortices arise from the lateral geniculate body. The commissural fibers through the corpus callosum connect both the visual

Table 3.1 Details regarding the types and features of aphasia

Features	Global	Broca's	Wernicke's	Transcortical motor	Transcortical sensory	Mixed transcortical	Anomic	Conduction
Comprehension	No	Yes	No	Yes	No	No	Yes	Yes
Fluency	No	No	Yes	No	Yes	No	Yes	Yes
Repetition	No	No	No	Yes	Yes	Yes	Yes	No
Spontaneous speech	Scant, reduced to few words, mutism possible	Short, slow, effortful, agrammatical, can pass ideas, words often mispronounced	Meaningless but fluent	Non-fluent	Meaningless but fluent	Telegraphic in nature with short, missing function words, brief noun-verb combinations	Grammar, fluency, and phrase length normal	Fluent, expression better than repetition
Accompanying neurological features	Right-side hemiplegia, hemisensory loss, and homonymous hemianopia	Hemiparesis with more involved of the right arm and face; dysarthria	Alexia, apraxia, Hemianopia or quadrantanopia	Leg weakness	Features are variable	Right side weakness or sensory loss	Features are variable	Features are variable
Lesion site	Large perisylvian infarcts involving receptive and expressive speech areas, primary sensorimotor cortices and primary visual and auditory cortices	Broca's area and regions inferior and posterior to Broca's	Wernicke's area, superior temporal gyrus	Area surrounding Broca's: Anterior association cortices	Area surrounding Wernicke's: posterior association cortices	Isolated damage to anterior and posterior eloquent cortex	Variable, possible lesions in the dominant hemisphere	Arcuate fasciculus that connects the Broca's and the Wernicke's areas
Prognosis (for vascular origin)	Poor	Generally good and typically resolves into anomic aphasia	Generally worse than Broca's but may resolve to transcortical sensory aphasia	Generally good and typically resolves into anomic aphasia	May evolve to anomic aphasia	Generally poor but variable	Good, often resolves to word-finding difficulty	May evolve to anomic aphasia

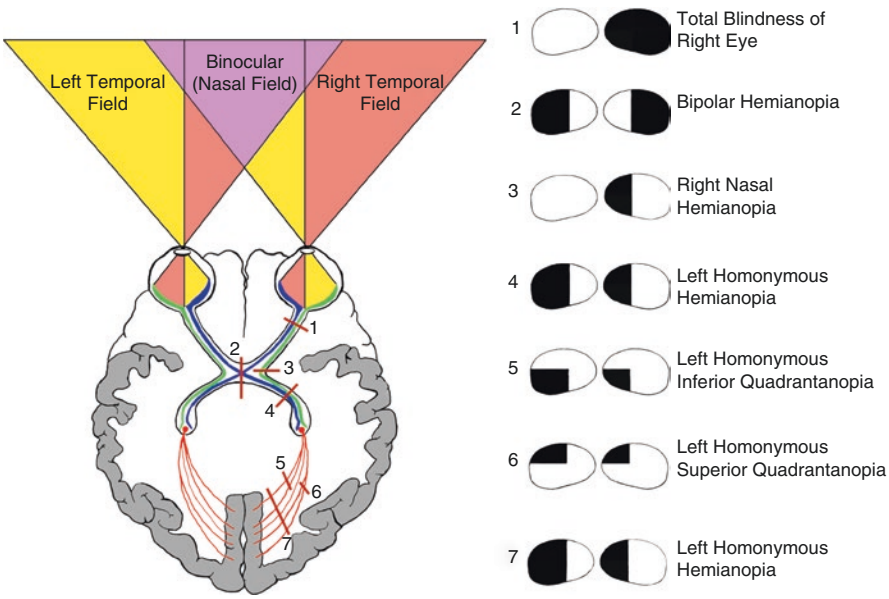

Fig. 3.12 An illustration of visual field deficits due to lesions of the visual pathway

cortices. Once the primary visual area discerns edges, shapes, and movements of the objects, the secondary visual cortex and the visual association areas synthesize these into a recognizable structure or pattern. These areas are also essential for visual depth perception. Damage to these areas disrupts motion perception and causes optic ataxia and may interfere with the learning of visuospatial tasks. In addition to the above, the patients with visual association cortex involvement may develop visual hallucinations (characterized by stereotyped images which seldom resemble fragments of memory), distortion of visual images (metamorphopsia), and failure to recognize the nature of objects seen (visual agnosia).

3.3.5 Internal Capsule

The internal capsule, a broad white matter band, composed of bundles of myelinated fibers located in the inferomedial portion of each cerebral hemisphere, separates the head of the caudate nucleus and thalamus from the lentiform nucleus (Fig. 3.13). The capsule consists of ascending and descending fibers that connect the cerebral hemispheres with the subcortical structures, the brainstem, and the spinal cord, i.e., it is a two-way track for information transmission to and from the cerebral cortex. Fibers projecting cephalically toward the cerebral cortex in a radiant pattern from the internal capsule, known as the corona radiate, are located above the superior border of the lentiform nucleus. The internal capsule has an anterior limb, genu, posterior limb, and retrolenticular and sublenticular segments (Fig. 3.14).

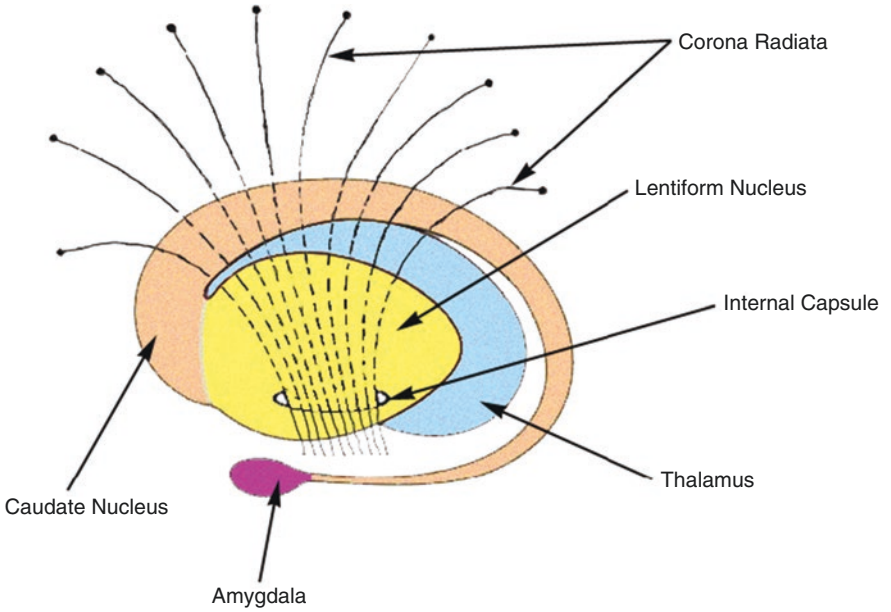

Fig. 3.13 The internal capsule separating the head of the caudate nucleus and thalamus from the lentiform nucleus

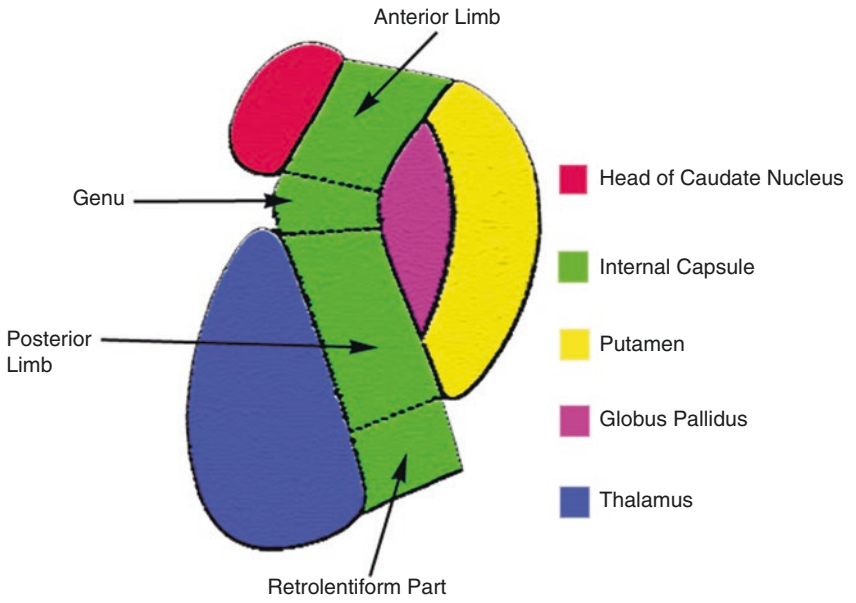

Fig. 3.14 The anterior limb, genu, posterior limb, and retrolenticular part of the internal capsule

Each of the regions mentioned above carries distinct ascending and descending tracts with their discrete functions.

The anterior limb of the internal capsule is bounded by the head of the caudate nucleus medially and the lentiform nucleus laterally. The anterior limb of the internal capsule contains frontopontine fibers arising from the frontal lobe. The fibers synapse with cells in the pontine nuclei, and through the middle cerebellar peduncle, the axons of these cells project to the opposite cerebellar hemisphere. The anterior limb also contains thalamic radiations which interconnect the thalamic and hypothalamic nuclei and limbic structures with the frontal cortex. The anterior limb fibers are known to be associated with emotional, cognitive, decision-making, and motivational processes. The genu of the internal capsule contains corticobulbar fibers derived mainly from the primary motor cortex and some from the premotor and supplementary motor areas to terminate at the appropriate cranial nerve nuclei located on the contralateral side of the brainstem. These fibers are concerned with the voluntary movements of facial muscles, mastication, and deglutition.

The posterior limb of the internal capsule is surrounded by the thalamus medially and the lentiform nucleus laterally. The posterior limb of the internal capsule contains the corticospinal tract. The corticospinal fibers representing the upper limb are located anteriorly. The fibers concerned with voluntary movements of the trunk and lower limbs are more posteriorly located. The descending axons of frontopontine fibers, specifically from the primary and supplementary motor areas of the frontal lobe and the corticorubral fibers that connect the frontal lobe to the red nucleus, are located within the posterior limb of the internal capsule. The somatosensory relays from the thalamus to the primary somatosensory cortex are located in the posterior one-third of the posterior limb. The retrolenticular segment of the internal capsule contains occipitopontine, occipitotectal, and parietopontine fibers. In addition to the above, this segment also contains thalamic radiation and optic radiation arising from the lateral geniculate body and interconnections between the occipital and parietal lobes and the thalamus. The sublenticular segment predominantly contains temporopontine fibers and auditory radiation from the medial geniculate body to the primary auditory cortex.

The vascular supply for the internal capsule arises from the perforating branches of the main cerebral arteries, including the middle cerebral artery, the anterior choroidal artery, and the internal carotid artery. The superior regions of the anterior limb, genu, and posterior limb get their vascular supply from perforating branches of the middle cerebral artery. Whereas, the inferior regions of the anterior limb receive supply from the recurrent artery of Heubner, the largest of the perforating medial lenticulostriate arteries arising from the anterior cerebral artery, and the inferior regions of the genu receive from the perforating arteries of the internal carotid and anterior choroidal arteries. In addition to the above, the perforating arteries of the anterior choroidal artery also supply the inferior regions of the posterior limb, the retrolenticular and sublenticular segments of the internal capsule. Occlusion of any of the perforating arteries supplied by the major vessels can predispose to cerebrovascular accidents known as lacunar strokes. Absence of cortical deficits

including aphasia, agnosia, dysgraphia, apraxia, alexia, and amnesia are typical for such deep strokes.

The neurological deficits due to infarctions of the internal capsule correlate with the fiber located within each limb. The anterior limb infarction presents with confusion, impaired attention, agitation, and dysarthria. The genu infarction presents with face and tongue weakness and dysarthria. The posterior limb infarction can cause pure motor hemiparesis contralateral to the lesion site, and the infarction of the posterior one-third of the posterior limb can cause contralateral hemisensory deficits with ataxic hemiparesis. Since the retrolenticular segment largely contains optic radiation fibers, infarctions of this region can lead to visual field deficits like homonymous hemianopia and superior or inferior quadrantanopia. The involvement of auditory radiations creates auditory deficits in lesions of the sublenticular segment.

3.4 Epidemiology

The World Health Organization (WHO) defines stroke as "rapidly developing clinical signs of focal or global disturbance of cerebral function, with symptoms lasting 24 hours or longer or leading to death, with no apparent cause other than of vascular origin." Stroke is one of the leading causes of long-term disability in adults worldwide and is the second leading cause of death after ischemic heart disease. Earlier studies estimated that stroke accounted for 9.6% of all deaths, and 40% of the patients were aged less than 70 years. Existing epidemiological data suggest that one out of four people are at risk of stroke in their lifetime. Beyond 65 years of age, the incidence of stroke increases dramatically with age. Around 30% of strokes occur in people below 65 years of age. Until the age of 85 years, women have a lower predisposition for stroke as compared to men. Approximately 70% of all strokes occur in low- and middle-income countries. The incidence of stroke in high-income countries has declined by around 40%. However, it has doubled in low- and middle-income countries. Demographic data of the developed countries reveals that the average age at which stroke occurs is around 73 years, and the probability of a first stroke is approximately 2 per 1000 population per annum.

Stroke patients are at the highest risk of death during the first week, and the risk of death can be 20% to 50% during the first month depending on the type, location, and size of the lesion, age, comorbidities, level of consciousness, complications, and effectiveness of treatment. Stroke survivors may be left with apparently no disability or with a mild, moderate, or severe disability. Survivors have a 10% risk of a subsequent stroke during the first year, and thereafter the risk reduces to 5% every year. In many of the survivors, a considerable extent of spontaneous recovery is anticipated during the first 6 months, and around 60 to 80% may achieve independence in self-care within a year after stroke. Some of the common disabilities noted among elderly stroke survivors after 6 months include hemiparesis, inability to walk unassisted, dependency in activities of daily living (ADL), aphasia, and depression.

The type of stroke is important in determining survival. Hemorrhagic stroke accounts for around 35% and ischemic stroke accounts for around 10% of deaths in one month. Analysis of the worldwide data indicates that Caucasians have approximately 80% ischemic strokes, 10 to 15% hemorrhagic strokes, and the remaining due to other causes of strokes. The hospital-based studies in the eastern Mediterranean region indicated that the stroke pattern is similar to that of western countries. Studies conducted in Asian and African countries indicate a higher proportion of hemorrhagic stroke (20 to 30%) among them as compared to Caucasians.

Regardless of the type of stroke, it is an accepted fact that stroke is caused by many different disease processes and not a single homogenous disease. Epidemiological studies have identified many risk factors for stroke including medical factors like prior history of stroke, ischemic heart disease, atrial fibrillation, and glucose intolerance; inherent biological traits like age and sex; physiological traits like serum lipoprotein and fibrinogen; behavioral traits like smoking, alcohol consumption, diet, and physical inactivity and social characteristics such as education, social class, and ethnicity; and environmental or geographic factors like altitude and temperature. Table 3.2 depicts the association of common conditions predisposing to stroke among adolescence to elderly population. Certain medical, behavioral, and environmental factors are classified as modifiable risk factors, whereas age, sex, and genetics are non-modifiable factors. Epidemiological studies have revealed that raised blood pressure is the single most important risk factor for ischemic stroke. In addition to the above, the risk of hemorrhagic stroke is directly proportional to the blood pressure and level of low-density lipoprotein. Regular use of anti-hypertensive medications has shown around a 40% reduction in the risk of stroke. Tobacco use, a

Table 3.2 Listing the common conditions causing stroke among adolescence to elderly population

Age category	Common conditions causing stroke
Adolescence and early adult life	Vascular malformations Pregnancy and puerperium Valvular heart disease Premature atherosclerosis Sickle cell anemia Migraine Arteritis Coagulopathies Arterial dissections Cerebral amyloid angiopathy
Middle age	Atherosclerotic thrombosis and embolism Cardiogenic embolism Hypertensive cerebral hemorrhage Rupture of saccular aneurysm Dissecting aneurysm
Late adult life	Atherosclerotic thrombotic occlusive disease Embolic occlusive disease Lacunar stroke Cerebral amyloid angiopathy

modifiable risk factor, increases the risk of both ischemic and hemorrhagic strokes by about twofold, indicating that heavy smokers are at a higher risk of stroke than light smokers. Epidemiological evidence reveals that many of the risk factors, including blood pressure and tobacco use, are somewhat similar in developed and developing countries.

3.5 Etiology

Among the vital organs, the brain depends continuously on an adequate volume of oxygenated blood. The total cessation of blood flow to the brain for more than 5 minutes can cause permanent brain damage. Under the control of brainstem centers, baroreceptors and vasomotor reflexes assure constancy in the cerebral circulation. The majority of strokes have a rapid or sudden evolving onset. Occlusion of an artery by thrombus or embolus deprives the brain of oxygenated blood, due to which the brain tissues may undergo ischemic necrosis or infarction. Occlusion or stenosis of the vessels to the brain can be the result of disease of the arterial wall, embolism from the heart, and hematological disorders. Leak or rupture of an artery can release blood into the brain, causing a hematoma within the brain substance and/or ventricles or subarachnoid spaces within the cranium. As the blood leaks or spills into the brain, the area the artery supplies will be deprived of oxygenated blood, causing a hemorrhagic stroke. The pressure created by the enlarging hematoma or the blood within the intracranial space and the biochemical substances released by the hemorrhage may adversely affect the adjacent vascular and brain tissues creating further localized or generalized injury to the brain tissue. Though the classification of stroke mechanism depends on the presence of risk factors and etiologies for stroke, the presence of more than one risk factor and etiology are frequently seen among stroke patients. Thrombosis, embolism, and decreased perfusion are considered the three commonly recognized mechanisms of ischemic stroke.

Thrombosis refers to an obstruction of blood flow due to a localized occlusive process formed by a thrombus or an atherosclerotic plaque (with or without a superimposed thrombotic occlusion). Atherosclerosis is the most common vascular pathology in which fibrous and muscular tissues overgrow in the endothelial lining layer of tunica intima of the arterial walls. The platelets which adhere to the atherosclerotic plaque can form clumps that serve as “nidi” for the deposition of fibrin, thrombin, and formation of the clot. The gradually growing atherosclerotic plaque can progressively occlude the lumen of the artery. Though atherosclerosis tends to affect the larger extracranial and intracranial arteries, the plaques are typically seen near the vessel-branch points where the turbulence of blood flow is maximum. The most common sites for atherosclerotic plaque formation are the carotid bifurcation, the origin of the middle or anterior cerebral arteries, vertebral artery origin, or vertebrobasilar junction (Fig. 3.15). Fibromuscular dysplasia, arteritis, dissection of the vessel wall, and hemorrhage into a plaque are the other less plausible causes for vascular pathological obstruction.

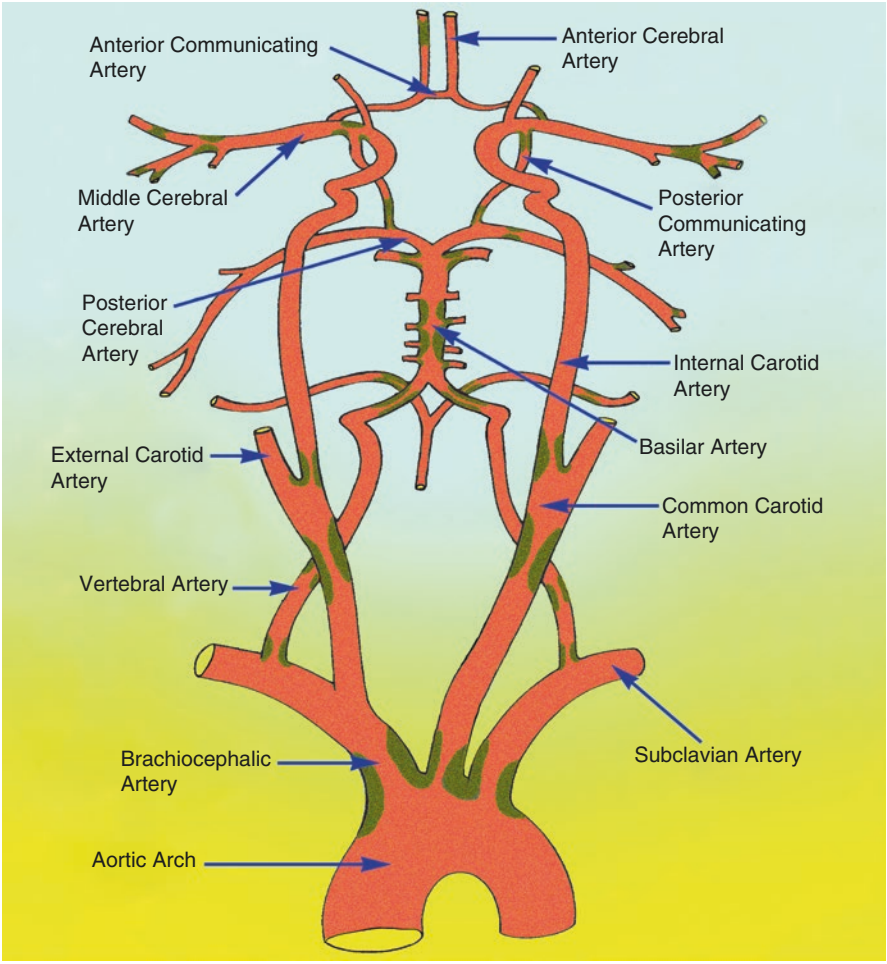

Fig. 3.15 Common sites of atherosclerotic plaque formation (grayish-green area) within the extracranial and intracranial arteries

In embolism, material or the embolus is formed elsewhere within the vascular system and dislodged to block the vasculature supplying the brain. The embolus typically originates from the heart, major arteries, and systemic veins. The frequently recognized cardiac sources for embolism include atrial fibrillation, sinoatrial disorder, acute myocardial infarction, bacterial endocarditis, and valvular disorders. For embolic strokes after acute myocardial infarction, the emboli are likely to arise from mural thrombi on the left ventricular wall, specifically in the anterior wall myocardial infarction. Large myocardial infarcts, left ventricular dilation, and congestive heart failure are risk factors for left ventricular mural thrombosis. The majority of the strokes tend to occur in the first weeks after the acute myocardial infarction. Atrial fibrillation can occur after acute myocardial infarction,

and fibrillation can be an independent risk factor for embolic stroke. The common source for the embolus is the left atrial appendage. In a patient with atrial fibrillation who has an acute stroke, recurrent stroke risk may be particularly high during the first week or two after the acute infarction. Cardioembolic stroke is a well-known complication of valvular disorders. The embolus can arise from the diseased native valve or the prosthetic valve. Rheumatic mitral stenosis with or without mitral regurgitation is commonly associated with thromboembolism. The risk of thromboembolism in rheumatic valve stenosis is related to age and low cardiac output and not to the severity of mitral stenosis, mitral calcification, or left atrial size. Atherosclerotic plaques of the aortic arch constitute another source of atherothrombotic embolism. Highly mobile thrombi in the lumen of the aortic arch and the presence of plaques of more than 4 mm size in the proximal arch are independent risk factors for atherothrombotic embolism.

Diminished blood flow to the brain tissue caused by systemic hypoperfusion either due to cardiac pump failure or systemic hypotension can critically affect the border zone or watershed regions at the periphery of the major vascular territories. In such cases, the effect of decreased perfusion can be more generalized, affecting the brain diffusely and bilaterally. Severe stenosis of the carotid and basilar artery and microstenosis of the small deep arteries are specific causes of stroke due to perfusion failure. More than 70% stenosis of the main artery by atherosclerotic plaque increases the risk of brain infarct in the distal zones. This arteriolar abnormality remains the most common defect in lacunar infarcts. Though the nature of the intrinsic arteriolar abnormality remains unclear, microatheroma, inadequate cerebral blood flow, or vasospasm may be the factors attributing or leading to lacunar infarcts.

The most common causes for hemorrhagic strokes are hypertension, arteriovenous malformation, aneurysm, bleeding disorders, use of cocaine, and anticoagulation therapy. For the prevention of recurrent thrombosis or thromboembolism, anticoagulation therapy is a crucial component of management of several medical conditions, including ischemic stroke, cardiac valvular disorders, and deep vein thrombosis. For those undergoing anticoagulant therapy, the International Normalized Ratio (INR) value of 4.9 and above increases the risk of hemorrhagic stroke.

3.6 Pathophysiology of Stroke

Brain tissue is easily vulnerable to the effects of ischemia due to its complete dependence on aerobic metabolism and low respiratory reserve. Tissue damage tends to occur more rapidly during hyperthermia compared to hypothermia. The spectrum of severity of tissue damage in the affected region depends mainly on the presence of patent collateral circulation, which in turn is determined by the normal variations in the circle of Willis, caliber of various collateral vessels, atherosclerosis, and other acquired arterial lesions. Ischemia of the brain tissue activates an ischemic cascade progressing from local depletion of oxygen or glucose to failure of production of

adenosine triphosphate (ATP), in turn adversely affecting the energy-dependent processes necessary for brain tissue survival and setting off a series of interrelated events ending in tissue injury and death. The extent of tissue injury generally depends on the severity, duration, and location of ischemia.

Thrombotic or embolic occlusion to the brain tissue creates an inner core of severe ischemia with blood flow below 10–25% and an outer layer of less severe ischemia known as the penumbra supplied by collaterals. The inner core area (zone of infarction) will display necrosis of both neuronal as well as supporting glial elements, whereas the neuronal cells in the penumbra may recover if reperfused in time. The extent of angiogenesis within the penumbral zone is mediated by the vascular endothelial growth factor and angiogenic growth factor secreted by the inflammatory infiltrates like leucocytes, blood platelets, and macrophages. Typically, the zone between the inner core area and the penumbral zone is critically hypoperfused, and appropriate treatment given within a few hours may salvage the same.

The various mechanisms involved in tissue damage are the following:

(i) Failure of mitochondria to replenish the depleting cellular energy store may trigger cell death.
(ii) Loss of ion pump functioning of the membrane causing loss of potassium in exchange of sodium, chloride, and calcium ions and the deleterious effects accompanied by the inflow of water and rapid swelling of neurons and glia (cytotoxic edema).
(iii) Release of excitatory neurotransmitters, mainly glutamate and aspartate. Uncontrolled release of glutamate into the ischemic areas mediates excitotoxic synaptic transmission via activation of N-methyl-D-aspartate (NMDA), which allows calcium and sodium influx causing disordered activation of a wide range of enzyme systems including proteases and lipases. These enzymes and their metabolic products including free radicals, damage cell membranes, genetic material, and structural proteins in the neurons, eventually leading to cell death.
(iv) Oxygen-free radicals and other reactive oxygen species produced in the ischemic zone will damage and destroy the cellular and extracellular elements, including the vascular endothelium.
(v) Apoptosis or programmed cell death may occur in the peripheral zone (penumbral zone).

In contrast to necrosis occurring in the ischemic core, apoptosis-inducing factors from mitochondria and proapoptotic molecules may trigger apoptosis in the peripheral zone. The ischemic cascade also activates various neuroprotective mechanisms against necrotic and apoptotic cell death such as the release of antiapoptotic and proapoptotic counter molecules, prion protein, and neurotrophin-3.

Cytotoxic or cellular edema tends to advance with time (minutes to hours) and can be potentially reversible. Failure of ATP-dependent calcium and sodium ion transport and release of oxygen-derived free radicals cause cellular edema of the brain cells, including neurons, glial cells, and endothelial cells. With the progression of infarction, cytotoxic edema caused by the initial acute hypoxia can give way to vasogenic edema. Vasogenic edema tends to occur over a few hours and days and

is generally irreversible. The extravasation of macromolecular serum proteins caused by increased permeability of brain capillary endothelial cells results in increased extracellular fluid volume along with increased intracranial pressure. The raised intracranial pressure may displace the brain hemisphere, shift the compartment of the brain, and compress the tracts, cranial nerves, and cerebral arteries. A sustained increase in pressure may cause irreversible damage to the brain and may lead to cerebral herniation and potential death.

Compared to ischemic strokes, hemorrhagic strokes are more dangerous. Intracerebral hemorrhage and subarachnoid hemorrhage are the two types of hemorrhagic stroke. The former generally occurs in small arteries or arterioles and is frequently due to hypertension, bleeding disorders, trauma, amyloid angiopathy, use of illicit drugs like amphetamines or cocaine, and arteriovenous vascular malformations. Typically, subarachnoid hemorrhage occurs due to rupture of an aneurysm near the base of the brain or a bleed from the vascular malformations near the pial surface.

3.7 Clinical Features of Supratentorial Stroke

Cerebrovascular accidents present clinically as neurologic deficits of sudden onset. The clinical features depend upon the area or the region of the brain affected, which in turn is defined by the arterial anatomy involved. Anatomically, the word supratentorial means the region of the brain located above the tentorium cerebelli, and almost 90% of strokes are supratentorial. The supratentorial area contains the cerebral hemispheres, subcortical structures including diencephalon and basal ganglia, optic nerve, limbic structures, and the lateral and third ventricles. The common symptoms of the left hemisphere stroke include right hemiparesis, aphasia, and right hemianopia. On the other hand, the symptoms of right hemisphere stroke include left hemiparesis, left hemispatial neglect, and left hemianopia. Infratentorial stroke due to posterior circulation impairment causes additional symptoms, including altered or reduced levels of consciousness, diplopia, bulbar palsies, dysmetria, and incoordination.

Though additional symptoms can be present in both supra- and infratentorial strokes, the most important historical feature is the “suddenness” of its onset. Sudden weakness or numbness in the face, arm, or leg (especially on one side of the body), confusion or trouble speaking or understanding speech, vision problems in one or both eyes, difficulty to walk, dizziness, loss of balance, or problems with coordination and severe headache with no known cause are the common five sudden warning signs of a stroke. Knowledge about the warning signs of a stroke can make a difference between recovery and disability. The BE-FAST algorithm (balance, eyes, face, arm, speech, and time) helps the public for the identification of the warning signs of persons having an acute stroke. Early recognition and timely medical or surgical management may save lives and certainly reduce the morbidity associated with the stroke.

Two terminologies that require special attention under this section are the transient ischemic attack (TIA) and the stroke in evolution or evolving stroke. The signs and symptoms of TIA usually resemble those of acute ischemic stroke. The presenting symptoms of TIA depend on the arterial territory involved and usually last for a few minutes to a maximum of 24 hours. Ischemic stroke and TIA share the same causative factors, including blockage caused by atherosclerotic plaques in the arteries or embolus from the heart. The TIA is often called a "mini-stroke," and several studies in diverse populations have shown that TIAs carry a very substantial risk of imminent brain infarction. Since TIA is a warning sign of impending stroke, seeking immediate medical attention following it can essentially prevent the imminent stroke.

Stroke in evolution is characterized by the deterioration of the neurologic deficits hours to few days after the initial clinical presentation of ischemic stroke. This concept of stroke in evolution or evolving stroke initially appeared in the 1950s. Several mechanisms might be potentially contributing to this phenomenon, including thrombus propagation, development of brain edema and its associated mass effect, metabolic disturbances such as hyperglycemia or hyponatremia, hemorrhagic transformation of the ischemic lesion, and pyrexia. Progressive damage of the ischemic penumbral zone due to the abovementioned factors could be the reason for the deterioration in evolving stroke.

The following section will cover the clinical features of common stroke syndromes occurring supratentorially, and the latter part of this chapter will cover the clinical presentation and management of infratentorial stroke (specifically, brainstem strokes).

The anterior cerebral artery stroke occurs due to the occlusion of the anterior cerebral artery, the smaller and the first terminal branch of the internal carotid artery. The artery supplies the medial aspect of the cerebral hemisphere, subcortical structures including the basal ganglia and anterior limb of the internal capsule, the anterior fornix, and the ventral four-fifths of the corpus callosum. The occlusions that occur in the first or A1 segment of the anterior cerebral artery (part of the artery between the internal carotid artery and the anterior communicating artery branch) are less likely to produce any symptoms because of patent collateral circulation, and occlusions in the distal segments of the artery are more likely to produce significant deficits. Table 3.3 lists the segments of the anterior cerebral artery. Table 3.4 gives the clinical features of the anterior cerebral artery syndrome and the structures involved.

The middle cerebral artery stroke occurs due to occlusion of the middle cerebral artery, the largest and the second terminal branch of the internal carotid artery. The artery supplies the whole of the lateral surface of the cerebral hemisphere and subcortical structures, including the basal ganglia, corona radiata, and posterior limb of the internal capsule. Table 3.5 lists the segments of the middle cerebral artery. Occlusion of the proximal segments of the middle cerebral artery can produce extensive brain tissue damage and significant cerebral edema. A large lesion with considerable cerebral edema can produce brain herniation and possible death. Table 3.6 depicts the clinical features of middle cerebral artery syndrome and the structures involved.

Table 3.3 The segments of the anterior cerebral artery

Name of the segment	Origin and termination
A1: Horizontal or Pre-communicating segment	• Originates from the terminal bifurcation of the internal carotid artery and extends approximately 15 millimeters in length • Terminates at the anterior communicating artery
A2: Vertical, post-communicating, or infracallosal segment	• Originates at the anterior communicating artery, extending anterior to the lamina terminalis; along the rostrum of the corpus callosum • Terminates either at the genu of the corpus callosum or the origin of the callosomarginal artery
A3: Precallosal segment	• Originates around the genu of the corpus callosum or the origin of the callosomarginal artery • Terminates where the artery turns directly posterior above the corpus callosum
A4: Supracallosal segment	• Originates above the body of the corpus callosum • Terminates anterior to the plane of the coronal suture
A5: Postcallosal segment	• Originates above the body of the corpus callosum posterior to the plane of the coronal suture

Table 3.4 The clinical features and the structures involved in the anterior cerebral artery syndrome

Clinical features	Location/structure involved
Contralateral hemiparesis; lower extremity more involved than upper extremity	Predominant involvement of motor area of lower extremity (paracentral lobule)
Paresis or lesser involvement of opposite arm	Partial involvement of motor area of the arm or corona radiata through which those fibers are descending
Contralateral hemisensory loss mainly involving lower extremity	Primary sensory area for lower extremity located on the medial aspect of cortex (paracentral lobule)
Frontal "gait apraxia" (broad-based gait, short stride, "magnetic" or shuffling type, freezing, falls or tendency to fall, en bloc turns, and an inability to walk and talk)	Probable location: inferomedial frontal-striatal projections
Urinary incontinence	Superior frontal gyrus: posteromedial region
Contralateral grasp reflex, sucking reflex, palmomental reflex, paratonic rigidity (subjects involuntarily resist passive movements)	Medial surface of the posterior frontal lobe
Akinetic mutism (abulia), lack of spontaneity, motor inaction, slowness, delay, whispering, Reflex distraction to sights and sounds	Probable location: superomedial lesion near subcallosum
Dyspraxia of left limbs; difficulties with imitation and bimanual tasks; tactile aphasia in left limbs	Corpus callosum
Perseveration and amnesia	Uncertain localization
Cerebral paraplegia	Bilateral occlusion of anterior Cerebral arteries and involvement of bilaterally motor area of the lower extremities

Table 3.5 The segments of the middle cerebral artery

Name of the segment	Origin and termination
M1: Horizontal or sphenoidal segment	• Originates at the terminal bifurcation of the internal carotid artery • Terminates either at the genu adjacent to the limen insulae (anteroinferior aspect of the insular cortical surface) or at the main bifurcation
M2: Insular segment	• Originates at the genu near the limen insulae or the main bifurcation • Terminates at the circular sulcus of insula
M3: Opercular segment	• Originates at the circular sulcus of the insula • Terminates at the external or superior surface of the Sylvian fissure
M4: Cortical segment	• Originates at the external or superior surface of the Sylvian fissure

Table 3.6 The clinical features and the structures involved in the middle cerebral artery syndrome

Clinical features	Location/structure involved
Contralateral hemiparesis; upper limb and face more involved than lower limb	Prominent involvement of motor areas of face and upper limb and partial involvement of the fibers descending from the leg area entering the corona radiata
Contralateral hemisensory loss mainly involving upper limb and face	Somatosensory area for the face and arm and thalamoparietal projections within the internal capsule
Contralateral homonymous hemianopia or homonymous quadrantanopia	Optic radiation in the internal capsule
Contralateral paralysis of conjugate gaze	Frontal eye field or projecting fibers
Sensory ataxia of contralateral limb or limbs	Parietal lobe
Motor speech disorder: Broca's aphasia	Broca and adjacent motor area of the dominant hemisphere
Sensory or receptive speech disorder: Wernicke's aphasia	Wernicke's area located posterior third of the superior temporal gyrus of the dominant hemisphere
Global aphasia	Large perisylvian lesions involving receptive and expressive speech areas
Perceptual deficits: Unilateral neglect, depth perception, inaccurate localization in the half field, impaired ability to judge distance, agnosia, amorphosynthesis, dressing apraxia, constructional apraxia	Usually parietal lobe sensory association cortex of the nondominant hemisphere.
Word deafness, anomia, jargon speech, alexia	Central language area and parieto-occipital cortex of the dominant hemisphere
Gerstmann's syndrome	Inferior parietal lobe of the dominant hemisphere
Pure motor hemiplegia	Lacunar stroke in the upper portion of the posterior limb of the internal capsule and the adjacent corona radiata
Gait apraxia or Bruns ataxia	Frontal lobes (bilateral)
Limb-kinetic apraxia	Premotor or parietal cortical damage
Loss or impairment of optokinetic nystagmus	Supramarginal gyrus or inferior parietal lobe

Table 3.7 The segments of the posterior cerebral artery

Name of the segment	Origin and termination
P1: Pre-communicating segment	• Originates at the termination of the basilar artery • Terminates at the posterior communicating artery
P2: Post-communicating segment	• Originates from the posterior communicating artery around the midbrain • Terminates as it enters the quadrigeminal cistern
P3: Quadrigeminal segment	• Travels posteromedially through the quadrigeminal cistern • Terminates as it enters sulci of the occipital lobe
P4: Cortical segment	• Originates within the sulci of the occipital lobe

The internal carotid artery syndrome occurs due to occlusion of the internal carotid artery. It frequently produces a massive zone of infarction in the region of the brain parenchyma supplied by the middle and anterior cerebral arteries, especially when the collateral circulations are weak. Extensive cerebral infarction in these areas can cause significant cerebral edema and mass effect, including uncal herniation, coma, and death.

The posterior cerebral artery syndrome occurs due to occlusion of the posterior cerebral artery, the terminal branch of the basilar artery. The artery supplies the respective occipital lobe and medial and inferior temporal lobe, the rostral part of the brainstem, and the posterior diencephalon, including most of the thalamus. Table 3.7 lists the segments of the posterior cerebral artery. Blockages of the proximal segment of the posterior cerebral artery (from the bifurcation site to the origin of the posterior communicating artery) can produce minor neurological deficits owing to the collateral blood supply from the posterior communicating artery. Blockages of the posterior cerebral artery beyond the proximal segment can cause a variety of deficits depending on the cortical and central branches occluded. The clinical features of posterior cerebral artery syndrome and the structures involved are mentioned in Table 3.8.

3.8 Complications of Stroke

Medical and neurological complications are frequent among post-stroke patients. Complications tend to increase the length of hospitalization, hinder functional recovery, interfere with rehabilitative therapies, and increase the costs of care. In addition to the above, the complications are a major cause of death in the early phase post-stroke. Those patients with severe disabling strokes are more vulnerable to develop complications. Pyrexia, systemic inflammatory response, hypoxia, hyperglycemia, and certain medications used for the management of the complications may have a damaging physiological effect on an injured brain or may compromise its capability for plastic change. Most of the complications develop during the acute phase (first few weeks) of stroke. Cardiac abnormalities, dysphagia, and pneumonia are a few of the early complications, whereas others, such as decubitus ulcers,

Table 3.8 The clinical features and the structures involved in the posterior cerebral artery syndrome

Territory	Location/structure involved	Clinical features
Cortical	Contralateral homonymous hemianopia	Calcarine cortex or optic radiation
	Bilateral homonymous hemianopia, cortical blindness, denial of blindness, inability to perceive objects not centrally located, apraxia of ocular movements, inability to count objects	Bilateral occipital lobe, possibly with involvement of parieto-occipital region
	Dyslexia without agraphia, color anomia	Calcarine lesion on the dominant hemisphere and posterior part of corpus callosum
	Memory defect	Lesion of inferomedial portions of temporal lobe bilaterally or less frequently the dominant side
	Visual agnosia	Dominant occipital lobe
	Prosopagnosia or face blindness (inability to recognize familiar faces)	Nondominant hemisphere fusiform or lateral occipitotemporal gyrus; visual association area
	Simultagnosia (inability to perceive more than a single object at a time)	Dominant visual cortex
	Topographic disorientation	Nondominant calcarine and lingual gyri
	Unformed visual hallucinations, metamorphopsia, photophobia	Calcarine cortex
Central	Thalamic syndrome: spontaneous pain and dysesthesias and loss of all sensory modalities	Ventral posterolateral nucleus of thalamus
	Choreoathetosis, hemiballismus	Subthalamic nucleus or its pallidal connections
	Intention tremor, contralateral ataxic, postural tremor	Dentatothalamic tract
	Claude's syndrome: ipsilateral third cranial nerve palsy, contralateral hemiparesis, and ataxia	Dentatorubral fibers, corticospinal and bulbar fibers, and oculomotor nerve fibers in midbrain
	Weber's syndrome: contralateral hemiplegia with third cranial nerve palsy	Midbrain infarction with involvement of third nerve and cerebral peduncle
	Contralateral hemiplegia	Cerebral peduncle
	Vertical eye movement paralysis, skew deviation, sluggish pupillary reaction to light, miosis, and ptosis	Supranuclear fibers to third cranial nerve; high midbrain tegmentum ventral to superior colliculus

deep vein thrombosis, and falls, can occur after several days. Many of the complications are preventable and early recognition and treatment can effectively ameliorate the deleterious effects of the same. Only the major systemic medical and neurological complications are discussed here.

Cerebrovascular accidents and cardiovascular diseases share several risk factors. As discussed earlier in the chapter, cardiac diseases such as atrial fibrillation, valvular disorders, or congestive heart failure can increase the risk of stroke, and this can

predispose patients to develop cardiac complications. Autonomic dysregulation and physiological stress induced by cerebrovascular accidents can be plausible causes for the development of cardiac complications. Timely and accurate recognition of patients who are at high risk for cardiac complications might help to prevent such complications. Stroke patients with established cardiovascular diseases, severe stroke, diabetes mellitus, and peripheral vascular disease are at high risk of myocardial infarction during the early recovery phase of stroke than those stroke patients without any of these comorbidities. Cardiac arrhythmias, including atrial fibrillation, ventricular tachycardia, supraventricular tachycardia, and ventricular ectopic beats have been reported post-stroke. Hemodynamic instability due to cardiac arrhythmias and thromboembolism due to atrial fibrillation can predispose to second or multiple strokes.

Bronchopulmonary pneumonia is a frequent medical complication of stroke and the most common cause of pyrexia within the first few days after stroke. According to the current evidence, pneumonia has a threefold increased risk of death. Most of the stroke-related cases of pneumonia develop as a result of aspiration. Empirical use of antimicrobial treatment for gram-positive and negative bacilli can effectively manage aspiration pneumonia. Severe stroke, old age (>65 years), speech impairment, dysphagia, cognitive dysfunction, reduced or altered level of consciousness, severe facial palsy, mechanical ventilation, brainstem strokes, and multiple strokes are certain independent risk factors of pneumonia. The presence of these conditions can alert the clinician and anticipate the risk of pneumonia in stroke patients to decide about further surveillance, diagnostics, and feeding. Weak expiratory muscles and poor or ineffective cough can also predispose to pneumonia. Paying close attention to oral care and dental hygiene can minimize the possibility of the development of pneumonia. The use of oral antiseptics and frequent dental hygiene regimen for mechanically ventilated stroke patients may lower the ventilator-associated risk of pneumonia.

Desaturation of oxygen and apnea within the first few days post-stroke can aggravate brain injury by compromising oxygen delivery to the penumbral zone of the brain tissues. The reasons for stroke patients developing hypoxemia can include alteration in the central regulation of respiration, weakness of the respiratory muscles, and added complications such as pneumonia, aspiration, atelectasis, and pulmonary emboli, which can impair air exchange at the pulmonary segments level. Stroke patients with larger brain lesions, old age, preexisting cardiac and pulmonary disease, and swallowing impairments are more likely to develop oxygen desaturation. Maintaining the normal oxygen saturation is the logical goal to minimize the desaturation effects, and acute stroke patients should be monitored by pulse oximetry maintained at a target oxygen saturation level of $\geq$92%.

Pyrexia is a complication seen among post-stroke patients, particularly during the first 72 hours, and is more common among hemorrhagic strokes than ischemic strokes. Fever can indicate systemic stress, underlying infection, or presence of intraventricular blood in a hemorrhagic stroke. Central thermoregulatory impairment can also be a reason for the fever. The central hyperthermia presents with rapid onset of high fever and notable temperature variation. It is commonly seen in

brainstem strokes associated with high mortality. Fever can increase the metabolic demands of the injured brain tissues and exacerbate the neuronal injury. Prompt treatment of hyperthermia with antipyretic medications can effectively improve neurological outcomes in many stroke patients. Though inducing hypothermia can be an alternative, it can lead to cardiac arrhythmias, infections, hemorrhagic transformation of infarcts, and venous thrombosis.

Hyponatremia is a common electrolyte disorder encountered in acute stroke patients. It develops either due to the syndrome of inappropriate secretion of antidiuretic hormone or cerebral salt wasting syndrome. Antidiuretic hormone is a posterior pituitary gland hormone that primarily regulates body water by acting on the kidneys. Normally, a drop in plasma volume or an increase in serum osmolality causes the release of the antidiuretic hormone to increase total body water. In the former syndrome, failure of the negative feedback mechanism that usually controls the release of the antidiuretic hormone causes persistent production of the same, despite body fluid hypotonicity and expanded circulatory volume. Cerebral salt wasting syndrome, described by Peter J.P. and co-workers in 1950, is characterized by the development of excessive natriuresis (excretion of an excessively large amount of sodium in the urine) and subsequent hyponatremia, particularly in patients with subarachnoid hemorrhage. Though theories for the pathophysiology of cerebral salt wasting include the release of brain natriuretic peptide or damage to the hypothalamus with subsequent sympathetic system dysfunction, the exact mechanism is largely unknown. The treatment strategies for either syndrome are not the same. For cerebral salt wasting syndrome, the patient is given fluids and sodium supplementation, whereas for the syndrome of inappropriate secretion of antidiuretic hormone, the patient is fluid restricted. Typically, with appropriate management, the hyponatremia tends to resolve within days to weeks but can remain a chronic issue.

Approximately 40% to 75% of stroke patients develop dysphagia. Reduced oral intake due to dysphagia increases the risk of poor nutrition and dehydration. As discussed earlier, dysphagia is a major risk factor for pneumonia. Usually, the nutritional requirements of stroke patients with dysphagia are met either by insertion and placement of the nasogastric tube (Ryle's tube) or surgical placement of percutaneous endoscopic gastrostomy (PEG). Evidence states that during the first few weeks post-stroke, nasogastric tube feeding is preferable as it can enable some patients to recuperate their swallowing functions. Ryle's tube is easy to insert but can be uncomfortable and get easily dislodged. On the contrary, PEG is an invasive procedure, and peritonitis and bowel perforation are a few complications of the same. However, both of these managements don't offer any protection against aspiration pneumonia.

Gastrointestinal hemorrhage has been reported in some prospective studies as a less common complication after stroke. The risk of bleeding is higher in stroke patients of Asian descent. The severity of the stroke, prior history of peptic ulcer disease, cancer, renal failure, sepsis, and presence of abnormal liver function are independent predictors of gastrointestinal bleeding among acute stroke patients. Late gastric emptying, stress ulcers, and mucosal irritation secondary to gastric

feeding are certain mechanisms speculated to cause gastrointestinal hemorrhage. Judicious prophylactic use of antacids, H2 receptor antagonists, and sucralfate is effective in reducing the risks of bleeding.

Approximately 30% to 50% of acute stroke patients develop bowel incontinence. Bowel and bladder incontinence can coexist in many of the patients. Old age, large and disabling strokes, and reduced or altered level of consciousness are certain important predictors of bowel incontinence. Stroke patients having reduced functional mobility, loss of manual dexterity, visual impairment, cognitive dysfunction, and communication difficulties are likely to have persistent bowel incontinence even at 3 months post-stroke. Certain medications with anticholinergic properties such as antipsychotics or antiemetics can considerably increase the risk of fecal incontinence. Encouraging early mobilization, improving toilet access, dietary modification, avoiding dehydration, and minimizing polypharmacy, may facilitate bowel control after a stroke.

Urinary tract infection is another important and frequent complication seen among acute stroke patients. Older age, use of urinary catheters, severity of the stroke, and female gender are independent predictors of urinary tract infections after stroke. The majority of the patients may develop uncomplicated urinary tract infections. Escherichia coli frequently cause urinary catheter-related urinary tract infections. Meticulous catheter care and avoidance of unnecessary catheterization can help prevent urinary tract infections. For suspected infection, urine cultures can confirm the diagnosis and guide appropriate antibiotic treatment. Urinary incontinence and retention occur in 30% to 60% of stroke patients. Urodynamic studies have revealed high rates of detrusor hyperreflexia post-stroke, and high detrusor hyperreflexia suggests damage to corticospinal pathways. Large infarcts, altered sensorium, cognitive impairment, aphasia, and severe functional impairments are independently associated with bladder dysfunction. The incontinence can adversely affect the self-esteem and confidence of stroke patients, impose burden on family members or caregivers, delay hospital discharge, and lead to institutionalized care. Methodical assessment and management of urinary symptoms by specialized professionals may tackle the issues of incontinence during the acute phase of stroke.

Deep vein thrombosis (DVT) is a major concern during the post-stroke stage, especially when the lower limb is paralyzed. The prevalence is as high as 50% during the first two weeks after stroke, in the absence of DVT prophylaxis. In a prospective study, magnetic resonance imaging (MRI) for the pelvis and lower extremities detected venous thromboembolism in 40% of acute stroke patients and pulmonary emboli in 12%, despite aspirin and graded compression stockings usage. In general, DVT develops early and possibly within the first few weeks post-stroke. The severity of paralysis, advanced age, and dehydration are important risk factors for DVT development. The most typical symptoms of DVT include edema of the paretic or paralyzed limb, painful and tender area, and a certain amount of redness and localized rise in the skin temperature. In approximately 50% of cases, such clinical symptoms are undetectable and can be identified only by Doppler ultrasonography, contrast venography, or impedance plethysmography. In undiagnosed and/or untreated proximal DVT, the most feared complication is the fatal pulmonary

embolism, often occurring between the second and fourth weeks post-stroke, which accounts for 15% of early death. Low molecular weight heparin or low dose unfractionated heparin used prophylactically can be effective in preventing DVT and pulmonary embolism and the risk of death; however, it can increase the risk of intracranial bleeding, especially in hemorrhagic stroke or hemorrhagic transformation of infarcts. Placement of an inferior vena cava filter can be an option for preventing pulmonary embolism in such high-risk patients, though this approach can encourage further DVT formation. The use of graded compression stockings and intermittent pneumatic compression can be effective for preventing DVT, particularly when the risks of bleeding associated with anticoagulants in stroke patients are high. However, in some patients, especially with a history of peripheral vascular disease, the use of compression stocking poses serious concerns including skin ulcers and necrosis, as it might further reduce the blood flow to the lower extremities.

Survivors are generally anxious about the possibility of recurrence of stroke, as it is an important cause of morbidity and mortality. Patients with a prior history of stroke are four times more likely to have another stroke compared to matched controls. Stroke patients are most at risk of recurrence during the first 6 months. Patients below 65 years of age are at lower risk as compared to older age groups. The absolute risk of recurrence is approximately 30% for all types of strokes during the first 5 years, and the risk of recurrence following primary hemorrhagic stroke is somewhat similar to that of ischemic stroke. Stroke recurrence can have a devastating effect on morale and can prevent patients from regaining independence. The underlying mechanisms of recurrent stroke are not very clear, and the etiology can be multifactorial. Patients with large vessel atherothrombosis and cardiovascular conditions are at higher risk of recurrent stroke. Stroke survivors need to seek information and guidance from the neurologist or physician about the risks of a recurrent stroke and measures required to prevent them. Based on the causative and risk factors for recurrent stroke, the preventative measures may include the use of antithrombotic agents, antihypertensive medication, hypoglycemic agents, statins for reduction of elevated low-density lipoprotein and triglycerides, anticoagulation for atrial fibrillation and cardiac conditions, and cessation of smoking.

Stroke patients are at increased risk for fractures, mostly involving the hip joint. In general, hip fractures are associated with high morbidity and mortality, especially among the elderly. There is a sevenfold higher risk of fractures for stroke patients during the first year. Elderly stroke patients are also likely to have age-associated osteopenia. Reduced lower limb muscle strength, inadequate weight-bearing, and use of anticoagulants accelerate bone loss after stroke, especially in the paretic limbs. Osteopenic bones are brittle and are more susceptible to fractures from trivial trauma or fall, and such fractures are frequent on the paretic side. Associated arm weakness, loss of protective and balance reactions, neglect, seizures, cognitive and motor impairments, sedative medications, or drugs affecting sensorium are the independent factors associated with falls and fractures. The use of hip protectors as shock absorbers, assistive and orthotic devices for ambulation, gait training for safe ambulation, and promoting balance reactions may prevent fractures in stroke patients.

Pain is another frequent complication seen among stroke patients. Among stroke patients, pain mainly involves the paretic limbs, especially the affected side shoulder. It can interfere with physiotherapy, interrupt sleep, and contribute to fatigue and depression. Almost one-third have moderate to severe pain in the first few months post-stroke. However, in most cases, the pain improves spontaneously, although in some it may worsen with time. Preexisting painful disorders like arthritis and decreased mobility, changes in gait, advanced age, and postural changes can contribute to pain. Complex regional pain syndrome, previously known as reflex sympathetic dystrophy, is a chronic painful condition that may affect the paretic extremity in some stroke survivors. The patient may complain about the severe and constant pain in the affected area and often describes it as burning or pins and needles type and may experience changes in skin temperature, skin color, and swelling of the joints. The affected limb may feel warmer or cooler compared to the unaffected limb. Changes in skin texture (skin may appear shiny and thin), abnormal sweating patterns, changes in nail and hair growth patterns, and stiffness in affected joints are certain common features of complex regional pain syndrome. Early identification, physiotherapy, and medical management using chemical sympathetic blocks and oral or intramuscular corticosteroids may help to minimize the consequence of this debilitating condition.

Approximately 10% of stroke patients can develop central post-stroke pain as a direct consequence of a lesion affecting the central somatosensory system or its pathway. The thalamus is believed to play a key role in the underlying pathophysiology of central pain. The central pain can be focal, segmental, or affecting half the body and can present as severe and persistent burning or aching pain, intermittent and spontaneous lacerating or shooting pain, evoked by mechanical or thermal stimuli. Typically, the central pain tends to develop during the first few months post-stroke, and medications like amitriptyline and lamotrigine may show certain efficacy in ameliorating it. Regarding pain involving the shoulder joint, muscle paralysis, spasticity, joint inflammation, soft tissue contracture, and nerve injury can be the possible causes. The hemiplegic shoulder pain syndrome can be due to the weakness of the vertical stabilizers of the shoulder joint needed for glenohumeral stabilization. In such patients, biceps and supraspinatus are frequently tender, and the "Neer Impingement Sign" is positive. Use of analgesics, pain management using electrotherapeutic modalities, foam supports for shoulder or shoulder strapping, and daily passive range of motion (ROM) exercises can help prevent shoulder pain.

Stroke patients may report fatigue as a frequent complaint. Fatigue can cause functional limitations and can contribute to depression. The cause of fatigue among stroke patients is multifactorial. General physical deconditioning, associated medical ailments, side effects of medications, and certain central mechanisms are the possible factors attributing to fatigue. Management of fatigue among stroke patients needs to be individualized with assessment and treatment of concomitant disorders, including infection, hypothyroidism, anemia, depression, and adrenal insufficiency.

Emotional incontinence can be a feature of lesions of the brain affecting the frontal lobe, hypothalamus, and limbic system. Stroke patients with pseudobulbar

affect (pathological laughing and crying) are known to develop emotional lability or emotional incontinence. Such patients demonstrate emotional outbursts of uncontrolled or exaggerated laughing or crying that are inappropriate with the mood. They may quickly change from laughing to crying with minimal provocation. Often the patients may express that they are unable to control or inhibit such episodes. Frequent crying may also accompany depression. Antidepressants and coping and support may help to control symptoms of pseudobulbar affect.

Based on the available literature, about 33% of stroke patients have depression as another complaint. Young stroke patients, women, aphasia, and those with severe stroke and greater disabilities are at a higher risk of developing depression. Post-stroke depression can contribute to mortality, including suicide. Depressed patients are less likely to participate in rehabilitative therapies, less compliant with medications, and tend to have poorer recovery following stroke. The use of antidepressants, counseling, and cognitive behavioral therapy has been attempted for treating post-stroke depression with variable results.

Stroke patients are susceptible to develop bedsores, particularly when bedridden for a prolonged time. Immobility or reduced mobility, age, bowel incontinence, bladder incontinence, poor nutrition, poor cognition, inadequate perfusion, presence of chronic diseases, and reduced skin sensation increase the risk of skin breakdowns. In stroke patients, pressure ulcers are frequently developed over prominent bony areas such as sacral, ischial, trochanteric, malleolar, and heel. Early mobilization, regular two-hour change of posture within the bed, use of padded heel boots, and alternating pressure air mattresses can prevent or minimize their development.

Stroke is the most common cause of seizures among the elderly population. Post-stroke seizure and epilepsy (multiple episodes of seizures) are common causes of hospital admissions or readmissions. Approximately 10% of the post-stroke population is at risk of developing seizures within the first 5 years, and around 5% of patients will have a seizure within the first few weeks post-stroke. Hemorrhagic stroke patients are more likely to have seizures after a stroke than those with ischemic stroke. One-third of cases can present with generalized tonic-clonic seizures and the remaining can present with partial seizures. Early-onset seizures usually present with a focal onset, while generalized tonic-clonic seizures are more common with late-onset seizures. Around 10% of post-stroke epilepsy patients can develop status epilepticus. Though the prognosis of status epilepticus is poor, the use of antiepileptic medications, including phenytoin, carbamazepine, and phenobarbital, remains the mainstay of management of seizures post-stroke.

3.9 Clinical Diagnosis

If approached systematically, the clinical diagnosis can be easier and more logical. The neurologist or the physician must decide on certain key questions to diagnose and plan the medical treatment: (1) What is the mechanism (hemorrhage or ischemia, including their subtypes) of stroke? (2) Where is the anatomical location of

the lesion? While gathering the information from history and medical records, the clinician should also identify whether the signs and symptoms are due to any non-vascular processes such as a brain tumor, infection, trauma, intoxication, metabolic abnormality, or seizure disorder, which mimics a stroke. The information (data collected from the patient, family members or bystanders, and medical records) regarding the past and present personal and family illnesses, presence and nature of past strokes or TIA, activity at the onset of the stroke, temporal course and progression of the findings and presence of any accompanying symptoms like headache, altered level of consciousness, vomiting, and seizures can aid in identifying the mechanism of stroke.

Analysis of the neurological symptoms and their distribution, neurological examination findings, and brain and vascular imaging findings provides information about the diagnosis of stroke location. Mechanisms of stroke and anatomical diagnoses need not be always absolute. In a stroke patient, hemorrhagic stroke may be by far the most possible diagnosis, but embolism and thrombosis are also likely and should not be eliminated from consideration. Whether it is the mechanism or the anatomical location, the process of diagnosis should include hypothesis generation and testing. The clinician must decide on ischemia versus hemorrhage before hypothesizing the subtypes and similarly thrombosis versus embolism versus reduced perfusion before distinguishing subtypes of thrombosis and thus proceeding systematically from the general to the specific. Raised blood pressure, murmurs or arrhythmia, vascular bruits, and cardiac enlargement are certain examples of physical findings that can influence the identification of the stroke mechanism.

A raised blood pressure of 250/130 mmHg would increase the likelihood of hemorrhagic stroke than an ischemic stroke. A normotensive patient with prior history of several intracerebral hemorrhages in different locations has a high probability of having cerebral amyloid angiopathy or bleeding diathesis as the plausible cause. The presence of fever and cardiac murmur suggests the likelihood of a systemic condition such as infectious endocarditis. The presence of diabetes mellitus and coronary artery disease strongly favors a diagnosis of associated atherosclerosis of the larger vessels and a thrombotic mechanism of stroke. A prior history of heart disease raises the possibility of arrhythmia, mural thrombosis, and valvular heart disease, which are potential sources of brain embolism. Repeated history of two or three strokes during the past one year, in the absence of heart disease, can suggest brain embolism probably from a hypercoagulable state or an aortic source. A young stroke (ischemic or hemorrhagic stroke occurring in adult subjects aged less than 65 years) is more likely to be a hemorrhagic mechanism than a thrombotic stroke. However, a middle-aged person with a history of tobacco smoking, a family history of cardiac disease, and a high level of low-density lipoproteins have the possibility of premature atherosclerosis and occlusion of the large artery as the mechanism of the stroke.

The nature of the symptoms and their suggested localization are important in making a diagnosis. With left hemiparesis and the presence of a left visual field deficit or left hemineglect, the anatomical location of the lesion can be to the right cerebral hemisphere. The presence of gaze palsy to the right, nystagmus, and

internuclear ophthalmoplegia would favor a brainstem site lesion in the pons or midbrain. Repeated TIAs in the same vascular territory are virtually diagnostic of thrombotic stroke. For instance, if a patient who presents with aphasia and right limb weakness had an attack of right face and numbness of right hand one week earlier and a TIA characterized by right-hand weakness three weeks prior, then it could be relatively sure that the stroke is a result of thrombotic occlusion of the left anterior circulation. If the same patient also gives a history of a black shade descending over the left eye, causing temporary blindness, then it would be certain that the left internal carotid artery before its ophthalmic artery branch is the anatomical location of the occlusive lesion.

During the night or a nap, the circulation is least active and most sluggish, and many of the thrombotic stroke patients may notice the symptoms soon after the sleep or nap. On the contrary, patients with embolic or hemorrhagic strokes tend to develop the symptoms when the circulation is more active or when the blood pressure is high. However, the current data state that ischemic and hemorrhagic strokes tend to occur during the morning hours till noon, especially when the patient has begun the daily activities. Though hemorrhagic stroke can occur at night and thrombotic stroke can occur during the active time of the day, chances of developing a thrombotic or a lacunar stroke during vigorous physical activity are less likely. The Valsalva maneuver or vigorous sneeze or coughing can loosen an embolic section, resulting in brain embolism. Physical trauma to the neck and sudden neck movements including neck manipulations, following weight lifting, and during the postpartum and after labor should raise the suspicion of arterial dissection.

Clinical improvement shortly after the onset of the neurological deficit argues strongly against an intracerebral hemorrhage. The gradual and progressive development of focal neurological deficit accompanied by the gradual and progressive development of symptoms of raised intracranial pressure suggests intracerebral hemorrhage. The maximal neurological deficit at the onset unassociated with headache is most likely an embolic mechanism. Sudden and severe headache, accompanied by vomiting and an altered level of consciousness right at the onset, suggests a subarachnoid hemorrhage. Unlike intracerebral hemorrhage that tends to have a progressive focal neurological deficit at the onset, subarachnoid hemorrhage causes sudden raised intracranial pressure responsible for the severe headache and the decreased level of consciousness. Altered sensorium is unlikely in lacunar stroke, a subtype of thrombotic stroke. Loss of consciousness is not uncommon in large ischemic stroke and bilateral brainstem stroke. The presence of seizures in the early period after stroke onset argues for intracerebral hemorrhage or an embolic stroke. Vomiting is rare in anterior circulation ischemic strokes. However, it is common in posterior circulation strokes, presumably because of the involvement of the vomiting center located on the floor of the fourth ventricle.

To diagnose and treat cardiogenic stroke, which is different from the intrinsic disease of the intracranial and extracranial arteries, a carefully detailed history of cardiac symptoms, myocardial infarction, rheumatic heart disease, palpitations or arrhythmia, and congestive heart failure, and a detailed examination of the cardiac system is essential. Clinical examination of the systemic and extracranial arteries

may provide clues regarding the presence of atherosclerosis or diminished blood flow, possibly supported by the eye examination findings, as the eyes provide further clues regarding the body's vascular system and the possible stroke mechanism.

The localization of the brain lesion is mainly from the patient's historical description of the neurological symptoms and the neurological examination findings. The most important and frequent brain dysfunctions that need special emphasis are the higher cortical function, level of alertness, visual and oculomotor systems, and gait. If the patient has symptoms or signs present in the right limbs or the right visual field, the bedside tests of the higher cortical function should include the examination of language functions like asking the patient to write a few lines about the town where he or she lives in, to read a paragraph from a newspaper, to name objects in the environment, and to repeat the spoken language. When the symptoms and signs of dysfunction are present in the left limbs or the left visual field, it is specifically important to check the visuospatial functions and look for neglect of the left side of space. Bedside tests like drawing a clock or a house, copying a single two-dimensional figure, reading a brief paragraph or headline, and identifying objects in a picture may reveal findings like omitting words, phrases, or people on the left side of the picture suggesting left neglect.

Memory can be affected by a focal brain lesion involving the posterior cerebral artery territories. Large lesions in the posterior aspect of the cerebral hemispheres may produce only visual impairments and may leave speech, motor, and somatosensory systems unscathed. The confrontation test may provide further clues regarding the visual field deficit. The presence of conjugate-gaze paralysis during clinical examination indicates a frontal or deep hemispheric lesion opposite the side of gaze palsy. The presence of dysconjugate palsies or paralysis of eye movement muscle(s) and the presence of horizontal or vertical nystagmus during gazes are usually diagnostic of vertebrobasilar stroke. If an apparently normal clinical examination finding is revealed in a recumbent or seated position, with the absence of any bulbar findings, and the same patient demonstrates incoordination and imbalance while walking, such a finding may suggest cerebellar hemorrhage or infarction. Table 3.9 depicts the general categories of neurological signs and symptoms and the vascular territories or areas involved.

3.9.1 Neuroimaging and Laboratory Diagnosis

Neuroimaging is an integral part of the evaluation for all stroke patients. For such a potentially devastating condition, clinicians need all of the objective data available to diagnose, prognosticate, and treat individual stroke patients. Rapid identification of the occluding artery or hemorrhage, including its size, shape, and extent of the brain lesion, and estimation of the central necrotic core size and the penumbra can guide the clinician in the appropriate management of stroke. Various neuroimaging modalities, including computed tomography (CT) and MRI, are available for the evaluation of patients specifically presenting with acute ischemic stroke,

Table 3.9 Listing the general categories of neurological signs and symptoms and the vascular territories or areas involved

Location	Vascular territory/area involved	Features
Left hemisphere (anterior hemisphere) lesion	In the territory of the internal carotid artery or its middle and anterior cerebral arteries tributaries	Right hemiplegia, right limb hemisensory loss, aphasia, visual field defect of right, right conjugate gaze palsy, difficulty reading, writing, and calculating
Right hemisphere (anterior hemisphere) lesion	In the territory of the internal carotid artery or its middle and anterior cerebral arteries tributaries	Left hemiplegia, left limb hemisensory loss, left visual space neglect, difficulty drawing and copying, visual field defect of left, left conjugate gaze palsy, left tactile extinction, left visual extinction
Left posterior cerebral artery lesion	Posterior cerebral artery territory	Visual field defect of right, difficulty reading with retained writing ability, difficulty naming colors and objects presented visually, normal repetition of spoken language, numbness, and sensory loss in the right limbs
Right posterior cerebral artery lesion	Posterior cerebral artery territory	Visual field defect of left, often with neglect, left limb numbness, and sensory loss
Vertebrobasilar infarction	Vertebrobasilar territory	Vertigo, diplopia, vomiting, headache in the occiput, mastoid or neck region, weakness or numbness of all four limbs or bilateral regions, crossed motor or sensory findings (i.e., weakness or numbness of one side of the face and the opposite side of the body), ataxia of limbs, bilateral blindness or reduced vision, nystagmus, dysconjugate gaze, gait or lower limb ataxia out of proportion to weakness, crossed signs, bilateral visual-field defects, amnesia
Pure motor stroke or ataxic hemiparesis	Lacunar etiology; vascular supply for internal capsule or basis pontis	Weakness of the face, arm, and leg of one side of the body, without higher cortical dysfunction, sensory or visual dysfunction, or reduced alertness, may present with mixed weakness and incoordination or ataxia on the same side of the body
Pure sensory stroke	Lacunar etiology; usually thalamic area; non-thalamic areas include brainstem, internal capsule, and somatosensory cortex	Numbness or reduced sensibility of the face, arm, and leg on one side of the body, sensory ataxia, absence of motor weakness, visual or higher cortical function abnormalities

intracerebral hemorrhage, and subarachnoid hemorrhage. Both CT and MRI are noninvasive and safe new generation scanners of which the former one is readily available in many of the hospitals. Both for CT and MRI, the image findings depend on the time of the scan and the clinical event.

3.9.1.1 CT and MRI in Stroke

CT scan will show well-circumscribed areas of high density (hyperdense) with smooth borders immediately after the onset of intracerebral bleeding. In case of continued bleed, the sequential scans taken hours to days later may demonstrate enlarging hematoma. Edema developed during the first few days may present as a dark rim around the hyperdense (white in appearance) hematoma. For minor subarachnoid hemorrhagic bleed, CT cannot be a reliable diagnostic tool. When subarachnoid hemorrhage is suspected, lumbar puncture spectrophotometry may reveal xanthochromia (a yellowish tinge color of the Cerebrospinal Fluid [CSF] due to degeneration of red blood cells) in the CSF which is generally confirmatory for the same. The absence of blood in the CSF on lumbar puncture excludes the diagnosis of subarachnoid hemorrhage.

For large ischemic lesions, due to large-artery occlusive disease or cardiogenic brain embolism, a CT scan may show infarction as a low density (hypodense) lesion. Often during the first few hours (up to 6 hours) of the onset of symptoms, the scan may appear normal. CT scans taken at a later time for such large hemispheric ischemic lesions will show a clearly demarcated hypodensity and surrounding edema with mass effect. A CT axial image showing diffuse hypodensity in the cerebral cortex suggestive of ischemic stroke is depicted in Fig. 3.16. Following 2 to 3 weeks after the stroke onset, some infarcts that had been hypodense may become isodense and may obscure the lesion for some time. This phenomenon is called the "fogging effect," and within weeks, a repeat scan will turn the image back to hypodense. CT is generally not useful in detecting and delineating brainstem, cerebellar, and spinal cord infarcts and is not accurate in delineating lesions adjacent to bony surfaces.

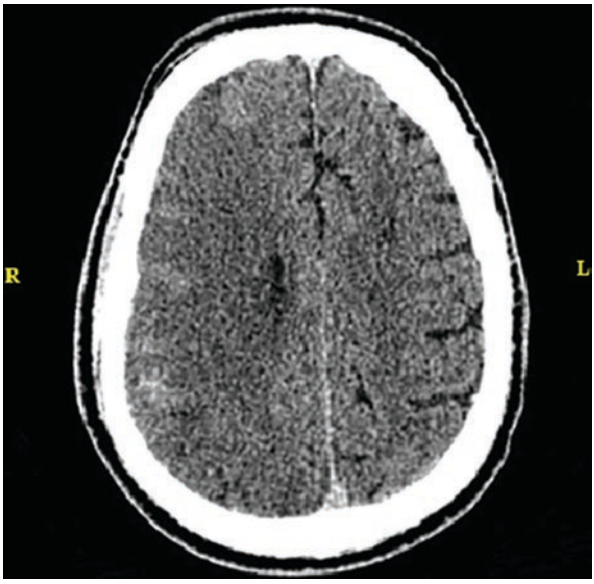

Fig. 3.16 CT axial image showing diffuse hypodensity in the right cerebral cortex with loss of gray white matter interface and cortical effacement suggesting ischemic stroke

Well-defined borders, considerable hypodensity, and shrinkage of the infarcted brain region all suggest months' old chronic infarct, whereas poorly defined hypodensity with surrounding edema and mass effect usually suggests an acute process. For those patients admitted with pure motor hemiparesis due to a lacunar infarct, the CT and MRI may not show any lesions involving the descending corticospinal system.

MRI is the investigation of choice for acute onset stroke and patients with TIAs. MRI is more sensitive than CT in detecting early ischemic changes. Cerebral ischemia alters water content in the affected region, prolonging the T1 and T2 relaxation constants and due to which the lesions will appear as a dark region (hypointense) in T1-weighted image sequences and as bright region (hyperintense) in T2-weighted images. With time (a few days later), lesions will become more hypointense on T1-weighted and more hyperintense on T2-weighted images. Diffusion-weighted MRI or diffusion-weighted imaging (DWI) is specifically sensitive for the detection of acute brain infarcts as it can detect the water shift (extracellular to intracellular) occurring in the cytotoxic edema phase within minutes of stroke onset. Infarct areas appear bright on DWI (same as hyperintense on T2-weighted images) and dark on apparent diffusion coefficient (ADC) images. The DWI and the ADC images of an early stage infarct (hyperacute stage) are depicted in Fig. 3.17. DWI can detect the earliest ischemia, and some hours later, T2 fluid-attenuated inversion recovery (FLAIR) can also show hyperintensity. Figure 3.18 shows patchy hyperintensity on the T2 and FLAIR images suggesting early (acute infarct) stroke. For those TIAs with no residual symptoms, the DWI scans may show brain infarcts, and such lesions are regarded as "small strokes." Both for the anterior and posterior circulations, the location, pattern, and multiplicity of DWI sequences of the lesions can help in suggesting the causative stroke mechanism, including the cardiogenic multiple embolic strokes or multiple strokes due to hypercoagulable state.

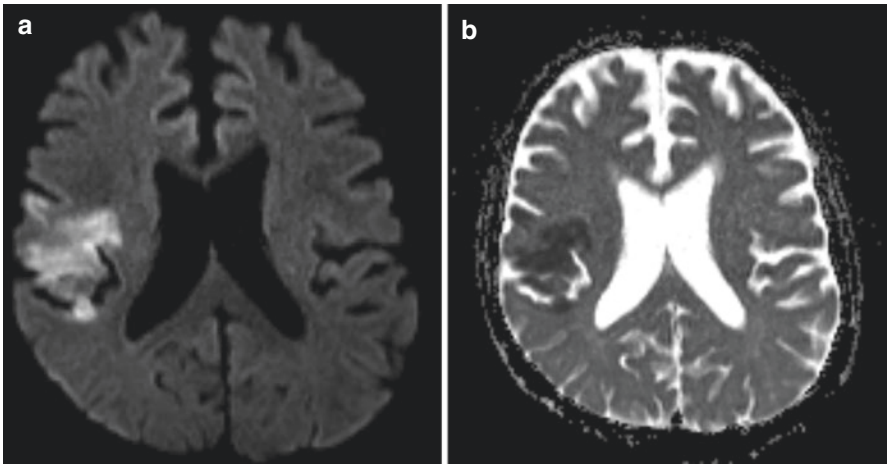

Fig. 3.17 Depicting the DWI and the ADC images of an early stage infarct. (**a**) Magnetic resonance (MR) axial images showing patchy zones bright on DWI suggesting early stage infarct. (**b**) MR axial images showing patchy dark zones on ADC suggesting early stage (hyperacute) infarct

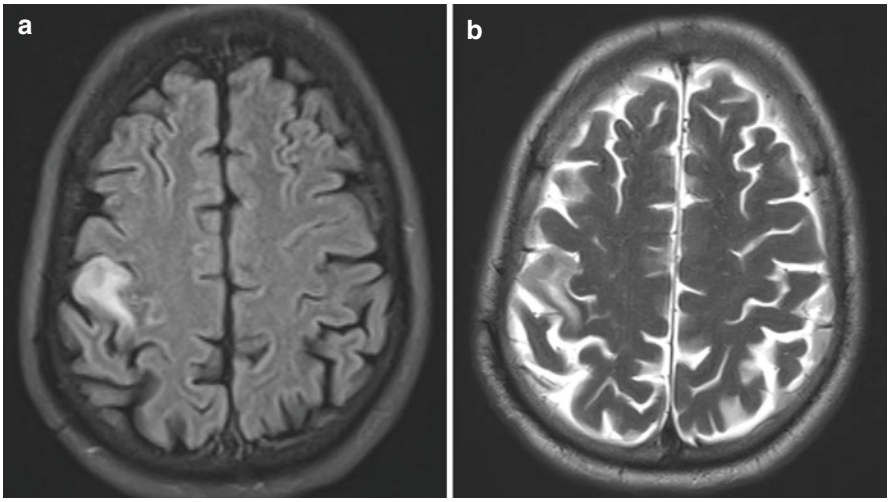

Fig. 3.18 MR axial images showing patchy hyperintensity on the T2 (**a**) and FLAIR (**b**) images, respectively, suggesting early (acute infarct) stroke

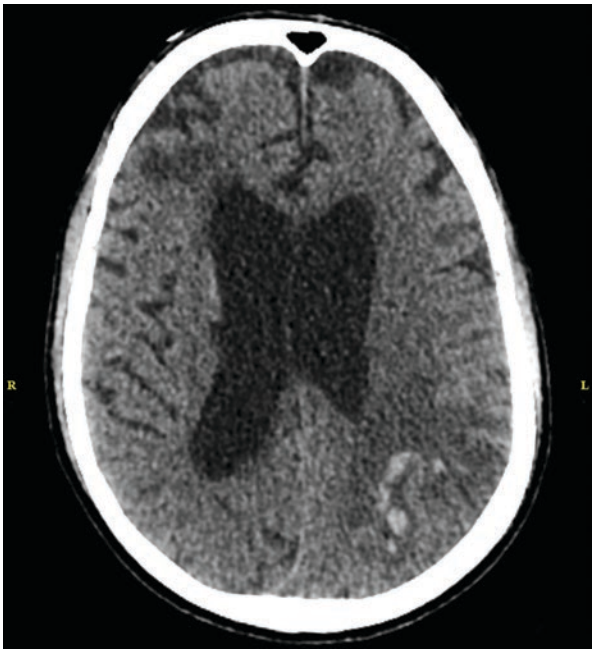

Fig. 3.19 CT axial image showing ill-defined hypodensity in the left posterior cerebral cortex with hyperdensities within suggesting hemorrhagic infarct

Figure 3.19 demonstrates a CT image of a hemorrhagic infarct. A CT axial image showing a large hemorrhage with ventricular extension is depicted in Fig. 3.20. Though CT scans of intracerebral hemorrhage are easier to interpret than MRI scans, susceptibility-weighted imaging (SWI), a sensitive technique under MRI,

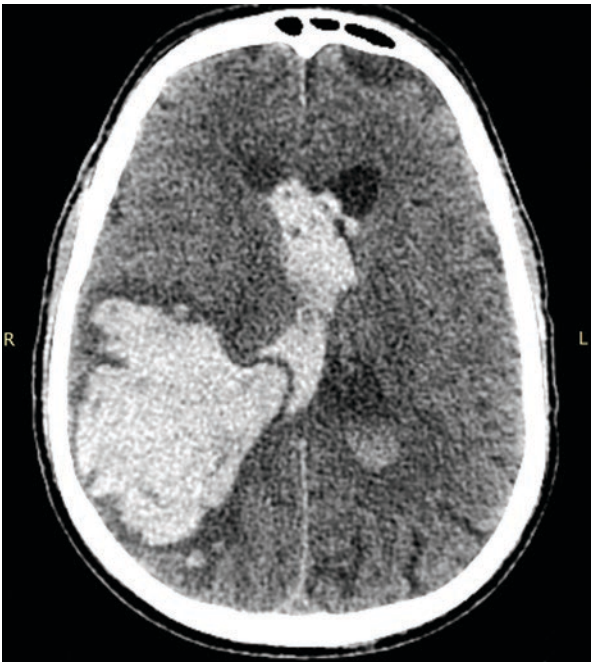

Fig. 3.20 CT axial image showing large hemorrhage in the right cerebral cortex and ventricle extension

can accurately demonstrate the bleed as an area of blooming lesion. Hemoglobin derivatives (oxyhemoglobin, methemoglobin, hemosiderin, or ferritin) at the site of bleed have a paramagnetic effect, and the appearance of the image (hypointense or hyperintense) depends on the nature of the compound, which is time-bound.

Though the size of the lesion has considerable prognostic value, as large lesions in the anatomical area are more likely to cause severe deficits than small lesions in the same area, the severity of the clinical deficit need not always be directly proportional to the infarct size as shown on CT or MRI. While comparing the CT or MRI findings with the clinical deficits, it would not be surprising to see a discrepancy between them. A discrepancy might suggest that certain tissues, although not morphologically damaged enough to show on scans, are not functioning normally. MRI and perfusion CT studies can give detailed information about the neuronal tissue at risk of death by a given vascular lesion. While comparing the DWI and the perfusion CT images, if the perfusion defect appears larger than the diffusion-weighted zone of infarction, the tissue displaying perfusion deficit is at imminent risk of infarction unless blood flow improves to that zone. The clinician should also confirm whether the location of the lesion(s) based on CT and MRI and the location based on the clinical findings are proportionate and correlating.

CT and MRI images can provide details about the mass effect, including the displacement of midline structures, effacement of gyri, cisternal spaces encroachment, brain tissue herniation, and brainstem displacement. Cerebral edema can develop around infarcts and can even be secondary to the reperfusion of a blocked

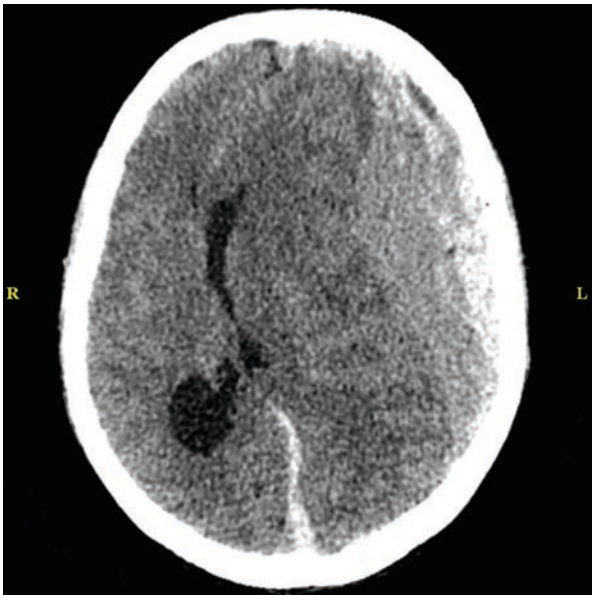

Fig. 3.21 CT axial image showing large subdural hemorrhage with the displacement of midline structures and effacement of gyri

artery. In young patients, edema can be more threatening than in aged patients as the atrophied brain in the latter can provide extra space for brain expansion.

The mass effect created by enlarging hematomas (Fig. 3.21) can be a more serious concern than the edema and mass effect following brain infarction. CT angiography can reveal a "spot sign" a feature indicating the dynamic nature of intracerebral bleed. The spot sign is a unifocal or multifocal contrast enhancement(s) within an acute primary bleed, disjointed from adjacent normal or abnormal blood vessels, and it corresponds to the site(s) of active hemorrhage and is a signature of active intracerebral bleed. The presence of a "spot sign" indicates a greater risk of hematoma expansion, especially if it drains into the CSF space and is an adverse prognostic factor. Infratentorial bleeds have a worse prognosis than supratentorial bleeds. The mass effect produced by the hematoma within the brain and blood within the ventricular system can obstruct the flow of CSF and cause hydrocephalus, which requires surgical decompression by temporary drainage or permanent shunting of CSF.

Blood may accumulate around a bleeding aneurysm or the subarachnoid spaces and cisterns and can provide clues about the site of bleeding. Bleeding from the anterior communicating artery aneurysm causes the presence of blood in the suprasellar cisterns and frontal interhemispheric fissure. Bleeding from the right or left middle cerebral artery bifurcation aneurysm leads to blood in the respective Sylvian fissure. A posterior fossa aneurysm bleeds into the pontine and cerebellopontine angle cisterns (Fig. 3.22). The thickness of blood seen on CT or MRI sequences will provide information about the degree of bleeding. Large subarachnoid bleeds can cause hydrocephalus, and the vasoconstriction of the blood vessels in the vicinity

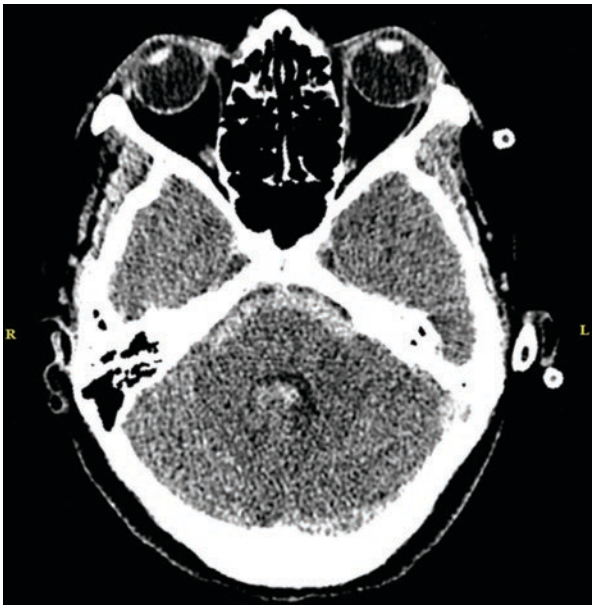

Fig. 3.22 CT axial image showing hyperdensities into the pontine and cerebellopontine angle cisterns

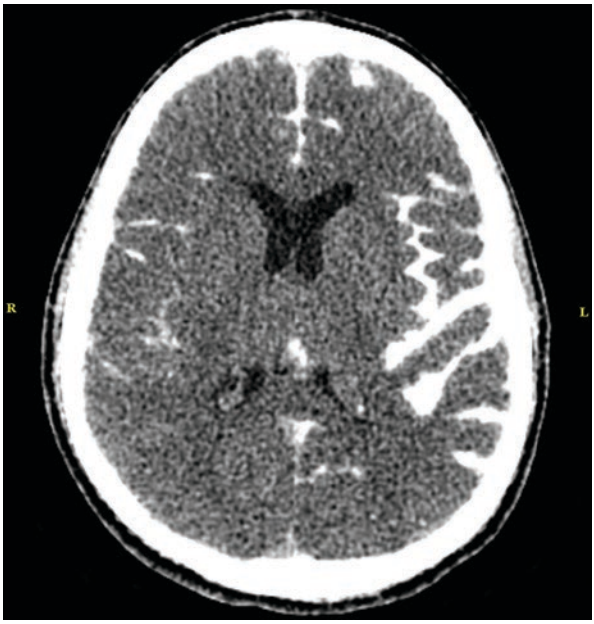

Fig. 3.23 CT axial imaging showing hyperdensities suggestive of large subarachnoid bleeds (left more than the right)

can cause delayed cerebral infarction. Figure 3.23 depicts a CT axial image showing hyperdensities suggestive of large subarachnoid bleeds. Both CT and MRI are not particularly sensitive for the detection of subarachnoid hemorrhage, especially when bleeding is minor or in the recent past. Restless, claustrophobic, or critically

ill patients often have difficulty holding still, the time required to produce high-quality MRI images, and for the same reasons, such patients will not be suitable candidates for MRI.

3.9.1.2 Ultrasound and Vascular Imaging in Stroke

Ultrasound and vascular imaging techniques can reveal the vascular structures involved. Transcranial Doppler ultrasonography, a noninvasive technique, is used to check the patency of extracranial components of the carotid arteries and the posterior circulation of the brain. MR angiography offers many advantages over other noninvasive methods of vascular imaging. Angiography in patients with hypertensive bleeds will show the predominance of microbleeds in the deep structures, including the subcortex, basal ganglia, lateral ganglionic region, pons, and cerebellum. In such a case, the likelihood of an etiology other than hypertension is relatively low. Angiography in amyloid angiopathy will demonstrate the predominance of microbleeds in the lobar regions of the cerebral cortex. Hematomas resulting from aneurysms are invariably contiguous to the aneurysms at the base of the brain or surface of the brain. Intracerebral hematomas secondary to anticoagulants are mostly located to the lobar or cerebellar and tend to evolve and enlarge gradually. Arteriovenous malformations can be located anywhere within the brain, particularly in subependymal regions. Figure 3.24 depicts the MR and T2 axial angiography showing a cluster of vessels suggesting arteriovenous malformation.

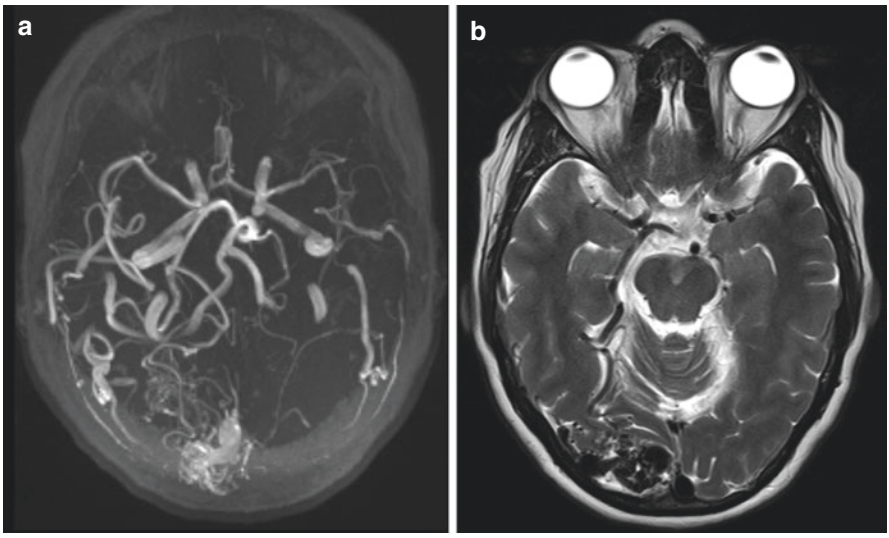

Fig. 3.24 MR and T2 axial angiography showing a cluster of vessels suggesting arteriovenous malformation. (**a**): MR axial angiography showing a cluster of vessels with arterial feeders suggesting arteriovenous malformation in the posterior occipital cortex (**b**): MR axial T2 angiography showing a cluster of vessels as signal voids suggesting arteriovenous malformation

3.9.1.3 Blood Investigations

Based on the nature, location, and severity of the vascular lesion, it is crucial to find out whether the abnormalities of blood constituents are causing or contributing to ischemic or hemorrhagic strokes. Abnormalities in the clotting system can predispose to hypercoagulability and thrombosis. Infections, cancer, and other diseases can release acute phase reactants that may alter coagulability sufficient to promote thromboembolism. Increased viscosity of blood can alter blood flow, especially in small arterioles and capillaries of the brain, and can cause or contribute to regional decreases in blood flow and potentiate cerebral ischemia. Box 3.1 displays the standard set of blood analyses consisting of hematological studies, serum electrolyte levels, and renal and hepatic tests essential to identify the hematological, metabolic, renal, and hepatic abnormalities underlying the stroke or TIA.

Box 3.1 The Standard set of Blood Tests for Stroke and TIA

Standard set of blood tests for stroke and TIA
• Blood sugar
• Hemoglobin
• Hematocrit
• Homocysteine
• Total and differential WBC count
• Platelet count
• Erythrocyte sedimentation rate
• Activated partial thromboplastin time
• Prothrombin time-international normalized ratio (INR)
• Serum fibrinogen level
• Total cholesterol and lipid profile
• Serum calcium
• Blood urea nitrogen
• Blood electrolytes
• C-reactive protein

3.10 Medical and Surgical Management of Stroke

Technological advancements in diagnostics have made it possible to quickly and safely determine the cause of most strokes. Even though socioeconomic and psychological factors can influence stroke treatment, the presence of comorbid medical conditions can often limit the choice of available treatments. For instance, the clinician may not recommend surgery for stroke patients with advanced cancer. Abnormalities of coagulation functions and blood constituents can indicate certain therapeutic strategies and contraindicate other treatments. However, age is never an absolute contraindication for stroke management provided that the elderly are cautiously handled.

The nature, location, and severity of the vascular lesions are important factors determining the selection of possible and optimal therapeutic strategies. In addition to the above, the choice of treatment also critically depends on the nature of the causative cerebrovascular process, i.e., management of internal carotid artery occlusion will undoubtedly be different from the management of severe internal carotid artery stenosis or carotid atherosclerotic plaque without stenosis. Reperfusion can be potentially hazardous when the vascular territory is necrotized. On the contrary, if the vascular territory is ischemic and the neurons are stunned and dysfunctional, reperfusion or augmenting the blood flow can substantially save the neuronal tissue from the imminent infarction.

The pace of progression also dictates the urgency and speed of stroke management. For instance, a patient with an acute episode of TIA has a greater probability of stroke tomorrow as compared to a patient with three-month-old case of a single TIA. A stroke patient worsening under medical supervision requires more urgent management than a stable patient with a week old stroke. Box 3.2 displays the reasons for the worsening of acute stroke symptoms. The therapeutic strategies are either general or specific. The general strategies (Box 3.3) apply to all stroke patients, whereas specific therapeutic strategies focus on the specific concerns of the patient, which include management of raised intracranial pressure, potential threat for a second embolism, and recurrent threat of subarachnoid hemorrhage. The general care goals for the clinicians are to limit suffering, give comfort, and prevent complications. Even when no specific therapy seems warranted, stroke patients deserve excellent nursing and general care. Since stroke patients are at risk for future stroke, prevention strategies should begin as early as possible.

Box 3.2 Reasons for Worsening of Acute Stroke Symptoms

Reasons for worsening of symptoms
• Hemorrhagic transformation of an ischemic infarct
• Failure of collateral circulation
• Progressive occlusion of the arterial lumen
• Expansion of hemorrhage
• Reduced perfusion due to hypovolemia or systemic hypotension
• Cerebral edema
• Embolization or propagation of thrombus
• Seizures
• Infections, particularly pneumonia and urinary tract infection
• Cardiac arrhythmias
• Pulmonary embolism
• Depression

Box 3.3 General Therapeutic Strategies for Stroke Management

General therapeutic strategies

1. Prevent stroke complications such as urinary infections, DVT and pulmonary embolism, phlebothrombosis, bedsores, and contractures and joint stiffness.
2. Prevent further strokes and vascular disease by controlling the stroke risk factors.
3. Maintain adequate blood pressure to minimize the risk of hypotension and hypertension.
4. Restore/maintain fluid and electrolyte balance.
5. Maintain nutrition.
6. Maintain blood glucose levels within the normal range.
7. Maintain bowel and bladder function, which may include a urinary catheter.
8. Improve neurological function and facilitate recovery.

Different medical and surgical strategies are available to improve the circulation to the ischemic region distal to the occluding lesion. During the early stage of acute care, a reduction in blood flow caused by postural changes can reduce the cephalad flow through the stenotic vessel or collateral channels and may worsen the ischemia. For minimizing the possibility of inadequate blood perfusion, ischemic stroke patients are generally advised to be treated in the supine position as sitting, standing, and propped up position of head may increase ischemic symptoms. The therapeutic strategies used for reperfusion include angioplasty, stenting, thrombolysis, mechanical clot retrieval or clot aspiration, surgical endarterectomy, and vasodilator treatment.

Neurointerventional techniques have become an important therapeutic strategy for managing many stroke conditions. Since the 1980s, interventional procedures have used devices including coils, catheters, balloons, and glues, for the treatment of patients with intracranial aneurysms and arteriovenous malformations. Percutaneous transluminal angioplasty, with or without vascular stents, first developed for coronary artery, has become an important option for cervical and cerebral atherosclerotic arteries and fibromuscular dysplasia. Vascular stenting, in conjunction with percutaneous balloon angioplasty, has shown significant improvement in outcomes including lowering the rate of recurrent stenosis and event-free survival post 1 year, compared to angioplasty alone. In addition to the above, stents generally reduce the risk of plaque dislodgement and significant intimal dissection and improve the elastic recoil of the vessel wall.

Increasing evidence is available regarding the benefits and effectiveness of angioplasty, with or without stenting, to treat carotid atherosclerotic disease occluding the vessel or generating embolic debris. An important technical advancement was the development of embolus protection devices (distal protection devices) that consist of baskets or umbrellas placed distal to the target atherosclerotic lesion to trap debris dislodged while performing the angioplasty. Angioplasty and vascular stenting can manage occlusive lesions and symptomatic lesions in neck arteries and intracranial arteries. Patients with long and smooth lesions in the large arteries,

especially with coronary artery disease, tend to do better with neurointerventional techniques. On the contrary, patients with focal irregular ulcerated atherosclerotic lesions, tortuous arteries, and arteries with catheter access difficulty tend to do better with surgical endarterectomy.

Thrombolysis means the chemical lysing of the clots within the vessel. Following thrombus formation, the vascular system stimulates the formation of endogenous fibrinolytic mechanisms for thrombolysis. At the sites of fibrin deposition, the release of tissue plasminogen activator (Factor XII) and other chemical substances converts plasminogen to plasmin, the active fibrinolytic enzyme. The endogenous formation of plasmin is probably responsible for the spontaneous recanalization of thrombosed arteries. Recombinant tissue plasminogen activator (r-TPA), streptokinase, and urokinase are certain thrombolytic drugs used in the past. These drugs were administered either intra-arterially into the clots or intravenously. The presence and extent of reperfusion depended mostly on the location of the clot and the stroke mechanism. The main stem and divisional middle cerebral artery occlusions respond better to intra-arterial thrombolysis, compared to internal carotid artery occlusions. The proximal segment of the middle cerebral artery responds better than distal branch occlusions. Embolic occlusions were more successfully recanalized compared to thrombosis engrafted upon atherosclerotic narrowing. For those thrombolysis cases with re-occlusion, transluminal angioplasty might help to maintain the patency of the lumen. Recanalization is generally better when angiography suggests good collateral circulation prior to administration of r-TPA. Though the recommended therapeutic window ranges from 3 to 4.5 hours, patients treated with r-TPA) within 3 hours have shown better response compared to those treated between 3 and 6 hours and controls. Significant infarction, mass effect, edema, hemorrhage, and the onset of stroke beyond 4.5 hours are specific contraindications for r-TPA. Overall, the thrombolytic drug will not facilitate recovery if it fails to recanalize the vessel. Intracranial hemorrhage and death are a few of the complications associated with thrombolysis.

Catheter-based mechanical thrombectomy techniques have made rapid advances in the recanalization of the occluded arteries during the past few decades. Compared to thrombolytic drugs, mechanical thrombectomy is more effective and substantial in reperfusion, more effective in the removal of large clots in the proximal vessels, and less risky for intracerebral and systemic hemorrhages. During mechanical thrombectomy, to minimize the possibility of vasoconstriction, intra-arterial infusion of papaverine, an alkaloid antispasmodic drug, may be used. The endovascular mechanical intervention devices can be categorized as angioplasty and/or stent devices, suction thrombectomy devices, and thrombus retrieval devices. Angioplasty and/or stenting are highly effective in the recanalization of local atherosclerotic occluded brain arteries. Suction thrombectomy devices use vacuum aspiration to remove the occlusive clot out of the artery. Stent retrievers are the most recent and most successful among the family of thrombectomy devices. The mesh columns of the stent retrievers expand and entangle inside the target clot, and while withdrawing the stent, the mesh will bring out the thrombus. The two major advantages of stent retrievers over the rest are their substantially higher recanalization rate and

instant restoration of blood flow immediately upon deployment within the target artery.

The local reconstruction endarterectomy or surgical endarterectomy is the common direct surgical method of unblocking an occluded vessel. Surgical endarterectomy not only augments the blood flow but also removes the source of intra-arterial emboli. Endarterectomy is considered when arterial stenosis is between 60 and 99% as it can reduce the risk of imminent stroke by as much as 55%. Revascularization following endarterectomy of symptomatic carotid stenosis has a much higher therapeutic index as compared to asymptomatic occlusive lesions. However, symptomatic patients with arterial stenosis between 50 and 69% tend to have modest benefits. A careful selection of patients can significantly lower the risk of neurological and cardiac morbidity and mortality associated with this procedure. The complications associated with endarterectomy include stroke, cerebral hyperperfusion syndrome, bleeding, and infection.

The search for effective neuroprotective agents is mostly unsuccessful. Neuroprotective agents are likely to stabilize the threatened tissues and salvage vulnerable brain tissues before the thrombolytic agents are administered. Magnesium sulfate and nitroglycerin are a few of the neuroprotective agents studied. Researchers have been exploring the potential beneficial effects of hypothermia in combination with thrombolysis. In addition, they are also exploring the beneficial effects of standard anticoagulants and antiplatelets as adjuncts to thrombolytic drugs. The use of ultrasound to increase the transport of r-TPA) into the thrombus and binding of r-TPA to fibrin has also been explored.

Unlike brain infarction, a large hemorrhagic stroke or subarachnoid hemorrhage always adds extra volume into the rigid and closed cranial cavity. Such extra volume often increases the pressure inside the cranium. Herniations into other dural compartments, midline shifts in the intracranial contents, and the generalized rise in the intracranial pressure are the common causes of death in such strokes. Treatment should include strategies to control and prevent elevation of intracranial pressure. Subarachnoid hemorrhage patients almost always have increased intracranial pressure due to the expanding intracranial volume. Increased volume, either in the form of blood or edema surrounding the hematoma, can obstruct the CSF flow and drainage or reduce the cerebral perfusion pressure, further escalating the brain parenchymal damage. Serial neuroimaging studies have reported that hematomas tend to expand during the first few hours (3–6 h) after the onset of the symptoms, whereas edema around the hematoma tends to develop around the first 48 hours. Usually, larger hematomas have more surrounding edema. Hemoglobin products and thrombin are a few factors that may promote edema formation. The aim of therapy should be to limit the size of the hemorrhage by limiting the bleeding or draining the hematoma and treating the accompanying edema. In case of hemorrhage caused by arteriovenous malformation or aneurysm, removing the offending vascular lesion prevents recurrent hemorrhage. Another important strategy to stop the bleeding is to reduce arterial tension.

Endovascular coiling, surgical clipping, or bypass or flow diversion are the frequent procedures performed for intracranial aneurysms. Endovascular coiling is a

minimally invasive fluoroscopy-aided procedure, where the platinum coils are released into the aneurysm through a catheter. These coils induce embolization of the aneurysm and seal off the opening of the aneurysm, thus preventing blood from getting into the aneurysmal sac. The number of coils required depends on the size of the aneurysm and are left permanently inside the aneurysm. Endovascular coiling procedures are generally indicated for unruptured brain aneurysms than ruptured aneurysms, as well as for older patients and for those patients for whom surgery is contraindicated.

Clipping is a surgical procedure to treat an aneurysm. Following a craniotomy, the goal is to isolate and clip the aneurysm from the rest of the vasculature without blocking any of the adjoining large or small arteries nearby. The clip, made of titanium, is placed permanently across the base or neck of the aneurysm to prevent the blood from filling the aneurysm. Clips are made in a variety of shapes, sizes, and lengths due to variations in size and shape of the neck of aneurysm (part of aneurysm near the origin on the main artery) among patients. A ruptured aneurysm is life-threatening, and the risk of repeated bleeding is high during the next few weeks and is an indication of surgical clipping within 72 hours of the first bleed. Vasospasm, stroke, seizure, bleeding, and an imperfectly placed clip are a few of the complications which could arise from aneurysm clipping. Size, location, and neck geometry of the aneurysm, age of the patient, general health, and comorbidities are certain essential factors that decide the various treatments like surgical clipping or bypass or endovascular coiling or flow diversion.

3.11 Physiotherapy Management of Supratentorial Stroke

A coordinated interdisciplinary team consisting of neurologist, nurse, physiotherapist, speech therapist, occupational therapist, dietician, clinical psychologist, and medical social worker should oversee the comprehensive plan of care to address various issues of stroke patients and the concerned family members. To provide a supportive environment and assist patients and their family members, communication between the interdisciplinary team members is critical. Among the health professions, physiotherapy plays an important role in promoting functional abilities, reducing disabilities, preventing or minimizing the complications of stroke, promoting independence, and achieving a better quality of life.

Patients who sustained cerebrovascular accidents may have several impairments. These impairments can interfere with their functional capabilities, and the extent and severity of impairments will depend on the anatomical location of the lesion and the mechanism of stroke. A detailed assessment of the patient covering the medical history, course of the disease, impairments, activity limitations, and participation restrictions helps to identify the priorities and concerns of the patient, his or her abilities, and the resources available. The intervention should include restorative strategies to reduce impairments, activity limitations and participation restrictions. Preventive strategies will minimize the complications and the secondary sequels of

the disease, and compensatory strategies will improve function by modification of the task or environment. For patients with more disabling sensorimotor deficits, which is common among moderate-to-severe strokes, long-term planning is essential as they are less likely to be resolved during the acute or subacute phase management in a hospital-based setup (acute care or inpatient rehabilitation).

The acute phase therapeutic intervention should begin as soon as the patient is medically and hemodynamically stable, which is generally 48–72 hours after the onset of symptoms. During this phase, rehabilitation consists of low-intensity exercises. By reviewing the medical record and communicating with the medical team, the therapist should regularly update himself about the neurological and medical status of the patient. Early mobilization during this phase prevents or reduces the ill effects of bed rest and deconditioning, may improve the level of consciousness of the patient, and speed up the return to independence. Encouraging the use of the paretic side and facilitation or stimulation of the same side are likely to promote functional reorganization, reduce the possibility of learned nonuse of the paretic extremities, and minimize the potential for maladaptive patterns of movement. Interventions should not only include early mobilization, positioning, bed mobility, transfer techniques, ROM exercises, splinting, and encouraging the ADL but also instructing, educating, and training the patient and family members.

The acute phase is a quite stressful time for the patient and the family members, and therefore the information and instructions given to the patient and family members need to be sorted. Appropriate, but effective, communication is crucial during this stage. Providing a less distractible environment may encourage the patient to draw his or her attention for effective communication. The location from which the therapist interacts with the patient should also consider visual field defects like homonymous hemianopia and quadrantanopia or perceptual disorders like spatial neglect if any. In this phase, the physiotherapist should be vigilant about the potential risk of medical emergencies and complications, including uncontrolled or accelerated hypertension, cardiac arrhythmias, DVT, and aspiration.

During the subacute phase of rehabilitation, patients usually require more intense intervention, particularly when they have considerable residual impairments. Generally, an inpatient rehabilitation facility will be more suitable for such patients. The intensity and timing of rehabilitation efforts, medical stability, severity of cognitive and perceptual deficits, and patient's motivation and endurance are certain factors influencing the functional outcomes. Many of the interventions begun during inpatient rehabilitation tend to continue and progress beyond the inpatient rehabilitation facility. Typically, post-discharge, the rehabilitation services are continued and progressed either on an outpatient basis or for subjects with limitations in accessing hospital facilities; the same services are rendered through a community outreach program. The intervention programs focusing on flexibility, strength, balance, locomotion, endurance, and upper limb function have shown meaningful outcomes for those treated in the outpatient facilities.

Historically, motor functional recovery was thought to be complete within 3 to 6 months post-stroke. However, recent evidence proves that functional recovery

after a stroke can continue for months or years. The acute phase functional gains following stroke is generally attributed to improved perfusion or recanalization of the ischemic or penumbral zone, reduction of cerebral edema, and absorption of damaged tissue. However, improvement during the long-term functional recovery is ascribed to neuroplastic mechanisms like collateral sprouting, synaptogenesis, and unmasking of neural pathways, believed to circumvent rather than repair the damaged brain tissue.

Regarding training programs for stroke patients, the specificity of training and increased intensity of training are important factors in the process of learning. Practice sessions beyond the therapy session are recommended for better learning. It is also important to create an enriched environment that supports learning and provides the typical challenges of everyday life. The enriched environment tends to improve the activity level and enhance recovery. The strategies include computers with Internet access, reading material, puzzles, board games, tablets, access to music and books, encouraging family members to bring in hobbies and activities, and access to communal areas. In cases of severe sensorimotor deficits with a limited scope of recovery or stroke with multiple comorbidities, compensatory training strategies should be encouraged to resume functions using the less involved extremities and alternate movement patterns.

During the chronic phase, 6 months following the onset of symptoms, the patient and family are instructed about the home exercise program and are educated about the importance of maintaining physical fitness, health promotion, lifestyle modification, modification of risk factors, fall prevention, and safety. For those patients who cannot avail outpatient rehabilitation facilities due to severe disability or immobility or being bed-confined, home care rehabilitation services can provide the intervention strategies. Finally, the patient's recovery and eventual outcome not only depends on the location of the vascular lesion, mechanism of stroke, and time and type of medical or surgical intervention but also on the presence of preexisting medical conditions, appropriate rehabilitation strategies, and amount of family support and financial resources available.

3.11.1 Clinical Examination

A detailed clinical examination helps to identify the impairments pertinent to the stroke syndrome, identify the stroke patients that require extensive rehabilitation intervention programs, and develop an appropriate plan of care, which includes the anticipated goals, interventions, expected outcomes, and prognosis. In addition to the above, the clinical examination also helps to monitor the recovery and treatment progress toward projected goals and expected outcomes, prescription of appropriate walking aids, splints, and adaptive equipment and develop a home-based exercise program at the time of discharge.

Lesions in the frontal and temporal lobes of the dominant hemisphere can lead to specific communication deficits. Around 30% of stroke patients will have a certain

degree of language dysfunction. Different types of aphasia and its features have already been addressed earlier in this chapter. Evaluating and treating patients with receptive and global aphasia can often be challenging as they may fail to comprehend spoken words and gestures. Appropriate speech therapy, along with time and patience, may help to develop some basic communication methods to interact with these patients. Other communication deficits like dysarthria and emotional lability may also affect the patient's ability to interact with individuals.

The clinical examination should include tests and measures to quantitatively or qualitatively assess the impairments, functional performance, and activity limitations. Subjective information obtained by interviewing the patient or family members and from the medical records should cover details such as general demographic data, present and past medical and surgical history, socioeconomic status, risk factors, and social and health habits. The presence of blurring of vision, visual field deficits, facial muscle weakness, hemisensory loss of facial sensation, hearing difficulties, swallowing difficulties, and slurring of speech necessitates the examination of cranial nerves. Since, perceptual dysfunctions like visual neglect or inattention can mimic visual field defects, it is essential to rule them out before the test for visual field is executed using the confrontation method. Examination of the third, fourth, and sixth cranial nerves may disclose ocular motility disturbances, including diplopia, strabismus, oscillopsia, and conjugate gaze paralysis.

Many of the stroke patients may have partial impairments, as opposed to a total loss of sensory perception. These sensory impairments may affect the patient's ability to control and coordinate movement. Such patients may also lose the ability to perceive upright postures and may face difficulty in performing normal weight shifts and sequenced motor activities. Deficits in type and extent of somatic sensation impairments among stroke patients depend on the location and size of the vascular lesion. Usually, for cortical lesions, sensory impairments will be specific to a local area, unlike the diffuse involvement of one side of the body which suggests deeper lesions involving the thalamus, internal capsule, or nearby structures. In addition to the above, cortical lesions are characterized by a loss of combined and cortical sensations and relative preservation of the primary modalities of sensations. Crossed sensory loss, i.e., anesthesia over ipsilateral face and contralateral trunk and limb, is characteristic of a brainstem stroke. Profound involvement of the somatosensory system will adversely affect the motor performance, motor learning, and therapeutic outcomes and contribute to hemineglect and learned nonuse of limbs. During the evaluation, it is also essential to look for central post-stroke pain as it can debilitate and limit the participation of the patients during the rehabilitation programs. Lesions affecting the somatosensory cortex or its pathways can result in sensory ataxia with the muscle strength somewhat preserved. Vascular lesions involving the cerebellum typically produce cerebellar ataxia and hypotonicity. The presence of Romberg's sign, absence of incoordination with eyes open, and the presence of incoordination with eyes closed (finger-to-finger or finger-pointing test with eyes closed) distinguish sensory ataxia from cerebellar ataxia. A detailed sensory evaluation may not be practical or possible in many patients when cognition and communication skills are affected.

The degree and the extent of motor recovery depend on several factors, including the anatomical location of the lesion, severity of the lesion, mechanism of stroke, rehabilitation strategies used, and motivation of the patient. The muscles of the involved side, soon after the onset of stroke, due to cerebral shock, will be typically flaccid (flaccid stage-stage 1 of Brunnstrom recovery stages). This early stage is short-lived, and during stage 2 of recovery, the flaccidity will be replaced by progressive development of spasticity, hyperreflexia, and the appearance of basic limb synergies (mass movement patterns). These synergistic movements are generally stereotypical and primitive and can either be elicited reflexively or volitionally. Muscles involved in the basic limb synergies are strongly linked and are relatively easier to recruit during the early and middle stages (stage 2 and stage 3) of recovery. The mass movements produced by the synergistic action of these muscles are highly stereotypic among stroke patients. The spasticity will accentuate, and movements outside the basic synergies will not be possible (stage 3) until the recovery progresses further. In the later stages of recovery (stage 4 and stage 5), the spasticity progressively wanes, and the strong linkage between the muscles of synergies reduces characterized by movement patterns away from the basic synergies. Isolated joint movements (fractionation of movements) and normal synergies with near-normal coordination and speed are the features of stage 6, and the last stage (stage 7) is characterized by the return of normal motor function, including fine motor skills, speed, and coordination. The extent of recovery need not be the same for the upper and lower extremities as the vascular territories for the concerned areas are not the same. Though the pattern of motor recovery is somewhat similar among most of the supratentorial strokes, individual variations are common that include the speed of recovery (from few days to several months) and extent of recovery (full recovery to incomplete recovery). The presence of basic limb synergy, increased tone, hyperreflexia, and Babinski sign warrants the need for noting the Brunnstrom recovery stage for the upper and lower extremities of the involved side. For further details about the basic limb synergies and stages of recovery, refer to Brunnstrom's approach under the chapter titled "Therapeutic Approaches." In addition to identifying the motor recovery stage of the stroke patient, the ability of the patient to voluntarily move those muscles which are not associated with basic limb synergy needs to be evaluated, including latissimus dorsi, serratus anterior, finger extensors, and ankle evertors. Evaluation of fine motor and dexterity skills (writing, dressing, and feeding) is a part of the examination when the stage of motor recovery is above 5.

Evaluation of the patient's deep tendon reflexes will provide valuable information about the presence of abnormal muscle tone. The reflexes are hypoactive in hypotonia and absent in flaccidity. The presence of hyperreflexia, one of the features of upper motor neuron lesion, generally tends to emerge when the cerebral shock dissipates soon after the onset of stroke. A percussion or knee hammer can help the therapist to grade the hyperactivity of the deep tendon reflexes, and while examining the tendon reflex, it is wise to know whether there is any presence of sustained or non-sustained clonus. The most common sites for eliciting clonus are the ankle, patellar, and wrist. The grades and descriptions of deep tendon reflexes are mentioned in Box 3.4. The clinical examination may reveal the presence of released

postural reflexes like tonic neck reflex and tonic labyrinthine reflex and associated reactions. Associated reactions are readily elicitable when hyperreflexia, spasticity, and mass synergies are present.

Box 3.4 The Grades and Descriptions of Deep Tendon Reflex

Deep tendon reflex grades
0: No response or not elicitable
1+: A diminished or slight response or a response brought out only with reinforcement
2+: A normal response; same as the contralateral unaffected side
3+: A brisk response; more than seen on the contralateral unaffected side
4+: A hyperactive, exaggerated or very brisk response; presence of clonus
It is preferable to document the clonus site (ankle, patellar, or wrist) and the type of clonus (sustained and non-sustained) separately. More than 5 beats is considered as a sustained clonus.

Spasticity, a feature of upper motor neuron lesion, tends to occur predominantly in the antigravity muscles like scapular retractors and depressors, shoulder adductors and internal rotators, elbow flexors, forearm pronators, wrist and finger flexors, pelvic retractors, hip adductors, extensors, and internal rotators, knee extensors, plantar flexors, and invertors. Abnormal processing of the sensory (afferent) input reaching the spinal cord level and defect in inhibitory modulation from higher cortical centers and spinal interneuron pathways are the possible mechanisms for spasticity in many of these patients. In addition to the hindrance created to voluntary movements, the spastic muscles can cause typical posturing of the affected limb(s), painful spasms, degenerative changes, and fixed contractures. Lack of phasic activity of these spastic muscles also causes impairment in the automatic postural adjustments required for normal transitions and mobility, translating to balance impairments and increased risk for falls. Therefore, bedside examination of tone is essential to determine the presence of flaccidity, hypotonicity, or spasticity. If clinical examination reveals the presence of spasticity, the severity of spasticity can be graded based on the resistance offered to the passive stretch. In the acute stage, the severity of the spasticity is graded by an ordinal scale named the modified Ashworth scale (Table 3.10). In subacute and chronic stages, to differentiate spasticity from soft tissue changes (possible tightness and contracture,) the modified Tardieu scale is the most appropriate.

The use of a standard or electronic goniometer can provide the passive ROM reading of joints as flexibility can be affected by tonal abnormalities of the muscles and soft tissue changes. Testing the active ROM may be practically less important, especially when the spasticity is profound, and the influence of basic limb synergies on isolated voluntary movements is considerable. Regarding the starting position opted, the released postural reflexes like tonic neck and tonic labyrinthine reflexes may cause inconsistent active ROM measurement findings. Contractures can develop as a result of poorly managed or unmanaged spasticity, and further when it

Table 3.10 Grades of modified Ashworth scale

Grade	Features
0	No increase in muscle tone
1	Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of the range of motion when the affected part(s) is moved in flexion or extension
1+	Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the ROM
2	More marked increase in muscle tone through most of the ROM but affected part(s) easily moved
3	Considerable increase in muscle tone, passive movement difficult
4	Affected part(s) rigid in flexion or extension

Courtesy Professor Richard Bohannon

progresses, the edema and pain can worsen the mobility of the joint(s). For subacute and chronic stroke patients, the evaluation should include examination for tightness and contractures, especially the shoulder adductors, elbow flexors, wrist and finger flexors, forearm pronators, hip adductors, knee flexors, and plantar flexors.

Patients may exhibit difficulty in performing purposeful movements in the absence of sensory impairments or motor weakness. Motor praxis, the ability to plan and execute coordinated movement, is often impaired or absent in premotor frontal cortex lesions of right or left hemispheres, corpus callosal lesions, and injuries of the left inferior parietal lobe. Generally, apraxia is more prevalent in the left hemisphere lesion as compared to the right hemisphere lesion. Apraxia can be evident when the stroke patient attempts to perform self-care activities, and these patients may not remember how to hold and use a comb or a toothbrush or don a piece of cloth. Some of these patients may perform the functional movements spontaneously but may fail to perform on command or remember the steps or sequence of movements necessary to achieve the goal. Types, locations, and features of apraxia are listed in Table 3.11.

A considerable number of stroke patients will have paresis of the affected side, which is a major factor for motor function impairment, activity limitation, and participation restriction. The force necessary for initiating and controlling movement is affected, and increased effort and fatigability are the frequent complaints of those patients demonstrating weakness. The degree of muscular weakness is related to the anatomical location of the lesion and the severity of the lesion. It can range from total inability to produce any contraction, as seen in hemiplegia, to a reduction in-force production, as seen in hemiparesis. Most stroke patients will not be able to generate normal levels of muscular force, tension, or torque necessary to initiate and control functional movements and posture. Maintenance of a constant level of force production to control extremity movements can also be difficult. Generally, weakness is more profound in the distal aspect compared to proximal. The normal phasic activity of the proximal and distal muscles is replaced by tonic activity. Changes can also be noticed in the muscle fibers characterized by selective loss of Type II muscle fibers associated with a percentage-wise increase in the number of Type I muscle

Table 3.11 Details regarding types, locations, and features of apraxia

Type	Location	Features
Ideomotor apraxia	Damage to parietal association areas; lesions of intrahemispheric white matter fibers interconnecting parietal and frontal areas; less frequently, lesions of the premotor and prefrontal cortices and supplementary motor area	On verbal instruction, unable to perform complex commands like a salute, snap the fingers, and wave goodbye usually with the involved extremity; unable to pantomime how to use common objects like hammer, toothbrush, and comb or how to kick or throw a ball; may substitute a hand or finger for the imagined object; unable to carry out the act on command but may imitate it
Ideational apraxia	Damage to the left posterior temporoparietal junction	Able to carry out individual components of a complex motor task but unable to perform the entire sequence properly; while attempting to sequence, the patient omits steps or gets the steps out of order; inability to plan the series of steps; for instance, trying to put the car in drive before starting the engine or sealing an envelope before inserting the letter
Buccofacial apraxia	Usually, affecting the infero-anterior portion of the left supramarginal gyrus or the infero-posterior portion of the left posterior central gyrus	On request, unable to execute complex motor acts involving the lips, mouth, and face; unable to execute activities like whistling, pursing the lips, sticking out the tongue, pretending to blow out a match stick, or demonstrate a flying kiss
Constructional apraxia	Most commonly associated with lesions in the parietal-occipital lobes	Impaired visuospatial skills causing inability to copy geometric forms of any complexity; able to draw individual shapes with inability to draw more complex geometric figure: for instance, able to draw a square but not a cube; inability to comprehend spatial relationships; often associated with hemineglect
Dressing apraxia	Usually right parietal lobe lesions	Inability to don or doff clothing correctly; inability to manipulate the clothing in space and understand its 3D relationships; often associated with hemineglect
Limb kinetic apraxia	Mild lesions involving the corticospinal tract that are not severe enough to cause detectable weakness	Difficulty with fine motor control in the absence of obvious weakness; characterized by difficulty to type on the computer or tying shoelaces; not a true apraxia due to damage of the primary motor pathway
Gait or Bruns apraxia	Bilateral frontal lobe disorders	Inability to initiate the process of walking, despite the power and coordination of the legs being normal; characterized by broad-based gait, short stride, “magnetic” or shuffling type gait, freezing, falls or tendency to fall and en bloc turns

Table 3.11 (continued)

Type	Location	Features
Apraxia of speech	Left premotor and supplementary motor areas	Patients have forgotten how to make the sounds of speech; no weakness of the vocal tract; prosody may be impaired and speech may be stuttering; speech pattern may change and sounds like a foreign accent; may be able to repeat short common words but fails to repeat polysyllabic words: for instance, if asked to repeat "potatoes," he or she may repeat it as "tapotos or posatos"; grammar and syntax intact

fibers. In addition to the above, a reduced number of functional motor units, abnormal recruitment of motor units, and decreased firing rates account for lesser force production and difficulty in initiating rapid voluntary movements. Though muscle strength evaluation is an integral component of neurological examination, the presence of spasticity, hyperreflexia, released postural reflexes, and dominant basic limb synergies, and an inability to produce isolated joint movement will pose problems in executing the traditional manual muscle test (MMT) for many of the stroke patients. Till the motor recovery stage 6, when the isolation of joints is not possible, it is preferable to use functional strength testing instead of MMT. Activities like the 30 sec sit-to-stand test; Timed Up and Go (TUG) test; observing the contribution of paretic upper extremity while bridging, hitching, or hiking the pelvis; and swinging of the paretic upper limb while walking are ways of assessing the functional strength of the paretic upper limb and lower limb muscles. Beyond the motor recovery stage 5, when isolations are possible, traditional MMT and objective tools like the handheld dynamometer and the computerized isokinetic dynamometer are more appropriate for grading or quantitatively measuring the muscle strength.

Postural impairments in alignment, symmetry, and stability and impairments in dynamic balance control are common issues seen in stroke patients. Poor reactive postural control to destabilizing external forces and reduced proactive or anticipatory postural control during self-initiated movements predispose these patients to lose balance. During changing tasks and environmental demands, disruptions in the central sensorimotor processing further contribute to ineffective recruitment of postural strategies and inappropriate postural movements. Errors in timing and sequencing of muscle activity, abnormal coactivation of muscles, and delays in initiating motor activity will result in disorganized postural reactions. Stroke patients will typically demonstrate uneven weight-bearing, increased postural sway, and a tendency to fall toward the affected side. The routine examination should cover both static and dynamic balance control in sitting and standing. The usual postural deviations seen among stroke patients are presented in Table 3.12. Asking the patient to reach while sitting or standing will provide information about the limits of stability.

Table 3.12 Presenting the usual postural deviations among stroke patients

Part of the body	Postural deviations
Head and neck	Forward head posturing; lateral flexion of the head with rotation away from the affected side
Trunk	Flattened lumbar curve with an exaggerated thoracic curve (round back posture); lateral flexion with trunk shortening on the affected side
Pectoral girdle	Shoulders are of unequal height and affected shoulder depressed; if the upper extremity is more involved, presence of humeral subluxation with scapula facing downwards and adducted; presence of scapular winging
Upper limb	Affected side shoulder held in flexion, adduction, and internal rotation, elbow in flexion, forearm in pronation, and wrist and fingers in flexion (typical attitude of the hemiplegic upper limb)
Pelvic girdle	Asymmetrical weight-bearing; majority of the weight borne by the unaffected side; fear or reluctance to shift body weight toward the affected side; sacral sitting with posterior pelvic tilt; affected side pelvis retracted and elevated during standing
Lower limb	While sitting, the affected side hip has a tendency to be held in abduction and external rotation with knee in flexion; while standing, the affected side hip is held in extension, adduction, and internal rotation, with knee in extension, and ankle and toes in plantar flexion; unequal weight-bearing on feet and more weight borne by the unaffected side

The therapist should also note the alignment of body segments and trunk control while performing the weight shifts. Berg Balance Scale (BBS), Brunel Balance Assessment (BBA), Tinetti Performance-Oriented Mobility Assessment (Tinetti-POMA), Function in Sitting Test (FIST), Functional Reach Test (FRT), Lateral Reach Test (LRT), and TUG test are some of the qualitative or quantitative scales available to measure the static and dynamic balance of stroke patients. Trunk Control Test (TCT), Trunk Impairment Scale (TIS), and Postural Assessment Scale for Stroke (PASS) are tests or scales developed to examine the postural abilities of stroke patients. The components, grading or scoring methods, procedure, and psychometric properties for these tools are not within the scope of this book. Tables 3.13 and 3.14 present the details of the BBS and Tinetti-POMA scale, respectively.

Pusher syndrome, also known as lateropulsion or contraversive pushing, common after right hemisphere damage, due to altered perception of the body's orientation with respect to gravity, is not uncommon among stroke patients. Typically, the patients with pusher behavior use the stronger extremities of the non-paretic side to push themselves toward their hemiparetic side. These patients have a postural preference toward the affected side, and any attempt to transfer weight over to the non-paretic side or return the body to a neutral or midline position will be strongly resisted. Depending on the severity, the syndrome can substantially hamper maintenance of lying, sitting, or standing postures and transitions. While standing, the strong pushing behavior creates significant postural instability, and these patients demonstrate no fear when active pushing is inducing instability. During ambulation, in addition to the lateropulsion, the affected side lower extremity may scissor, and the cane or walker may pose added trouble than support. Functional skills are

Table 3.13 Berg Balance Scale

Name: _____ Date: _____
Location: _____ Rater: _____
Item Description Score (0–4)
1. Sitting to standing _____
2. Standing unsupported _____
3. Sitting unsupported _____
4. Standing to sitting _____
5. Transfers _____
6. Standing with eyes closed _____
7. Standing with feet together _____
8. Reaching forward with outstretched arm _____
9. Retrieving object from floor _____
10.Turning to look behind _____
11.Turning 360 degrees _____
12.Placing alternate foot on stool _____
13.Standing with one foot in front _____
14.Standing on one foot _____
Total _____

General Instructions

Please document each task and/or give instructions as written. When scoring, please record the lowest response category that applies for each item. In most items, the subject is asked to maintain a given position for a specific time. Progressively, more points are deducted if:

- The time or distance requirements are not met.
- The subject's performance warrants supervision.
- The subject touches an external support or receives assistance from the examiner.

Subject should understand that they must maintain their balance while attempting the tasks. The choices of which leg to stand on or how far to reach are left to the subject. Poor judgment will adversely influence the performance and the scoring. Equipment required for testing is a stopwatch or watch with a second hand, and a ruler or other indicator of 2, 5, and 10 inches. Chairs used during testing should be a reasonable height. Either a step or a stool of average step height may be used for item # 12.

(continued)

Table 3.13 (continued)

1. Sitting to standing
Instructions: Please stand up. Try not to use your hand for support.
( ) 4 Able to stand without using hands and stabilize independently
( ) 3 Able to stand independently using hands
( ) 2 Able to stand using hands after several tries
( ) 1 Needs minimal aid to stand or stabilize
( ) 0 Needs moderate or maximal assist to stand

2. Standing unsupported
Instructions: Please stand for two minutes without holding on.
( ) 4 Able to stand safely for 2 minutes
( ) 3 Able to stand 2 minutes with supervision
( ) 2 Able to stand 30 seconds unsupported
( ) 1 Needs several tries to stand 30 seconds unsupported
( ) 0 Unable to stand 30 seconds unsupported
If a subject is able to stand 2 minutes unsupported, score full points for sitting unsupported. Proceed to item #4

3. Sitting with back unsupported but feet supported on floor or on a stool
Instructions: Please sit with arms folded for 2 minutes.
( ) 4 Able to sit safely and securely for 2 minutes
( ) 3 Able to sit 2 minutes under supervision
( ) 2 Able to able to sit 30 seconds
( ) 1 Able to sit 10 seconds
( ) 0 Unable to sit without support 10 seconds

4. Standing to sitting
Instructions: Please sit down.
( ) 4 Sits safely with minimal use of hands
( ) 3 Controls descent by using hands
( ) 2 Uses back of legs against chair to control descent
( ) 1 Sits independently but has uncontrolled descent
( ) 0 Needs assistance to sit

( ) 4 Can reach forward confidently 25 cm (10 inches)
( ) 3 can reach forward 12 cm (5 inches)
( ) 2 can reach forward 5 cm (2 inches)
( ) 1 reaches forward but needs supervision
( ) 0 loses balance while trying/requires external support

9. Pick Up Object from the Floor from a standing position
Instructions: Pick up the shoe/slipper, which is placed in front of your feet.
( ) 4 Able to pick up slipper safely and easily
( ) 3 Able to pick up slipper but needs supervision
( ) 2 Unable to pick up but reaches 2–5 cm(1–2 inches)
From slipper and keeps balance independently
( ) 1 Unable to pick up and needs supervision while trying
( ) 0 Unable to try/needs assist to keep from losing
Balance or falling

10. Turning to look behind over left and right shoulders while standing
Instructions: Turn to look directly behind you over toward the left shoulder. Repeat to the right. Examiner may pick an object to look at directly behind the subject to encourage a better twist turn.
( ) 4 Looks behind from both sides and weight shifts well
( ) 3 Looks behind one side only other side shows less weight shift
( ) 2 Turns sideways only but maintains balance
( ) 1 Needs supervision when turning
( ) 0 Needs assist to keep from losing balance or falling

11. Turn 360 degrees
Instructions: Turn completely around in a full circle. Pause. Then turn a full circle in the other direction.
( ) 4 Able to turn 360 degrees safely in 4 seconds or less
( ) 3 Able to turn 360 degrees safely one side only 4 seconds or less
( ) 2 Able to turn 360 degrees safely but slowly
( ) 1 Needs close supervision or verbal cuing
( ) 0 Needs assistance while turning

5. Transfers
Instructions: Arrange chair(s) for pivot transfer. Ask subject to transfer one way toward a seat with armrests and one way toward a seat without armrests. You may use two chairs (one with and one without armrests) or a bed and a chair.
( ) 4 Able to transfer safely with minor use of hands
( ) 3 Able to transfer safely definite need of hands
( ) 2 Able to transfer with verbal cuing and/or supervision
( ) 1 Needs one person to assist
( ) 0 Needs two people to assist or supervise to be safe

6. Standing unsupported with eyes closed
Instructions: Please close your eyes and stand still for 10 seconds.
( ) 4 Able to stand 10 seconds safely
( ) 3 Able to stand 10 seconds with supervision
( ) 2 Able to stand 3 seconds
( ) 1 Unable to keep eyes closed 3 seconds but stays safely
( ) 0 Needs help to keep from falling

7. Standing unsupported with feet together
Instructions: Place your feet together and stand without holding on.
( ) 4 able to place feet together independently and stand 1 minute safely
( ) 3 Able to place feet together independently and stand 1 minute with supervision
( ) 2 Able to place feet together independently but unable to hold for 30 seconds
( ) 1 Needs help to attain position but able to stand 15 seconds feet together
( ) 0 Needs help to attain position and unable to hold for 15 seconds

8. Reaching forward with outstretched arm while standing
Instructions: Lift arm to 90 degrees. Stretch out your fingers and reach forward as far as you can. (Examiner places a ruler at the end of fingertips when arm is at 90 degrees. Fingers should not touch the ruler while reaching forward. The recorded measure is the distance forward that the fingers reach, while the subject is in the most forward lean position. When possible, ask subject to use both arms when reaching to avoid rotation of the trunk.)

12. Place alternate foot on step or stool while standing unsupported
Instructions: Place each foot alternately on the step/stool. Continue until each foot has touched the step/stool four times.
( ) 4 Able to stand independently and safely and complete 8 steps in 20 seconds
( ) 3 Able to stand independently and complete 8 steps in > 20 seconds
( ) 2 Able to complete 4 steps without aid with supervision
( ) 1 Able to complete >2 steps needs minimal assistance
( ) 0 Needs assistance to keep from falling/unable to try

13. Standing unsupported one foot in front
Instructions: (Demonstrate to subject.) Place one foot directly in front of the other. If you feel that you cannot place your foot directly in front, try to step far enough ahead that the heel of your forward foot is ahead of the toes of the other foot. (To score 3 points, the length of the step should exceed the length of the other foot and the width of the stance should approximate the subject's normal stride width.)
( ) 4 Able to place foot tandem independently and hold 30 seconds
( ) 3 Able to place foot ahead independently and hold 30 seconds
( ) 2 Able to take small step independently and hold 30 seconds
( ) 1 Needs help to step but can hold 15 seconds
( ) 0 Loses balance while stepping or standing

14. Standing on one leg
Instructions: Stand on one leg as long as you can without holding on.
( ) 4 Able to lift leg independently and hold >10 seconds
( ) 3 Able to lift leg independently and hold 5–10 seconds
( ) 2 Able to lift leg independently and hold ≥3 seconds
( ) 1 Tries to lift leg unable to hold 3 seconds but remains standing independently
( ) 0 Unable to try of needs assist to prevent fall

( ) **Total Score** (maximum = 56)

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Table 3.14 Tinetti performance-oriented mobility assessment scale

Tinetti Performance-Oriented Mobility Assessment (POMA) – Balance Tests Initial instructions: Subject is seated in hard, armless chair. The following maneuvers are tested		
1.	**Sitting balance**	Leans or slides in chair =0 Steady, safe =1 _____
2.	**Arises**	Unable without help =0 Able, uses arms to help =1 Able without using arms =2 _____
3.	**Attempts to arise**	Unable without help =0 Able, requires >1 attempt =1 Able to rise, 1 attempt =2 _____
4.	**Immediate standing balance** (first 5 seconds)	Unsteady (swaggers, moves feet, trunk sway) =0 Steady but uses walker or other support =1 Steady without walker or other support =2 _____
5.	**Standing balance**	Unsteady =0 Steady but wide stance(medial heals >4 inches apart) and uses cane or other support =1 Narrow stance without support =2 _____
6.	**Nudged** (subject at maximum position with feet as close together as possible, examiner pushes lightly on subject's sternum with palm of hand 3 times)	Begins to fall =0 Staggers, grabs, catches self =1 Steady =2 _____
7.	**Eyes closed** (at maximum position of item 6)	Unsteady =0 Steady =1 _____
8.	**Turing 360 degrees**	Discontinuous steps =0 Continuous steps =1 _____ Unsteady (grabs, staggers) =0 Steady =1 _____
9.	**Sitting down**	Unsafe (misjudged distance, falls into chair) =0 Uses arms or not a smooth motion =1 Safe, smooth motion =2 _____
Balance score:		_____/16
Tinetti performance-oriented mobility assessment (POMA) – gait tests Initial instructions: Subject stands with examiner, walks down hallway or across room, first at "usual" pace, then back at "rapid, but safe" pace (using usual walking aids)		
10.	**Initiation of gait** (immediately after told to "go")	Any hesitancy or multiple attempts to start =0 No hesitancy =1 _____
11.	**Step length and height**	Right swing foot Does not pass left stance foot with step =0 Passes left stance foot =1 _____ Right foot does not clear floor completely with step =0 Right foot completely clears floor =1 _____ Left swing foot Does not pass right stance foot with step =0 Passes right stance foot =1 _____ Left foot does not clear floor completely with step =0 Left foot completely clears floor =1 _____

Table 3.14 (continued)

Tinetti Performance-Oriented Mobility Assessment (POMA) – Balance Tests Initial instructions: Subject is seated in hard, armless chair. The following maneuvers are tested		
12.	**Step symmetry**	Right and left step length not equal (estimate) =0 Right and left step length appear equal =1 _____
13.	**Step continuity**	Stopping or discontinuity between steps =0 Steps appear continuous =1 _____
14.	**Path** (estimated in relation to floor tiles, 12-inch diameter; observe excursion of 1 foot over about 10 ft. of the course)	Marked deviation =0 Mild/moderate deviation or uses walking aid =1 Straight without walking aid =2 _____
15.	**Trunk**	Marked sway or uses walking aid =0 No sway but flexion of knees or back or spreads arms out while walking =1 No sway, no flexion, no use of arms, and no use of walking aid =2 _____
16.	**Walking stance**	Heels apart =0 Heels almost touching while walking =1 _____
Gait score = **_____/12**		
Total score (gait + balance) =		**_____/28**

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significantly impaired, and patients are vulnerable to repeated falls toward the affected side. The disorder typically is associated with unilateral lesions of the left or the right posterolateral thalamus. Early identification of lateropulsion helps the physiotherapists to plan and guide the therapeutic strategies for managing this unique motor dysfunction. Though the clinical assessment scales, Burke Lateropulsion Scale (BLS) and Scale for Contraversive Pushing (SCP), are few of the outcome measures available for the Pusher syndrome, currently there are no gold standard, reliable, and valid outcome measures for clinicians and researchers to consistently quantify the degree and presence of contraversive pushing.

Though the majority of stroke patients learn to walk independently by 6 months post-stroke, gait abnormalities tend to persist throughout the chronic stages of the disease. Primary muscle weakness, disinhibited and hyperexcitable brainstem descending pathways (reticulospinal and vestibulospinal tracts), and influence of spastic synergistic activation organized into fewer motor synergies are the plausible reasons for the impaired postural stability and mobility. Gait impairments seen among stroke survivors are the mechanical consequences of interactions of weakness, spasticity, and spastic synergistic activations of the trunk, pelvis, and lower limb muscles. During the stance phase of gait, the hip and knee extension is generated by the synergistic activation of the spastic muscles, which prevents the flexion of the hip and knee for foot clearance. To overcome these impairments, during the

swing phase, the patients will typically hike the pelvis and circumduct the affected lower limb (circumduction gait) for foot clearance. Depending on the severity of weakness and spasticity and the extent of spastic synergistic activations, a wide spectrum of gait impairments can be clinically observed.

In routine clinical practice, to identify the gait deviations, the observational analysis should cover the movements occurring at the trunk, pelvis, hip, knee, ankle, and foot during the gait in different planes of motion. Gait assessment should address the stance phase control of the involved lower limb during the initial weight acceptance, midstance, and weight advancement on the involved limb. Similarly, the assessment should address knee and foot control during the swing phase for the toe clearance and position of the foot. The common gait deviations with respect to body part or location and the possible reasons are summarized in Table 3.15. Quantitative analysis of the distance, time, cadence, velocity, and step and stride length and

Table 3.15 Lists of common gait deviations with respect to body parts and the possible reasons

Location	Deviation	Possible reasons
Trunk	Forward trunk leaning during stance	Weak hip extensors/flexion contracture
Pelvis	Retraction	Weak abdominals and increased tone in extensor synergy muscles of lower extremity
	Hiking	Inadequate hip and knee flexion; increased tone in the trunk and lower extremity extensor muscles
Hip	Circumduction	Increased extensor tone, weakness of the hip and knee flexors, increased plantar flexion in the ankle or foot drop
	Inadequate hip flexion during swing phase	Increased tone in hip and knee extensors, weakness or flaccidity of lower limb flexor muscles, poor proprioception
	Trendelenburg limp	Weakness of hip abductors
	Tendency for affected leg scissoring	Spasticity of hip adductors
	Exaggerated hip-knee-ankle flexion during swing phase seen in some stroke patients	Use of strong flexor synergy, spastic hamstrings
Knee	Decreased knee flexion during swing	Increased lower extremity extensor (mainly quadriceps) tone, weak hip flexion
	Excessive flexion during stance	Weak hip and knee extensors, poor proprioception, flexion contracture
	Hyperextension during stance	Severe spasticity in quadriceps, hip retraction, plantar flexion contracture, impaired proprioception; knee extensors weakness and compensatory locking of knee in hyperextension
Ankle	Equinus gait and presence of foot drop	Spasticity or contractures of gastrocnemius and soleus; weakness of dorsiflexors
Foot and toes	Inversion of the subtalar joint	Hyperactive or spastic tibialis anterior and posterior muscles, weak peroneal muscles
	Clawed toes and hammer toes	Toe flexors spasticity; weak toe extensors

kinematic and kinetic analysis of various joints in different planes of motion using various instruments including GAITRite®, 2D or 3D video motion analyzers, optical motion analyzers, electrogoniometers, EMG sensors, and force plates have helped researchers to analyze gait deviations among stroke patients. In addition to the above, several mobility and gait assessment tools are in use for both clinical and research fields including Dynamic Gait Index (DGI), Functional Ambulation Profile (FAP) Community Balance and Mobility Scale (CB & M), Functional Gait Assessment (FGA), Wisconsin Gait Scale (WGS), and Modified Gait Abnormality Rating Scale (GARS-M).

Stroke patients spending more time in bed, diminished consciousness levels, severe neurological deficits, and very elderly patients with delicate and thinner skin are more susceptible to develop pressure ulcers. Spasticity and contractures contributing to increased friction, faulty transfer techniques contributing to sheer force, and bowel or bladder incontinence causing maceration can be causative factors for pressure sores. Studies have shown that poor nutrition and pressure sores can increase the morbidity and mortality of stroke patients. Poor nutrition can retard wound healing due to the reduction of fibroblasts, angiogenesis, collagen synthesis, granulation tissue formation, and tissue remodeling. Periodic use of the Braden scale, a scale to predict bed sores, can prevent or minimize the possibilities of hospitalized stroke patients developing pressure ulcers. Inculcate routine inspection of pressure-prone areas among nurses and caregivers for those patients who are vulnerable to developing pressure sores.

For those stroke survivors with associated cardiovascular disease, a supervised exercise tolerance test is advisable during the early phase of rehabilitation. Standard methods of monitoring the vital signs, electrocardiogram (ECG) monitoring for any ECG changes, use of a pulse oximeter for oxygen saturation, and use of Borg's Rating of Perceived Exertion (RPE) can be the measures to assess the exercise tolerance. The Borg's RPE is depicted in Table 3.16. Standard cycle ergometry, semirecumbent cycle ergometry, and treadmill walking with or without harnesses are certain modes used for testing. The test protocols should be individualized, submaximal, with intermittent rest if required, and gradually progressed in intensity. The presence of dysrhythmias, systolic blood pressure more than 200 mm Hg, diastolic blood pressure more than 120 mm Hg, ST-segment elevation or depression of $\geq$2 mm, and severe fatigue are considered to be the clinical endpoints of testing. For independent ambulatory stroke patients with or without walking aid, 2- or 6-minute walk test can measure the walking endurance. The therapist should note the total distance covered, the number of rest periods taken, and any symptoms reported while walking or at rest.

Most stroke survivors experience difficulties with self-care activities and mobility tasks, and some may experience the inability to perform activities like feeding, bathing, bed mobility, sitting up, and walking. These functional limitations are the result of sensorimotor dysfunctions caused by the stroke. Many functional outcome measurement tools are used to objectively measure the functional gains and abilities of the patient during the phases of rehabilitation and post-discharge from the hospital. The outcome measures used for functional abilities help to determine the impact

Table 3.16 Borg's rating of perceived exertion scale

Borg RPE Scale®	
Use this scale to tell how strenuous and tiring the work feels to you. The exertion is mainly felt as fatigue in your muscles and as breathlessness or possibly aches. When the exercise is hard it also becomes difficult to talk. It is your own feeling of exertion that is important. Don't underestimate it, but don't overestimate it either. For common exercise, such as cycling, running or walking, 11-15 is a good level. For strength and high-intensity interval training (HIIT), 15-19 is good. If you are sick follow your doctor's advice. Look at the scale and the descriptions and then choose a number. Use whatever numbers you want, even numbers between the descriptions.	
6 **No exertion at all**	No muscle fatigue, breathlessness or difficulty in breathing.
7 **Extremely light** 8	Very, very light.
9 **Very light**	Like walking slowly for a short while. Very easy to talk.
10	
11 **Light**	Like a light exercise at your own pace.
12 **Moderate**	
13 **Somewhat hard**	Fairly strenuous and breathless. Not so easy to talk.
14	
15 **Hard**	Heavy and strenuous. An upper limit for fitness training, as when running or walking fast.
16	
17 **Very hard**	Very strenuous. You are very tired and breathless. Very difficult to talk.
18	
19 **Extremely hard**	The most strenuous effort you have ever experienced.
20 **Maximal exertion**	Maximal heaviness.
	Borg RPE Scale© Ratings (R) of Perceived (P) Exertion (E). © Gunnar Borg, 1970, 1998, 2017 English

The Borg RPE scale (R) ((C) Gunnar Borg, 1970, 1998, 2017). Scale printed with permission.

of impairments and activity limitations, decide the short- and long-term goals and objectives of treatment, plan and monitor the effectiveness of treatment, and predict community reintegration and return to work. Functional mobility skills including bed mobility, transfers, locomotion, basic and instrumental ADL, personal care and hygiene, and communications are some of the components that are included in most of the functional scales. The Barthel Index (BI), Functional Independence Measure (FIM), Fugl-Meyer Assessment (FMA), Wolf Motor Function Test (WMFT), Action Research Arm Test (ARAT), Stroke Rehabilitation Assessment of Movement (STREAM), and Stroke Impact Scale (SIS) are some of the common functional assessment tools used in clinical and research areas. The outcome scales are either impairment-based or are activity limitation or participation restriction based. Most of the tools mentioned above have demonstrated excellent reliability, validity, and sensitivity. Readers should note that there is sufficient literature available dealing exclusively with the components, standard procedures, and psychometric properties of such scales which are not within the scope of this book.

3.11.2 Physiotherapy Treatment

Before going into the details of physiotherapy management, the author would like to specify some valid points. A shotgun approach is not an appropriate method to tackle the sensorimotor dysfunctions of the stroke patient. Instead, an eclectic program that is tailor-made to the individual patient's problems is more meaningful. The therapist must have adequate knowledge, both theoretical and practical, about various therapeutic approaches to provide an eclectic treatment program for stroke patients. Just the way each motor control theory explains a certain number of observations and fails to explain other motor behaviors, neurodevelopment-based and non-neurodevelopment-based approaches (discussed in chap. 2) when delivered as a standalone has provided therapeutic benefits but not for all the motor dysfunctions. The fact as mentioned above has been substantiated by current evidence, which states that none of the therapeutic approaches, as a standalone, is superior or more effective in promoting recovery of extremity function or postural control post-stroke, as compared to the other. Since all these approaches have strengths and weaknesses of their own and collectively have the edge over a standalone, an eclectic approach makes it all the more meaningful to tackle most of the stroke patient's sensorimotor dysfunction issues.

The existing gamut of research with regard to the physiotherapeutic management of stroke patients, if carefully inspected, will reveal several shortcomings including selection criteria, inappropriate outcome measures, and biasing at various levels of the study to prove the superiority of the experimental group over the conventional or control group. Deliberately underperforming conventional or routine therapy group in certain instances and claiming benefits produced by an existing clinically or statistically proven intervention while piggyback riding on it are common

findings in many works. All the above aspects have to be kept back of one's mind before instinctively selecting or following any technique.

Unlike patients with other neurological disorders, stroke patients belong to a diverse group with variable levels of function. Based on the patient's abilities and requirements, the therapists need to carefully select the strategies that have the greatest chance of successfully remediating existing impairments and promoting functional recovery. Pitting a few of the effective techniques of specific approaches will not convert a standalone approach into an eclectic approach. As discussed earlier, a true eclectic model needs sound knowledge, both theoretical and practical, about both the traditional and recent approaches. The author believes that the knowledge earned from each scientific therapeutic approach must serve as a reservoir from which a clinician can wisely choose the necessary tools to customize the eclectic treatment program to suit the specific needs of a patient. Choosing the best treatment methods to address the patient's sensorimotor issues must be the most rational approach when substantial evidence for the effectiveness of any single approach over the others is unavailable. The choice of interventions should also consider other factors, including the stage or phase of stroke, age of the patient, existing comorbidities, potential discharge plans, and social and financial status.

The therapist, based on an accurate examination, should interpret the clinical findings and utilize educational skills and manual handling techniques to retrain movement and solve the problems. The therapist needs to develop the expertise of recognizing correct and incorrect responses to therapeutic strategies so that positive outcomes are encouraged and unwanted results are avoided. In many stroke patients, with a potential for recovery, functional task-specific training should be the mainstay of treatment to regain control of the functional movements. In the early phase of management, the focus should be to improve the motor control and strength of the trunk and proximal girdle muscles, emphasizing more on the affected side, through specific treatment strategies. With respect to gaining trunk control, the author would like to mention the relevance of a textbook titled *Right in the Middle*, a comprehensive work by an eminent physiotherapist, Patricia M. Davies. According to Davies, gaining control over the hemiplegic arm and leg is dependent upon the patient's ability to control the trunk. The trunk and the girdles act as stable anchorages for the extremity muscles, and the lack of trunk control predisposes to the development of primitive and stereotypic mass patterns. She advised a series of exercises explicitly emphasizing the trunk like "bridging and tentacle" exercises on stable and unstable surfaces to activate the core muscles, specifically the abdominal obliques. For a better understanding of the selective trunk and extremity control, the author advises the readers to refer to the textbook mentioned above.

The aim of physiotherapy management following a stroke is to maximize the return of functional movement and independence and to minimize the possibilities of secondary complications. Emphasizing early functional independence improvement provides an important source of motivation for both the patient and the family members. Though the intervention commences in the acute stage following admission to the hospital, active participation in the relearning of mobility and independence generally takes place during the subacute and the chronic stages post-stroke.

Despite the common patterns, the acute, the subacute, and the chronic stages are rarely distinct, frequently tend to overlap, and do not follow the same time frame for every patient. In the acute stage, the stroke patients' levels of consciousness and neurological deficits can vary, ranging from unconscious to fully conscious, intubated or on oxygen support to room air-breathing, and complete communication breakdown to sound communication and from total paralysis to normal muscle strength and control. Depending on the presentation, during the acute stage, the treatment strategies should ensure normal respiratory function, general mobility, and facilitation of movements of the paretic side, encourage motor relearning, and prevent sequels including DVT, pressure sores, and painful shoulder.

3.11.2.1 Pulmonary Care

Respiratory muscle weakness among acute stroke patients may increase the frequency of chest infections and contribute to weak cough and poor airway clearance. Strategies to improve the strength and endurance of the diaphragm include the application of manual resistance by placing the therapist's hand on the patient's upper abdomen in the semi-recumbent position and then, while inspiring, instructing the patient to lift the weight of the therapist's hand. The therapist can apply a quick stretch to the diaphragm before an active inspiration to facilitate a stronger contraction of the inspiratory muscle. By increasing the manual resistance or changing the patient's starting position, the exercise should be made more challenging to improve the patient's performance. Segmental breathing exercises can also be encouraged to minimize the possibility of atelectasis of the lower lobes of the lungs. Expansion exercises for the lateral lobes should be encouraged by placing the therapist's hands on the patient's lateral lower rib cage, and the patient should be instructed to "breathe in" against the manual pressure. Activities to improve pulmonary function should include the use of incentive spirometers, paper blowing exercise, blow bottle exercise, and inspiratory or expiratory muscle trainers. In addition to the above, general trunk flexibility or mobility exercise may also improve breathing efficiency, especially when there is a presence of lateral chest wall tightness. For those stroke patients who are unarousable following cerebrovascular accidents, routine care should include chest manipulations and physiotherapy to maintain pulmonary hygiene.

3.11.2.2 Bed Positioning

Proper positioning is an important component of physiotherapy during acute stage management, and it is the responsibility of all the members of the interdisciplinary team to ensure that the appropriate position is maintained throughout. Proper positioning minimizes the possibilities of abnormal tonal development, discourages mass movements, stimulates normal motor functioning, improves sensory awareness, enhances oromotor and respiratory functions, and prevents the possibilities of

ROM restrictions, musculoskeletal deformities, and pressure ulcers. The position of the patient should be changed every two hours between lying on the back, the affected side, and the unaffected side. Both the girdles require extra attention and need to be placed in mild protraction, as the rhomboids and gluteus maximus tightness secondary to immobility and tonal abnormality can contribute to retraction of the shoulder and pelvic girdles.

A 1.5-inch thick towel roll spanning from the affected scapula to the pelvis can promote protraction in the supine position. If the thickness of the towel roll is considerably more than the recommended, it can be counterproductive and may encourage postural asymmetry. In supine, keep the shoulder in 10–20° abduction with external rotation, elbow in extension and forearm supinated, and the wrist and fingers in a functional position. Protracted pelvis with mild hip knee flexion (a pillow kept under the knee joint) minimizes the likelihood of extensor synergy dominating the lower limb. The use of an additional pillow for the head and neck in the supine position can promote cervical flexion and worsen forward head posturing and therefore is not recommended.

While lying on the unaffected side, the stroke patient's trunk should be in neutral (neither bent forward nor unduly extended), and with the support of a pillow or two, the affected upper extremity should be in protraction, elbow extended, and forearm in neutral with the wrist and fingers in a neutral or extended position. With the support of a pillow, position the affected lower extremity with the pelvis in protraction, hip and knee in flexion, and ankle in neutral (Fig. 3.25). A similar kind of position is recommended for the affected upper extremity, without pillows, while lying on the affected side. Ensure that the affected shoulder is protracted sufficiently to

Animated photograph of model with permission

Fig. 3.25 Recommended bed posture in side-lying on the unaffected side. Note: The affected upper extremity is kept in protraction, the elbow in extension, and the forearm in neutral with the wrist and fingers in a neutral or extended position

minimize the possibility of impingement and excessive weight falling over the shoulder if directly under. The affected side pelvis should be protracted, with the hip in extension and knee in slight flexion. Since neglect is often a feature of cortical stroke, increased sensory input provided by side-lying position on the affected side reduces the effects of neglect in these stroke patients.

3.11.2.3 Management of Sensory Impairments

Patients with significant sensory impairments may demonstrate impaired or absent spontaneous voluntary movements of the hemiplegic side limbs. Encouraging the patient to use the affected side provides a greater chance of improving the awareness and function of the side limbs. Refusal by the patient to use the affected side accentuates the problems caused by the absence of normal sensorimotor experience and contributes further to the learned nonuse phenomenon. Sensory retraining or sensory stimulation approaches are certain interventions used to address these sensory impairments. Mirror therapy, repetitive functional activity practice augmented by sensory cues, and bimanual movements are examples of sensory retraining approaches. Weight-bearing techniques, manual compression, use of inflatable pressure splints, mobilizations, electrical stimulation, and use of thermal stimulation are examples of sensory stimulation approaches. With the current evidence neither proving nor refuting these techniques, a careful and judicious way of choosing one or other techniques based on the therapist's knowledge and experience may help to improve the sensory impairments in such patients.

The physiotherapist should encourage the stroke patients to weight bear and undergo compression (joint approximation) of the sensory-deficient limbs during the functional training in sitting (Fig. 3.26), standing, or modified plantigrade position. Care has to be taken to keep the joints in proper alignment while performing weight-bearing or joint approximation in various positions, to minimize the possibility of injuries to the affected sensory-deficient limb. Besides, a safety education program has to be instituted for the patient and family members to improve sensory awareness and protection of the affected limbs, mainly while performing transfers and wheelchair mobility.

3.11.2.4 Management of Visual Field Deficit and Unilateral Neglect

Visual field deficits and perceptual impairments can often worsen the poor awareness of the paretic side. Though more prevalent among patients with right hemisphere stroke, hemispatial neglect and anosognosia, two distinguishable and dissociable syndromes which often coexist, can cause total unawareness of the disability or the extent of the problems. Such patients may benefit from strategies that encourage awareness of paretic limbs, use of the environment on the paretic side, and use of the paretic limbs. The use of visual, verbal, and motor cues, promoting active visual scanning movements, encouraging the patient to look at his or her

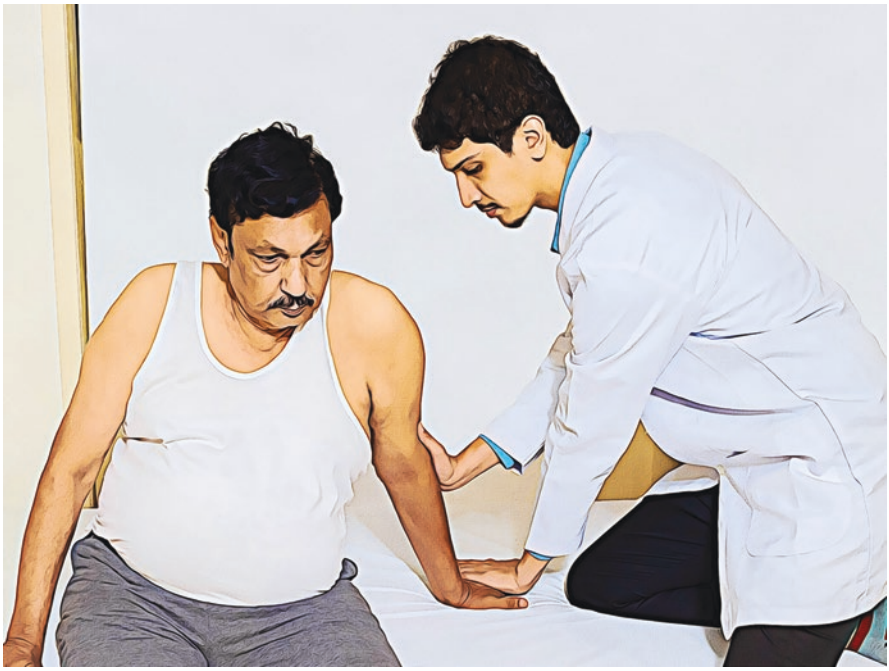


Fig. 3.26 Weight-bearing through the affected upper limb in sitting

paretic limbs, and exteroceptive and proprioceptive stimulation of the affected limbs by brushing, stroking, muscle tapping, and vibration are strategies to overcome poor awareness of the paretic limb. Reach out activities, Proprioceptive Neuromuscular Facilitation (PNF) diagonal patterns and chopping movements crossing the midline, and functional activities that encourage bilateral interaction of the paretic and non-paretic limbs can also be helpful to overcome the poor awareness of the paretic side. Even encouraging family members to address or interact from the paretic side and placing the commonly used objects like mobile phones, spectacles, napkins, and tissue wipes on the paretic side tends to increase the awareness and attention given to that side of the body.

3.11.2.5 Management of Joint Flexibility and Integrity

About two-thirds of stroke patients develop spasticity and may result in muscle tightness and joint stiffness, eventually affecting the mobility and functional abilities. Spasticity, in addition to muscle tightness and joint stiffness, can cause soft tissue pain and antagonist muscle weakness. Considerable evidence is now available, suggesting the beneficial effects of flexibility exercises among stroke survivors. The flexibility exercise program aims to relieve spasticity, improve ROM,

prevent tightness and contractures, and improve motor functions. The flexibility exercise program consists of stretching exercises and joint ROM exercises performed by an external force or self. Often the flexibility exercises are used for warm-up and may be combined with aerobic training and strength training that can help paretic patients improve the ROM. The flexibility exercises are preferably initiated in the early phase of management to maintain joint mobility and integrity. Providing terminal stretch for passive, active, or active-assisted ROMs, without inducing unbearable pain (except the bearable stretch pain), positional strategies to maintain the soft tissue length, and flexibility exercise program performed two to three times daily are a few strategies that can prevent or minimize the potential development of tightness and contracture. The proper positioning of the paretic extremities encourages normal or near-normal joint alignment and minimizes the potential development of typical abnormal attitudes of the limb seen among stroke patients.

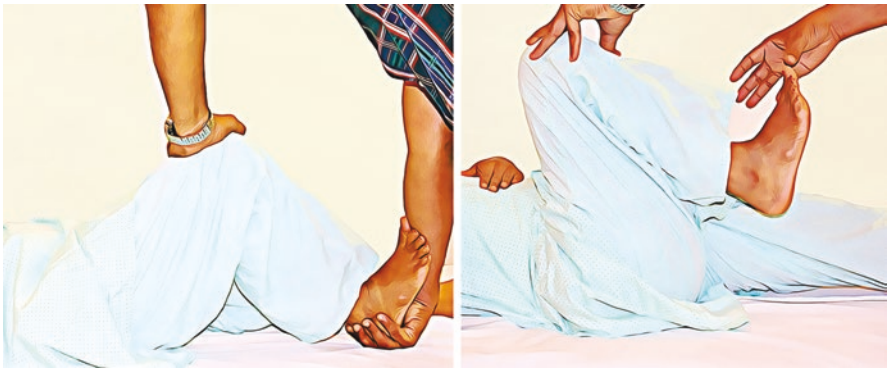
Most stroke patients will have impaired scapulohumeral rhythm due to tonal abnormalities of the scapular muscles and inactivity or poor strength of the muscles in the scapular region. Before shoulder ROM exercises, the passive mobilization of the scapula on the thoracic wall emphasizing upward rotation and protraction will help minimize the potential soft tissue impingement in the subacromial space while performing glenohumeral movements. Gentle traction to the shoulder with external rotation further minimizes the possibility of impingement. The use of overhead pulley, a self-ROM exercise, traditionally practiced in many centers, is unwarranted due to the possibility of impingement during overhead activity. Weight-bearing exercise given in sitting, with paretic shoulder kept in partial abduction and externally rotation, elbow in extension, forearm in supination, and wrist and fingers in extension can relieve the spasticity and improve the soft tissue flexibility, thus minimizing the potential for elbow flexors, forearm pronators, and long flexors developing tightness.

Self-performed ROM exercises of the trunk and the girdles, especially with cradling of the affected arm, well elaborated in chapter "Therapeutic Approaches" under Brunnstrom's approach and Bobath approach, further encourages scapular mobility over the thoracic wall, a prerequisite for activities like bed mobility and functional activities in sitting. In case of inadequate scapulohumeral rhythm, overhead shoulder movements with hands clasped with the shoulder in flexion, elbow in extension, and forearm in supination is contraindicated. Promote normal scapulohumeral rhythm (scapular mobilization techniques) before passive ROM can be commenced. Despite the regular flexibility exercises, a certain number of patients may find it difficult to maintain the joints in proper alignment, particularly when spasticity is considerable. For such patients, the use of a resting splint, which maintains the position of the forearm, wrist, and fingers in the functional position, is recommended.

In the case of paretic lower extremity, many stroke patients may find it challenging to dorsiflex their foot due to spasticity of plantar flexors and/or weakness of the dorsiflexors. The traditional stretching technique for the tight or spastic plantar flexors, prolonged positional stretches using toe or foot wedges, weight-bearing

activities with affected lower extremity maintained in proper alignment, and weight-shifting activities in standing and modified plantigrade position can be beneficial to regain the ankle ROM. Following stretching of the plantar flexors, procedures like passive positioning of the hip and knee in near-complete flexion in the supine lying may encourage patients to voluntarily activate the dorsiflexors and reciprocally inhibit the activity of the spastic plantar flexors (Fig. 3.27).

Similarly, overactivity and/or tightness of the gluteus maximus with spasticity of quadriceps may make it difficult for many stroke patients to flex the hip and knee in the supine position. For such patients, in the crook lying or supine lying position, passive stretching (15–20 sec) of the hip extensors with the knee held in flexion, followed by commands to activate the hip flexors in the inner range, may help to initiate the hip flexors voluntarily. Commands like "do not allow your leg to drop" or "'move your thigh toward the chest" with or without manual assistance or support may encourage the patient to gain control over the hip flexors (Fig. 3.28). The



Animated photographs of model with permission

Fig. 3.27 Passive stretching of the plantar flexors in supine followed by the placement of hip and knee in near-complete flexion to facilitate the voluntary activation of the dorsiflexors

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Fig. 3.28 Passive stretching of hip extensors with the knee held in flexion, followed by commands to encourage activation of hip flexors in the inner range

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Fig. 3.29 Placement of affected side hip in abduction, knee in flexion, and leg off the side of the bed with foot flat on the stool/stepper

technique mentioned above may be repeated certain times to encourage the patient to eccentrically or isometrically contract the hip flexors before he or she can advance to concentric contraction. Following the initial phase of gaining control over the inner range, progress toward gaining control over the middle and outer ranges of motion. Eventually, when the patient gains sufficient control and strength, he or she can be advised to perform hip and knee flexion for tasks like crossing the affected leg over the flexed unaffected leg in a crook lying position.

For those patients who are wheelchair-bound, regular stretching of the hip flexors is advisable. If the extensor synergy dominates, position the affected lower limb of the patient with the hip in abduction, knee bent, leg off the side of the bed, and foot flat on the floor or stool (Fig. 3.29). Such a placement of the paretic lower limb may help to break the strong dominance of extensor synergy, which is typical in many patients, and prevent the possibility of limb scissoring while attempting to stand or walk.

3.11.2.6 Spasticity Management

Since normal joint flexibility and integrity is an amalgamation of independent factors like the normalization of tone, soft tissue extensibility, and the volitional activation of antagonistic muscle groups, the readers can expect a certain degree of overlap in the strategies or techniques utilized for encouraging joint flexibility and integrity and those aimed at mitigating spasticity. Familiarizing themselves with the contents in the preceding sections of the text will ensure a complete understanding of the nuances in the management of tone.

Early mobilization and daily stretching exercises help to maintain the length of spastic muscles and soft tissues. Once the flexibility exercise program has achieved full ROM, positioning the limb in the lengthened position reduces the hyperactivity of the spastic muscles. The use of weight-bearing exercises in kneeling or quadruped positions on a therapeutic mat helps to normalize the muscle tone of the paretic limb. The PNF techniques like rhythmic initiation or rhythmic rotation incorporating axial rotation and the various truncal mobility exercises mentioned in literature

like “right in the middle” can reduce truncal stiffness and improve trunk control. Sustained or prolonged stretching helps to relax the spastic muscles through the mechanisms of autogenic inhibition. Slow vestibular stimulation induced by slow rocking movements can provide relaxation effects and may relieve the hyperactivity of the spastic muscles. It is advisable to teach and encourage the family members to perform the right method of flexibility and stretching exercises to ensure that the flexibility exercise program extends beyond the training given by the therapist.

As discussed previously, facilitating or activating the weak antagonist will also reduce the hyperactivity of the spastic muscles. The use of exteroceptive or proprioceptive techniques like quick stretching, muscle tapping, and brushing over the weak antagonist muscles can facilitate or enhance the action of the same. Such facilitation techniques need to be given only for those muscles which are weak and antagonist to the spastic group and should be discarded once the patient gets voluntary control over those muscles. While attempting to facilitate or voluntarily initiate weak muscles, avoid excessive effort from the patient’s side which can often worsen the tone of the spastic muscles.

Modalities including the application of cold and use of neuromuscular electrical stimulation (NMES), can also help temporarily relieve the spasticity of the muscles. A cold or ice pack applied over the spastic muscle for 10–15 minutes duration tends to slow the nerve conduction and reduce the hyperactivity of the muscle spindle. Electrical stimulation of the weak antagonist muscles may help to reduce the tone of spastic muscles through reciprocal inhibition. However, it is essential to ensure that electrical stimulation does not activate the spastic muscles. In addition to the above, certain monophasic currents like interrupted galvanic currents are not advisable for the stimulation of weak antagonists as they may pose safety concerns due to possibilities of inducing chemical burns. The use of orthotic devices, including resting splints, serial casts, and inflatable pressure splints to maintain the spastic muscle in its lengthened position, can reduce the hypertonia and improve or preserve the passive ROM.

The therapist should discourage the placement of soft or squeeze ball and cloth roll in the patient’s palm, especially when there is a likely chance of spasticity and tightness developing within the wrist and finger flexors. Such strategies may facilitate palmar grasp reflex, encourage imbalance between the flexors and extensors muscle strength, and predispose to a poor functional return of hand, which is typically characterized by difficulty to extend the wrist and open the hand, simultaneously.

3.11.2.7 Shoulder Dysfunction and Pain Management

Subluxation of the glenohumeral joint, spasticity, soft tissue impingement in the subacromial space, rotator cuff tears, adhesive capsulitis, and complex regional pain syndrome are specific reasons for hemiplegic shoulder pain. Flaccidity of the rotator cuff muscles, lack of normal tone and absence of voluntary movements, impaired proprioception, and inactivity of certain muscles like serratus anterior alter the

normal position of the scapula and shoulder biomechanics. In the absence of supporting musculature, glenohumeral movements like forward flexion or abduction with scapula rotated downward and depressed can predispose the humerus to sublux from the glenoid fossa. Mechanical stresses created by the repeated traction and gravitational forces eventually can change a painless subluxation to a painful one.

Even spasticity of certain muscles, like the scapular depressors and pectoralis major along with inactivity of muscles like serratus anterior, can contribute to depression, retraction, and downward rotation of the scapula and predispose to subluxation and restricted ROM. The friction and compression stresses occurring between the humeral head and superior soft tissues while performing the passive or active-assisted ROM in the absence of normal scapulohumeral rhythm can also predispose to shoulder pain. In stroke patients, undue tightness and thickening of the soft tissues and the joint capsule can lead to conditions like adhesive capsulitis. Improper handling techniques, poor positioning of the shoulder joint, improper alignment of upper limb joints during functional weight-bearing exercises, passive ROM with inadequate scapular mobilization, and undue traction of affected upper extremity during transfers can cause microtrauma to the soft tissue structures and lead to shoulder pain. Use of scapular mobilization prior to glenohumeral passive ROM, preventing undue weight falling on the shoulder joint while lying on the involved side, minimizing traction on the shoulder during transfer techniques, taping techniques, NMES of the weak shoulder muscles, and use of slings are some strategies recommended to minimize or prevent the shoulder pain.

Impaired glenohumeral joint biomechanics and trauma to the affected shoulder have been implicated in the development of complex regional pain syndrome post-stroke. Glenohumeral subluxation is a common finding in stroke patients with complex regional pain syndrome. Complex regional pain syndrome, also known as Sudeck's atrophy or shoulder hand syndrome in stroke patients, is characterized by pain; edema; stiffness and limitation in ROM; vasomotor and sudomotor changes; trophic changes in the hair, nails, and skin; and patchy bone demineralization of the affected extremity. Muscle weakness, spasticity, sensorimotor deficits, and coma during the initial days post-stroke are a few clinical factors associated with its development. In the early stage (first stage), the pain is typically intermittent and limited to the shoulder, and later, the pain intensifies and extends to the whole extremity. Due to joint stiffness and limitation, the wrist tends to assume a flexed posture, and while attempting to extend the wrist, the patient may report intense pain and guard against any movement attempts. Pale-pink discoloration of the skin and cold skin are vasomotor changes. The skin can be hypersensitive to touch, pressure, or temperature variations. Pain subsides during the second stage. Dystrophic changes of the muscle, atrophy of the skin, vasospasm, hyperhidrosis, coarse hair and nails, and radiographic evidence of patchy osteoporosis are the characteristics of the second stage. In the third stage, pain and vasomotor changes are rare, and pericapsular fibrosis and articular changes and progressive atrophy of the skin, muscles, and bones are its features. The hand is typically held in a clawed position due to the contracture of muscles, with marked atrophy of the thenar and hypothenar muscles. If identified during the early phase, the signs and symptoms of this disabling

condition are quite reversible. On the contrary, the beneficial effect of interventions is least likely once the condition advances to the third stage.

The therapeutic interventions are based on the clinical examination findings of complex regional pain syndrome. The goals of treatment are to reduce pain, maintain joint mobility, and restore function. Gentle mobilization of the joints, passive ROM exercises, strengthening of the affected extremity muscles, management of edema, and desensitization techniques are the mainstay of care for these patients. Preventative strategies to minimize early joint injury can reduce the likelihood of complex regional pain syndrome. The strategies should include providing support for the affected arm during the flaccid stage, proper positioning and handling of the extremity, preventing traction injury, promoting passive ROM following scapular mobilization, and advising the concerned nursing staff to minimize venous infusions into the hemiplegic hand. A gradual process of desensitization (by applying systematically different sensory stimuli on the skin over the affected area) may help normalize the sensation by resetting the altered central processing in the nervous system. For the restoration of the upper extremity function, promoting active movements of the affected shoulder, strengthening the weak muscles, and edema relieving techniques need to be considered.

Flaccidity or hypotonicity of the shoulder muscles can increase the risk of traction injury to the soft tissues in and around the glenohumeral region. Slings can minimize or prevent soft tissue injuries, relieve pressure on the neurovascular bundle, improve the anatomical alignment of the shoulder, and ease the stress and the gravitational pull on the glenohumeral joint. Conventional triangular sling, Bobath sling, Rolyan humeral cuff sling, and arm pouch sling are some of the shoulder slings used for hemiplegic patients with glenohumeral subluxation. During the initial phase transfers and gait training, the therapist can focus his attention on the patient's posture or trunk control, if the sling is worn to support and safeguard the affected shoulder. However, many of the slings have a modest effect in reducing the subluxation of the glenohumeral joint or improving the shoulder function. Most of the slings can encourage typical upper limb posturing characterized by shoulder adduction and internal rotation, elbow flexion, forearm pronation, and wrist flexion, promote tightness and contracture, discourage active voluntary contraction of shoulder muscles, encourage learned nonuse, increase flexor hypertonicity, and contribute to body schema dysfunction and neglect. The Rolyan humeral cuff sling has an arm cuff on the humerus supported by a "figure of eight" harness. It encourages better symmetry, maintains elbow in extension, provides a certain reduction of subluxation, does not restrict the distal limb functions, and can be worn for a longer period. Such humeral slings are preferred over those conventional slings encouraging shoulder adduction and internal rotation and elbow flexion. Taping techniques for minimizing shoulder subluxation, neuromuscular electrical stimulation of weak rotator cuff muscles (especially supraspinatus) and the middle fibers of the deltoid, use of proper support to the shoulder and elbow while sitting or standing, and use of lateral elbow guard and/or straps while the patient is on a wheelchair are the other alternatives to the use of a sling.

3.11.2.8 Strategies to Improve Voluntary Movement Control

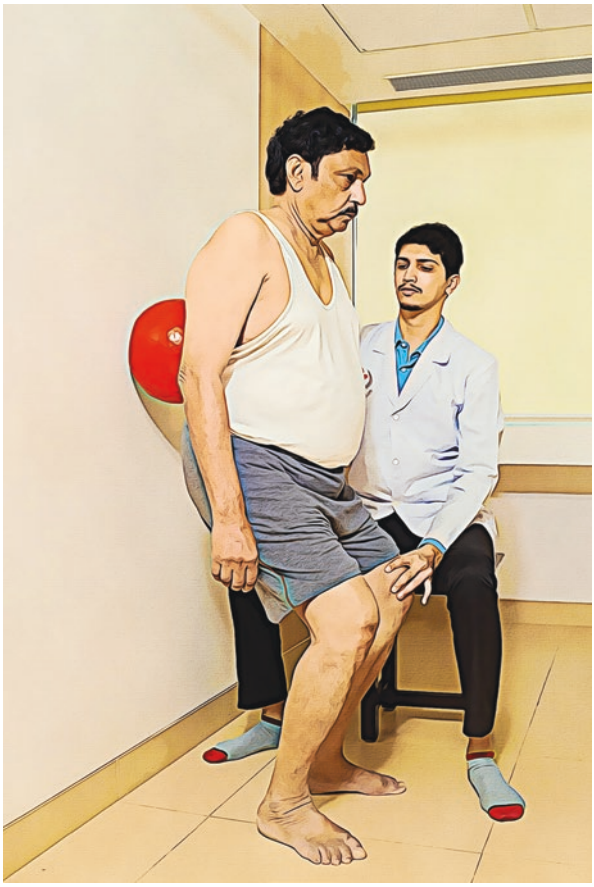
In the initial phase post-stroke, the affected limbs are flaccid or hypotonic. Following the cerebral shock, when the deep tendon reflexes return and patients progressively develop control over the basic limb synergies, activities that promote voluntary movement control, postural control, and functional use of the extremities should be the primary focus of training. The presence of obligatory basic synergy patterns can hinder the patient in re-attaining the fractionated movements of the extremities. Released postural reactions, including associated reactions, can affect the inter-and intra-limb movement control. During the training phase, if the stroke patient can initiate voluntary movements, the focus of therapy should be toward encouraging movement patterns away from the basic synergy and fractionation or dissociation of body segments. Use of reflex inhibiting pattern and key point of control, selection of appropriate postures, adequate stabilization of the body part, and guidance can help the patient to perform movements without inducing excessive tone or abnormal synergy pattern. Advice the patient not to perform movements too quickly or with excessive effort as it may accentuate the hypertonia of the muscles.

For those patients with the inability to initiate movement, techniques or strategies discussed in chapter "Therapeutic Approaches" (such as the facilitatory techniques of Rood's approach and exteroceptive and proprioceptive stimulation and use of postural reflexes as stated in Brunnstrom approach can be an option. In addition to those techniques, stretching of the spastic antagonists prior to the facilitation of agonists or NMES of the weak agonists can be tried. The aim of the treatment should be to gain adequate control over the movement initiated by the patient so that it can be further fractionated or dissociated for functional movements.

The functional movements, including reaching, walking, and climbing, require phasic activity of the muscles, smooth and coordinated actions of muscles in different roles (agonist, antagonist, synergist, and fixator), and variation in the type of contractions (eccentric, isometric, and concentric). Reversal of direction of movement encourages the phasic switching over action of muscles, and practice of tasks utilizing variations of contractions helps in improvising the interaction between agonists and antagonists crucial for normal coordination and function. For instance, practicing partial squats or modified wall-squats (Fig. 3.30) within a limited range with or without the support or assistance of the therapist helps to sequentially contract the hip and knee extensors between eccentric to isometric to concentric. For those stroke patients with considerably weak muscles, to encourage better recruitment of motor units, eccentric and isometric contractions are emphasized before concentric contractions.

3.11.2.9 Strategies to Improve Muscle Strength

Hemiparesis, the most common impairment seen in stroke patients, can persist for months to years, and the weakness can significantly contribute to activity limitations and participation restriction. The inability of muscles to generate adequate



Animated photograph of model and therapist with permission

Fig. 3.30 An illustration of the modified wall-squat

force post-stroke can be due to increased stretch reflex excitability, increased antagonist muscle coactivation, reduced rate of motor unit firing, and alterations in the muscle property (decreased muscle mass and muscle fiber length and changes in pennation angle and tendon length). Recent evidence on progressive resistance strength training of the hemiparetic side muscles has shown improvement in muscle strength and functional performance with no apparent worsening of spasticity or reduction of ROM.

The modalities used for strength training include manual resistance, free weights, elastic bands, and machines. Use of reeducation board, sling suspension therapy, or aquatic exercises is recommended for those patients who have muscle strength less than that required to move against gravity. The structured strength training exercise is advisable for those who demonstrate independent movement in the gravity-resisted plane. Resistance training consisting of 8–12 repetitions, 2–3 sets per treatment session, a minimum of 2–3 days per week performed with free weights, bands, or machines is adequate to improve the strength of the weak muscles of the paretic limbs. Task-oriented progressive resistance strength training has shown a carryover effect in functional activities in addition to improvement in lower limb strength.

Performing task-oriented functional activities or circuit training using body weight or wearing weighted cuffs with a progressive increase in the number of repetitions, complexity, or duration of exercise can improve muscle strength and endurance.

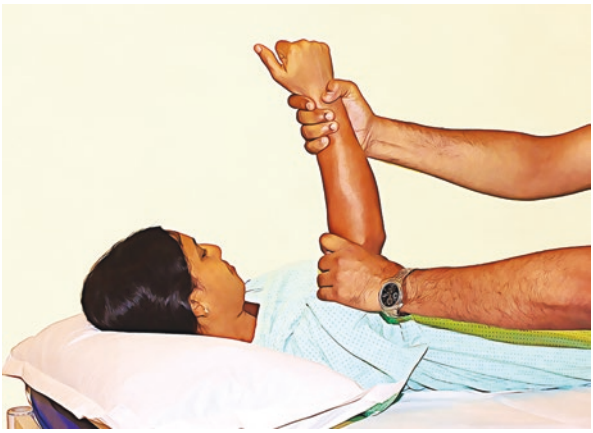
For recent stroke and stroke patients with comorbidities, including ischemic heart disease, valvular disorders, and poorly controlled blood pressure, high-intensity strengthening exercises are contraindicated. Strength training activities, especially isometric exercise, can induce breath-holding that can elevate the blood pressure to dangerous levels and impose excessive cardiac load. Low-intensity exercises (30–50% of 1 repetition maximum [RM]), dynamic exercises performed in sitting position, exhaling during the lifting phase and inhaling during the lowering phase of the strength training, adequate rest periods between the sets, and repeated monitoring of the blood pressure are certain strategies to avoid the abovementioned.

3.11.2.10 Improving Upper Limb Functions

Though sensorimotor impairments and functional issues vary from patient to patient, it tends to be more severe in the middle cerebral artery syndrome and the dense lesions affecting the internal capsule. In such conditions, since the sensorimotor recovery is often limited, compensatory training strategies and environmental modifications in addition to the standard treatment strategies, like early mobilization, flexibility exercise program, and positioning strategies, are preferable to maximize the functions. For those stroke patients with a better scope of motor recovery, the training strategies should focus on promoting fractionation of the movements, reversal of direction of movements, and repetitive task-specific practice.

To enhance proximal stabilization and to counter the effects of excessive flexor hypertonicity and dominance of flexion basic limb synergy, weight shift with weight-bearing on the affected side extended arm while stabilizing the hand on a supporting surface should be encouraged as an early activity. Such joint approximation activities performed while sitting, standing, or modified plantigrade position can help to increase the activity of pectoral girdle stabilizers, and, meanwhile, muscle tapping or exteroceptive stimulation over the triceps muscle belly can facilitate the elbow extensors to gain better control over the elbow joint.

The focus should be on encouraging the activity of the serratus anterior and upper fibers of the trapezius muscles to gain control over the scapular upward rotators. In the supine position, encouraging the patient to push the arm up toward the ceiling with shoulder flexed to 90°and elbow extended can initiate the serratus anterior muscle. The therapist will often have to support the arm in the abovementioned position (Fig. 3.31) to encourage him/her to initiate the movement. Commands like "do not allow me to push your arm downward" or "push your arm upward" just after a quick stretch to the serratus anterior in supine may activate the muscle. Once the patient can initiate the movement, manual resistance can be offered to further build the strength of the muscle. Overactivity of the pectoralis major muscle (sternal fibers) can hinder the voluntary initiation of serratus anterior in many stroke patients. Positional stretch, myofascial release, or traditional stretching techniques can be



Animated photograph of model with permission

Fig. 3.31 Proprioceptive stimulation and commands like "do not allow me to push your arm downward" to activate serratus anterior in supine position

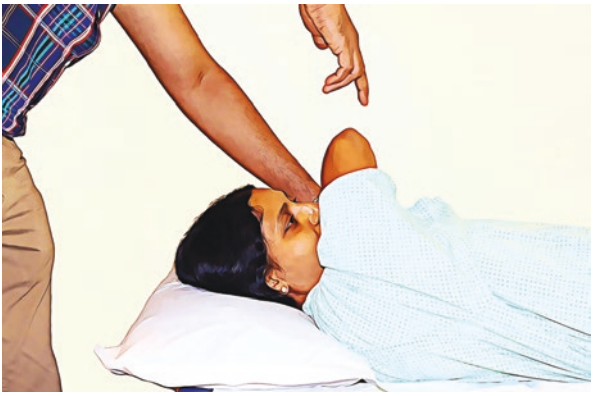


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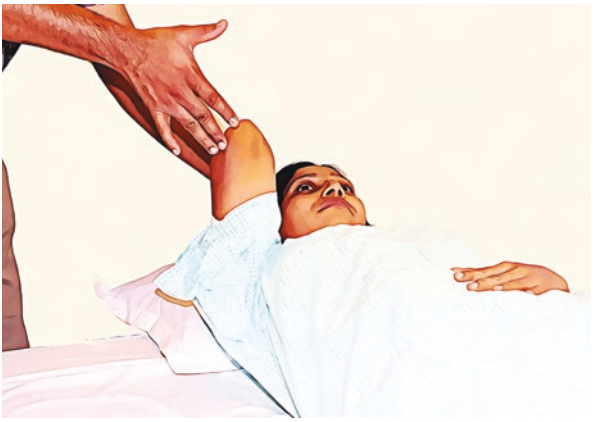
Fig. 3.32 Short lever exercises for the shoulder flexors in the supine position

attempted in such a situation to minimize the overactivity of the pectorals and encourage the initiation of serratus anterior muscle. The activity of the trapezius muscle can be initiated in the sitting position. Support and guidance to encourage shrugging of the shoulders (initially bilaterally and then advancing to affected side shoulder alone) and commands like "do not drop your shoulder" can help in initiating the same muscle.

To further build up the control over the affected side shoulder, short lever exercises for shoulder flexors (Fig. 3.32) and horizontal adductors (Fig. 3.33) should/can be encouraged in the supine position. Reaching out activities with the affected upper limb requires synergistic activity of the serratus anterior, shoulder flexors, and elbow extensors. To prepare for the same, facilitation techniques can be attempted to activate the elbow extensors with the patient's arm held in 90° flexion (Fig. 3.34). Once the patient can initiate the contraction, encourage the patient to eccentrically, isometrically, and concentrically control the elbow extensors. Following which attempts should be made to perform reach-out activities in supine with the elbow held in extension or near extension (Fig. 3.35). In the supine position, attempts to



Animated photograph of model with permission

Fig. 3.33 Short lever exercises for the shoulder horizontal adductors in the supine position

Animated photograph of model with permission

Fig. 3.34 An illustration of the facilitation technique (muscle tapping) to activate the elbow extensors with the subject's arm held in 90° flexion

Animated photograph of model with permission

Fig. 3.35 Reach-out activity performed in supine with the elbow held in near extension

perform circumduction movement in a short circle and later advance toward a larger circle with adequate support and guidance may further improve the control of the shoulder. Patients with limited control over the shoulder and elbow muscles, while sitting, should be encouraged to slide the hand forward over a tabletop or re-education board. Practicing wiping or polishing activities and pushing or sliding a ball forward-backward help to recruit the scapular protractors, shoulder flexors, and elbow extensors. Visual guidance will help in improving eye-hand coordination during the reach-out activities. The patient should practice independent lifting and multiplanar reaching with increasing resistance (weight of the object held by the hand or secured by the Velcro) and varying height and distance in sitting, modified plantigrade, and standing positions based on the motor recovery potential. Trick movements or substitution movements and encouraging patterns similar to basic limb synergies need to be avoided as they can hinder the potential for further recovery.

For hand function, grasp, manipulation, and release are the movements required for the finer motor activities of the hand. During the initial phase of hand function recovery, voluntary release can be difficult as compared to the grasp. Weight-bearing activities through the hand may normalize the tone of the overactive long flexors of the wrist and hand. Positional stretch, sustained stretch, and other inhibitory techniques for the spastic wrist and finger flexors along with facilitatory techniques for wrist and finger extensors may encourage extension movements. NMES for the wrist and finger extensors can also improve the function of the wrist and finger extensors, similar to the deltoid and supraspinatus muscle functions for shoulder subluxation. Task-oriented activities can be practiced with the weaker hand, which will encourage the patient to use the affected limb for ADL. For those patients with potential for further recovery, wrist stabilization exercises (initially with the elbow in extension and later in flexion) should be achieved prior to hand activities like finger extension, opposition, and manipulation of objects. Careful observation of functional tasks like manipulation of objects and use of utensils or tools may provide clues to eliminate unwanted muscle activities or movement patterns interfering with the functions. For those patients in stage 3 and above of Brunnstrom motor recovery stage, therapeutic approaches like constraint-induced movement therapy (CIMT) and motor relearning program can promote or enhance further hand function recovery. CIMT consists of a multifaceted intervention designed to promote upper extremity function by engaging patients in intense task-oriented practice wearing a mitt on the uninvolved upper extremity for $\leq$6 hours per day for 10 consecutive days. The physiotherapist uses feedback, shaping techniques, and encourages the patients to modify and progress performance during practice. The patients undergoing CIMT are encouraged to self-monitor the target behaviors and use problem-solving abilities to identify the obstacles and generate potential solutions. The less intense modified CIMT (mCIMT) and CIMT have shown neuroplastic changes, including a shift in motor cortical activation toward the areas in the contralesional hemisphere. Evidence does exist regarding the beneficial effects of CIMT and mCIMT for acute, subacute, and chronic stroke patients.

Certain literature reports that bimanual training is as effective as but not superior to unimanual training of the paretic upper extremity post-stroke. It is hypothesized that similar movement in the less involved upper limb facilitates movement in the involved upper limb. Functional task training (FTT), Bilateral arm training with rhythmic auditory cues (BATRAC), and robot-assisted training (RAT) are the three main types of bimanual training interventions. In terms of outcome measures, compared to standard or routine therapy, bimanual training was not shown to be significantly better than the former.

In the past, electromyographic (EMG) biofeedback has been extensively used to improve motor function in stroke patients. The EMG biofeedback helps to recruit motor units in weak or hypoactive muscles, train voluntary inhibition of spastic muscles, and increase the kinesthetic awareness of voluntary movements. Existing evidence reports that the benefit of biofeedback is considerable when it is used as an adjunct with task-specific training. For moderate to severe motor impairments of the upper extremity, task-oriented training with robotic devices has been attempted to reach and grasp and/or release movements. Rehabilitation using robotic devices enables high levels of intensive practice and is generally well-tolerated by stroke patients. Though it is a useful adjunct to therapy, the high cost is a major limitation for widespread use of these units worldwide.

For the past two decades, virtual reality has been used in the field of stroke rehabilitation. Virtual reality is the simulation of a real environment that enables the user to interact with certain elements in the virtual environment using a man-machine interface. It provides the user visual, auditory, tactile, and motion information and a variety of interfaces for interacting with the simulation setting, ranging from common devices like mouse or joystick to more complex devices with motion capture systems or haptic devices, providing users sensory feedback and experience close to the real task. The degree of immersion varies depending upon the virtual environment setting. The term "immersion" refers to the extent to which the user perceives the virtual environment as the real world. The virtual environment can be immersive, non-immersive, or semi-immersive. Semi-immersive and non-immersive systems are the most widely used systems for the management of motor symptoms among stroke patients. Various virtual reality programs have been developed and used for upper and lower limb training, gait training, and posture and balance training for subacute and chronic stroke patients. Current evidence suggests that virtual reality training for stroke patients is safe and cost-effective for improving upper and lower limb functions, dynamic balance, prevention of falls, stair climbing, ROM, muscle strength, and gait speed. Virtual reality is hypothesized to motivate and promote practice-dependent reorganization. Transcranial magnetic stimulation and functional MRI studies have demonstrated reduced ipsilateral cortical activation and increased contralateral cortical activation as a result of intensive practice of the affected limb using virtual reality. Further analysis has also revealed that the activation was more in the contralateral primary sensorimotor cortices than in the ipsilateral premotor cortex or contralateral supplementary motor areas.

3.11.2.11 Improving Lower Limb Functions

Activities specifically emphasizing the breaking up of the obligatory basic synergy patterns of the affected lower extremities are the key strategy for preparing the patient for the gait. PNF diagonal pattern (D1 extension pattern), which associates hip extensors and abductors with knee extensors, can encourage effective midstance, which otherwise is less likely to occur due to the strong linkage of the dominant extensor synergy. The use of elastic bands around the upper thigh to encourage hip abduction with the knee in extension in supine or standing positions and encouraging lateral sidesteps in standing are a few other alternatives. Hip adduction should be stressed during hip and knee flexion to facilitate the normal swing phase, which otherwise is characterized by abduction and flexion of the hip with knee flexion, a feature of flexor basic limb synergy. Activities like PNF D1 flexion pattern in supine and standing and crossing and uncrossing the affected lower limb over the unaffected limb in crook-lying position or sitting can associate hip adduction with hip and knee flexion and break the linkage between hip abductors and the flexors. Bridging and encouraging knee flexion in standing or modified plantigrade standing postures are activities that can promote hip extension along with knee flexion essential for the swing phase of the gait.

Several therapeutic exercises are available to improve the trunk and pelvic control of stroke patients. These exercises can be practiced on firm ground, therapeutic mat, or Swiss ball. Swiss ball exercises can be used for encouraging pelvic rolling in supine and pelvic shifting in sitting. Dissociation of the upper trunk to the lower trunk can be practiced in supine, crook-lying, and kneeling positions on a therapeutic mat or standing positions on firm ground. The smooth reversal of direction of movement produced by reciprocal muscle action initiated in supine (like foot sliding up and down in crook-lying position or leg sliding in and out in supine), then in sitting (like foot slides under the chair), and finally in standing (partial squats or partial wall squats) and the use of proprioceptive training (including dynamic squats and single-limb squats) for the affected lower limb initially on a firm surface and later on foam surface can improve eccentric and concentric muscle control of the knee musculature.

As a preparation for gait, it is also essential to initiate and strengthen the activity of the dorsiflexors and evertors of the foot. Stretching of the overactive plantar flexors is a prerequisite for initiating the activity of dorsiflexors and evertors. The use of exteroceptive or proprioceptive techniques can often help in initiating the evertors, which are neither a component of flexor nor an extensor basic limb synergy. Care has to be taken while selecting the technique or strategy to initiate and strengthen the muscles, as wrong selections can further create overactivity of the plantar flexors and invertors accentuating the muscular imbalance at the level of ankle and foot.

3.11.2.12 Strategies to Improve Postural Control and Balance Reactions

Sensorimotor dysfunctions can significantly change the postural control and balance reactions of stroke patients. Absence, delay, or variability in the balance reactions and impairments in latency, amplitude, and timing of muscle activity are certain typical features seen in these patients. Poor postural control and balance

reactions can predispose these patients to falls and fractures and can further reduce their level of confidence in balance and locomotor skills. Balance training programs progressively proceeding from less challenging to more challenging postures performed overground and on unstable surfaces like a gym ball or a wobble board will boost the patient's level of control and confidence. Gradually increasing the level of difficulty like the range of displacement and speed of displacement, encouraging self-initiated movements and emphasizing consistency, postural symmetry, and maximizing the use of the affected side should be the goals of training. To assist and instill confidence and prevent falls, during the early phase of balance training in standing, the therapist may encourage the patient to use appropriate supportive devices like a harness or gait belt. Indiscriminate and prolonged use of such supportive devices can often be counterproductive and needs to be discarded, especially when patients seem likely to improve their postural and balance control.

Following effective and consistent maintenance of symmetrical aligned static upright postures, the stroke patients should be encouraged to self-displace their center of mass to explore the limits of stability. The use of verbal and tactile cues and verbal instructions can progressively encourage the patient to achieve larger ranges of displacement to further improve the limits of stability. Provide an ample amount of practice for the patient to master the skill of safely moving in any direction while aligning the center of mass over the base of support. Encouraging weight shift activities in sitting and standing with more weight-bearing on the affected side pelvis and lower limb, respectively, and discouraging the overuse of unaffected limbs will provide the opportunity for the affected side musculatures to develop appropriate postural and balance responses.

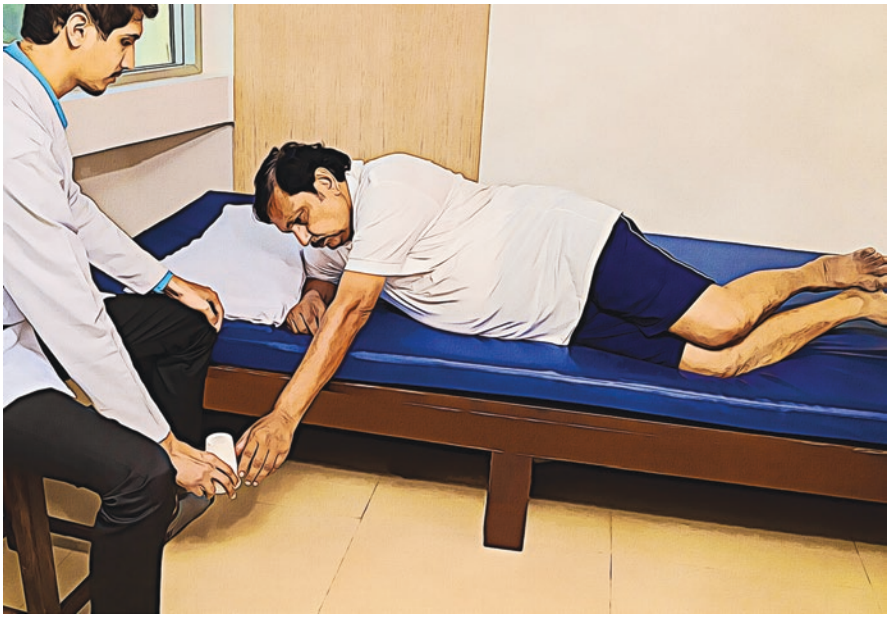
To regain balance following the unexpected displacement of the center of mass, ankle, hip, and stepping strategies are crucial. Single limb standing (affected side) on a foam surface with minimal upper limb support, preferably with the affected side knee maintained in minimal flexion, can encourage the ankle and foot muscles to contract actively. Standing on a wobble board or a half-foam roller can be the alternatives to activate these muscles to promote ankle strategies required for regaining balance; however, such exercises may be too advanced for stroke patients during early rehabilitation. Perturbations in the anteroposterior direction or weight shifts in the same direction can promote anteroposterior hip strategies. Tandem stance or near tandem stance on floor or foam can promote mediolateral hip strategies, whereas displacement of the center of mass beyond the base of support can promote stepping strategies.

Advancing the exercises from stable to an unstable surface, increasing the amplitude of displacement of the center of mass within and beyond the base of support, reducing the base of support, progressing from uniplanar to multiplanar reaching outs, incorporating head and trunk rotation for the task, encouraging dual-task activities, walking sideways, backward and braiding, and proceeding from a closed to an open environment are certain strategies to improve the level of difficulty for the balance training program. To maintain and regain balance during the balance training program, the stroke patient should be allowed to identify the potential problems and encouraged to actively solve the problem by recruiting the appropriate safety strategies.

3.11.2.13 Treatment Strategies for Pusher Syndrome

The postural and balance issues among the Pusher's syndrome patients are distinct from those seen among the rest of the stroke patients. Asymmetry in sitting and standing, excessive weight-bearing through the affected side, tendency or strong urge to push consistently toward the affected side using the unaffected limbs, apprehension about falls toward the unaffected side with absolute "no botheration" about the repeated instability, and falls toward the affected side are certain hallmarks of "Pusher's behavior" or ipsilateral pushing. Typically, the therapist's efforts to passively correct the tilted posture will result in a stronger resistance from the patient and often can be counterproductive. Visual stimuli, environmental prompts and boundaries, self-initiated corrections, active efforts to achieve vertical (midline/neutral) position, use of mirrors, verbal, and tactile cues for postural orientation, training activities on a Swiss ball to promote symmetry, and the use of air splints and walking aids are the strategies available to correct the pushing behavior. However, the author (Abraham M. Joshua) believes that strategy emphasizing overcorrection along with carefully guided and instructed activities can be a better practical solution than the abovementioned strategies. The strategy developed and encouraged by the author is distinct from those training programs which emphasize upright positions with active movement shifting toward the stronger side or for vertical orientation.

During the early phase of management of Pusher's behavior, a clear and concise explanation about the pushing behavior and its consequences needs to be addressed to the stroke patient and the family members. The information should even emphasize the safety issues and the roadblocks of rehabilitation. Advising and encouraging the patient to lie on the unaffected side for as much time as possible should be the first component of the strategy. Meanwhile, emphasis should be given to discourage the patient from lying on the affected side. Once the patient becomes comfortable with side-lying on the uninvolved side, he or she should be encouraged to lie on the uninvolved side propped up on the elbow. The posture mentioned above can be actively maintained, if not by pillows. Once the patient can actively maintain the posture, encourage him or her in a controlled fashion to perform repeated short-range propping up from the side-lying position. Reach-out activities for the affected upper limb can be introduced in the side-lying with propped up elbow position (Fig. 3.36). The direction of reach-outs should be toward the uninvolved side. If the patient tends to flex and rotate the neck toward the affected side, the therapist should dissuade those attempts and should encourage rotation toward the unaffected side. Once the pushing tendency subsides, the patient should be advised and/or assisted in bringing both legs off the side of the bed. The reach-out activities should continue for this posture (Fig. 3.37) before advancing to sitting up over the side of the bed. While sitting, a partial amount of lean toward the unaffected side should be encouraged. Instructions like "shrug your shoulder" for the affected side and "drop your shoulder" for the uninvolved side can further encourage the partial tilt toward the uninvolved side. An alternate way to encourage the leaning toward the uninvolved side is to instruct or guide the patient to bring the shoulder or forehead closer toward the therapist or family member sitting by the side of the uninvolved side.



Animated photograph of model and therapist with permission

Fig. 3.36 Reach-out activity performed in side-lying with propped up elbow of the uninvolved side

Animated photograph of model and therapist with permission

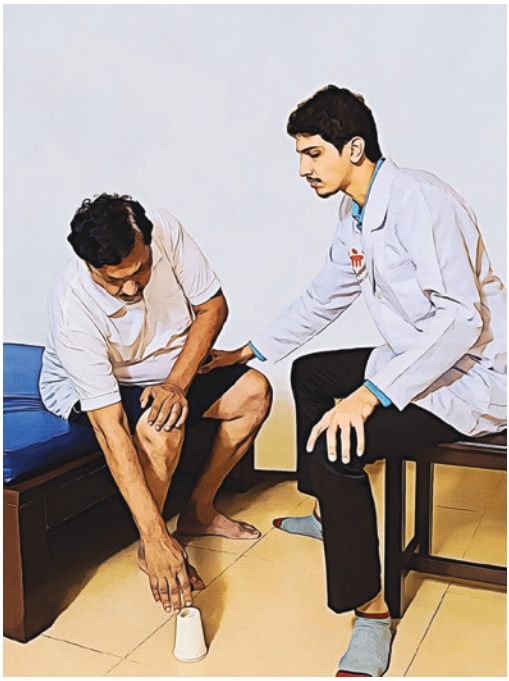
Fig. 3.37 Reach-out activity in side-lying with the legs off the bed

Animated photograph of model and therapist with permission

Fig. 3.38 Reach-out activity toward the uninvolved side with the uninvolved upper limb folded across the chest

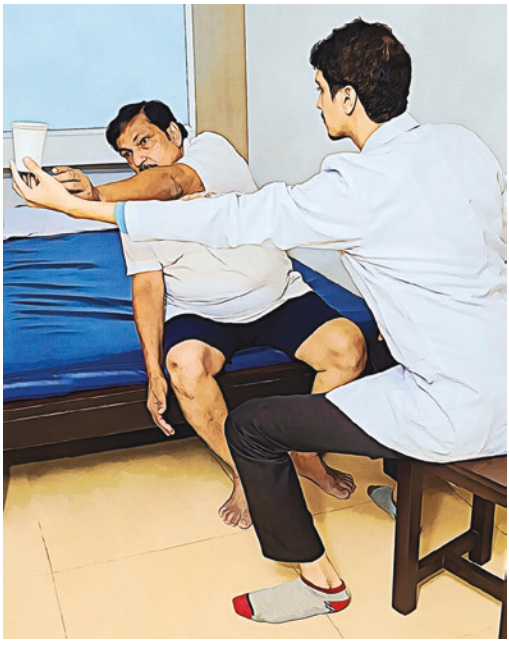
Throughout the training, attempts by the patient to use the uninvolved upper limb to push himself or herself up toward the affected side should be discouraged. The tendency to use the uninvolved upper limb for pushing is more when the uninvolved side shoulder is kept in abduction and elbow in extension. Asking the patient to keep his uninvolved upper limb folded across the chest (Fig. 3.38) or reach-out toward the uninvolved side using the same side upper extremity (Fig. 3.39) or rest the uninvolved arm hang by the side (Fig. 3.40) or over the thigh are certain strategies to minimize the usage of the uninvolved upper limb for pushing. While sitting in a wheelchair, for those patients with severe pushing behavior, encourage sitting with partial lean toward the uninvolved side with head and neck turned to the same side.

Often the therapist needs to repeatedly educate and encourage the patient on the relevance of not pushing and actively leaning and turning toward the uninvolved side. To encourage active participation of the patient, the therapist should ask questions like “which direction are you tilted?” and “which direction you need to move to overcorrect and prevent falling?” Visual scanning toward the uninvolved side and asking the patient to give details of the objects placed near and around the uninvolved side of the patient can further encourage the overcorrection. Once able to sit unsupported with partial leaning, the patient should be encouraged to perform



Animated photograph of model and therapist with permission

Fig. 3.39 Reach-out activity in sitting using the upper extremity of the uninvolved side



Animated photograph of model and therapist with permission

Fig. 3.40 Reach-out activity in sitting with the uninvolved arm hanging by the side

reach-out only toward the uninvolved side. Depending upon the strength and control, the patient can use either of the upper limbs for reach-outs. The therapist should avoid making any tactile contact with the involved side trunk as the patient can lean toward the therapist's manual contact. For those patients who have achieved independent sitting, small increments of self-initiated movements can be encouraged to bring the trunk to the midline and then back to the leaning posture toward the uninvolved side. As the postural control improves, self-initiated to and fro movements passing beyond the midline toward the involved side can be encouraged.

For sit-to-stand task, cues and manual guidance can be given to encourage the patient to lean toward the uninvolved side while transferring (Fig. 3.41). The therapist's hand can act as a cue and instruction, including "touch your head on my hand" and "follow my hand," can help the patient to lean and get up toward the uninvolved side. While standing, the same strategy used in sitting, including overcorrection, reach-out activities, and weight shifts toward the uninvolved side, needs to be encouraged (Fig. 3.42). Once the subject can stand with partial lean to the uninvolved side, self-initiated to and fro movements initially up to the midline and later beyond midline toward the involved side need to be incorporated. In order to make the task more interesting, the therapist can ask the patient to do "head butting" on

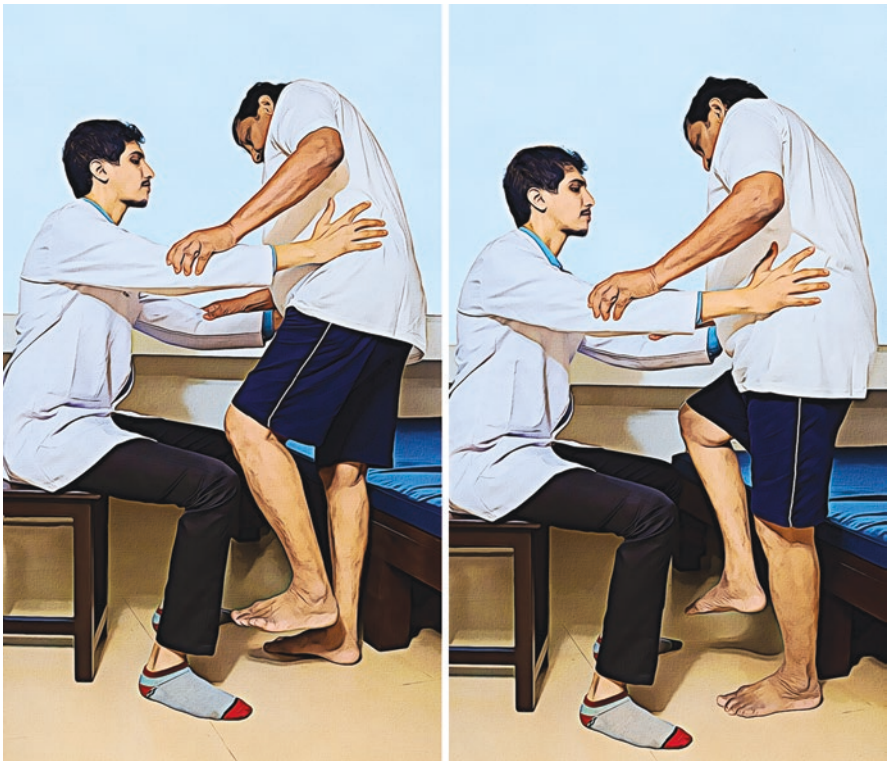
Animated photograph of model and therapist with permission

Fig. 3.41 Guidance to encourage the subject to lean toward the uninvolved side during the sit-stand transition. Note: The upper limb of the uninvolved side is kept in supination over the thigh to minimize the tendency of pushing

Animated photograph of model and therapist with permission

Fig. 3.42 Providing visual guidance to maintain leaning toward the uninvolved side while standing

the therapist's hand to encourage a faster correction. The position of the patient's head, neck, trunk, and extremities, appropriate instructions, the therapist's hand that serves as a cue, and manual guidance and support provided will ensure the correct way of maintaining and regaining the erect posture without pushing. Once the patient can maintain an erect posture, weight shifts and marching in place (Fig. 3.43) and stepping forward, backward, and sideways can be attempted as preparation for walking. In case if patient tends to revert to pushing behavior in standing, overcorrection strategies can be reattempted to minimize the behavior. For those patients with a tendency to push while attempting to walk, instruct and guide them to walk with mild-to-moderate lean toward the uninvolved side. Figure 3.44 illustrates how



Animated photograph of model and therapist with permission

Fig. 3.43 Marching in place with the trunk mild to moderately tilted toward the uninvolved side

the patient can be guided to walk while maintaining the mild-to-moderate lean toward the uninvolved side. Typically, within 2–3 weeks, with the strategy mentioned above, the pushing behavior will subside, and the patient will be able to perform sitting up from lying and the sit-to-stand transfer without losing balance or falling consistently to the involved side.

The overall strategy is to overcorrect the pushing behavior until he or she can maintain an erect posture without the tendency to fall toward the affected side. For instance, if the patient tends to push toward the right side and backward, the overcorrection should be toward the left side and forward. In the initial phase of training, the use of mirrors may not provide much benefit. For patients with minimal pushing behavior, cognitive strategies and mirrors can be helpful in self-initiating weight-bearing toward the uninvolved side. Even motor learning strategies can be of help in reducing ill effects. Demonstrating correct vertical orientation, providing consistent feedback about the body's orientation, practicing correct weight shifts, and involving the patient for problem-solving may help in tackling the pushing behavior.

Animated photograph of model and therapist with permission

Fig. 3.44 An illustration depicting the strategy to walk with a mild-moderate lean toward the uninvolved side

3.11.2.14 Promoting Functional Independence

Restoring or improving functional independence in ADL is an important factor for the quality of life post-stroke. Stroke survivors who need assistance for ADL always feel themselves as a burden for their family members and often are socially isolated, overwhelmed, and abandoned. In addition to restoring the sensorimotor function, the therapy should include effective strategies aimed at improving functional independence in basic, instrumental, and everyday activities. The sensorimotor dysfunction of the paretic side can impose a tremendous challenge for relearning functional mobility and independence. During the early phase of therapy, strategies focusing on improving truncal symmetry and usage of both sides of the body should be the priority. For independent control, exercise should progress from assisted to guided movements and then eventually active movements.

Concerning bed mobility, rolling to either side should be encouraged right from the early stage of rehabilitation. Attempts to roll to the unaffected side can be difficult as compared to rolling to the affected side. While practicing to roll, care must be taken not to leave the affected upper extremity behind; instead, the patient must

be encouraged to clasp the hands together to bring the affected extremity forward to initiate the roll. Rolling onto the affected side and encouraging the side-lying on-elbow position helps to promote early weight-bearing through the affected upper limb. Stroke patients should practice supine-to-sit after rolling from both sides, but during the bed mobility training, more emphasis needs to be given to the supine-to-sit using the involved side. Verbal commands, manual guidance, and assistance need to be given during the transition, especially while moving the affected leg over the edge of the bed and while attempting to push up to sit using the affected upper extremity.

The joints need to be kept in normal alignment during the transition and undue weight falling directly over the affected shoulder has to be avoided as it may predispose to shoulder impingement and pain. Rather than practicing the task as a whole, block practicing should be encouraged. Attempts also should be made to perform controlled lowering as it may help in gaining better motor control during the side-lying-to-sit transition. The rhythmic initiation and diagonal patterns that encourage bed transitions can also be incorporated. Activities like bridging and tentacle exercises need to be promoted as those exercises can help in developing trunk and hip control necessary for early weight-bearing through the foot. In addition to the above, such activities also promote scooting, bed mobility, sit-to-stand transfers, use of bedpan, pressure-relieving technique for the buttocks, and movements away from the basic lower limb synergies. If the patient has difficulty maintaining the crook-lying position while attempting bridging, the therapist may need to stabilize the foot and the affected side lower limb manually. If the hip extensors are weak, bridging the affected lower limb and unaffected lower limb in a tentacle position can be difficult. In such situations to encourage the contribution of affected side hip extensors, placing a small ball or a soft roll under the unaffected foot or crossing the unaffected limb and placing it over the affected limb can be tried for plain bridging before bridging along with tentacle exercise.

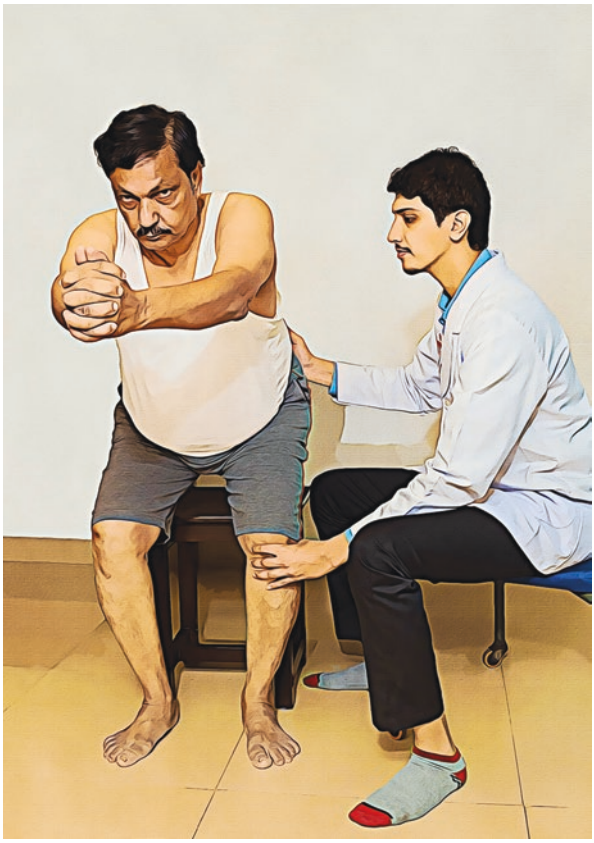
Achieving a symmetrical posture with the spine and pelvis in proper alignment should be the focus during the early phase of training in sitting. The therapist should encourage the patient to hold his or her spine erect, pelvis in neutral, and feet flat on the floor. Most of the stroke patients will sit with asymmetric posture bearing more weight on the unaffected side, pelvis posteriorly tilted, and kyphosis of the upper trunk with or without lateral flexion of the trunk to the affected side. Verbal or tactile cues, use of visual feedback, and guidance can encourage these patients to correct such posturing. Encouraging early sitting with bilateral support of upper extremities on the tabletop, large gym ball or the therapist's shoulder is meaningful. Exercise for encouraging the sitting control should progress from static to dynamic, stable to the unstable surface (gym ball or wobble board), dissociations of upper trunk from the lower trunk movements, and eventually dynamic challenges like reach-out activities in multiplanar direction.

Bridging and tentacle exercises and gym ball exercises encourage the activity of the abdominal obliques, generally improve trunk and pelvic control, and translate to better weight shifts while sitting. Practicing scooting in sitting or "butt walking" at the side of the bed and encouraging push-ups in sitting with both upper extremities

(provided the affected upper limb has a certain amount of strength and control) can be attempted before sit-to-stand transitions are introduced for functional training.

Sit-to-stand transitions should be practiced with a focus on symmetrical weight-bearing, coordinated muscular responses, and appropriate timing. Placement of feet well behind, with the heel still in contact with the floor or the supporting surface, allows the ankle dorsiflexors to assist the transition. Improper placement of feet, poor alignment of the lower limbs, reduced forward momentum, premature activation of spinal extensors, asymmetrical weight-bearing through the feet, and the improper or poor sequencing of body components required for the sit-to-stand transition can make transfers unsafe or difficult. During the initial days of training for sit-to-stand transfers, to minimize the demand and extensor force required for the transition, a higher bed or seat is preferred to lower platforms. During the training, stroke patients will be instructed and/or assisted to place both feet on the floor right under the knees at hip-width apart. Verbal instructions like "sit tall" will encourage the patient to sit erect. Verbal and tactile cues are required to encourage the patient to flex his or her erect spine over the hips before initiating standing up by extending the lower limbs and then by extending the spine over the hips. Tactile and proprioceptive cues can also assist knee extension during the transition. The patient can be either advised to keep both upper extremities forward with hands clasped together or placed over the therapist's shoulders with the therapist sitting directly in front. Premature extension of the spine during this training process will make standing up unsafe or difficult. Similarly, uneven or excessive pushing off with both hands on the supporting surface should be discouraged as it may lead to uneven weight-bearing or excessive forward momentum. For smooth execution of sit-to-stand transitions, proprioceptive training (including static and dynamic partial squats) on a firm surface (Fig. 3.45) advancing toward a foam surface, weight shifts, single-limb stance using stall bars or manual assistance, and strengthening exercises for the affected lower extremity muscles are recommended.

During the sit-to-stand training, it is not uncommon to see stroke patients relying heavily on the unaffected lower limb. The tendency to lean to the unaffected side, more weight-bearing through the unaffected lower limb, and placement of the normal foot behind the weak foot indicate the patient's overdependence on the unaffected side for such tasks. Encouraging the patient to weight-bear more through the affected limb can only be possible if the affected foot is positioned behind the normal foot. Strengthening of affected side lower limb muscles and proprioceptive training for affected lower limb, including single limb partial squats with adequate support, can further enhance weight-bearing and confidence of the patient. Repeated practice, encouraging the patient to execute the movement with increasing speed, paying attention to the sequence of the components for sit-to-stand, and avoiding pauses between the components, need to be encouraged to improve the sit-to-stand transition. Proper positioning of the upper extremity with the affected elbow in extension and hands clasped together and learning to control the lower extremities eccentrically for a gradual descend to sit-down can further improve the overall control of sit-to-stand transition.



Animated photograph of model and therapist with permission

Fig. 3.45 Performing dynamic partial squats on a firm surface to improve knee control and strength

In the early phase of rehabilitation, while training transfer techniques (transfers like the bed to a wheelchair and wheelchair to a tub seat), the stroke patients may require maximal assistance. Adjusting the height of the hospital bed or wheelchair can reduce the difficulty level for transfer. Placing the wheelchair next to the unaffected side, instructing the patient to scoot the buttock forward to the edge of the bed, and assisting the patient to stand and pivot a quarter turn on the unaffected lower limb before sitting down is the sequence of components required for safe transfer from one surface to other. Though this strategy promotes early transfers, it neglects and minimizes the contribution of the affected side and encourages learned non-use. To avoid such compensatory strategies, the therapist should encourage the patient to transfer to both sides, with the emphasis more toward the affected side. If the upper extremity control is poor, with or without shoulder subluxation or pain, stabilization of affected arm in extension and external rotation against the therapist's body, cradling the hand and holding in forward flexion, and placement of the affected upper extremity over the therapist's shoulder are the appropriate strategies for the same. The therapist's knee can be used to exert a counterforce on the patient's knee during the transfer if the knee control is inadequate.

Both standing and modified plantigrade standing postures can facilitate functional activities. Modified plantigrade can be a safe posture to teach weight-bearing activities for both upper and lower limbs, especially when prone or prone progression postures are not advisable. If the potential for motor recovery is good and symmetrical weight-bearing over the base of support is possible, the patients should be encouraged to practice standing with one hand support and eventually with no upper extremity support. Activities that promote weight shifts, multiplanar reach outs, and activities that encourage the dissociation of the upper trunk to the lower trunk are strategies to improve dynamic stability. PNF techniques like rhythmic stabilization and slow reversal hold technique may help to improve poor postural stability in some stroke patients. Use of additional postures such as prone on elbows, quadruped, kneeling, and half kneeling on the therapeutic mat can increase the level of difficulty and improvise the postural control further. However, such postures can be inappropriate or unsafe for those patients with comorbidities such as ischemic heart disease, chronic obstructive pulmonary disease, and severe osteoarthritis of the hip or knee joint.

3.11.2.15 Strategies to Improve Gait

To develop effective gait training strategies, the therapist should understand the impairments primarily determining the walking ability of stroke patients. Muscle strength, motor control, and balance are the common impairments strongly related to gait. Of all the treatment strategies, exercise is the most common therapeutic intervention used to improve gait in hemiplegic patients. Graded muscle strengthening can improve the ability to generate force but generally does not transfer to improved walking ability. Providing task-specific practice in addition to resistance training to those stroke patients with impaired sensorimotor coordination may help to extract the benefits of strength gains. Early task-specific gait retraining with more repetition may facilitate the development of new motor programs or refinement of existing programs necessary to accommodate these deficits. Treadmill training with or without a harness system and task-specific overground locomotor training (practicing a wide variety of functional mobility tasks such as walking, sit-to-stand transfer, turning, obstacle training) are two main approaches for task-specific practice.

Treadmill practice can be considered as a “forced use” that maximizes the use of the hemiparetic limb by encouraging more number of steps, a greater amount of paretic limb loading, and better activation of the paretic muscles at different speeds. The treadmill with a harness or bodyweight support system enables lower functioning stroke patients to practice early walking when supervised traditional techniques are unsafe to practice. Concerning improvements in gait performance, the evidence is conflicting between treadmill training with or without bodyweight system and standard treatments.

Overground gait training provides a more natural stimulus to challenge the different components necessary for walking. Overground gait training promotes anticipatory postural control like avoiding an obstacle or changing the course to avoid

bumping and reactive control, like responding to a slip or nudge. Treadmill training may not allow participants to experience the normal postural demands or visual sampling that occur during walking. In addition to the above, treadmill training will not offer many of the functional variations in gait, such as turning, rising from a chair to walk, and starting or stopping of gait. The successful overground practice of a variety of tasks that represent community walking, including stepping up a curb, walking in a crowded hallway, and walking on even and uneven surfaces may enhance self-efficacy and consequently walking ability. A combination of treadmill and overground task-specific locomotor training should be encouraged to gain the benefits of both approaches if centers have such facilities or infrastructure.

In addition to strengthening exercises for the lower limb muscles, stretching of the appropriate muscles, particularly, the calf muscles should be considered. Functional task-specific skills should include walking forward, backward, and sideways, braiding, and side-stepping. Throughout the training program, care has to be taken not to encourage those moves that can reinforce the basic limb synergies. As the patient gains more locomotion control, he or she should be encouraged to improve the rhythm and speed of walking. Verbal and auditory cues can facilitate rhythm and speed. For those patients with a scope of better motor recovery, prolonged and indiscriminate use of parallel bars and ambulation aids like hemi walkers and quadripod sticks are not advisable as those strategies can hinder the patient's potential to walk without the device, develop appropriate balance mechanisms, and encourage postural symmetry. Patients who are entirely dependent on such aids tend to walk slow with impaired locomotor rhythm.

The therapeutic interventions for gait training should incorporate the repetitive practice of a wide variety of mobility tasks. Such intensive mobility training should contain the components of graded strengthening using functional tasks including repetitive rise from a chair, stepping up and down a stepper, aerobic component including graded walking activity, cycle ergometer, and performing continuously moderate intensity functional tasks and a variety of challenging walking activities with substantial postural control demands. The use of a circuit of workstations with the components mentioned above and adequate rest will generally improve the performance of gait. In the back of the mind, the therapist also should realize that the optimal gait training program should include balance and agility training, which is in addition to the practice of upper extremity and trunk mobility tasks.

During the stance phase, knee hyperextension, an abnormal movement away from the anatomical neutral position into extension, is reported in approximately 65% of stroke subjects. Decreased ability to activate the knee extensor muscles, reduced eccentric control of the knee extensors, spasticity of quadriceps and plantar flexors, reduced strength of hamstrings, and proprioceptive deficits are the possible causes for the same. Longstanding knee hyperextension can lead to the posterior capsule and anterior cruciate ligament laxity predisposing to early degenerative changes of the knee joint, poor proprioceptive control during terminal knee extension, joint deformity, chronic knee pain, and reduced independence in daily

activities. During the swing phase, knee hyperextension makes knee flexion difficult for effective ground clearance, in turn promoting circumduction and excessive energy consumption while walking. A novel technique "prowling along with proprioceptive training" developed by the author had shown significant improvement in reducing knee hyperextension, improving the ankle dorsiflexion range, and augmenting spatiotemporal parameters of gait. The word "prowling" means walking in a predatory manner, characterized by walking with bilateral knee bent attitude, which provides the dual advantage of activating the quadriceps muscles and changing the direction of moment-arm of quadriceps and increasing mechanical advantage. The proprioceptive training given for the involved lower limb consists of partial squats, single limb stance, and single limb dynamic partial squats while standing on firm ground as well as on a foam mat. The technique can be beneficial for reducing knee hyperextension while standing and walking for those stroke patients who are cognitively sound with the Brunnstrom recovery stage of ≥ 3, with no severe plantar flexor tightness.

The author believes that the optimal program to improve gait should involve the repetitive and intensive practice of tasks that loads more weight on the paretic limb, functional strengthening, and balance training, which is progressively incremented in difficulty according to the tolerance of the stroke participant. For community ambulation, tasks like walking on different terrains, negotiating curbs, climbing up and down the stairs, stepping over obstacles, turning, and quick stops and starts while walking should be incorporated. If space or resources are the constraints, the use of virtual reality simulating realistic visual stimuli can be an effective alternative.

3.12 Brainstem Stroke Syndromes

Brainstem stroke accounts for approximately 10% of all strokes. Brainstem stroke syndromes are a group of syndromes that are classically caused by the occlusion of small perforating arteries of the posterior circulation supplying the brainstem. Typically, these syndromes occur in the territories of the basilar or vertebral arteries. Posterior cerebral artery, superior, anterior inferior, and posterior inferior cerebellar arteries, basilar artery, and vertebral artery are main vessels that provide arterial supply to the brainstem. Except for basilar artery occlusion, all the remaining posterior circulation strokes have a low mortality of 5%. The outcome for bilateral vascular lesions of the brainstem due to occlusion of the basilar artery is generally bleak.

The brainstem plays a crucial role in controlling balance, coordination, eye movements, hearing, speech, and swallowing, and strokes within the regions of the brainstem will have a different set of clinical symptoms and challenges as compared to the hemispheric strokes. Ipsilateral cranial nerve palsy(ies) and contralateral

weakness and/or hemisensory loss are the typical characteristic clinical pictures of all brainstem strokes. Dysarthria and dysphagia, headache, vomiting and nausea, ocular eye movement abnormalities and diplopia, vertigo, nystagmus, hemiplegia or quadriplegia, ataxia, and change in the level of consciousness are some of the specific impairments resulting from brainstem strokes. Hemineglect, perceptual dysfunction, aphasia, and apraxias are absent as the hemispheric cortical and subcortical areas are spared in brainstem stroke syndromes. In the following section, the author would like to brief some of the common brainstem stroke syndromes occurring at the midbrain, pons, and medullary levels.

3.12.1 Weber's Syndrome (Medial Midbrain Syndrome)

Medial midbrain syndrome (Fig. 3.46) occurs due to occlusion of the penetrating branches of the posterior cerebral artery that supplies the medial aspect of the midbrain. Contralateral paralysis of the lower part of the face, tongue, arm, and leg are its clinical manifestations. The third cranial nerve involvement results in ipsilateral gaze palsy or ophthalmoplegia. It is characterized by the inability to voluntarily move the ipsilateral eye upward and inward with an abnormal downward and outward resting position of the eye. In addition to the above, the involvement of the third cranial nerve also results in unresponsive dilated pupil, diplopia, and ptosis.

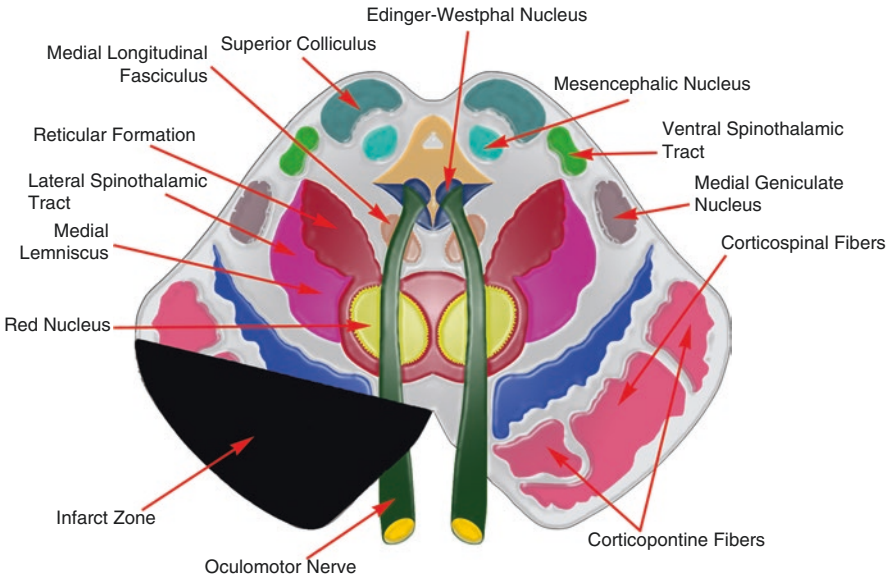


Fig. 3.46 An illustration of midbrain lesion in Weber's syndrome (medial midbrain syndrome)

3.12.2 Benedikt Syndrome

Lateral midbrain syndrome, also known as Benedikt syndrome (Fig. 3.47), is another brainstem stroke caused by the occlusion of the penetrating branches of the posterior cerebral artery. The location of the vascular lesion is somewhat lateral to the location for Weber's syndrome and involves the medial lemniscus and red nucleus. Occlusion of those penetrating branches causing Benedikt syndrome produces contralateral hemianesthesia, involuntary movements of the opposite side limbs (involvement of red nucleus), and tremor (involvement of dentatorubrothalamic tract) which is in addition to third cranial nerve palsy. Radiologically, it can often be difficult to distinguish Benedikt from Weber's syndrome.

3.12.3 Locked-In Syndrome

Also known as cerebromedullospinal disconnection or pseudo-coma or de-efferented state is caused by the occlusion of the basilar artery, which supplies the pons. The syndrome is easy to identify as a collection of bilateral long tract signs (motor and sensory) supplemented by fifth to eighth cranial nerve dysfunctions, which result in quadriplegia and inability to speak. Locked-in syndrome patients are aware and awake but cannot move or communicate verbally due to

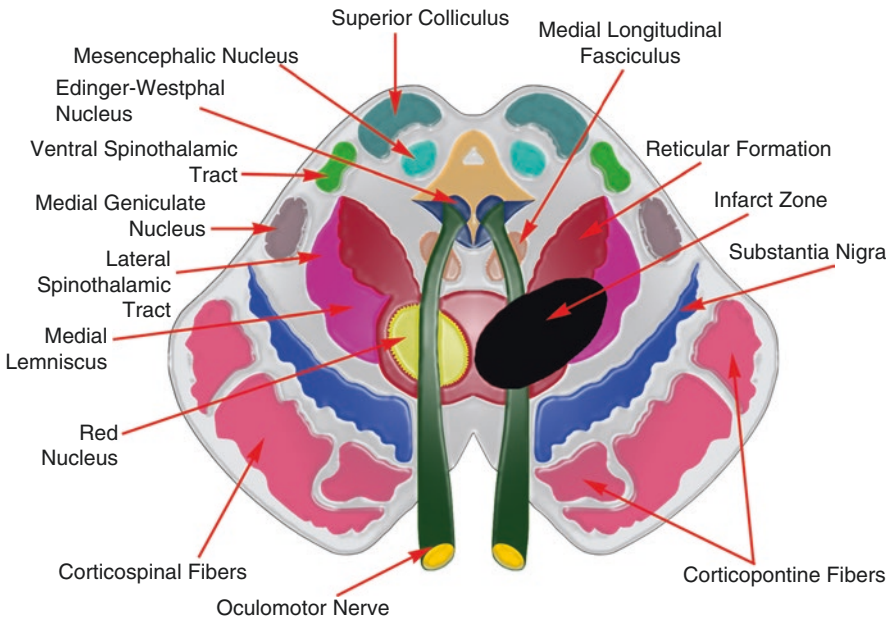


Fig. 3.47 An illustration of midbrain lesion in Benedikt syndrome

complete paralysis of nearly all voluntary muscles in the body except for the eyes. These patients may use their spared movements of the eyes and blinking to communicate with others. Bilateral corticospinal tracts involvement causes weakness or paralysis of both upper and lower extremities, and bilateral corticobulbar tracts involvement causes facial muscle weakness, dysarthria, and dysphagia. Extensive lesions involving bilateral sixth cranial nerves will cause horizontal gaze weakness, and in such cases, the vertical gaze will be the only eye movement possible. Respiratory failure is the common cause of death for many patients with locked-in syndrome.

3.12.4 Medial Pontine Syndrome (Foville's Syndrome)

The medial pontine syndrome (Fig. 3.48) most commonly results from the occlusion of the paramedian branches of the basilar artery, which supplies the medial territory of the pons. Except for the different distinguishing cranial nerve features, the clinical features of patients presenting with the medial pontine syndrome is similar to those presenting with the medial medullary syndrome. The spastic weakness of the contralateral arm and leg (involvement of corticospinal tract), loss of vibration, kinesthetic and position sense of the contralateral arm and leg (medial lemniscus involvement), and strabismus and lateral gaze palsy (involvement of ipsilateral sixth cranial nerve) are the common features of this syndrome.

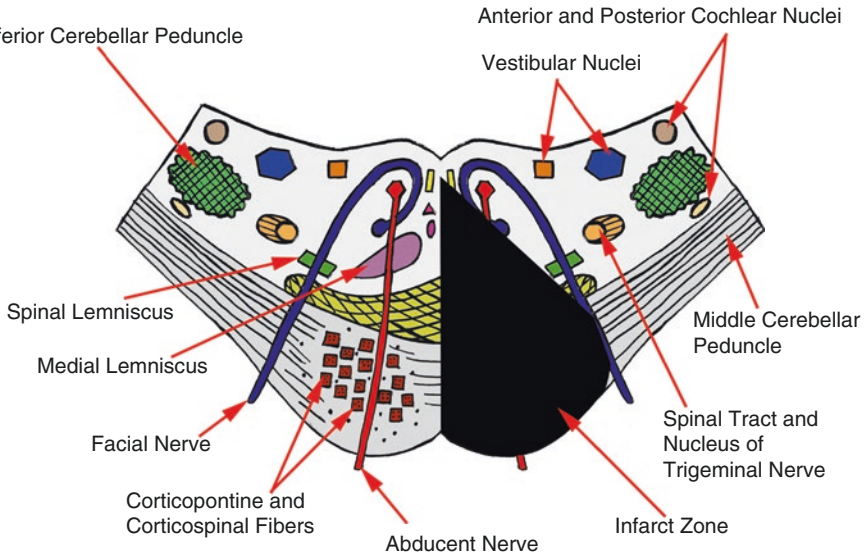


Fig. 3.48 Illustrating the pontine lesion in medial pontine syndrome

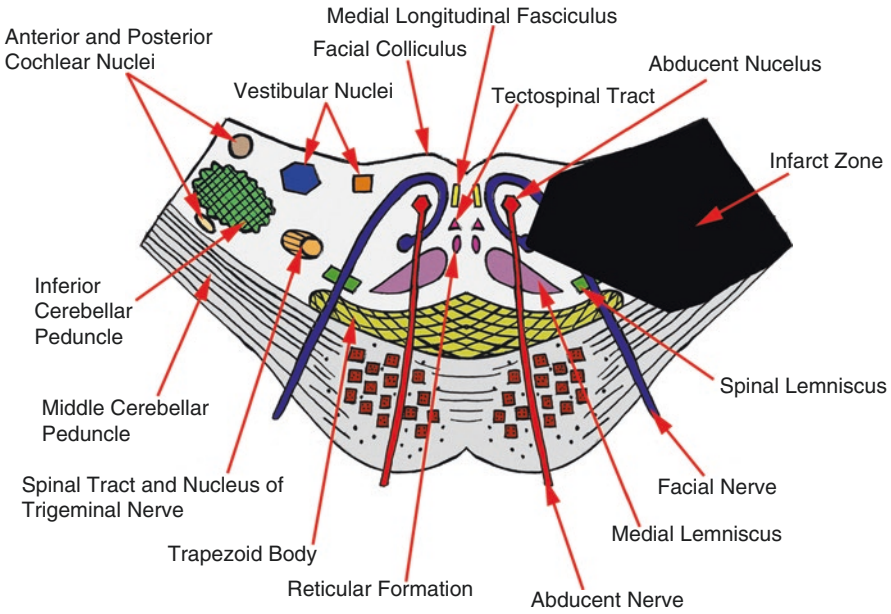


Fig. 3.49 Illustrating the pontine lesion in lateral pontine syndrome

3.12.5 Lateral Pontine Syndrome (Marie-Foix Syndrome)

The occlusion of the anterior inferior cerebellar artery that supplies the lateral territory of the pons causes the lateral pontine syndrome (Fig. 3.49). The clinical manifestations of this syndrome are very similar to those of lateral medullary syndrome, except for different distinguishing cranial nerve features. Lateral pontine syndrome patients typically present with pain and temperature loss of the contralateral trunk and extremities, ataxia of the ipsilateral extremities, gait ataxia, ipsilateral pain and temperature loss over the face, ipsilateral lower motor neuron facial palsy, reduction of ipsilateral lacrimation and salivation, loss of taste from the anterior two-thirds of the tongue, loss of corneal reflex, ipsilateral hearing loss, nystagmus, nausea, vertigo and vomiting, and Horner's syndrome.

3.12.6 Millard-Gubler Syndrome

Millard-Gubler syndrome, also known as ventral pontine syndrome (Fig. 3.50), is caused by the unilateral vascular lesion of the ventrocaudal pons involving the basis pontis and the fascicles of sixth and seventh cranial nerves. The occlusion of the short circumferential or paramedian branches of the basilar artery results in this

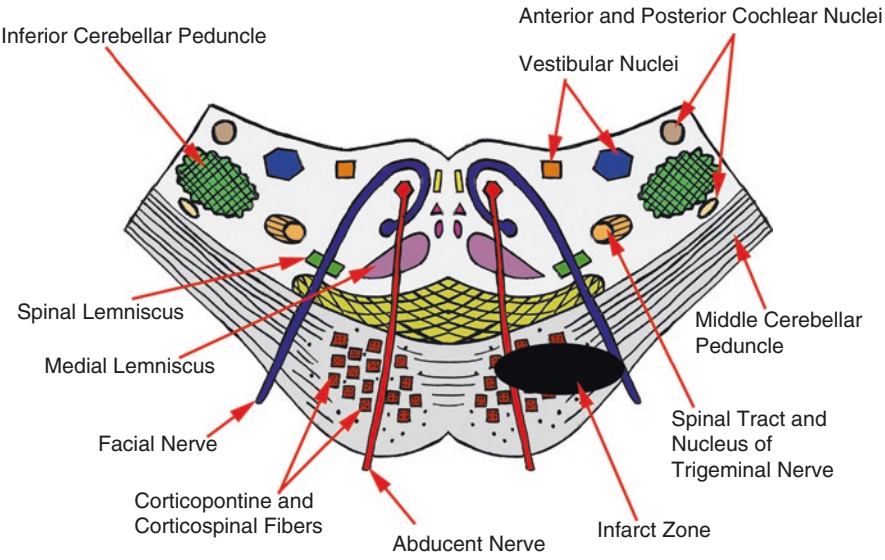


Fig. 3.50 An illustration of pontine lesion in Millard-Gubler syndrome

syndrome. Paralysis of the sixth cranial nerve leads to diplopia, internal strabismus, and inability to rotate the affected eye outward. The involvement of the seventh cranial nerve causes ipsilateral lower motor neuron facial paralysis with loss of corneal reflex and contralateral hemiplegia (sparing the face) due to pyramidal tract involvement. The unique feature of the Millard-Gubler syndrome is the "crossed hemiplegia," i.e., ipsilesional lower motor neuron facial paralysis and contralesional hemiplegia.

3.12.7 Lateral Medullary Syndrome of Wallenberg

Lateral medullary syndrome (Fig. 3.51), also known as Wallenberg syndrome, usually occurs due to the occlusion of the posterior inferior cerebellar artery, if not the vertebral artery branch supplying the vascular territory. It is the commonest of brainstem strokes and involves the dorsolateral medulla and the cerebellum. The clinical features of the lateral medullary syndrome consist of ipsilateral hemisensory loss of pain and temperature sensation of the face due to involvement of the fifth cranial sensory nucleus (the spinal nucleus of the trigeminal nerve) and contralateral hemisensory loss of pain and temperature due to involvement of lateral spinothalamic tract. The involvement of the inferior cerebellar peduncle leads to ipsilateral ataxia of the extremities, gait ataxia, and postural asymmetry. In addition to nausea, vomiting, and vertigo, the involvement of the vestibular nucleus causes ipsilateral nystagmus. Paralysis of the ipsilateral palatal and laryngeal muscles

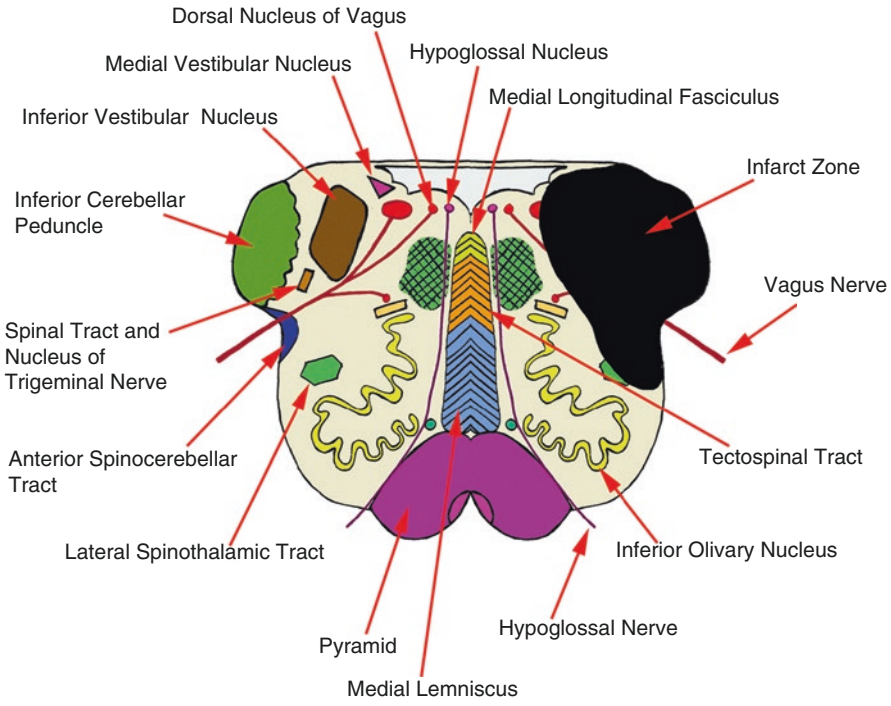


Fig. 3.51 Illustrating the medullary lesion in lateral medullary syndrome of Wallenberg

causes dysphagia and dysarthria, which is in addition to hoarseness and uncontrollable hiccups caused by the involvement of the nucleus ambiguus. The lateral medullary syndrome patients will also have ipsilateral Horner syndrome due to the involvement of the descending sympathetic fibers.

3.12.8 Medial Medullary Syndrome of Wallenberg

Inferior alternating syndrome, hypoglossal alternating hemiplegia, lower alternating hemiplegia, and Dejerine syndrome are the synonyms of the medial medullary syndrome. The medial medullary syndrome (Fig. 3.52) syndrome commonly occurs due to the occlusion of the vertebral artery, which supplies the medial aspect of the medulla. The syndrome presents with contralateral hemiparesis due to the involvement of the pyramidal tract. Medial lemniscus involvement causes contralateral impairment of position and kinesthetic sensations and discriminative sensations. The 12th cranial nerve involvement results in ipsilateral paralysis of tongue muscles and deviation of the tongue to the ipsilateral side.

Table 3.17 provides information about some of the selective brainstem stroke syndromes. Many times the infarctions within the brainstem are patchy and neither

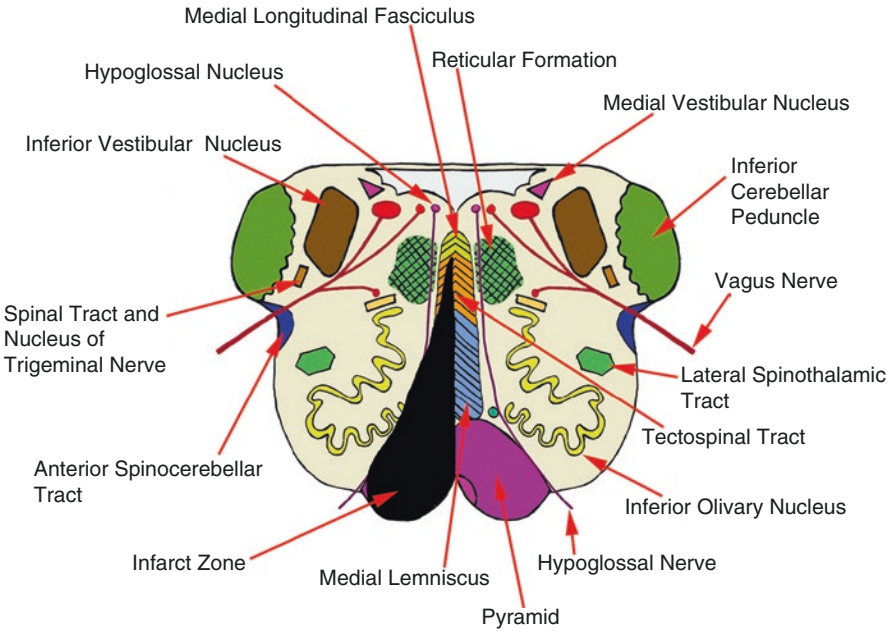


Fig. 3.52 An illustration of medullary lesion in medial medullary syndrome

Table 3.17 Details of certain selective brainstem stroke syndromes

Syndrome name	Lesion location	Artery involved	Cranial nerves involved	Tracts involved	Signs/features
Weber	Base of midbrain	Paramedian branches of posterior cerebral artery or perforating branches from basilar bifurcation	3rd cranial nerve	Corticospinal	Oculomotor palsy, crossed hemiplegia
Claude	Midbrain tegmentum	Perforating branches of posterior cerebral artery	3rd cranial nerve	Red nucleus and brachium conjunctivum	Oculomotor palsy, contralateral cerebellar ataxia and tremor
Benedikt	Midbrain tegmentum	Paramedian penetrating branches of posterior cerebral artery or basilar artery	3rd cranial nerve	Red nucleus	Oculomotor palsy, contralateral abnormal movements

Table 3.17 (continued)

Syndrome name	Lesion location	Artery involved	Cranial nerves involved	Tracts involved	Signs/features
Nothnagel	Midbrain tectum	Penetrating branches of basilar artery	Unilateral or bilateral third cranial nerve	Superior cerebellar Peduncles	Oculomotor palsy, ipsilateral cerebellar ataxia
Locked-in	Bilateral pons	Basilar artery	5th to 12th cranial nerves	Corticobulbar and corticospinal tract	Quadriplegia and fifth to 12th cranial nerve palsies
Millard-Gubler	Caudal ventral medial pons	Short circumferential or paramedian branches of basilar artery	Fascicles of the 6th and seventh cranial nerves	Corticospinal tract	Abduction and peripheral facial palsy, contralateral hemiplegia
Foville	Caudal tegmental medial pons	Perforating branches of basilar artery	6th and 7th cranial nerves	Corticospinal tract, medial lemniscus, medial longitudinal fascicle	Gaze and peripheral facial palsy, contralateral hemiparesis (and hypesthesia, internuclear ophthalmoplegia)
Marie-Foix	Lateral caudal pons	Anterior inferior cerebellar artery	Nil	Middle cerebellar peduncle, corticospinal and spinothalamic tracts	Ipsilateral ataxia, contralateral hemiparesis and spinothalamic sensory loss
Wallenberg	Medulla, lateral tegmentum	Posterior inferior cerebellar artery or rarely vertebral artery	Spinal nucleus of 5th nerve, 9th to 11th cranial nerves	Lateral spinothalamic tract, descending sympathetic fibers, spino- & olivo-cerebellar tracts	Ipsilateral 5th and 9th to 11th cranial nerve palsies, Horner's syndrome, cerebellar ataxia; contralateral pain and temperature deficit
Déjerine	Medial medullary region	Vertebral artery and/or paramedian branches of the anterior spinal artery	12th cranial nerve	Corticospinal, medial lemniscus	Ipsilateral tongue palsy, contralateral hemiplegia, and dorsal column sensory loss

follow the theoretic distribution of a major artery nor present the classical features of complete clinical syndromes. Individual variations in collateral circulation and overlap between the arteries that supply the brainstem are the possible reasons for such diversities.

3.13 Physiotherapy for Brainstem Stroke Syndromes

Though there is extensive literature on physiotherapy for strokes affecting the cerebral hemispheres, there is a paucity of literature on the physiotherapy of brainstem strokes. Overall the brainstem strokes are less frequent than hemispheric strokes and many of the patients with brainstem strokes may experience minimal neurological or functional deficits after stroke. Alternatively, a certain percentage of patients may experience catastrophic brainstem stroke, typically affecting bilateral pontine vascular territory, with low survival rates or profound neurological or functional deficits that preclude their participation in rehabilitation programs. Though conditions like locked-in syndrome have a bleak prognosis, the current literature indicates that within the first year post-stroke, the brainstem stroke survivors have a 35% probability of returning to independent living compared to a 22% probability of hemispheric stroke survivors.

In addition to gaze palsies and cranial nerve deficits, brainstem strokes can lead to several physical impairments, including hemiplegia or quadriplegia, somatosensory deficits, and ataxia leading to gait deviations, reduced balance, and safety. Sensorimotor deficits can also reduce the strength of muscles of the affected side extremities. The brainstem stroke patients can have dissociated sensory loss like pain and temperature loss in lateral medullary syndrome or loss of vibration and kinesthetic sensations in medial medullary syndrome. The impairments mentioned above can lead to functional limitations in bed mobility, transfers, self-care, and gait.

The therapist should perform a detailed evaluation of the patient to derive the treatment objectives and goals and means of treatment. Since the sensorimotor impairments and functional deficits are diverse in various brainstem stroke syndromes, the strategies selected for treatment should be based on the clinical presentation, the problem list, and the functional limitations of the patient. To a certain extent, the standard physiotherapy exercises designed to improve sensation, ROM, muscle strength, postural control, balance, transfers, locomotion, and stair climbing can address most of the issues seen in these patients. The treatment strategies should incorporate activities that encourage postural correction, trunk and pelvis activation, sitting and standing balance, and shoulder and scapular mobility. The overground exercises should advance from the stable to the unstable surfaces, and if the facility for aquatic therapy is available, the latter can be used for improving the posture, balance, and strength of the patients. Task-related motor training can improve the patients' balance capability while sitting, standing, and performing reaching activities. For those patients with mild to moderate paresis, a restorative approach focusing on improvement of the motor function should incorporate strengthening

exercises, overground exercises, treadmill training with partial body weight support, and functional electrical stimulation. The use of a biofeedback unit, training using a mirror and balance training with a balance board can be attempted for those patients with a scope of further balance improvement. Facial muscle strengthening exercises and exercises to improve the tongue muscle strength may reduce the dysarthria of the patients.

Approximately 70% to 80% of brainstem stroke patients are likely to be ataxic and these patients suffer from movement inaccuracies including errors in timing, irregular trajectories, delayed initiation of movements, movement decomposition, and imprecision during reaching activities. The therapeutic exercises specific to cerebellar ataxia can include Frenkel's exercise, other exercise strategies for dysmetria, postural and balance control training, and strengthening exercises. For further understanding of cerebellar ataxia and the strategies related to it, the author advises the readers to read the chapter "Cerebellar Dysfunction".

Locked-in syndrome patients will be near completely dependent with considerably reduced mobility and are most likely to develop serious complications due to immobilization. Though the benefits of exercise after stroke have been widely reported, the present evidence concludes that exercises have no adverse effects and may benefit locked-in syndrome patients. The quality of the existing evidence for the benefits of exercise for the locked-in syndrome is relatively low, as the study designs and interventions were heterogeneous; therefore the outcomes cannot be generalized. Typically, these patients require the same care and management strategies that are given for those patients who have quadriplegia following higher cervical spinal cord injuries.

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