

Stroke is defined as a sudden neurological deficit caused by focal vascular lesion in the brain.

The vascular lesion can be either a hemorrhagic or ischemic involving the blood vessels supplying various parts of the brain. The extent of neurological involvement may range from mild motor deficit to gross involvement of various function namely sensorimotor, perceptual, emotional, behavioral, memory, intelligence, speech and language function.

ETIOLOGY

Different mechanisms have been found to cause vascular insufficiency to the brain resulting in stroke. However, the most common causes are:

1. *Thrombus*: This is mainly due to the presence of atherosclerotic plaque in the cerebral arteries as a result of severe platelet adhesion, fibrinous coagulation and decreased fibrinolysis activity.
2. *Emboli*: These are free flowing bodies in the cerebral blood stream in the form of dislodged thrombus, fats, air, tissue particle, etc. which gets trapped at any point along their course, frequently at the bifurcation of the arteries, and cause occlusion to the cerebral circulation.
3. *Hemorrhage*: It occurs due to rupture of the blood vessels in the brain. Following hemorrhage tissue death occurs due to both ischemia and mechanical injury to the brain substance as a result of compression by the clot. Hemorrhage usually occurs either due to hypertension, arteriovenous malformation or even due to trauma.

There are some risk factors that can predispose to stroke. The common ones are diabetes, high blood pressure, and cardiac disorders. The chances of these three occurring in combination

increases with age. Patients with increased hematocrit count are at a higher risk of getting stroke. Cardiac disorders like valvular heart diseases, endocarditis, atrial fibrillation, or postcardiac surgery patients are at a higher risk of contacting stroke. Other secondary risk factors that can increase the chances of stroke attack are cigarette smoking, obesity, sedentary lifestyle, increased consumption of high fat diet, psychological stress, and excessive alcohol intake.

PATHOPHYSIOLOGY OF CEREBRAL ISCHEMIA AND CONSEQUENT INFARCTION

The two pathophysiological changes leading to cerebral infarction are loss in the supply of oxygen and glucose due to vascular occlusion, and various changes in cellular metabolism consequent as a result of collapse of energy producing processes with disintegration of cell membranes.

Complete occlusion to brain substance causes severe damage to it with a zone of infarction, which is however found to be smaller than the actual area supplied by the involved artery. *The margin of this infarcted zone consists of cells that are alive but metabolically less active. These surrounding areas are termed as ischemic penumbra.* These areas are nourished by meningeal collateral.

The necrotic tissue swells rapidly mainly due to excessive intercellular and intracellular water content. Also, lack of O_2 is another factor that can contribute to swelling.

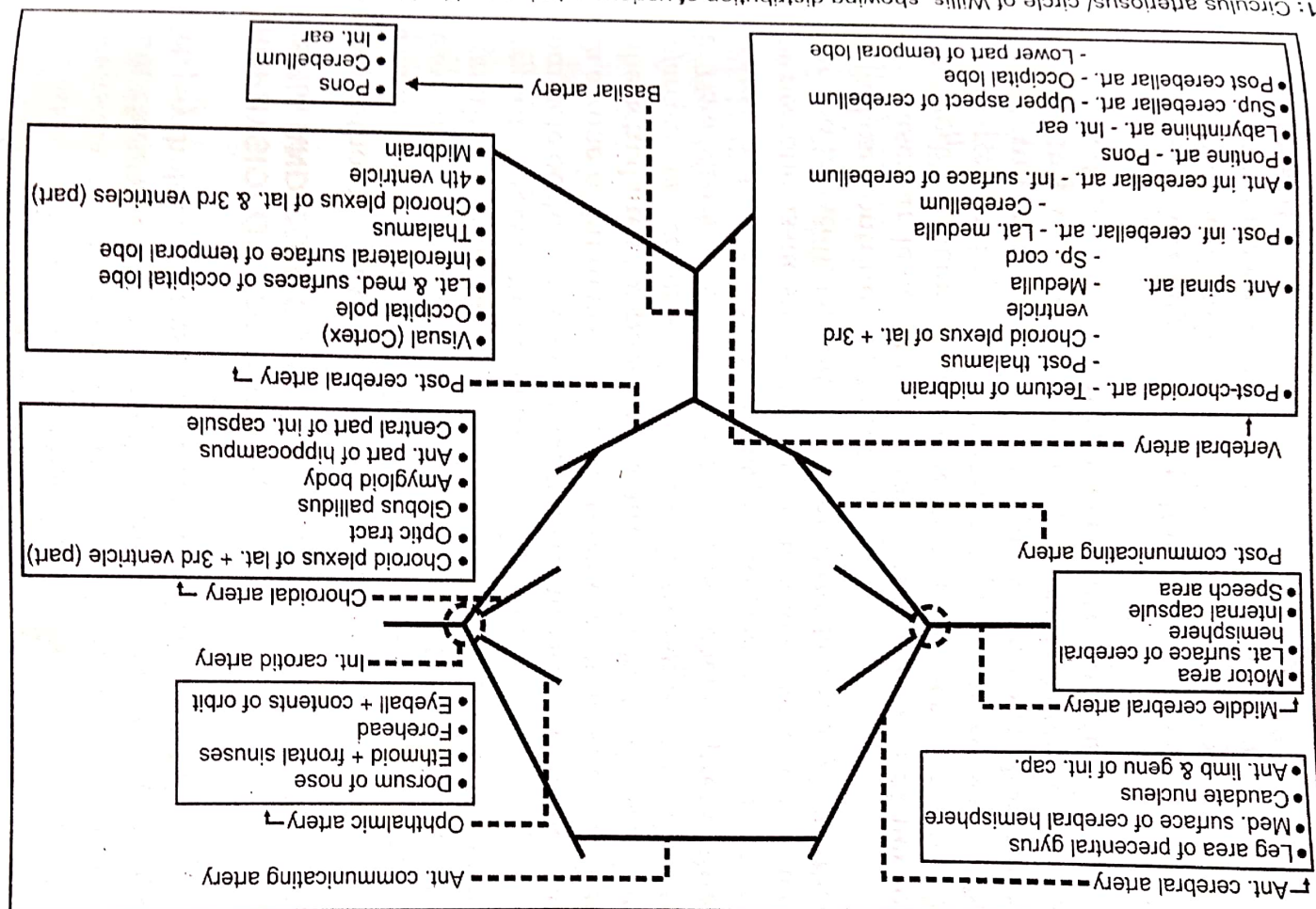
This vascular lesion to the brain causes release of neurotransmitters like glutamate and aspartate by the ischemic cells, which excites neurons and produces an intracellular influx of Na and Ca leading to irreversible cell damage. Thus recent research attempts at blocking this action of glutamate and aspartate on nearby cells, which will reduce the secondary involvement of surrounding viable cells.

Cerebral edema begins within few minutes and reaches a maximum by about 4 days, however it mostly disappears by 3 weeks. This edema can increase the intracranial pressure and can even cause contralateral and caudal shift of brain structure (Fig. 2.1).

Neurovascular Syndromes

Internal carotid artery occlusion in its proximal segment immediately after its generation from the common carotid artery is often silent.

1: Circle of Willis, showing distribution of various arteries and its blood supply to various areas in the brain



Middle Cerebral Artery

<i>Structures involved</i>	<i>Neurological deficit</i>
Motor area of face, arm, and fibers descending from leg area to enter the coronal radiata	Paralysis of contralateral face, arm and leg
Somatosensory area of face, arm and face leg	Sensory impairment over contralateral and leg
Motor (Broca's) area on dominant hemisphere	Motor speech disorder
Central language area and parieto-occipital cortex of the dominant hemisphere	Central aphasia, word deafness, anomia, jargon speech, alexia, agraphia, acalculia, and finger agnosia
Non-dominant parietal lobe	Perceptual disorder like unilateral neglect, anosognosial unawareness of hemiplegic side, apraxia, and spatial disorganization
	Homonymous hemianopia and loss of conjugate gaze to the opposite side
Parietal lobe	Ataxia of contralateral limbs
Bilateral frontal lobe	Brun's ataxia or apraxia of gait
Supramarginal gyrus or inferior parietal lobe	Loss or impairment of optokinetic nystagmus
Posterior limb of internal capsule and adjacent corona radiata	Pure motor hemiplegia without sensory and visual involvement

Infarction secondary to proximal MCA occlusion often produces significant cerebral edema with increased intracranial pressures and may lead to brain herniation, coma or death.

Anterior Cerebral Artery

<i>Structure involved</i>	<i>Neurological deficit</i>
Motor leg area	Paralysis of opposite foot and leg
Involvement of arm area	Involvement of opposite arm although this is rare
Sensory area of foot and arm	Cortical sensory loss over foot and leg

Contid...

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Structure involved	Neurological deficit
Bilateral involvement of posteromedial part of superior frontal gyrus	Urinary incontinence
Medial surface of posterior frontal lobe	Contralateral grasp reflex, sucking reflex and gegenhalten (paratonic rigidity), frontal tremor
Severe frontal lobe infarction	Memory loss and behavioral impairments
Supplementary motor area of dominant hemisphere	Aphasia
Corpus callosum	Apraxia and agraphia
Bilateral motor area of leg	Cerebral hemiplegia

Homonymous hemianopia never occurs in ACA stroke.

Posterior Cerebral Artery

Structure involved	Neurological deficit
Thalamus	Hemianesthesia (contralateral sensory loss) or thalamic sensory syndromes (unpleasant hemibody sensation with spontaneous pain)
Occipital cortex	Homonymous hemianopia, visual agnosia, prosopagnosia
Bilateral occipital cortex involvement	Cortical blindness
Temporal lobe ischemia	Amnesic syndrome with memory defect
Midbrain	Skew deviation, athetoid posturing, postural tremor, hemiballismus, Contralateral hemiplegia
Cerebral peduncle	Decerebrate attacks
Motor tract between red and vestibular nuclei	

Vertebrobasilar Artery

The basilar artery supplies the pons, inner ear and cerebellum. Complete occlusion is often fatal. Progressive lesion often starts with occipital headache, and then causes diplopia, hemiplegia, quadriplegia and finally coma.

Locked in syndrome is seen in lesion involving the ventral pontine nuclei with a complete basilar syndrome without affection of the reticular system and is described as anarthria, quadriplegia, but preserved consciousness. Alertness and gaze are intact, infact the only communication that can be established is through vertical eye movements. Prognosis of this condition is very poor and the residual disability in surviving population is severe.

Vertebrobasilar artery system occlusion can produce either ipsilateral or contralateral symptoms depending upon whether the tracts involved indirectly have already crossed or uncrossed. Cranial nerve abnormalities along with cerebellar symptoms can occur. Symptoms such as visual loss, homonymous hemianopia, diplopia, facial numbness, or weakness, tinnitus, dysarthria, or dysphagia may be seen.

Hemiparesis of one or more limbs can occur with involvement of corticospinal tract.

Paresthesia of the face or limbs can occur with involvement of the lemniscal system. Pain and temperature sensation on the opposite side will be involved in the ischemia of the spinothalamic tract involvement.

Reticular spinal tract involvement causes alteration in the level of consciousness. Horner's syndrome due to sympathetic tract involvement is rare. *Sudden loss of tone in the lower limbs as a result of involvement of the medullary pyramids is termed as Drop attack* can also result in vertebrobasilar artery syndrome.

TIA (transient ischemic attack) due to affection of vertebrobasilar artery is very common than carotid artery.

CLINICAL MANIFESTATION

Sensory

The sensory involvement in a stroke patient depends upon the site and the extent of the lesion. Touch, pain, temperature, proprioceptive sensation may be involved to a variable degree. The affection of

these sensation is more in case of the lesion involving the thalamus, which can even cause *contralateral hemianesthesia*. Involvement of the somatosensory area of the cortex causes loss of cortical sensation, namely *tactile localization*, *tactile discrimination*, *stereognosis*, and *tactile extinction* (which is inability to appreciate the stimulus on the affected side when it is applied simultaneously with the stimulus on the unaffected side, although the patient does perceive the stimulus on the side when the stimulus is applied alternately).

A common visual disturbances seen is *homonymous hemianopia* in which there is loss of vision in the nasal half of opposite eye and the temporal half of the eye on the hemiplegic side. This usually occurs due to lesion of the optic radiation in the internal capsule or primary visual cortex.

Crossed anesthesia can be seen if the lesion involves the upper part of the pons in which there is anesthesia on the same side of the face and on the opposite side of the trunk and limbs.
Lack of *Conjugate gaze* may be noted.

Motor

Immediately after the onset of stroke, there is a stage of cerebral shock with flaccidity and areflexia. Gradually, this is replaced by development of spasticity, hyperreflexia, and abnormal mass movement pattern, which is termed as synergy.

The duration of flaccidity may vary from days to weeks to infinite. In some rare cases (conditions in which there is pure pyramidal tract lesion) signs of mild spasticity may be seen in certain groups of muscles like elbow flexors, wrist flexors, quadriceps and calf, but total predominating spasticity never sets in.

However majority of the cases go through a common sequence of prognosis, which according to Brunstorm comprised of five stages (Table 2.1). This was further simplified by Bobath into only three stages (Table 2.2). Thus it is clear that 95 percent of hemiplegics may vary depending upon various factors, and the progress may become stagnant at any point of time.

This development of hypertonicity has been termed as a positive reaction by Jacksonian as it enables the patient to gain some level of dependency. The other response is a negative response, which occurs due to disruption of the higher facilitatory influence on the spinal

Table 2.1: Stages according to Brunstom

Stage 1	Flaccid and no movements
Stage 2	Spasticity begins and basic limb synergies may be seen as associated reaction
Stage 3	Limb synergies are produced voluntarily
Stage 4	Some movement combination not belonging to synergy may appear, spasticity starts declining
Stage 5	More difficult movement combinations are learned if improvement
Stage 6	Spasticity disappears, isolated movement possible and coordination also can become normal

Table 2.2: Bobath's stage

1. Flaccid
2. Spastic
3. Stage of spontaneous recovery

nerves. The negative response gives rise to weakness or paresis, which is however masked by the predominant spasticity in the patient.

Spasticity

Severe degree of spasticity will make movements impossible; moderate spasticity will allow for some slow movements but they will be performed with too much effort and abnormal coordination; mild spasticity will allow for gross movements with normal coordination but fine movements of the limbs especially involving the distal portion will be difficult or impossible.

In hemiparesis there are some muscles which are always involved and which consequently give rise to a peculiar abnormal mass movement pattern and attitude that is characteristic of many hemiparesis patient. The spasticity invariably affects the antigravity muscle for reasons not clearly understood. It is however presumed that this antigravity muscle are relatively more stretched than progravity muscles in neutral position hence stimulating the stretch reflex giving rise to spasticity.

The distribution of spasticity in the upper and lower extremities is as follows (Table 2.3):

Table 2.3: Distribution of spasticity in upper and lower extremities

Upper extremities	Lower extremities
1. Shoulder girdle: depressor and retractor	1. Pelvic girdle: retractor
2. Shoulder: internal rotators and adductors	2. Hip: extensor, adductor, and internal rotators
3. Elbow: Flexors	3. Knee: Extensors
4. Wrist and finger: Flexor	4. Ankle and toes: Plantar flexors
5. Forearm: pronators	and supinators

Spasticity also involves the muscles of the trunk and neck on the opposite side causing lateral bending of patient towards the spastic side

Synergy

Synergy patterns are abnormal, stereotyped, primitive, mass movement pattern associated with spasticity and which can be triggered either reflexly or voluntarily.

Synergy can be either flexor or extensor. They involve the action of certain muscles in combination that gives rise to an abnormal movement pattern not useful for functional activities. This abnormal synergies are presented in Table 2.4.

Table 2.4: Abnormal synergies in upper and lower limbs

	Upper limbs	Lower limbs
Flexion synergy	Shoulder girdle retraction and elevation, shoulder abduction external rotation, supination flexion of elbow, wrist and finger flexion	Hip flexion abduction and lateral rotation, knee flexion, dorsiflexion and inversion
Extension synergy	Shoulder girdle protraction and depression, shoulder adduction, internal rotation, elbow extension, pronation wrist and finger flexion	Hip extension, adduction, internal rotation, knee extension, ankle plantar flexion, inversion and toe plantar flexion

Thus it can be observed that the following muscles does not take part in either of the synergies.

- Latissimus dorsi
- Teres major
- Serratus anterior
- Wrist and finger extensors
- Ankle evertors

It is highly essential to understand that synergies are different from the abnormal attitude seen in a hemiparesis patient. The abnormal hemiplegic attitude is due to the combination of strongest component of the flexor and extensor synergy in both upper and lower limbs. The strongest components for the upper limb in the flexor synergy are shoulder girdle retraction, elbow flexion, wrist and finger flexion. The strongest components of the extensor synergy for the upper limb are shoulder girdle depression, shoulder adduction, internal rotation, and pronation.

The strongest components of the flexor synergy for the lower limb are lateral rotation, and inversion. The strongest components of the extensor synergy for the lower limbs are pelvic retraction, hip adduction, hip extension, knee extension and plantar flexion.

Hence, a hemiplegic patient has an attitude of shoulder retraction and depression, shoulder adduction and internal rotation, elbow flexion, pronation and wrist and finger flexion. The lower limb adopts an attitude of pelvic retraction, hip extension, adduction and external rotation, knee extension, and ankle plantar flexion and inversion. This is shown in the Figure 2.2 (Plate 1).

Reflexes

In the stage of flaccidity all the reflexes are suppressed or absent. As the patient enters into the stage of spasticity, the reflexes reoccur. The deep tendon reflexes become hyperactive, in fact, in case of increased spasticity there may be presence of clonus especially of the tendo Achilles and wrist flexors. Superficial reflexes are lost but plantar responses show Babinski sign positive.

Release of primitive reflexes: Due to the lesion in the brain the other primitive reflexes that are produced by the lower level of CNS are released from the inhibitory influences of the higher centers. The common primitive reflexes that are seen in a hemiplegics are ATNR

- ii. *Symmetrical tonic labyrinthine Reflex (STLR)*) Supine position causes a increase in extensor tone and prone position increase the tone in the flexor muscles.
- iii. *Tonic lumbar reflex (TLR)* Rotation of the trunk to the hemiplegic side over the pelvis causes flexion of the hemiplegic upper limb and extension of the hemiplegic lower limbs. Rotation to the opposite side causes the opposite effect.
- iv. *Flexor withdrawal* Sensory stimulation to the sole of the foot causes a sudden hip-knee flexion. Infact in some patient this may be so dominating that it may interfere in the assessment of plantar response.
- v. *Positive supporting reaction* Pressure at the bottom of the foot causes co-contraction of the lower limb muscles making the limb like a rigid pillar.

Associated Reaction

Associated reactions are tonic postural reaction in muscles. They are abnormal reaction, which usually manifest itself as an attenuation of the hemiplegic's attitude in response to movement either on the same side of the body or occurring in any other parts. These abnormal positioning of the limb becomes very obvious during ambulation. These abnormal associated reactions should be differentiated from the normal associated movements (also called synkinetic movements), e.g. swinging of the arm while walking.

Impairment of Higher Level Reactions

Reflexes that are controlled by higher level of the brain like, righting reactions, equilibrium reactions, automatic adaptations of the muscles to the change of postures are all impaired.

Righting reactions: These are automatic responses which maintain the normal position of the head, trunk and limbs with respect to their alignment to each other. They help in carrying out various functions like rolling, getting up, sitting and standing.

Equilibrium reactions: These are normal responses, which help us in maintaining the balance during all our activities especially when we are in a danger of falling.

Automatic adaptation in postural tone: The automatic adjustment that takes place in order to allow for normal movement or maintenance of posture is termed as automatic postural tone. Examples: The contraction of erector spinae while bending forward and thus preventing a fall. Eccentric contraction of the quadriceps muscle while squatting is another example of automatic adjustment of the muscles. Similarly synergistic fixation of the proximal parts to allow movement to take place distally is also an example of postural adaptation of muscles.

Also in hemiplegics there may be impairment of *protective reactions* like extension of the upper limbs to avoid injuries during a fall.

Speech and Language Disorders

This occurs due to lesion involving the dominant parietal lobe. Three types of speech disorders have been seen following a stroke.

Broca's/motor/expressive/non-fluent aphasia: In this type of aphasia the patient's ability to understand is intact, but his ability to respond is affected due to involvement of the Broca's area. He has difficulty in articulating his speech.

Wernicke's/sensory/receptive/fluent aphasia: The patient's ability to speak is intact, in fact his speech is very fluent, but he doesn't understand or comprehend. His speech is totally irrelevant.

Global/conductive or total aphasia: In this type there is loss of understanding as well as production of speech. This occurs due to lesion of both the Broca's and Wernicke's area or may be due to the involvement of the conduction zone between these two.

Apraxia and Agnosia

Apraxia is inability to carry out learned purposeful movement despite the presence of a good motor, sensory, or coordination function. It is caused due to lesion of the dominant parietal lobe. Bilateral apraxia commonly involves lips and tongue. When the patient is asked to purse the lips or protrude the tongue, he won't be able to do it. But the patient may be found doing the same thing having no awareness of the error.

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Ideomotor is the type in which patient understands the purpose of movement but is unable to carry out on command but does it automatically.

Ideational is characterized by extreme absent-mindedness. Patient fails to perform purposeful movement both spontaneously and on command.

Constructional apraxia is difficulty in spatial organization of movement or objects, e.g. inability to imitate simple arrangements of blocks or a simple drawing.

Agnosia is failure to recognize objects despite having an intact visual, auditory, and tactile sensation, due to the lack of association pathways to arrange sensory images, memory and disposition towards the action.

Visual agnosia is inability to recognize common objects which is seen clearly by the patient due to lesion in the dominant parieto-occipital regions.

Auditory agnosia is inability to recognize familiar sounds or music due to lesion of dominant temporal lobe although the patient has

The perceptual impairment could be in the form of visuospatial disorders, unilateral body neglect, somatognosia or visual perceptual impairment.

Visuospatial relation disorder: It is difficulty in determining the distance, size, position, and relationship of various parts to the whole. An individual reaching out for an object may over estimate or underestimate the distance, e.g: If the individual has to wash his hand, he may hold the hand next to the tap instead of keeping under it. Also as he is unable to judge the distance the individual have been found to constantly collide with the wall or doorway.

Unilateral body neglect or inattention: The individual does not use the involved limb, i.e. the non-dominant limb, to its available control and infact ignores it. An individual with unilateral body neglect will wash the unaffected side of the body properly but not the affected side. Similarly, other activities like combing, clothing, shaving, face wash, etc. are done in same fashion.

Visuospatial impairment: In this impairment the individual does not respond to or recognize any meaningful stimuli which is present in the side contralateral to the affected non-dominant hemisphere, e.g. for a right dominant individual, the left side of the space appears not to be seen. The patient may eat only that part of the food kept on the right side of the plate. Also, while reading the newspaper the patient will read only that matter which is present on the right side of the page.

Somatognosia: This is also called body scheme or body image dysfunction. The patient will be unable to differentiate between mirror image and self. The individual may attempt to comb his mirror image instead of himself. Also, the patient may not be able to differentiate between his own body parts and that of others, hence, he may try to wear a watch in someone else wrist.

Visual perceptual impairment: Distortion in the perception of vertical and horizontal plane, which leads to frequent falling. Also, the patient have topographical disorientation (difficulty in finding way from one place to another). He will also have difficulty in telling time, visualizing mental picture of familiar person and figure ground impairment (inability to differentiate an object from the back ground).

Cognitive and behavioral changes occurs in lesion involving either of the cerebral hemisphere. *Patients with left hemisphere damage are usually very depressed, low profile, anxious and have a negative attitude towards life.* They are often cautious, insecure, slow in their movement and hesitant in their approach towards any activity. These patients need lot of encouragement and positive feedback from the therapist.

Patients with right hemisphere lesion are often euphoric, overconfident, impulsive and over estimate their capability. They often deny that they are disabled and hence the chance of causing injury is more in these individuals. They have to be trained to perform the movement with more caution and slow manner.

Cognitive dysfunction also manifests as difficulty in orientation, attention, conceptual abilities, memory and learning. Short-term memory is usually affected.

Dysphagia

The correlation of stroke with dysphagia remains unclear. However, it has been proved that dysphagia is quite frequently noted in patients with bilateral cerebral hemisphere or brainstem lesion. It is caused only when stroke involves larger cerebral vessels. Dysphagia frequently causes aspiration, which may need hospitalization or even death. The most common reason for aspiration is incomplete laryngeal elevation and closure. Altered sensation, palatal paralysis, defective lip closure and postural imbalance have all been contributing towards dysphagia.

Bladder and Bowel Impairment

In flaccid stage there is overflow incontinence hence continuous catheterization is advisable. Anterior communicating artery strokes involving paracentral lobule can cause loss of voluntary control of micturition. The patient will have sudden uncontrolled evacuation of urination irrespective of the time and place. Also there may be presence of constipation. However, no detail work has been carried out on this disability.

Sexual Dysfunction

Sexual activity is important for many couples throughout their lives. Many individuals are worried about their sexual life but are hesitant or shy to discuss or seek advice for the same.

Stroke may interfere with the sexual activity by affecting the individual desire, libido, erectile or lubrication, orgasm or ejaculation. The disability could be due to depressed state, sensorimotor dysfunction, etc.

Secondary Manifestation

Psychological Dysfunction

Psychological dysfunction are very common after an attack of stroke and are usually manifested as depression, social withdrawal, anxiety, sleep disorder (insomnia), secondary mania, emotional lability (pathological crying and laughing, emotional incontinence), behavior problem (aggressiveness, verbal abusing or yelling, resistive behavior or over dependency) and even personality changes like an introvert can become extrovert and may appear very happy and over talkative.

Musculoskeletal Complications

Musculoskeletal complications set in 95 percent of stroke patients if proper physiotherapy treatment is not given. It occurs mainly due to the combination of muscular imbalance and inactivity. Pain and stiffness are the common ones that affect various joints. However, certain joints are frequently involved like wrist flexors, elbow flexors, shoulder adductor, pronator, plantar flexor, knee extensor, and unilateral tightness of neck and trunk lateral flexors on the spastic side.

Decreased tone in the flaccid stage commonly causes subluxation of the glenohumeral joint on affected side if adequate care is not taken. This subluxation if allowed to persist may even give rise to periarthritis or frozen shoulder in subacute stages.

Pain at the shoulder can trigger the incidence of RSD (reflex sympathetic dystrophy or shoulder hand syndrome), which is abnormally increased sympathetic overflow. It occurs in three stages. Stage I manifested by burning pain and paresthesia, pitting edema (commonly on the dorsum of hand), decrease in wrist flexion range, cyanosis of hand due to vasoconstriction and increased sweating. Stage II in which there is increased pain, decrease in sweating, warm and more firmer hand, osteoporosis involving the bones of the limbs. Stage III gives rise to indurated edema, atrophy of the bones and the muscles. Pain is usually absent in this stage, the hand becomes

cool and deformity occurs due to involvement of the soft tissue around the joints. Claw hand deformity is very commonly seen. Hence close observation by the therapist is essential to prevent the progression into the third stage.

Scalenus anticus syndrome occurs because the patient continuously assumes a posture of head flexion, shoulder internal rotation, scapula retraction. It is manifested as neck and shoulder discomfort with tingling numbness in the hand and fingers.

Deep Vein Thrombosis (DVT)

Deep veins thrombosis in stroke is not as common as in spinal cord injury. It occurs due to accumulation of the clot in the distal blood vessels. It is manifested as pain or tenderness in the calf muscle, swelling, and discoloration of the leg. It can occur in hemiplegia mainly if the patient remains in comatose state for a long time.

Cardiac and Respiratory Deconditioning

Although the cardiac and respiratory systems are not directly affected, decreased physical activity has been found out to be the main cause of deconditioning of these systems. The patient's endurance level is drastically reduced which may adversely affect the rehabilitation process.

Pain

Pain is very common only in stroke affecting the thalamus and is termed as thalamic syndrome, which is characterized by very intense burning pain on the opposite side of body. Pain can also occur as a result of various musculoskeletal complications as discussed above.

Gastrointestinal and Renal Systems

Although, these systems may not be directly involved, rarely, some patients with stroke do exhibit constipation and urinary tract infection or calculus. However the frequency of involvement is very low.

Comparison of Normal and Hemiplegic Gait

Walking is an independent, automatic, symmetrical and economical event. During a normal walking there is both vertical and horizontal

displacement of CG in the form of a sinusoidal wave that causes horizontal displacement of 1.7 inch and vertical displacement of 1.8 inch. The gait determinants are as follows:

- Knee flexion at heel strike on loading which lowers the CG
- Pelvic tilting
- Pelvic rotation which increases with increase in the heel strike
- Foot ankle mechanism
- Knee flexion on heel rise or heel off
- Lateral displacement of pelvis.

Under normal circumstances walking is an automatic process which requires contribution and integration of various mechanism, however after the brain damage like in cases of stroke all the automatic adaptation is lacking and the patient tries to compensate for it by putting more voluntary efforts. The phasic action of muscle groups seen normally is replaced by abnormal muscular combination characteristic of primitive reflexes. The flexion and extension synergy interferes with the walking and greatly influence the hemiplegic gait.

The abnormality of gait in hemiplegic patient can be compared with normal gait taking into consideration the contribution of the joints for various phases of the gait cycle.

Ankle Joint

Early stance (heel strike to foot flat): In normals when the heel strike the ground, the angle at the ankle is 90 degrees once the weight is transmitted to the fore foot, the sole is gradually lowered which is controlled by eccentric contraction of the dorsiflexors to prevent slapping of the foot.

In hemiplegics, the anticipation of the weight bearing excites the extensor synergy which induces plantar flexion instead of dorsiflexion because dorsiflexion is not a part of the extensor synergy. The hemiplegic limb comes down on the entire sole and if the plantar flexion is marked then on the toes. Thus the heel strike is completely missing in hemiplegics. If there is pronounced inversion there is more weight borne by the lateral aspect of the foot and when it is moderate it gets corrected on weight bearing.

Mid stance: In normal when the sole is firm on the ground the leg starts pivoting forwards above the ankle causing a relative dorsiflexion. This happens in response to the weight shift from hind-

foot to the forefoot. The movement of relative dorsiflexion is controlled by eccentric work of the plantar flexors which gets gradually elongated.

In hemiplegics when the calf muscle is stretched by body weight it stimulates the stretch reflex causing increase spasticity of plantar flexors and it prevents any further stretch. Thus the forward shift of the body weight is prevented in hemiplegics.

Late stance (unloading to push off): In normals towards the end of the stance phase the heel raises from ground and the knee flexes. This is achieved by forceful concentric contraction of the plantar flexors for initiating a forward swing.

In hemiplegics, the knee does not flex due to lack of dorsiflexion at the ankle in the earlier phase and also due to the influence of quadriceps spasticity. Sometimes even if it does flex it flexes gradually. Moreover the combination of plantar flexion with knee flexion neither belongs to flexion or extension synergy hence is very difficult for a hemiplegic gait. Due to the lack of push off in hemiplegics the force required to initiate a swing is very less that causes slowness in walking.

Knee Joint

Early and midstance: In normals as the heel strikes the ground the knee is extended but as the body weight shifts on to the loading foot there is 15 degree flexion at the knee under the influence of body weight and it is controlled by eccentric contraction of the quadriceps to prevent buckling. Immediately afterwards due to concentric work of the quadriceps there is extension at the knee.

In hemiplegics patient in flaccid stage there can be buckling due to weight transmission. In stage of spasticity, the initial flexion does not take place and the knee continues to remain in hyperextension throughout. Some patient do have a tendency to buckle initially but immediately rectify by going into hyperextension.

Late stance: In normals the knee flexes with plantar flexion to a give momentum to the swing phase.

In hemiplegics the strong linkage between quadriceps and plantar flexors prevents the swing and the affected limb is moved forward by other compensatory mechanism.

Hip Joint

Early to midstance: In normals during weight bearing the abductors of that hip prevent the pelvis from sagging on the opposite side along with trunk flexors. The hip extensors work from heel strike to midstance. This short burst of activity reduces just before midstance.

In hemiplegics due to activation of the extensor synergy, the adductors contract in place of abductors and hence there is Trendelenburg sign. Moreover when the adductor spasticity is very severe the affected leg is placed very close to the normal limb or may even cross over interfering grossly with the forward propulsion.

Swing Phase

In normals this begins in late stance and is due to combined effort of muscles, gravitational force and momentum. EMG studies have shown that the muscle activity level is minimal. The hip flexors, knee flexor and dorsiflexors act to help in adequate ground clearance.

In hemiplegics the extensor synergy does not let go its grip and if does then it is very slowly so that the body moves forward in slow manner. Hip and knee flexion is absent because of plantar flexion at the ankle and retracted pelvis. Thus there is inadequate ground clearance. In the absence of forward rotation of the pelvis and hip flexion the limb is brought forward by circumduction. Occasionally the limb may be dragged behind with the pelvis posterior, hip in external rotation and toes facing laterally.

Assessment

The physiotherapist should be competent in assessment of a patient with stroke to find about the extent and severity of the involvement, to detect the influence of the secondary musculoskeletal changes that will determine the ultimate functional outcome of the patient.

History

The history of the patient in terms of the onset, duration of the illness, progression, associated features, previous treatment and the present status of the patient should be noted.

ON OBSERVATION

Posture: The patients general posture, the attitude of the upper and lower limb, and the facial symmetry should be checked.

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Other secondary manifestation like skin changes, presence of edema should be noted.

ON EXAMINATION

Higher function testing: The higher function testing includes assessing the memory, intelligence, consciousness, behavior, orientation and speech.

Cranial nerve testing: The cranial nerve should be assessed for their involvement as their affection may interfere in the rehabilitation process.

Sensory Examination

Evaluation of the sensory involvement gives an indication about the severity of the primary lesion and also determines the extent of improvement in these patients.

The following sensory involvement should be carried out:

- *Superficial:* Crude touch and fine touch
- *Deep:* Pressure, joint position sensation, kinesthetic sensation and vibration sensation.
- *Cortical sensation:* Tactile localization and stereognosis.
- Involvement of only the cortical sensation indicates that the lesion in the sensory cortex area. Involvement of the other sensation indicates an extensive subcortical lesion. Absence of sensory involvement indicates that the lesion is only localized to the motor areas of the brain or a reversible lesion with a good prognosis.

Motor Examination

Tone: The tone of the muscles is tested by fast passive movement to determine the distribution and severity of spasticity. It should be done at all the joints. Usually the muscles exhibit clasp knife type of spasticity. Spasticity may be graded as mild, moderate or severe based upon the restriction to passive movement.

Range of motion should be assessed at each joint. The range may be restricted due to the presence of spasticity, contracture, or adhesion within the joint.

Contracture or deformity: It is necessary to find out the muscles or the group of muscles, which has developed contracture. It should be noted whether the contracture is a mild or severe one.

One should know to distinguish between tightness and spasticity. This is essential because the physiotherapy approach will be different in both of these conditions.

Voluntary Control Testing

The brain appreciates movement performed and not the muscles hence it is very essential that in patients with upper motor neuron involvement the control of the patient for isolated movement at each joint should be checked. Earlier concepts of voluntary control testing comprised of six grades.

Grade 0 — No contraction or flicker or initiation.

Grade II — Flicker of contraction present or initiation of movement.

Grade II — Half range of motion in synergy or abnormal pattern.

Grade III — Full range of motion in synergy or abnormal pattern.

Grade IV — Initial half range is performed in isolation and the latter half in pattern.

Grade V — Full range of motion in isolation but goes into pattern when resistance is offered.

Grade VI — Full range of motion in isolation against resistance.

This voluntary control testing should be performed at all the joints and for all the movements. The starting position given to the patient should be either neutral or anti-synergist position and the therapist should ask the patient to perform a specific movement without any other associated movement performed at the same joint or other joint. Presence of contracture or deformity indicates the presence of a mechanical factor and hence should not be considered as a pattern.

However, the (STREAM) scoring of the voluntary movements which is as follows has made the concept of voluntary control testing very simpler.

- 0 — Unable to perform the test movement through any appreciable range (includes flicker or slight movement).
- 1 — Able to perform only part of the movement or complete the full movement with marked deviation from normal pattern.
- 2 — Able to complete the movement in a manner that is comparable to the unaffected side.

Functional Assessment

The assessment of hemiplegic is incomplete without finding the patients level of functional independence. The functional activities should be checked in lying, sitting, and standing posture.

Lying: The activities that need to be assessed are pelvic rotation, bridging, turning, rolling and various reaching out activities.

Sitting: The activities that need to be assessed are weight shifts, turning, bending, lateral bending, as well as functions of upper extremities like eating, brushing, combing, buttoning and reaching out activities. Balance of the patient can be checked by giving perturbation or by making the patient sit on a vestibular ball.

Standing: Static balance, weight shift, turning, and walking should be assessed.

Gait analysis should include observation of whether the patient walks independently, his balance in walking, turning around, and the various gait determinants should be checked for.

Information about various activities for personal hygiene that includes, washing face, bathing, and toilet activities should be taken from the patient.

MANAGEMENT

There are various physiotherapy approaches to the rehabilitation of stroke patient with hemiplegia.

Bobath: This is based on normal movement or neuro-development approach. The main aim is to prevent abnormal movement and adverse plastic adaptations and to facilitate normal movement and subsequent plastic changes (Bobath 1990).

Brunstorn (1970): He makes use of abnormal synergies and incorporate them into functional activities.

Conductive education (Peto): This was developed by Cotton and Kinsman (1983). The patient is encouraged to verbalize the activities as they perform them. This focuses on function.

Johnstone (1989): Follows developmental sequence, i.e. focussing on proximal stability. It makes use of orally inflated pressure splints.

Motor relearning program: This was described by Carr-Shepherd in the year 1987. This training of motor control is based on an understanding of kinematics and kinetics of normal movement, motor control process and motor learning.

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Proprioceptive neuromuscular facilitation (PNF): This was developed by Knott and Voss in the year 1968. To maximize sensory stimuli on the pool of AHC in order to stimulate purposeful muscle contraction.

Roods: This was first practiced by Stock and Meyer in the year 1966. This was mainly done to achieve purposeful muscular contraction by stimulating the skin through facilitating strokes on the skin. The stimulus given through the skin can have facilitatory and inhibitory effects.

Recent studies have shown that no single treatment can produce the desired response in different patients and that different therapist relies on different methods in treating hemiplegia. Thus one specific technique alone may be inadequate to produce the beneficial effect in stroke patient. The author uses an integrated approach using, Bobath, Burnstorm, Motor relearning, Roods, PNF and other form of sensory integration in treating patient with hemiplegia. The combination of treatment used differs from patient to patient.

The management of hemiplegia is different based upon the stage of the hemiplegic patient.

Acute Stage

The goals of the treatment in acute stage are:

- a. Prevent ignorance or unawareness of the hemiplegic side.
- b. Decrease the tendency to develop synergy in the chronic stage.
- c. Prevention of any joint restriction or stiffness.
- d. Prevention of complications due to immobilization like chest complication, deconditioning of the bone and muscles, etc.
- e. Early weight bearing.
- f. Psychological counselling.
- g. Education to the family.

These goals can be achieved through the following treatment.

Arrangement of the Patient's Room (Fig. 2.3)

Due to the lesion the patient suffers from sensory deprivation that leads to neglect of the hemiplegic side which can be greatly influenced by the patient's head position. Hence all the forms of the stimulus like the entrance to the room, the relatives, television, etc. should be present on the hemiplegic side so that the patient is forced to turn to that side which will stimulate awareness of the hemiplegic side.

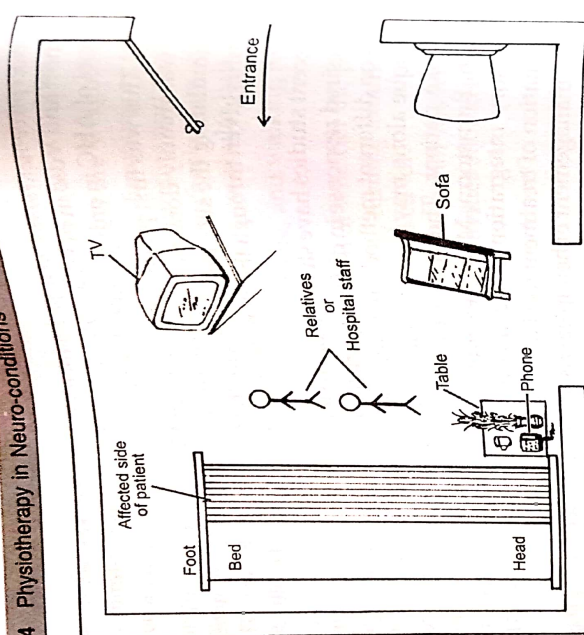


Fig. 2.3: Arrangement of the room for stroke patient

Positioning (Figs 2.4 to 2.6)

Positioning of the patient in an appropriate way is essential to control the development of spasticity and to help in faster improvement in the later stages. Preferably the patient is positioned sidelying and supine generally avoided.

On the affected side: The shoulder should be protracted and flexed. The elbow and the wrist should be extended. The forearm should be supinated. The pelvis should be in protracted position. The hip and knee should be in slight flexion and the ankle should be in neutral position.

On the sound side: The arm should be rested on the pillow kept in front of the patient. The shoulder girdle should be kept in protraction and slight elevation. The shoulder is kept in slight abduction and flexion with the elbow and wrist in extension position. The forearm should be in supine position. The pelvis should be kept in protraction. The hip should be slightly abducted and flexed. The knee should be slightly flexed and the ankle should be in neutral position.

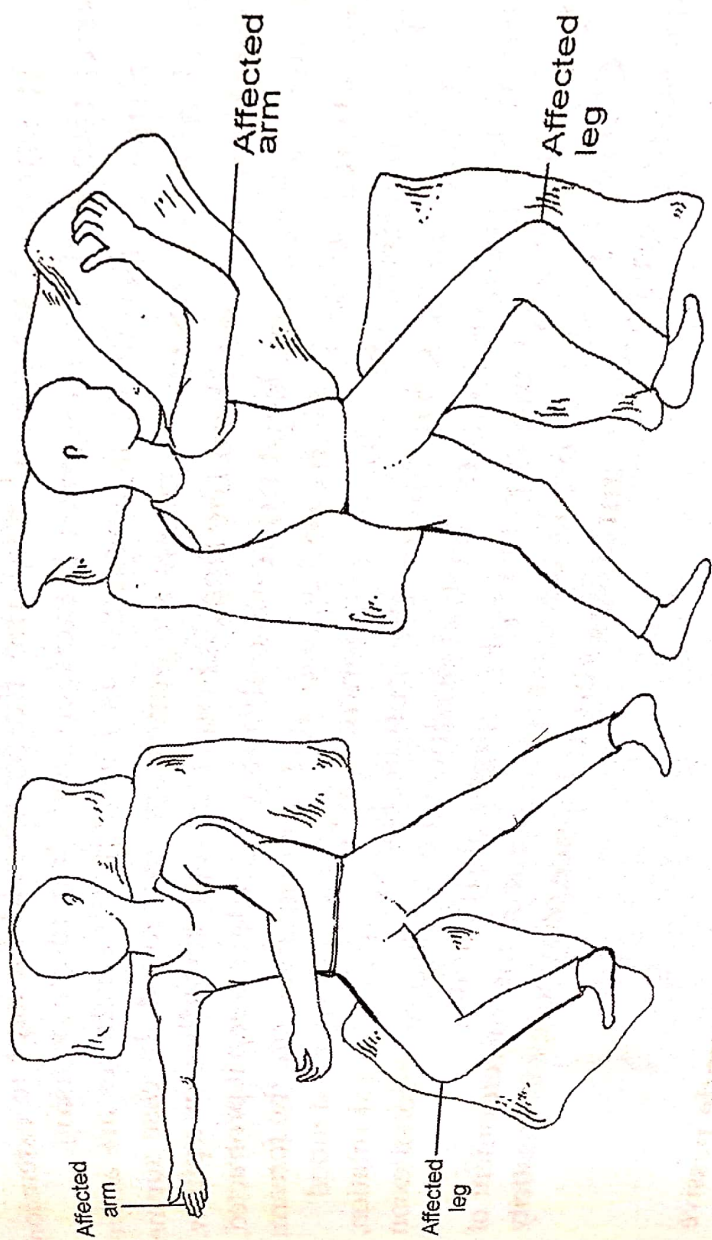
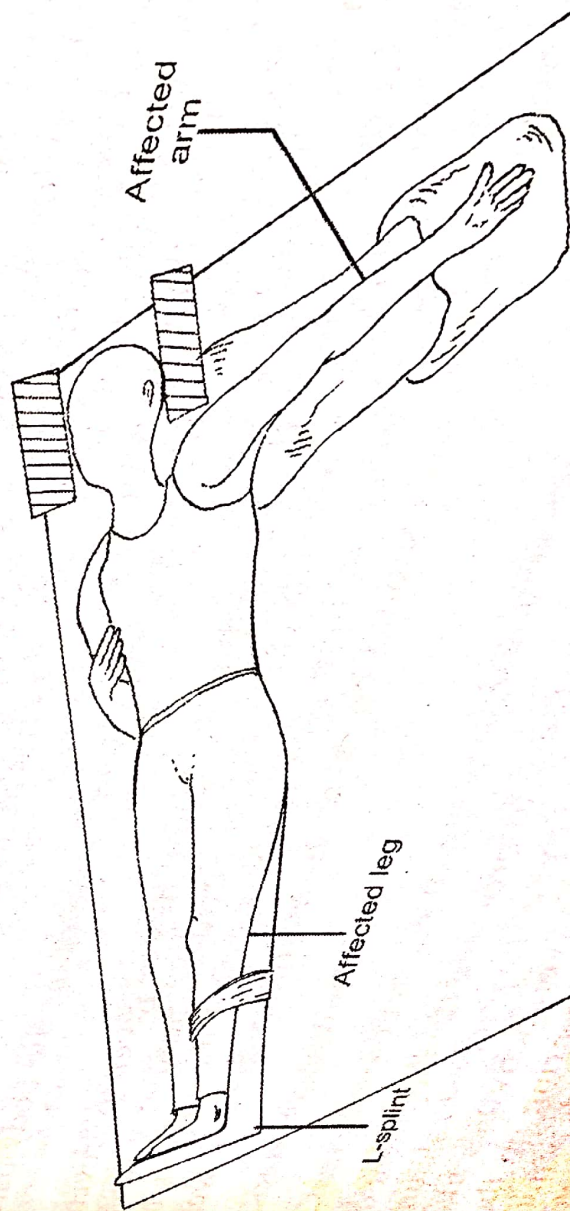


Fig. 2.4: Lying on affected side

Fig. 2.5: Lying on unaffected side



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It should be noted that the finger should be kept in extension and the web spaces maintained on both the above occasion.

Supine position is avoided as the primitive reflexes are active and also chances of pressure sores are increased. In case supine position is given then the head should be kept in midline on a pillow. Pillow should be kept under the shoulder girdle to keep it protracted, the shoulder is kept in abduction and external rotation, the forearm in supination, the elbow is extended, wrist and finger extended. A pillow is kept under the pelvis, leg kept in neutral rotation, the ankle maintained in neutral position, i.e. 90 degree of dorsiflexion by a pillow and the hip is kept in slight abduction.

Correct positioning is necessary to control the development of spasticity and also to minimize the influence of synergy in spasticity stage.

Mobilization and Stretching

During flaccid stage mobilization in the form of gentle passive exercises and stretching of various biarticular muscles should be given as they are very prone to develop tightness. Thus muscles like tendon Achilles, hamstring, quadriceps, adductors, tensor fasciae lata, biceps, wrist flexors, etc. should be stretched. Passive exercises should be given of all the movements at all the joints for at least 10 repetitions three to four times in a day.

Some forms of splints may be given to maintain the body parts in the desired position.

Commonly dorsiflexion splint or L splint may be given to prevent the foot from going into plantar flexion attitude. Similarly wrist extension splint is given to maintain the wrist and the fingers in extension position. Care should be taken to maintain the first web space.

Weight-bearing Activities (Fig. 2.7)

Weight-bearing exercises are necessary to promote development of tone in the muscles and also to maintain the absorption of calcium into the bones. Thus the patient should be given activities like bridging, supine on elbows, sitting with weight bearing on the affected arm, and standing should be given as soon as possible within the limitation of the patient's general medical status.

Subluxation of the glenohumeral joint is a very common complication in stroke patient, which can be prevented by proper

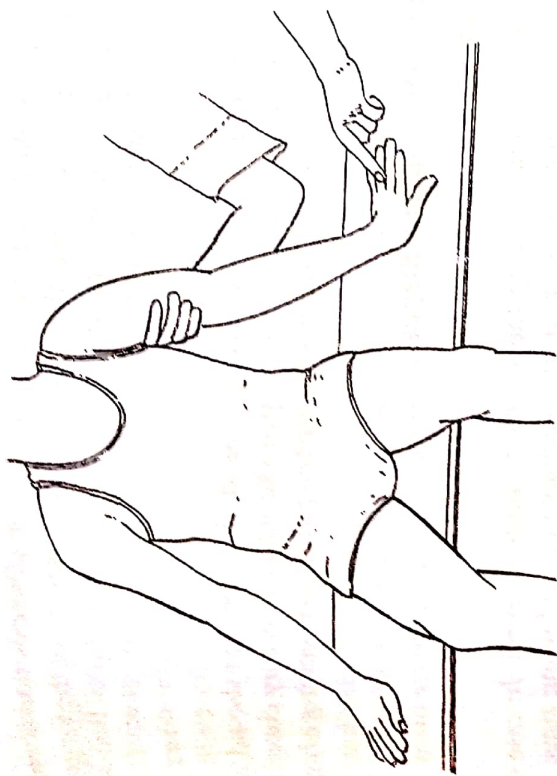


Fig. 2.7: Weight-bearing through affected upper limb

positioning and handling. Some form of support may be given to prevent distraction of the joint when the patient assumes an erect position. Generally a shoulder sling or Bobath splint is given to prevent this complication. Skillful taping also helps in preventing the subluxation very effectively and in addition also gives room for free movement. It also gives a tactile feedback which helps in faster development of tone in the shoulder muscles. Weight bearing exercises for the involved upper limb has also been found to be beneficial in preventing this. Shoulder sling is usually avoided as it facilitated the hemiplegic attitude, which the patient may develop in later stages.

Chest Physiotherapy

Chest physiotherapy in the form of inspiratory breathing exercises should be given to maintain the lung compliance and to prevent any chances of secretion accumulation. In cases of necessity nebulization or postural drainage can be given.

Oropharyngeal Retraining

Swallowing difficulty, which the patient has, can be counteracted by giving a proper positioning and by developing proper head

control. Slouched positioning or feeding in supine position should be avoided. Stroking over the neck area, ice massaging of the tongue, stimulating lip closure are some of the various techniques that are used to facilitate swallowing.

Counseling

As the patients are usually very depressed, the therapist should try and motivate the patient. The therapist should have a very cheerful and confident attitude, which will help the patient in keeping a high moral.

Treatment in Spastic Stages

Spasticity

It is always better to keep the spasticity under control. In case of spasticity the therapist should try to achieve the following aims:

- Normalize the tone.
- Development of normal functional pattern.
- Prevention of contracture and deformity.
- Train the patient to be functionally independent.
- Achieve highest possible physical security for the patient.

Normalization of the tone can be achieved by reducing the tone of the spastic muscles and simultaneously strengthening the weak antagonist muscles. Spasticity in the muscles can be reduced by the following methods:

- Gentle rhythmic passive movement.
- Sustained gradual stretching either manually or by using splints.
- Prolonged icing over the spastic muscle bulk for about 15 to 20 minutes. Studies have shown that prolonged icing and sustained stretching are equally effective in inhibiting spasticity.
- Faradic stimulation to the weak antagonist muscles can reduce the spasticity of the agonist muscle by the principle of reciprocal inhibition. However, this should be given only when the antagonist muscle is not active voluntarily.
- Reflex inhibiting postures or pattern. Intact, almost all the exercises given in the stage of spasticity are on the basis of reflex inhibiting pattern and postures.
- Biofeedback : This can be used to relax spastic muscle as well as to activate its antagonist.

Activities in Spastic Stage

Exercises in Lying (Figs 2.8A to L)

- **Scapular movement:** The patient scapula should be mobilized passively and also the patient should be asked to perform protraction and elevation movement of the scapula.
- **Touching the opposite shoulder:** The patient is trained to take his arm from extension, abduction and external rotation position toward his opposite shoulder into flexion, adduction, supination and external rotation.
- **Touching the head:** The patient is trained to touch the head by maintaining external rotation and supination.

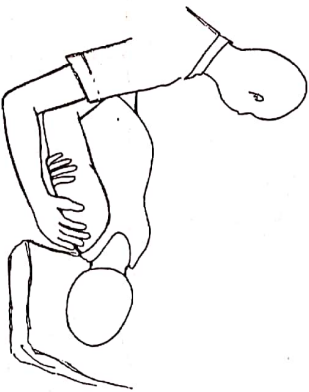


Fig. 2.8A: Guiding shoulder girdle movement



Fig. 2.8B: Protraction-retraction of shoulder girdle

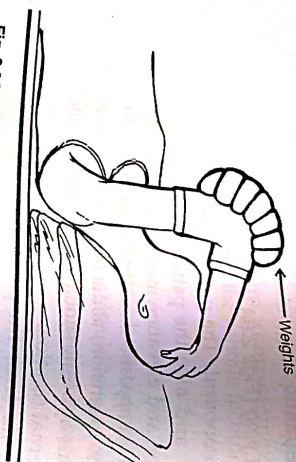


Fig. 2.8C: Developing shoulder control by balancing weight

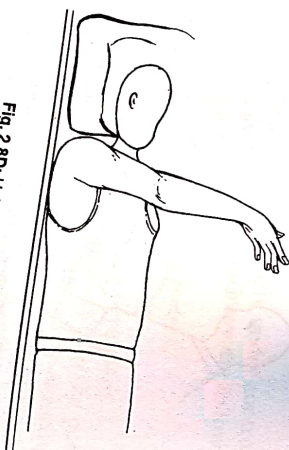


Fig. 2.8D: Holding arm in space



Fig. 2.8E: Extension abduction to flexion adduction pattern

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- The therapist maintains the hand in extension of the fingers with the thumb in abducted position and then she moves the hand into abduction and elevation maintaining the elbow in extension. This exercise can be progressed to active as the patient learns to control the movement.
- Elbow extension with shoulder in 90 degree of flexion.
- The patient is then trained to maintain the arm in space in different directions. This exercise will help the patient in developing good control of the upper limbs and also increases the proximal fixation.
- *Bridging*: Bridging should be done by weight bearing on the affected limb only. The therapist maintains the normal limb in flexion and encourages weight bearing through the hemiplegic lower extremities.

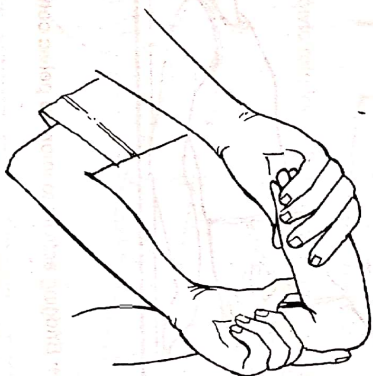


Fig. 2.8F: Correct way of holding hand to inhibit long flexor spasticity

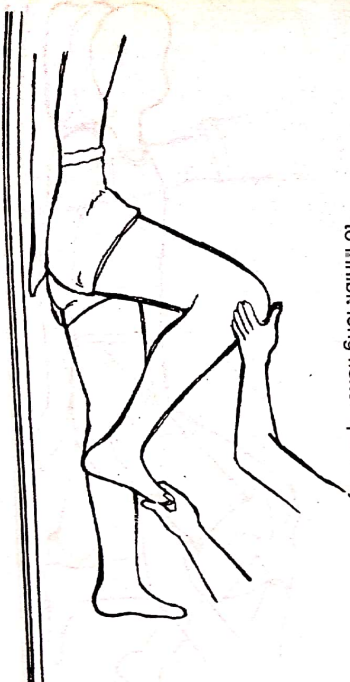


Fig. 2.8G: Hip-knee extension with ankle dorsiflexion

- *Unilateral rotation of the pelvis:* The patient performs hip-knee flexion of the affected lower limb and then rotated the pelvis to the opposite side. The patient tries to maintain this posture so that the spasticity of the trunk is inhibited. This exercise also help in encouraging forward rotation of the pelvis and correcting pelvic retraction.
- The patient is encouraged to perform flexion adduction and extension abduction pattern of the lower limb keeping the knee in extension position throughout.
- The affected lower limb is kept in abduction at the edge of the plinth such that the knee is in flexion. In this position the patient is encouraged to perform knee extension and flexion without

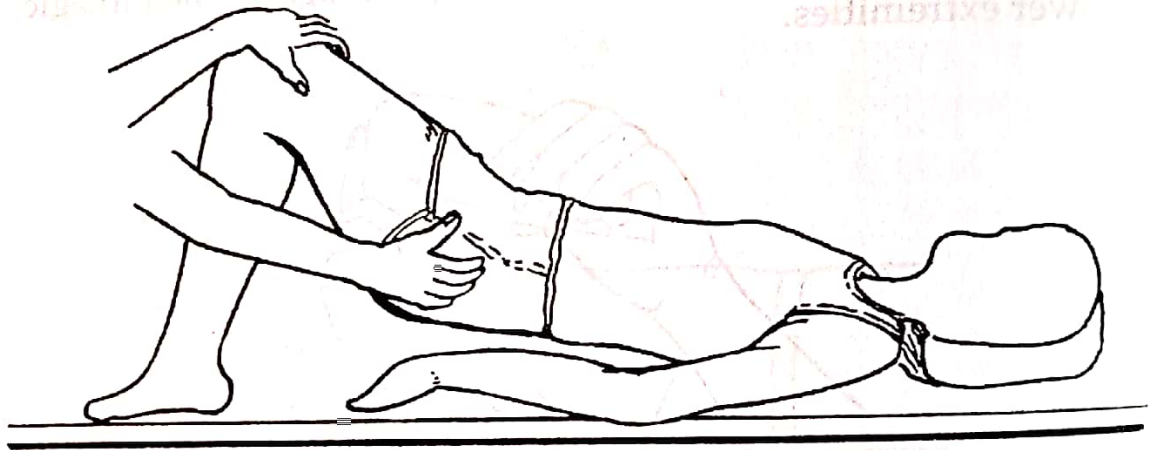


Fig. 2.8H: Bridging activity to improve pelvic control

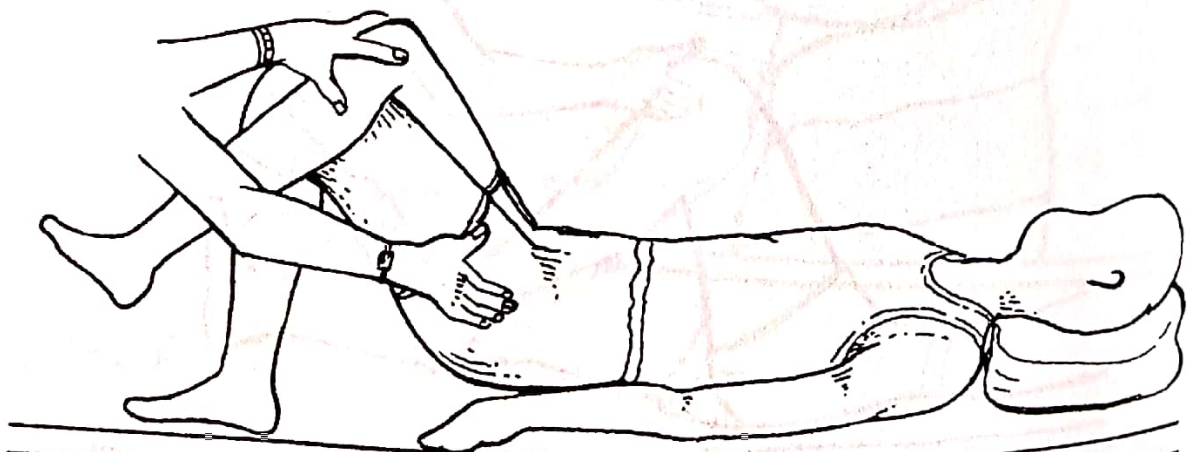




Fig. 2.8J: Pelvic rotation with hip knee flexion to inhibit trunk spasticity

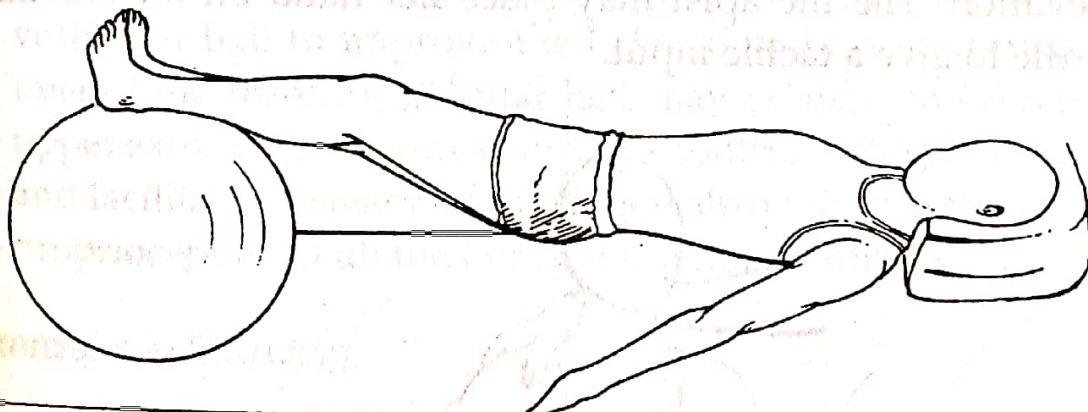
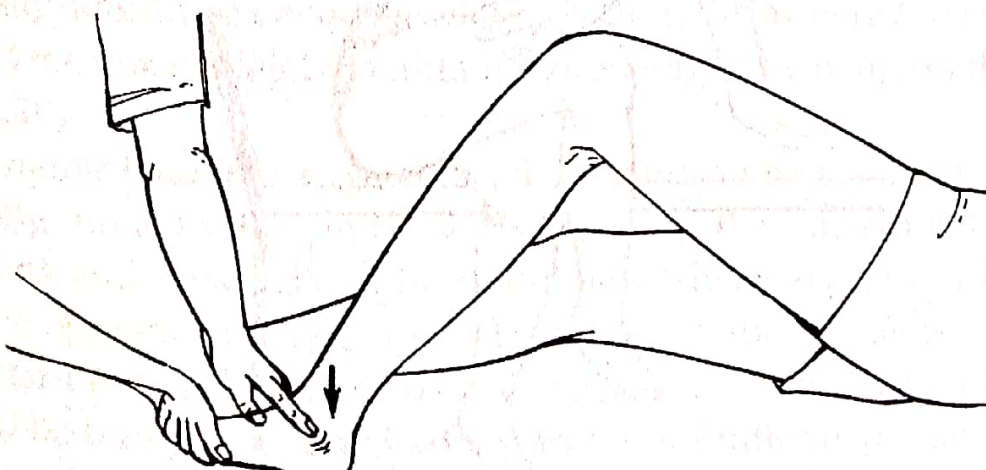


Fig. 2.8K: Bridging on vestibular ball to improve pelvic control



any adduction or flexion movement at the hip (the patient will have a tendency to perform hip adduction along with knee extension and hip flexion along with knee flexion).

- The patient may be trained to perform dorsiflexion with hip extension and plantar flexion with hip flexion. In case the patient is unable to perform dorsiflexion he may be trained to do with hip-knee flexion and the flexion at the hip and knee gradually reduced.

Exercises in Sitting

- *Weight bearing on the affected upper limb:* The therapist sits behind the patient and maintains the arm in slight abduction and extension with elbow extension. The patient should be encouraged to weight bear with the arm in this position. As the patient improves the support given by the therapist should be gradually reduced (Fig. 2.9A).
- *Shoulder shrugs:* This exercises help the patient to achieve scapular elevation. The therapist may place her hand on the shoulder girdle to give a tactile input.

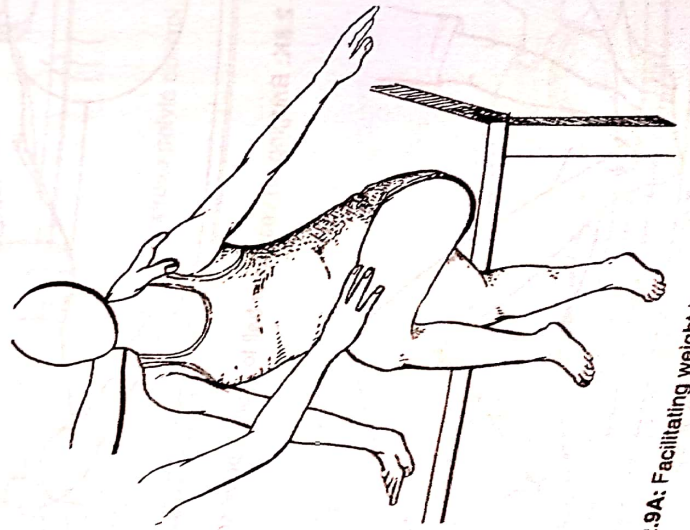


Fig. 2.9A: Facilitating weight transmission through affected side

- *Touching the opposite shoulder and placing the hand on the sacrum:* The patient is trained to touch the opposite shoulder in supination and then take it behind the back as far across as possible to the opposite side.
- *In sitting position some exercises for the hand can be given emphasizing more on the extension components.* Visual feed back exercises like holding a paper glass with water in it such that the water level should not increase or holding a clay mould without causing hand impression are some of the ways to train control of the hand.
- *Supination pronation* keeping the elbow flexed at 90 degrees.
- *Knee extension and flexion of the knee:* The patient should be asked to flex the knee by taking the foot as much as possible below the chair and then straightening it.
- *Getting up:* The patient is trained to perform getting up by moving the foot closer to the chair, bending forward, holding the hand rest and then getting up.
- *Vestibular exercises:* The patient may be made to sit on the vestibular ball to improve the balance and also normalize the tone of the trunk. Vestibular ball may be used to exercise the upper extremities mainly to achieve control at the proximal joint and facilitate extension of the fingers through stimulation of the proprioceptors at all the joints of the upper limb (Fig. 2.9B).

Exercises in Standing

In standing the patient is trained to achieve static balance by giving perturbation or by making the person stand on a wobble board or tilt board. Balance should also be trained in step stance. The patient is trained to shift weight alternately on both the lower limbs. Standing should be encouraged by keeping the affected leg slightly behind the normal leg to facilitate more weight-bearing on that side (Fig. 2.10).

Dynamic balance can be trained in standing by teaching various reaching out activities and also by teaching the patient rotation of the trunk and forward bending exercises. These exercises also help in development of automatic adaptation of the muscles which is important for performance of many activities. The patient balance should be trained for anticipatory and automatic response.

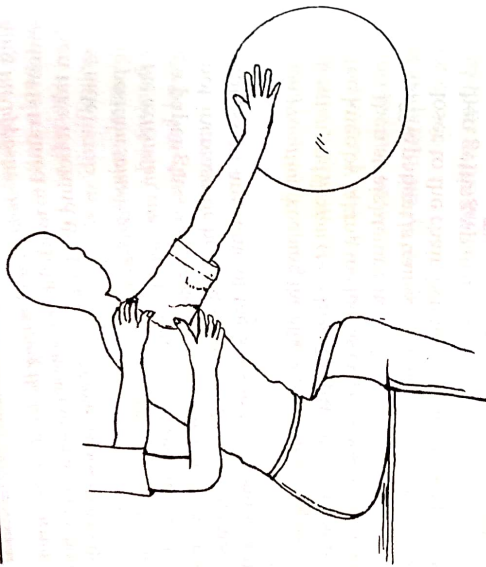


Fig. 2.9B: Approximation exercise for upper limb on vestibular ball. The patient can also be asked to move the ball with affected arm to develop control

- The following exercises may be given in standing:
- The therapist stands behind the patient and hold the pelvis such that the affected pelvis is kept in protraction. The patient is then encouraged to place the affected leg forward and then take it back. This is repeated for 10 times. Similarly the patient is encouraged to place the affected leg to the sides (abduction) and then come to the neutral position for ten times. This activities help the patient during swing phase of the gait cycle.
 - In the same position, the therapist trains the patient to take weight on the affected leg and place the normal leg forward and then back. This is repeated ten times. Similarly abduction movement can also be trained for 10 times. This activity encourages proper weight bearing of the affected limb.
 - Symmetrical throwing activities should be encouraged to increase the balance reaction and also to increase the stability limit which will help the patient in proper postural control.
 - Unilateral stance on the affected leg should be given to the patient which will facilitate isometric work of the abductors to prevent Trendelenburg's sign.

Fig. 2.10: T

Once the patient may be encouraged to walk on normal gait also be given ground. Treadmill hemiplegic improve the with part rehabilitat

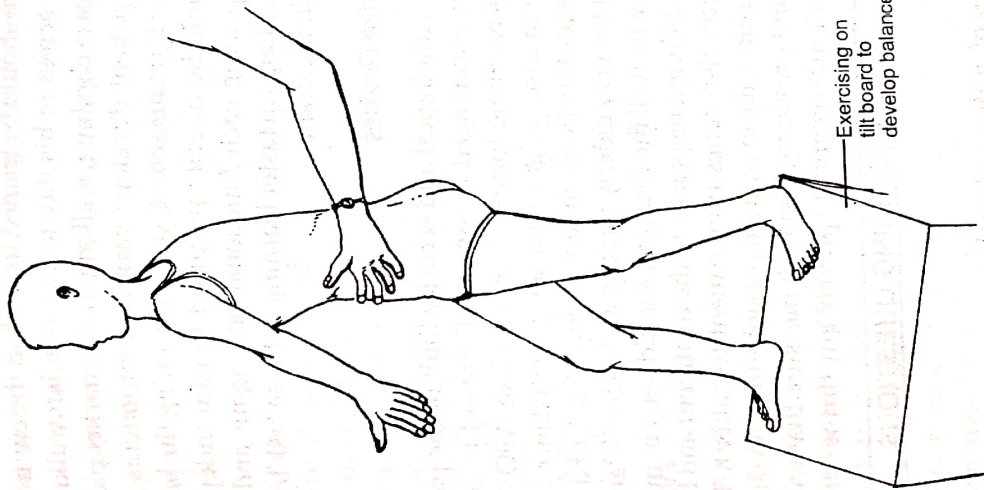


Fig. 2.10: Tilt board exercises for improving balance and control of lower limb

Once the patient is comfortably doing the above exercises then he may be trained to walk in between the parallel bars in a near normal gait pattern. Side walking and backward walking should also be given. Then progressed to walking unaided on the level ground.

Treadmill training or using a static cycle to train the gait in hemiplegic patient, especially in the early stages, has been found to improve the gait pattern to a remarkable extent. Treadmill walking with partial weight supporting harness have been useful in rehabilitating gait in hemiplegics.

The patient should be trained to walk in the environment which he will come across in his day-to-day life like walking on uneven surface, staircase climbing, walking in the market place, etc.

Once the patient develops various components of movement available to perform his activities of daily living the basic functional activities should be directly practiced to gain independency and accuracy. Repeating each functional task again and again over a period of times helps in faster learning of those task by formation of the engrams in the brain.

Use of Assistive Devices

Based on the pathological and security limit of the patient, he may or may not be encouraged to use a walking aid or other forms of assistive devices. Some forms of orthosis like AFO or knee brace may be given in cases of gross muscular imbalance that interferes with the patient's functional independence. Cane or a walking stick may be given for very elderly patient with poor balance reaction.

One point which all the therapist should keep in mind is that the patient show different responses to the treatment and hence the goal of rehabilitation also varies from patient to patient. The main aim should be to achieve the maximum possible functional independence for a patient and providing him with measure that will help him in achieving maximum security to prevent any further damage to him.

SELF-ASSESSING QUESTIONS

Long Essay

- Give causes of stroke and explain the management.
- Describe the circle of Willis and give its importance.
- Explain the blood supply to the brain and describe various stroke syndromes.
- Explain the assessment of patient with stroke
- Discuss the principle of assessment of a 60 years old stroke patient with hemiplegia of non-dominant side.
- Explain the clinical presentation of a stroke patient
- Describe the various methods of functional retraining for the various tasks of the upper limb like eating, reaching out, weight-bearing.
- Describe various techniques of rehabilitating a hemiplegic gait

- Describe the principles of underlying physiotherapy management of stroke patient.
- Explain the significance of reflex inhibiting position and pattern in treatment of stroke.
- Physiotherapy role in flaccid stage of stroke.

Short Essay

- Circle of Willis.
- Management of spastic hand.
- Perceptual disorders in stroke.
- Motor abnormality in stroke.
- Management of spastic foot.
- Management of spasticity.
- Hemiplegic gait.
- Exercises to improve gait in hemiplegia.
- Exercises to improve various hand functions.
- Voluntary control testing.

Short Answers

- What is apraxia?
- What is agnosia and its types?
- Types of apraxia.
- What is Gerstmann's syndrome?
- Define autotopagnosia?
- What is somatognosia?
- Aphasia and its types.
- What is hemianopia?
- Principles of road approach.
- What is motor relearning?
- What is reflex inhibiting pattern?
- Difference between associated reaction and associated movements.