STROKE REHABILITATION

DISSERTATION SUBMITTED IN FULFILLMENT OF THE REGULATIONS FOR THE AWARD OF M.D. PHYSICAL MEDICINE AND REHABLITATION

PSG INSTITUTE OF MEDICAL SCIENCES AND RESEARCH

THE TN DR MGR MEDICAL UNIVERSITY

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GUIDE
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CHENNAI, TAMILNADU

MAY 2011

CERTIFICATE

This is to certify that the thesis entitled "**STROKE REHABILITATION**" is a bonafide work of **Dr. V RAMAMOORTHY** done under my direct guidance and supervision in the Department of Neurology, PSG Institute of Medical Sciences and Research, Coimbatore in fulfillment of the regulations of the TN. Dr MGR Medical University, Chennai for the award of MD Degree in Physical Medicine and Rehabilitation.

GUIDE **PRINCIPAL**

ACKNOWLEDGEMENT

This dissertation project would be incomplete without the mention of some of the important people who have been pivotal in helping me to carryout this study.

I am grateful to my Guide Prof. **Dr. K. Ramadoss** M.D, DM. who has helped me starting from the designing of the study to the statistical analysis. Without his help it would have been impossible to complete this study successfully.

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CONTENTS

INTRODUCTION

Stroke is one of the leading causes of mortality and morbidity worldwide. Approximately 20 million people each year suffer from stroke and of these 5 million will not survive[1]. Developing countries account for 85% of global deaths from stroke. Stroke is also a leading cause of functional impairments, with 20% survivors requiring institutional care after 3 months and 15%-30% being permanently disabled.

Stroke is the most common causes of chronic disability. Of survivors, an estimated one third will be functionally dependent after 1 year experiencing difficulty with activities of daily living (ADL), ambulation, speech and so forth[2]. Stroke survivors represent the largest group admitted in inpatient rehabilitation hospitals. Another indicator of disability is the fact that approximately 26% of patients with stroke are institutionalized in a nursing home.

Modern advances in stroke research aid better management of stroke patients. What was once a disorder with a pessimistic prognosis has become a treatable disease. In the past, recovery from motor deficit after a stroke remains a puzzling scientific question. Now it has been found out that,

neuronal reorganization,
dynamic changes in the somatotopia of primary motor cortex,
recruitment of remote cortices and
participation of associative cortices

are clearly part of rearrangement process.[3] It is likely that such mechanisms represent the basis of clinical recovery of stroke patients.

Rehabilitation has an important role in improving functions beyond that explained by neurological recovery alone. Rehabilitation measures help to

restore lost activities, improve quality of life and decrease the long term economic cost of stroke. Proper patients' selection, realistic goal setting, active participation of both the patient and family and the use of an interdisciplinary team approach are important for the success of stroke rehabilitation. It is desirable to start rehabilitation programs to stroke patients once they are medically stable. Early intervention helps faster motor recovery.

Four programs of treatment, for patients with motor control problems caused by brain damage, were developed in the mid- 1950 and early 1960 based on neurophysiological and developmental research of the time as well as careful observation of the responses made by patients when being handled, positioned, touched or moved in various ways. These traditional therapeutic approaches are,

1. Rood's Approach
2. Bobath Neuro Development Treatment
3. Brunnstrom's Movement Therapy
4. Proprioceptive Neuro muscular Facilitation (PNF) Technique.

In this study, **'Intense sensory motor stimulation'** in the form of electrical stimulation, oil massage and reeducation exercise program are instituted to augment motor recovery in stroke patients.

AIMS AND OBJECTIVES

The **Primary objective** of this study is

- to investigate the effect of intense sensory motor stimulation given in the form of electrical stimulation, olive oil massage and reeducation exercise program in augmenting motor recovery in stroke patients.

The **Secondary objectives** of this study are to assess the various factors which influence the stroke recovery like,

- the time interval between the onset of stroke and the initiation of rehabilitation program
- the risk factors for stroke
- the areas of the brain affected and
- the pathology of stroke.

REVIEW OF LITERATURE

EPIDEMIOLOGIC ASPECTS

DEFINITION:

The world Health Organization (WHO) defines stroke as "**rapidly developing clinical signs of focal (or global) disturbance of cerebral function, with signs lasting 24 hours or longer or leading to death with no apparent cause other than of vascular origin".** This definition includes subarachnoid hemorrhage but excludes transient ischemic attack and hemorrhage or infarction related to infection or tumor.

STROKE MORBIDITY AND MORTALITY IN INDIA[4, 5]

- Prevalence 55.6 per 100,000 all ages
- 0.63 million deaths(WHO 2005)
- 1.44 – 1.64 million cases of new strokes every year
- 6,398,000 Disability Adjusted Life Years (DALYs)
- 12% of strokes occur in the population aged<40 years
- 28-30 day case fatality ranges from 18-41%

AGE:

The incidence of first stroke rises exponentially with age. In the 55-to59-year-old age group, the risk of stroke is about 5% per year, whereas in the 80-to 84–year–old group the risk is almost 25% per year.[6]

GENDER

Gender has been shown to affect stroke risk; men have a 30% increased risk until the later decades of life, when women have higher risk. Stroke is a

significant cause of morbidity and mortality in women, especially young women in the pregnancy and post-partum periods. More women under the age of 45 years die from stroke than from myocardial infarction. A disproportionate percentage of subarachnoid hemorrhages occur in women.

RACE:

Race related differences in incidence and mortality of strokes have also been demonstrated. When compared with whites, blacks have a higher incidence and mortality of stroke which may be in part attributable to a relatively late presentation for medical care. Asians have a higher incidence of stroke than whites but suffer less from heart disease.

GEOGRAPHY:

In India, the crude prevalence rate appears to be higher in urban compared to rural populations. The Parsi population in Mumbai appears particularly at risk, compared with the Indian population. The Parsi people migrated from Iran in the 7^{th} Century to India it is argued that they are ethnically distinct from the Indian population.

PATHOLOGY OF STROKE

Stroke can be classified as hemorrhagic or ischemic in origin.

A) HEMORRHAGIC STROKE:

Hemorrhagic strokes, constituting approximately 10% of all strokes, are either intra cerebral or subarachnoid in location. Hypertension, particularly uncontrolled hypertension, is the most common cause of brain hemorrhage. Clinical features depend on the location and severity of the brain hemorrhage. Brain hemorrhage is not preceded by transient ischemic attacks. Signs and symptoms develop acutely with altered level of consciousness, severe headache and usually elevated blood pressure.

SUBARACHNOID HEMORRHAGE:

Subarachnoid hemorrhage (SAH) also is characterized by sudden, severe headache which may result in loss of consciousness. Vomiting is frequent. Patients can be younger and do not necessarily have preexisting hypertension. Most patients will have a stiff neck (Positive Kernig's or Brudzinski's signs). Head CT is most likely to reveal subarachnoid hemorrhage in the first 24 hours; thereafter, the chance of finding blood diminishes. Patients with no clear cause for SAH tend to have the best prognosis. [7] Mortality from subarachnoid hemorrhage is high.

B) ISCHEMIC STROKE:

Ischemic causes of stroke are related to thrombotic, embolic or hemodynamic factors. Thrombotic infarction in large vessels occurs when a thrombus forms on an atherosclerotic plaque. Lacunar infarcts occur in the deep, penetrating arteries where they branch at 90º from main intra cerebral arteries.

Ischemia may be diffuse or localized depending on cerebral blood flow (CBF). Normal CBF is 50 to 55ml/100g/min. At 18ml/100g/min, synaptic transmission failure occurs. These cells still have the potential for recovery. However, when CBF drops to 8ml/100g/min, membrane pump failure occurs and cell death results.

ISCHEMIC PENUMBRA:

The "Ischemic penumbra" refers to the region of brain with CBF between 18/ml/100g/min to 8ml/100g/min which is still viable but has loss of electroencephalogram, flat evoked potentials, but normal ATP stores and intracellular potassium concentrations[8]. This region may be salvageable or progress to infarction.

DIAGNOSTIC AND THERAPEUTIC INTERVENTIONS OF ACUTE STROKE

Stroke diagnosis, prevention and treatment have dramatically improved over the past several years. New imaging techniques, use of anticoagulants and antiplatelet drugs and surgical advances are all recent additions to the armamentarium for stroke treatment. However, therapeutic nihilism regarding stroke treatment persists and can delay early treatment. Only about 40% of patients present to the hospital within 24 hours of experiencing symptoms. This delay can be critical, given the need to administer thrombolytics within 3 to 6 hours after onset of symptoms - the" therapeutic window" to salvage the penumbra. Unfortunately, patients with milder symptoms who would benefit most are least likely to arrive in time for therapy. Therefore, education of both the public and professional is needed to reduce the time from symptom onset to hospital arrival [9].

DIAGNOSIS OF ISCHEMIC STROKE:

A. Diagnostic approaches to ischemic stroke include the use of sonographic characterization of carotid artery plaques. MRI with or without angiography can also characterize the plaque to identify "craters" increase the likelihood of embolization.

B. Transesophageal echocardiograhic findings can also be helpful for detecting potential cardiac sources of embolism in patients with clinical risks for cardioembolism of unexplained stroke.

C. Functional MRI and Diffusion-weighted MRI may also be promising new techniques to enhance our imaging capabilities.

I. MEDICAL MANAGEMENT

THROMBOLYTIC THERAPY:

With the development of thrombolytic therapy, cerebral ischemia is being termed "brain attack" to convey the same urgency to the situation as is evoked by the term "heart attack".

A. **Tissue plasminogen Activator**: Tissue plasminogen activator (t-PA) has become the agent of choice for thrombolysis since the National Institute of Neurological Disorders and Stroke (NINDS). t-PA study group first published results in late 1995. Clinical outcome was improved in patients treated within 3 hours with t-PA despite an increased risk if intracranial hemorrhage.

B. **Streptokinase**: A second major trail used streptokinase as a thrombolytic agent in ischemic stroke (the Multicentre Acute Stroke Trail, MAST) but found excess mortality in the streptokinase – treated group.

C. **Heparin**: Heparin is used in patients with acute infarction in an effort to limit neurologic progression or to prevent recurrent embolism in cardio embolic stroke. Low molecular weight heparin has been shown to be effective in reducing the deficit in ischemic strokes.

Treatment of ischemic stroke can be divided into measures to promote reperfusion and that promotes cytoprotection. Reperfusion involves the use of thrombolytics. Cytoprotection is the use of pharmacologic agents that reduce neuronal injury. The "reperfusion window" based on animal studies, is about 3 hours, whereas the "cytoprotective window" is probably about 1.5 to 3 hours. Cytoprotection has an impact on the injury cascade by affecting failure of energy supply, release of neurotransmitters and accumulation of intercellular calcium, buildup of nitric oxide and cellular edema which ultimately cause cell death[10].

Various drugs have been tried including calcium channel blockers, antagonists of N-methyI-D-asparate, sodium channel blockers and inhibitors of excitatory amino acids. Lubeluzole, a compound that prevents intracellular glutamate concentrations has shown promise in early trials and may be useful in combination with other drugs or as an agent.

D. **Aspirin**: Aspirin has been used to treat ischemic stroke, both as primary prevention in patient with transient ischemic attacks or high-grade carotid stenosis and as secondary prevention after stroke occurrence.

II. SURGICAL MANAGEMNT

A. Carotid endarterectomy

Treatment of stroke also includes the use of carotid endarterectomy for symptomatic stenosis with 70% to 99% obstruction of the vessel. Restenosis after successful carotid endarterectomy can occur.

B. Percutaneous transluminal carotid angioplasty:

Another invasive approach in treating ischemic stroke is the use of percutaneous transluminal carotid angioplasty.

Management of Hypertension:

Hypertension occurs in 75% of patients with infarction or hemorrhage. Two-thirds of these patients had preexisting hypertension. Currently published recommendations are “to gradually lower the blood pressure to 180 /105mg Hg if the patients have a premorbid history of hypertension and to 160/95 mm Hg in patients with no history of hypertension”[11]. Patients with hemorrhagic stroke should probably be managed similarly, but the range of acceptable systolic and diastolic pressure can be 5mm Hg higher in each category mentioned above. Medications used for acute treatment of hypertension can include labetalol, enalapril, and clonidine.

CO-MORBIDITIES AND COMPLICATIONS

The following co-morbidities and complication are associated with stroke patients:

1. Coronary artery disease
2. Hypertension
3. Diabetes mellitus
4. Deep vein thrombosis
5. Dysphagia
6. Infection
7. Depression
8. Falls
9. Spasticity
10. Sleep apnea
11. Shoulder pain
12. Seizures

FACTORS THAT PREDICT MORTALITY AND DISABILITY AFTER STROKE

I. RISK FACTORS FOR ACUTE STROKE MORBIDITY

1. Stroke severity
2. Low level of consciousness
3. Diabetes mellitus
4. Cardiac disease
5. Electrocardiographic abnormalities
6. Old age

7. Delay in medical care
8. Elevated blood sugar in non-diabetic
9. Brainstem involvement
10. Hemorrhagic stroke

II. RISK FACTORS FOR DISABILITY AFTER STROKE

1. Severe stroke (minimal motor at 4 weeks)
2. Low level of consciousness
3. Diabetes mellitus
4. Cardiac disease
5. Electrocardiographic abnormalities
6. Old age
7. Delay in rehabilitation
8. Bilateral lesions
9. Previous functional disability
10. Poor sitting balance
11. Global aphasia
12. Severe neglect
13. Sensory and visual deficits
14. Impaired cognition
15. Incontinence (>1-2 weeks)
16. Depression
17. Poor social support

SCANDINAVIAN STROKE SCALE (SSS)

The neurological status of the stroke patient is assessed by the Scandinavian stroke scale. The Scandinavian stroke scale evaluates levels of consciousness, eye movements, power in the arm, hand and leg, orientation, aphasia, facial paresis and gait. The total score ranges from 0 to 58 points.

1. CONSCIOUSNESS:

Fully conscious	- 6
Somnolent but can be aroused to full conscious	- 4
Reacts to verbal command but not fully conscious	- 2

2. EYE MOVEMENTS:

No gaze palsy	- 4
Gaze palsy present	- 2
Conjugate eye	- 0

3. ARM MOTOR POWER:

Raises arm with normal strength	- 6
Raises arm with reduced strength	- 5
Raises arm with elbow in flexion	- 4
Can move but not against gravity	- 2
Paralysis	- 0

4. HAND MOTOR POWER:

Normal strength	- 6
Reduced strength in full range	- 4
Some movements, fingertips do not reach palm	- 2
Paralysis	- 0

5. LEG MOTOR POWER:

Normal strength	- 6
Raise straight leg with reduced strength	- 5
Raised leg with flexion of knee	- 4
Can move but not against gravity	- 2
Paralysis	- 0

6. ORIENTATION:

Correct time, place and person	- 6
2 of these	- 4
1 of these	- 2
Complete disorientation	- 0

7. SPEECH:

No aphasia	- 10
Limited vocabulary or incoherent speech	- 6
More than yes or no, but not longer sentences	- 3
Only yes/no or less	- 0

8. FACIAL PALSY:

No facial palsy	- 2
Facial palsy present	- 0

9. GAIT:

Walks 5 minutes without aids	- 12
Walks with aids	- 9
Walks with the help of another person	- 6
Sits with support	- 3
Bedridden / wheel chair bound	- 0

SEVERITY OF NEUROLOGICAL LESION

SSS SCORE		STROKE GRADE
45- 58	-	Mild
30- 44	-	Moderate
15- 29	-	Severe
0- 14	-	Very severe

Thus the neurological status is assessed by Scandinavian stroke scale. The Scandinavian stroke scale has been found to be reliable and its validity has found support in high correlations with other stroke scales.[12]

FUNCTIONAL INDEPENDENCE MEASURE (FIM)

Early prediction of functional recovery remains a crucial factor in client oriented practice, discharge planning and utilization of rehabilitation resources.

Prediction of length of stay of stroke rehabilitation in-patient was developed based on patient's age and functional admission. (scored on the FIM). FIM scores at admission are strong predictors of patient's length of stay with the "*Transfer items*" having the greatest predictive power. [13]

In FIM, there are **six items** like Self-care, Sphincter control, Transfers, Locomotion, Communication and Social cognition and **seven levels** like Total assist, Maximal assist, Moderate assist, Minimal assist, Supervision, Modified Independence and Complete Independence.

The FIM requires direct observation of patients to determine functional status. [14] FIM score is based on task performance. [14] Another feature of comprehensiveness of FIM is that it takes only 30minutes to complete. FIM does not lend itself to inconvenience. [14]

In a study conducted by Oczkowski and Barreca, patients with admission FIM scores above 96 invariably went home, whereas patient with admission FIM scores less than 37 always requires institutionalization.

It remains difficult to predict the place of discharge for stroke survivors with admission FIM scores between 37 and 96. These patients with intermediate FIM scores between 37 and 96 continued to exhibit wide fluctuations in their level of obtainable independence making discharge planning difficult. [15]

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

Self –Care	ADMIT	DISCHG	FOL-UP
A. Eating	☐	☐	☐
B. Grooming	☐	☐	☐
C. Bathing	☐	☐	☐
D. Dressing- Upper Body	☐	☐	☐
E. Dressing- Lower Body	☐	☐	☐
F. Toileting	☐	☐	☐
Sphincter Control			
G. Bladder Management	☐	☐	☐
H. Bowel Management	☐	☐	☐
Transfers			
I. Bed, Chair, Wheel Chair	☐	☐	☐
J. Toilet	☐	☐	☐
K. Tub, Shower	☐	☐	☐
Locomotion			
L. Walk /Wheelchair	☐	☐	☐
M. Stairs	☐	☐	☐
Motor Subtotal Score	☐	☐	☐
Communication			
N. Comprehension	☐	☐	☐
O. Expression	☐	☐	☐

Social Cognition			
P. Social Interaction	☐	☐	☐
Q. Problem Solving	☐	☐	☐
R. Memory	☐	☐	☐
Cognitive Subtotal Score			
Total FIM	☐	☐	☐

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

THE SEVEN STAGES OF STROKE RECOVERY

DEFINITION

The six original Brunnstrom stages are expanded to seven and the definitions, which formerly included only the limbs now incorporate postural control also. The "Chedoke" seven stages of motor recovery are defined as follows:

STAGE 1

Flaccid paralysis is present. Phasic stretch reflexes are absent or hypoactive. Hypoactive tonic stretch reflexes are demonstrated by a decrease in the resistance to passive movement. Active movement cannot be elicited reflexly with a facilitory stimulus or volitionally.

STAGE 2

Spasticity is present and is felt as a resistance to passive movement. No voluntary movement is present but a facilitory stimulus will elicit primitive movement patterns reflexly. These primitive patterns are the stereotyped flexion and extension synergies that relate to the spinal and brainstem reflexes.

STAGE 3

Spasticity is marked. The primitive, synergistic movements' patterns can be elicited voluntarily, but are obligatory. In most cases, flexion synergy dominates the arm, the extension synergy the leg.

STAGE 4

Spasticity decreases. Synergy patterns can be reversed if movement takes place in the weaker synergy first. Movements combining antagonistic synergies can be performed when the prime movers are the strong components of the synergy. Righting reactions develop, modify and partially integrate the spinal and brainstem reflexes.

STAGE 5

Spasticity wanes, but is evident with rapid movement and at the extremes of range. Synergy patterns can be reversed even if movement takes place in the strong synergy first. Movements with the weak components of both synergies acting as prime movers can be performed. Righting reactions have integrated the spinal and brainstem reflexes. Balance and equilibrium reactions are present and variety of functional movements is possible**.**

STAGE 6

Spasticity, as demonstrated by resistance to passive movements, is **no longer present.** Postures and movements are normal until rapid, complex actions are requested**.**

STAGE 7

Normal. A normal variety of rapid, complex movement patterns are possible.

The stages of stroke model provide a "Map with different routes" to effective outcomes, helping those within stroke rehabilitation to recognize, **where they are and where they are going**.[16]

NATURAL SPONTANEOUS NEUROLOGICALRECOVERY

There are two different but related ways that patients improve after stroke.

The first type of recovery, a reduction in the extent of neurological impairment can results from natural spontaneous neurological recovery from the effects of treatment that limit the extent of the stroke or from other interventions that enhance neurological functioning. This form of recovery presents as improvement in motor control, language ability or other primary neurological functions. [17]

The second type of recovery demonstrated by stroke patients is the improved ability to perform daily functions in their environment, within the limitations of their physical impairments [18.] The ability to perform these tasks can improve through adaptation and training in the presence or absence of natural neurological recovery. This is the element of recovery on which rehabilitation is thought to exert the greatest effect. Although most improvements in physical functioning occur within first 3-6 months, later recovery is also is commonly seen [19.]

RECOVERY OF MOTOR FUNCTION:

For most (but not all) stroke patients, the pattern of spontaneous recovery of motor function follows a relatively stereotyped sequence of events, in which lower extremity function recovers earliest and most completely, followed by upper extremity and hand functions. Return of tone usually precedes distal control and mass movement's patterns (or synergy patterns) precede specific, isolated, coordinated, volitional motor functions.

This sequence of recovery can stop at any stage. Some hemiparetic patients ultimately regain full or nearly complete use of all muscles in an isolated, coordinated fashion, independent of synergy patterns. Others experience incomplete improvement with partial voluntary use of the recovering extremity and a few patients demonstrate minimal or no recovery in motor control.

MECHANISMS OF RECOVERY IN NEUROLOGICAL FUNCTION:

Several mechanisms have been proposed to explain the clinically observed phenomenon of spontaneous recovery of neurological function[20]. These mechanisms generally can be divided into two broad categories. The first mechanism is resolution of local harmful factors, which usually accounts for early spontaneous improvement after stroke (usually within the first 3 to 6 months). These processes include resolution of local edema, resorption of local toxins, improved local circulation and recovery of partially damaged ischemic neurons.

The second mechanism to explain recovery is **neuroplasticity,** which can take place early or late. Brain plasticity is the ability of the nervous system to modify its structural and functional organization. The two most plausible forms of plasticity are collateral sprouting of new synaptic connections and unmasking of previously latent functional pathways. Other mechanisms of plasticity include assumption of function by undamaged redundant neural pathways, reversibility from diaschisis, denervation super sensitivity and regenerative proximal sprouting of transected neuronal axons. Experimental evidence indicates that **plasticity can be altered by several external conditions, including pharmacological agents, electrical stimulation, and environmental stimulation [21].**

FACTORS AFFECTING FUNCTIONAL OUTCOME AFTER STROKE

REVIEW OF REHABILITATION INTERVENTIONS

Review of medical literature shows that there is an association between rehabilitation interventions and outcomes after stroke. Outcome is defined as functional status, living situation, morbidity, mortality, costs, length of stay and quality life. In a critical review of Rehabilitation interventions by David & Deborah, the following rehabilitation interventions were evaluated for their relationship with functional outcome after stroke.

1. Functional deficits at rehabilitations admission
2. Timing of rehabilitation
3. Type of inpatient rehabilitation: interdisciplinary versus multidisciplinary.
4. Type of non-in patient rehabilitation : home health versus out patients versus day
5. Specificity of rehabilitation services
6. Intensity of rehabilitation services.

Outcome was defined as ***"Functional abilities at rehabilitation discharge and at follow-up (typically 3 to 12 months)''.*** These studies examined functional outcome with Barthel Index, Kennedy ADL scale and Functional Independence Measure.

RESULTS:

1. FUNCTIONAL DEFICITS AT REHABILITATION ADMISSION:

Functional deficits as predicators of functional outcome following stroke have been examined in 26 studies. All demonstrated an association between decreased functional abilities at admission to rehabilitation and poorer functional outcome.

Overall, the available literature demonstrates that decrease functional abilities in the first 1– 4 weeks after stroke are strongly associated with decreased discharge to home rates and decreased functional outcome at both rehabilitation discharge and up to 6 months of follow up. [22]

2. TIMING OF REHABILITATION:

The association of the timing of rehabilitation interventions typically defined as either multidisciplinary therapy or interdisciplinary rehabilitation services and outcome after stroke has been examined in 15 studies.

Overall, the available literature demonstrates that early rehabilitation interventions – within 3 to 30 days post stroke – are strongly associated with improved functional outcome. [23]

3. **TYPE OF INPATIENT REHABILITATION:**

INTERDICIPINARY VERSUS MULTIDISCIPLINARY

An interdisciplinary setting is defined as "one in which rehabilitation services are provided by diverse professionals who constitute a team that communicates regularly and used its varying expertise to work toward common goals".

Multidisciplinary settings usually included similar professional but regular communication and common goal orientation were less consistent. Overall, the available literature demonstrates that interdisciplinary rehabilitation following stroke is strongly associated with improved functional out come, shorter length of day, decreased costs and decreased mortality. [24]

4. TYPE OF NON–IN-PATIENT REHABILITATION:

HOME HEALTH VERSUS OUTPATIENT VERSUS DAY

The association between type of non-inpatient rehabilitation services, specifically day versus outpatient versus home therapy and functional outcome

after stroke has been examined in seven studies. Overall, the available literature demonstrates that participation in out-patient, home health and day rehabilitation programs is strongly associated with improved functional outcome after stroke. [25]

5. SPECIFICITY OF REHABILITATION SERVICES:

The association between the specificity of rehabilitation services, in particular the types of physical, occupational, speech and psychology therapy and functional outcome following stroke has been examined in eight studies.

Richards and associates randomized 27 acute stroke survivors to one or three treatment groups:

1. Intensive, early onset, task specific physical therapy
2. Intensive, early onset, generalized physical therapy or
3. Standard physical therapy

They showed that the intensive, early onset, task specific physical therapy demonstrated the greatest degree of gait recovery at 3 months. Overall, the available literature demonstrates that the task specific therapy services versus more generalized therapy are weakly associated with improved functional outcome after stroke. [26]

6. INTENSITY OF REHABILITATION SERVICES:

The association between the intensity of rehabilitation services, in particular, physical, occupational, speech and psychology therapy and functional outcome following stroke has been examined in five studies. Overall, the available literature demonstrates that the intensity of rehabilitation services is weakly associated with improved functional outcome after stroke. [27]

STROKE REHABILITATION

TRADITIONAL THERAPEUTIC APPROACHES

Current stroke rehabilitation practice throughout the world is based on a number of approaches that were developed independently by several pioneers in the 1950s.

Prior to the 1950s, the physical treatment of patients following stroke focused on encouraging the patients to use the unaffected side of the body to compensate for the disabilities of the affected side. The affected limbs were not a prime focus for treatment.

During the 1950s a number of new physiotherapy approaches emerged. The work of many of these pioneers e.g. Bobath (1969), Brunnstrom (1961, 1970) Knott & Voss (1968s) and Rood (1969) remains influential today. They developed their programs primarily from observation and experience.

I. NEURO PHYSIOLOGICAL APPROACHES:

1. BOBATH NEURO DEVELOPMENTAL APPROACH:

This approach is based on the assumption that increase tone and increased reflex activity will emerge as a result of lack of inhibition from a damaged postural reflex mechanism and that movement will be abnormal if it stems from a background of abnormal tone (Bobath 1990). A further assumption is that performing abnormal movements will reinforce more abnormal movements.

2. BRUNNSTROM:

This approach is based on the assumption that recovery progresses from sub cortical to cortical control of muscle function. Brunnstrom (1970) felt that normal selective movement did not recover unless patients first progressed through stages of abnormality. She therefore saw the use of mass flexion and extension synergies as a basis of retraining movement.

II. PROPRIOCEPTIVE EUROMUSCULARFACILITATION TECHNIQUE (PNF)

1. KNOT AND VOSS:

This approach is based on the assumption that people who move normally have passed through a developmental sequence and that after damage it is necessary to return to that developmental sequence before recovery will take place. It also assumes that maximal peripheral stimulation is required to recruit motor response. The use of total patterns of movement is believed to be a way of encouraging and reinforcing movement. This belief is based on the fact that the cortex is thought to control movements in patterns and not as singular muscular actions.

2. ROOD:

This approach is based on the assumption that postural stability and movement patterns are similar in all individuals and follow a developmental sequence. A return to this development frame work of motor sequence is fundamental to these motor responses. She suggests links between motor responses and somatosensory, autonomic and psychological functions. Therefore the relationship between sensory factors and motor functions assumes a major role.

3. JOHNSTONE:

The main aim of this approach is to control spasticity consistently overtime. The approach is based on the assumption that the damaged postural reflex mechanism can be controlled through positioning and splinting. The main problems identified are the imbalance of muscles tone and the resultant disabling postures. Treatment is based on a hierarchal model with manipulation and control of sensory and motor functions.

4. MOTOR RELEARNING PROGRAMME:

(Carr and Shepherd 1987)

This model is based on the assumption that the impaired learn in the same way as the unimpaired as the principles that they follow stem from work that has focused primarily on the learning responses of normal subjects. The program is based on four factors thought to be essential for motor relearning. They are

- elimination of unnecessary muscle activity,
- feedback,
- practice and
- the link between postural adjustment and movement.

Thus several different physiotherapy approaches are used in the management of patients who have suffered a stroke but these approaches are not mutually exclusive and are rarely delivered in a uniform or purist manner. All of the approaches aim to improve motor control and all of them demand that the therapist has a specialist training in the individual concepts.

In this study "Intense sensory motor stimulation" in the form of oil massage, re-education exercise program and electrical stimulation are instituted to augment motor recovery in stroke patients.

I. ROLE OF OIL MASSAGE IN STROKE REHABILIATTION

TECHNIQUE:

Effleurage or stroking massage involves lightly running the hand over the skin. This may be performed with either superficial or deep pressure and provides different effects depending on the depth of treatment. Deep stroking involves mechanical effects and should be performed with continuous contact of the hands from distal to proximal on the extremities, the back or the neck.

Once the proximal extent is reached the hands can be drawn back to the distal position applying light contact or no contact at all.

THERAPEUTIC USES:

In stroke rehabilitation, effleurage technique is effective

1) in assisting return of venous or lymphatic drainage in the paretic limb.
2) in increasing local circulation.
3) in increasing the stretchability of skin and flexibility of muscles. [28]

2. ROLE OF "ROM EXERCISES" IN STROKE MANAGEMENT

A. PASSIVE ROM

In stroke rehabilitation program, PROM exercises are instituted

a. to maintain joint and soft tissue integrity
b. to minimize the effects of the formation of contractures
c. to maintain mechanical elasticity of muscle
d. to assist circulation and vascular dynamics
e. to enhance synovial movement for cartilage nutrition and diffusion of materials in the joint
f. to decrease or inhibit pain
g. to help maintain the patient's awareness of movement. [29]

B. ACTIVE AND ACTIVE ASSISTIVE ROM

In stroke rehabilitation program, AROM and Active assistive ROM exercises are instituted to

a. to accomplish the same goals of passive ROM with the added benefits that result from muscle contraction.

b. to maintain physiologic elasticity and contractility of the participating muscle.

c. to provide sensory feedback from the contracting muscles

d. to provide a stimulus for bone and joint tissue integrity

e. to increase circulation and prevent thrombus formation

f. to develop coordination and motor skills for functional activities

g. to provide enough assistance to the muscles in a carefully controlled manner so that the muscle can function at its maximum level and progressively be strengthened.

3. ROLE OF ELECTRICAL STIMULATION IN MOTOR RECOVERY

HISTORY:

More than 30years ago, Liberson and colleagues introduced single channel electrical stimulation in stroke patients to prevent foot drop [30]. The technique is now generally known as FES because stimulation replaces or assists functional movements that are lost after injury to or disease of the CNS.

ELECTRICAL STIMULATION:

Fagri successfully used electrical stimulation to overcome the painful hemiplegic, subluxed shoulder[31] . Interestingly electrical stimulation also seems to speed up motor recovery, which is consistent with the observation of Fagri.

Clinical use of electrical stimulation is increasing. It is currently being used to restore and augment respiration, bladder, bowel and sexual function, hand grasp, standing and walking. [32]

PULSE:

Current is passed to the body tissues by means of wet pads, sponges or a bath of suitable solution. Thus the **conduction current** generated in the apparatus is changed to a **convection current** in a wet pads and tissue. The unit of stimulation current is called as a Pulse. This will cause one nerve impulse. If a 1 ms pulse is repeated every 10ms, they will cause a steady tingling sensation as they stimulate sensory nerve and a titanic muscle contraction as they stimulate motor nerves. Such current at higher intensities are used for muscle stimulation and are known as **Faradic Stimulation.**

The pulse rate can be expressed in Pulses/ Second (PPS) or the pulse frequency in hertz (HZ). In the Health care professions, electrical stimulation is used for 3 purposes;

a. to aid diagnosis
b. as a therapeutic tool and
c. to restore lost or damaged functions. [33]

Faradic type pulses:

Faradic type pulses are pulses of 0.1 - 1ms duration with repetition rates of 30 - 100HZ. The effective nerve stimulus is the **spike of voltage**, which can be about 1 ms in duration; the rest of the pulse having much lower does not cause nerve stimulation.

MECHANISM OF NERVE STIMULATION:

To apply electrical pulses to the tissues, a complete circuit is needed. So two electrodes with suitable conducting material are fixed to the skin.

For stimulating motor and sensory nerve i.e., large diameter fast conducting nerves, the pulse should be square wave and of short duration (0.5-1ms). Evenly alternating pulses have the advantage of avoiding any risk of chemical damage. Frequencies of around 80-100 HZ seem to be the best.[34]

MOTOR POINT:

In order to stimulate a normally innervated muscle effectively but painlessly the active electrode is applied to the motor point. This is a point on the skin surface at which maximum muscle contraction can be achieved because it is close to the point where the motor nerve trunk enters the muscle.

THE MOTOR UNIT:

The motor unit consists of an anterior born cell, the alpha motor neuron emanating from it and all the individual muscle fibres it supplies.

ELECTRICAL STIMULATION OF INNERVATED MUSCLE

Where voluntary active exercise is restricted, electrical stimulation may be substituted. It is usually applied by surging (or ramping) a series of short pulses at frequencies around 50-100 HZ i.e. a faradic type current. If the surge of pulses is made to last for say 2s and the interval between surges for 4s then a slow physiological muscle contraction and joint motion will be mimicked.

Electrical stimulation of muscle via its motor nerve has both immediate and long-term effects. Muscle contraction and vascular changes are examples of the former while muscle strengthening and structural changes in the muscle fibers may ultimately result from long term, chronic stimulation.

The flow of impulses in the motor nerve serves both to provoke immediate muscle contraction and in the long term to promote muscle fiber growth and change.

ROLE OF ELECTRICAL STIMULATION IN THE MOTOR RECOVERY AFTER STROKE

Electrical stimulation has been sporadically used in the treatment of hemiplegia. Reported benefits included decreasing spasticity, providing a supplementary means of Range of Movement (ROM) exercises, increasing strength and improving local blood flow in a paretic or paralyzed limb. There are also functional gains in the hemiplegic upper limb following treatment with electrical stimulation.[35] Electrical stimulation may facilitate motor recovery after stroke, reduce shoulder pain associated with hemiplegia and reduce cerebral spasticity [36] Electrical stimulation combined with voluntary activity led to similar or in a few cases even greater strength gains than that due to voluntary exercise alone.

Electrical stimulation has very favorable results that have been repeatedly presented [37]. Sonode and co-worker showed that the use of electrical stimulation could be a valuable complement to the normal training of the arm of hemiplegic patients in increasing motor functions significantly [38]. **Even modest improvement in ability may significantly enhance the quality of life**[39] . After electrical stimulation, there will be improvement of functions. Apart from muscle strengthening, the electrical stimulation effect led to an improvement of proprioception and supported relearning of motions. [40]

In weakened or weakening muscle, the value of electrical stimulation is much clearer and significant gains have been reported with improvement of muscle function. This has been successful in increasing the range of movement

in hemiplegic patients (Baker and parker, 1986) especially for the patient with shoulder pain and for the reduction of shoulder subluxation. [41]

Electrical stimulation has been applied as an alternative to manual passive movement to help prevent the loss of motion due to spasticity of the opposing muscles. This has been used successfully on the wrist and in finger movements of hemiplegic patients. The restoration of shoulder motion in plegic patients was successful by electrical stimulation [42]

Electrical stimulation is extensively used therapeutically to initiate and facilitate voluntary contraction of muscles in stroke patients.

ELECTRICAL STIMULATION FOR THE REDUCTION OF SPASTICITY

The effects of electrical muscle stimulation on spasticity are variable. In general, there have been three approaches. First, stimulation of antagonists to utilize the effect of **reciprocal inhibition.** Secondly, stimulation of the spastic muscle themselves and thirdly, alternately stimulating agonist and antagonist muscles. This latter approach has been tried using low frequency (3-35 HZ) 0.2ms pulses for some minutes daily over several weeks. The pulse frequencies are based on eutrophic stimulation and on the principle that afferent stimulation reinforces the presynaptic inhibition of motor neurons, thus reducing spasticity. After applying electrical stimulation, a statistically significant improvement was noted in all muscle tone / spasticity parameters in a clinical study.

MUSCLE FATIGUE AFTER ELECTRICAL STIMULATON

The rapid onset of muscle fatigue is one of the major limiting factors when using electrical stimulation to restore functional movement in individuals with a neurological deficit[43].

The choice of stimulation frequency is an important factor in reducing muscle fatigue. Although higher frequency stimulation generates stronger muscle contraction, muscle fatigue soon appears[44].

Benton and colleagues reported that rapid muscle fatigue occurs during continuous stimulation at frequencies higher than 30 to 40 HZ. Handa and colleagues [45] reported that a frequency of about 20 HZ would be recommended in clinical electrical stimulation. Based on these results, low frequency stimulation has been used in patients for continuous stimulation. Physiologically muscle fatigue has been sub grouped in to 2 types according to their physiological mechanisms;

Low frequency fatigue at 5 to 30 HZ and
High frequency fatigue at 50 to 100HZ. [46]

1. Low frequency fatigue – induced by low frequency stimulation and characterized by a subsequent long term loss of force.

2. High frequency fatigue - induced by high frequency stimulation and characterized by a subsequent rapidly recovering loss of force.

The mechanism of low frequency fatigue is thought to be an impaired excitation-contraction coupling so that less force is generated for each individual membrane excitation. [47]

The mechanisms of high frequency fatigue are thought to be a consequence of impaired membrane excitation, particularly associated with accumulation of potassium or conversely depletion of sodium in the interfiber space or more particularly in the extra cellular fluid contained in the transverse tubular system.[48]

TECHNIQUE OF APPLICATION

The patient is positioned so that the part to be treated is comfortably supported with muscles to be stimulated in a shortened position, although this may be modified when movement is to be produced, e.g. slight knee flexion allowing quadriceps stimulation to cause extension.

The skin surfaces to which the current will be applied must be examined and any cuts, abrasions or other lesions that might cause uneven current distribution is insulated (with a dab of petroleum jelly) or avoided. These areas should be washed to remove sebum and epithelial cells and left damp; using hot water warms the skin and helps to lower resistance further. The pads or sponges should be soaked in warm tap water, saline or sodium bicarbonate solution, which are somewhat better conductors particularly in soft water areas applied to the skin. Fixation is achieved with a rubber strap, a crepe or similar bandage or simply by body weight.

The nature of the treatment and the sensation to be expected, a tingling sensation and muscle contraction should be explained to the patient with reassurance that there is no way that any damage can be caused by this treatment.

PRECAUTIONS

The major resistance to current flow is the skin so that any break in the skin provides a low resistance pathway and therefore a high local current intensity may be painful.

If a current is applied at higher intensity very abruptly, the sensation and pain caused are likely to frighten the patient. If the current is increased

gradually over a few minutes, subjects become accustomed or accommodate to the electrical stimulation, so that the higher intensities are easily tolerated.

As current flows so the feelings become familiar and toleration rises. When applying treatment such fears must be taken into account. The patient must be carefully reassured and treatment applied initially at low intensity, which can be gradually increased.

MATERIALS AND METHODS

BACKGROUND AND PURPOSE:

Review of literature shows that there will be natural, spontaneous neurological recovery after stroke. The mechanism to explain recovery is **Neuroplasticity.** Brain plasticity is the ability of the nervous system to modify its structural and functional organization. The most plausible forms of plasticity are collateral sprouting of new synaptic connections and unmasking of previously latent functional pathways.

Experimental evidence indicates that plasticity can be altered by several external conditions, including pharmacological agents, electrical stimulation and environmental stimulation. Many different electrical stimulation applications have been explored since the pioneering work of Liberson et al on foot drop.

So in this study electrical stimulation is used to augment motor recovery in patients being rehabilitated after acute stroke, in addition to olive oil massage and reeducation exercise programme.

METHODS:

In this study, 20 hemiplegics admitted to PSG Hospitals, affiliated to the PSG Institute of Medical Sciences & Research, Coimbatore between March 2011 and 31st May 2011 were selected. No specific selection criteria with reference to age, severity of paralysis, aphasia, neglect, incontinence and cognitive function were applied. But stroke cases of longer duration were not included in this study. On admission to the rehabilitation facility, age and gender were documented for each patient as were the type and site of the cerebral lesion. Duration between stroke onset and rehabilitation admission were also recorded.

After thorough neurological examination, CT Brain was done to confirm the diagnosis. Then the neurological status was assessed by Scandinavian Stroke Scale and functional status was assessed by Functional Independence Measure. Then the treatment programme started.

ELECTRICAL STIMULATION

Electrical stimulation parameters:

Pulse width : 300ms
Intensity : 100v
Frequency : 50 Hz
Duty cycles:
On time: 5 sec
Off time: 10 sec

THE MUSCLES STIMULATED IN THE UPPER LIMB:

Deltoid, Supraspinatous, Biceps, Triceps, Common flexors of forearm, Common extensors of forearm and the Intrinsic muscles of hand.

THE MUSCLES STIMULATED IN THE LOWER LIMB:

Gluteus maximus, Quadriceps, Tibialis anterior

Each muscle is stimulated for 10 minutes. The active electrode is kept over the motor point of the muscle to be stimulated. The reference electrode is kept over the near by bony point. The muscle contraction / relaxation time ratio is kept at 5/10 seconds. The stimulation period was maintained throughout as 5 seconds. The patients receive **electrical stimulation** daily for 1½ - 2 hours. After electrical stimulation **olive oil massage** was done to the affected upper and lower limbs. The **PROM exercises** were done to the affected upper and lower limbs to prevent the joint contractures. Once the patient developed volitional movement, **strengthening program** was instituted. Then movement

pattern was taught. **ADL training** to the upper limb and **gait training** to the lower limb were taught.

OUTCOME MEASURES:

Outcomes were assessed in every two weeks.

The neurological recovery was assessed by Scandinavian stroke scale.

The functional recovery was assessed by Functional independence measure.

FOLLOW UP:

The patients were followed up till June 15th 2011; in the follow-up it was found that most patients were able to maintain their functions and make further improvements. Also their physiological well-being seemed quite good.

The benefit of this recovery was maintained for a long time perhaps because all patients continued to do exercise program at home.

STATISTICAL ANALYSIS & DISCUSSION

HYPOTHESIS- I

I. There will be significant level of **neurological recovery** in the treated group of stoke patients during the time of **discharge** and in the **follow-up** period, when compared to their neurological status at the time of **admission**.

Ia. There will be significant level of neurological recovery in the stroke patients during the time of **discharge** when compared to their neurological status at the time of **admission**.

Ib. There will be significant level of neurological recovery in the stroke patients during the time of **follow-up** when compared to their neurological status at the time of **admission**.

HYPOTHESIS- II

II. There will be significant increase in the **functional independence level** of the stroke patients during the time of **discharge** and the **follow-up**, when compared to the functional independence level at the time of **admission.**

IIa. There will be significant increase in the functional independence of the patients at the time of **discharge** when compared to their functional independence level at the time of **admission**.

IIb. There will be significant increase in the functional independence level of patients at the time of **follow-up**, when compared to their functional independence level at the time of **admission**.

STATISTICAL ANALYSIS

DISCUSSION:-

To analyze a data, two types of statistical techniques are used.

They are,

a) Descriptive statistical technique and
b) Inferential statistical technique.

In the descriptive statistics, Mean and Standard deviation are calculated for the scores and tabulated as follows:-

Table No: 1

Shows Mean, Standard Deviation of Scandinavian stroke scale scores of stroke patients at different stages

S.no	STAGES	NO. OF CASES	MEAN	STANDARD DEVIATION
1.	On admission	20	32.82	9.16
2.	On discharge	20	47.90	7.68
3.	On follow up	20	53.13	6.85

The above table shows the mean score of Scandinavian stroke scale (SSS) on discharge and follows up. The standard deviation also seems to be within the normal limits. It does not skewed very much. So this distribution can be said as normal distribution.

To analyze the significant differences between the three means, the "Analysis of Variant Technique" (ANOVA) was used.

Table No: 2

Shows "ANOVA of Scandinavian stroke scale scores of different groups of stroke patients"

S.no	Source of variations	Sum of squares	DF	Mean square	F	Level of significance
1	Between groups	6269.63	2	3134.81		
					37.31	>0.01
2	Within groups	5366.27	84	63.88		

The F ratio 37.31 was found to be significant at 0.01 level indicates that stroke level of different stages is significantly different from one another. The hypothesis I has been proved by this statistical finding.

To analyze further which group is different from another group, 'student't' test' was used.

Table No: 3

Shows Mean, Standard deviation and't' value of Scandinavian stroke scale scores on admission and discharge

S.no	Group	No. of cases	Mean	Standard deviation	T	P
1	On admission	20	32.82	9.16		
					6.79	>0.01
2	On discharge	20	47.90	7.68		

The above't' value 6.79 which is significant at 0.01 level reveals that the differences between the means of Scandinavian stroke scale score on admission and discharge are significantly different. It indicates that the patients condition have markedly improved at the time of discharge when compared to the condition at the time of admission. This finding proves the hypothesis Ia.

To analyze the mean score of admission and follow up, again the‘t’ test was calculated as follows.

Table No: 4

Shows Mean, Standard deviation and ‘t’ value of Scandinavian stroke scale scores on admission and follow-up

S.no	Group	No. of cases	Mean	Standard deviation	T	P
1	On admission	20	32.82	9.16		
					9.58	>0.01
2	On discharge	20	53.13	6.85		

The above ‘t’ value of 9.58 which is significant at 0.01 level, indicates that the mean score of follow up was significantly different from mean score of admission level. This finding proves the hypothesis Ib.

DISCUSSION:

The above results indicate that the condition of the stroke patients has significantly improved at the time of discharge and follow up when compared to the time of admission. It is possible to say that the different forms of ***“intense sensory motor stimulation”*** would have contributed much for the recovery of the stroke patients. Similarly after the discharge also, the improved condition was maintained significantly. This has proved the efficiency of the intense sensory motor stimulation in stroke rehabilitation.

Table No: 5

Shows Mean Standard deviation of functional independence measure scores of stroke patients at various stages

S.no	Group	No. of cases	Mean	Standard deviation
1.	On admission	20	75.79	19.22
2.	On discharge	20	105.17	12.75
3.	On discharge	20	115.35	12.00

The above table reveals that the mean and standard deviation of functional independence scorers of stroke patients. The mean score of functional independence levels of the patients during admission, is very low than the mean score of functional independence at the time of discharge and follow up. Similarly the measure of standard deviation indicates that there is not much deviation in the distribution.

To analyze further and to find out the significant differences between the means, the “analysis of variant” (ANOVA) was worked out. The results are as follows:

Table No: 6

Shows “ANOVA” of functional independence measure scores of stroke patients at different levels

S.no	Source of variations	Sum of squares	DF	Mean square	F	Level of significance
1	Between groups	24499.13	2	12249.56		
					43.94	>0.01
2	Within groups	23415.73	84	278.75		

The F ratio of the above table 43.94 which is significant at 0.01 level reveals that, there is significant difference in the functional independence levels among the three groups. This result proved the hypothesis II.

To analyze further which group is differently from another group, the "student't' test" was used. The results are tabulated as follows:

Table No: 7

Shows Mean, Standard deviation and "t" value of functional independence measure scores on admission and discharge

S.no	Group	No. of cases	Mean	Standard deviation	'T'	P
1	On admission	20	75.79	19.22		
					6.87	>0.01
2	On discharge	20	109.17	12.75		

The above't' value 6.87 which is significant at 0.01 level, indicates that the functional independence level of the patients have significantly improved when compared to the functional independence levels of the patients during admission. These findings prove the hypothesis IIa.

Similarly to find out the differences between the functional independence levels at the time of admission and follow up, the't' test was used. The results were tabulated as follows:

Table No: 8

Shows Mean, Standard deviation and "t" value of functional independence measure scores on admission and follow up

S.no	Group	No. of cases	Mean	Standard deviation	'T'	P
1	On admission	20	75.79	19.22		
					9.42	>0.01
2	On follow up	20	115.37	12.00		

The't' value 9.42 which is significantly at 0.01 levels indicate that, the functional independence level of the patients during follow up is significantly more when compared to the functional independence level at the time of admission. This result proves the hypothesis IIb

.DISCUSSION

The above findings prove that the functional independence of the stroke patients have improved very much at time of discharge and follow up.

The possible reason could be due to the attack of stroke, the movements of the extremities were lost. That could be the possible reason for their low level of functional independency at the time of admission. Since the stroke level is highly related with the motor function, it would have retarded the physical function. Once the severity of the stroke level is decreased, the functional independency will also improve significantly. Another possibility could be the beneficial effects of occupational therapy for the upper limbs and motor training for the lower limbs, which could have improved the functional independency of the stroke patients during discharge and follow up period. Since the stroke level has got direct bearing the motor function, the researcher wants to know the relationship between the magnitude of the stroke and the

level of functional independency at the time of admission, at the time of discharge and at the time of follow-up.

To find out the relationship between these two levels, the 'Correlation Coefficient Techniques' has been used. The results are given below:

Table No: 9

Shows the correlation between and Scandinavian stroke scale scores on admission and functional independence measure score on admission

S.no	Group	No. of cases	correlation co-efficient "r"	Level of Significance P
1	Scandinavian stroke scale score on admission	20		
			0.62	>0.01
2	Functional independence measure score on admission	20		

The 'r' value 0.62, which is significant at 0.01 level indicates that, there is a high level of relation between the severity of stroke and functional independence level.

Table No: 10

Shows the correlation between and Scandinavian stroke scale scores on admission and functional independence measure score on discharge

S.no	Group	No. of cases	correlation co-efficient "r"	Level of Significance P
1	Scandinavian stroke scale score on discharge	20		
			0.81	>0.01
2	Functional independence measure score on discharge	20		

The above table showing the correlation coefficient value of 0.81 which is significant at 0.01 levels indicates that the high level of relation between the recovery of stroke at time of discharge and functional independence level at the time of discharge.

Table No: 11

Shows the correlation between and Scandinavian stroke scale scores on admission and functional independence measure score on follow up

S.no	Group	No. of cases	correlation co-efficient "r"	Level of Significance P
1	Scandinavian stroke scale score on follow up	20		
			0.97	>0.01
2	Functional independence measure score on follow up	20		

The correlation value 0.97 which is significant at 0.01 levels shows the high level of relation between the recovery of stroke and functional independent level.

The above correlation coefficient value suggests that, when the severity of stroke level increases, the functional independence level significantly decreases. Whereas, when there is improvement of the stroke state, the functional independence level will also increase significantly.

It was decided to analyze further, the magnitude of stroke, the neurological recovery and the functional independency in different possible parameters like duration of treatment, time lag before admission, pathology of stroke, topography of the brain affected and vascular territory of different cerebral arteries affected.

Since the total number of cases in certain conditions is found to be less, the researchers could not use any parametric tests and confirmed only with the non parametric tests to analyze the data.

The percentage was calculated for each condition and the same was discussed.

Table No: 12

Shows the rate of neurological recovery (Scandinavian stroke scale score) of stroke patients who have undergone treatment for different duration.

S. no	Duration of Treatment	Neurological Recovery (SSS Score)		
		On admission	On discharge	On follow up
1	1-30 days	59%	92%	96%
2	1-60 days	59%	88%	94%
3	1-90 days	43%	69%	78%

It is learnt from the above table that the stroke cases with severe neurological damage (less Scandinavian stroke scale score) on admission required longer duration of treatment.

Table No: 13

Shows the rate of functional recovery (FIM score) of stroke patients who have undergone treatment for different duration.

S. no	Duration of Treatment	Neurological Recovery (SSS Score)		
		On admission	On discharge	On follow up
1	1-30 days	78%	93%	98%
2	1-60 days	60%	84%	93%
3	1-90 days	50%	74%	82%

From the above table, it is clear that the stroke cases with severe functional impairments (less FIM score) on admission required longer duration of treatment.

Table No: 14

Shows the rate of neurological recovery of stroke cases admitted for different time lag period

S. no	Time lag	Neurological Recovery (SSS Score)		
		On admission	On discharge	On follow up
1	1-30 days	56%	86%	95%
2	1-60 days	59%	77%	84%
3	1-90 days	53%	73%	87%

The above table shows the neurological recovery (percentage of SSS score) is maximum at discharge and follows up in the group of stroke cases which were admitted for treatment with short time lag.

Table No: 15

Shows the rate of functional recovery of stroke cases admitted for different time lag period

S. no	Time lag	Functional Recovery (FIM Score)		
		On admission	On discharge	On follow up
1	1-30 days	59%	85%	93%
2	1-60 days	65%	80%	89%
3	1-90 days	52%	77%	85%

The above table shows the functional recovery (percentage of FIM score) on discharge and follow up is maximum in the group of stroke cases which were admitted early for treatment (short time lag).

Table No: 16

Shows the rate of neurological recovery (SSS Scores) in different pathological groups

S. no	Pathology	Neurological Recovery (SSS Score)		
		On admission	On discharge	On follow up
1	Embolism	37%	81%	93%
2	Hemorrhagic	33%	38%	51%
3	Thrombotic	58%	84%	93%

Table No: 17

Shows the rate of functional recovery (FIM Scores) in different pathological groups

S. no	Pathology	Functional Recovery (FIM Score)		
		On admission	On discharge	On follow up
1	Embolism	64%	80%	96%
2	Hemorrhagic	45%	68%	68%
3	Thrombotic	60%	84%	92%

From the above two tables (16 & 17), the inference drawn is that neurological and functional recovery on discharge and follow up are greater in the embolic and thrombotic stroke cases; whereas neurological and functional recovery on discharge and follow up are less in hemorrhagic stroke cases.

Table No: 18

Shows the rate of neurological recovery (SSS Scores) in different levels of brain (Topograph)

S. no	Topograph	Neurological Recovery (SSS Score)		
		On admission	On discharge	On follow up
1	Cortical level	57%	85%	93%
2	Internal Capsular Level	53%	85%	85%
3	Brainstem level	63%	82%	100%

Table No: 19

Shows the rate of functional recovery (FIM Scores) in different levels of brain (Topograph)

S. no	Topograph	Functional Recovery (FIM Score)		
		On admission	On discharge	On follow up
1	Cortical level	60%	85%	93%
2	Internal Capsular Level	40%	78%	86%
3	Brainstem level	72%	92%	99%

From the above two tables (18 & 19), it is learnt that neurological and functional recovery on discharge and follow up are fair if the stroke is at the level of the internal capsule. The neurological and functional recovery on discharge and follow up are better if the stroke is at the levels of cortex or brainstem.

Table No: 20

Shows the rate of neurological recovery (SSS Scores) of stroke cases at various cerebral artery territory levels

S. no	Territory of cerebral artery involved	Neurological Recovery		
		On admission	On discharge	On follow up
1	ACA*	-	-	-
2	PCA*	-	-	-
3	Basilar artery	63%	83%	100%
4	MCA	56%	83%	91%

Table No: 21

Shows the rate of functional recovery (FIM Scores) of stroke cases at various cerebral artery territory levels

S. no	Territory of cerebral artery involved	Functional Recovery (FIM Score)		
		On admission	On discharge	On follow up
1	ACA*	-	-	-
2	PCA*	-	-	-
3	Basilar artery	72%	92%	99%
4	MCA	52%	83%	98%

*: No cases at these levels of study.

It is clear from the two tables (20 & 21) that the rate of neurological and functional recovery on discharge and follow up are better in stroke cases due to basilar artery territory involvement than middle cerebral artery territory involvement.

Table No: 22

Shows the rate of motor recovery in the extremities (SSS Scores) of stroke patients who have undergone treatment for different duration

Duration of treatment	Motor Recovery (SSS Score)		
	On admission	On discharge	On follow up
1-30 days UL	0%	75%	92%
LL	83%	97%	100%
1-60 days UL	24%	70%	87%
LL	42%	77%	93%
1-90 days UL	0%	43%	58%
LL	25%	55%	70%

Table No: 23

Shows the rate of motor recovery in the extremities (FIM Scores) of stroke patients who have undergone treatment for different duration.

Duration of treatment	Motor Recovery (FIM Score)		
	On admission	On discharge	On follow up
1-30 days UL	61%	80%	93%
LL	86%	100%	100%
1-60 days UL	40%	75%	90%
LL	37%	76%	92%
1-90 days UL	29%	43%	73%
LL	23%	60%	72%

From the above tables it is learnt that the neurological and functional motor recovery in both extremities on discharge and follow up are maximum in stroke cases whose admission Scandinavian stroke scale scores and functional independence measure scores were high in both limbs. The stroke cases which have low Scandinavian stroke scale scores and low functional independence measure scores on admission required longer duration of treatment.

Table No: 24

Shows the rate of motor recovery (SSS Scores) in the extremities of stroke cases admitted for treatment in different time lag period

Time lag	Motor Recovery (SSS Score)		
	On admission	On discharge	On follow up
0-30 days UL	24%	74%	88%
LL	40%	79%	95%
31-60 days UL	9.5%	51%	64%
LL	50%	68%	80%
61-90 days UL	5%	47%	72%
LL	30%	57%	80%

Table No: 25

Shows the rate of motor recovery (FIM Scores) in the extremities of stroke cases admitted for treatment in different time lag period

Time lag	Motor Recovery (FIM Score)		
	On admission	On discharge	On follow up
0-30 days UL	42%	76%	92%
LL	37%	77%	89%
31-60 days UL	48%	68%	81%
LL	47%	72%	84%
61-90 days UL	27%	62%	73%
LL	30%	32%	84%

The findings from the above tables (24 & 25) prove that neurological and functional motor recovery in both extremities on discharge and follow up are more in stroke groups which were admitted early for rehabilitation (short time lag).

Table No: 26

Shows the rate of motor recovery in the extremities (SSS Scores) in different pathological groups

S.no	Duration of treatment	Motor Recovery (SSS Score)		
		On admission	On discharge	On follow up
1	Embolism UL	0%	66%	75%
	LL	0%	72%	94%
2	Hemorrhage UL	0%	0%	0%
	LL	16%	16%	27%
3	Thrombosis UL	20%	70%	85%
	LL	47%	67%	91%

Table No: 27

Shows the rate of motor recovery in the extremities (FIM Scores) in different pathological groups

S.no	Duration of treatment	Motor Recovery (SSS Score)		
		On admission	On discharge	On follow up
1	Embolism UL	64%	94%	100%
	LL	14%	57%	85%
2	Hemorrhage UL	35%	42%	43%
	LL	28%	68%	68%
3	Thrombosis UL	41%	73%	87%
	LL	39%	79%	91%

It is clear from the above tables (26 & 27) that the neurological and functional motor recovery in both extremities on discharge and follow up are better in stroke groups due to embolism and thrombosis; recovery is less in hemorrhagic pathology.

Table No: 28

Shows the rate of motor recovery (SSS Scores) in the extremities in cases of stroke at different levels of brain (Topograph)

S.no	Topograph	Motor Recovery (SSS Score)		
		On admission	On discharge	On follow up
1	cortical level UL	21%	67%	90%
	LL	44%	82%	97%
2	Internal capsular level UL	7%	56%	73%
	LL	41%	62%	79%
3	Brainstem level UL	50%	75%	100%
	LL	28%	72%	100%

Table No: 29

Shows the rate of motor recovery (FIM Scores) in the extremities in cases of stroke at different levels of brain (Topograph)

S.no	Topograph		Motor Recovery (FIM Score)		
			On admission	On discharge	On follow up
1	cortical level	UL	40%	75%	99%
		LL	40%	75%	88%
2	Internal capsular level	UL	40%	63%	77%
		LL	32%	71%	83%
3	Brainstem level	UL	71%	100%	100%
		LL	45%	71%	97%

The above tables (28 & 29) prove that neurological and functional motor recovery in the extremities on discharge and follow up are better if the stroke occurs at cortical or brain stem level. Motor recovery is fair if stroke occurs at internal capsular level.

Table No: 30

Shows the rate of motor recovery (SSS Scores) in the extremities of stroke cases at various cerebral artery territory levels

S. no	Territory of cerebral artery involved		Motor Recovery (SSS Score)		
			On admission	On discharge	On follow up
1	ACA*		-	-	-
2	PCA*		-	-	-
3	Basilar artery	UL	50%	75%	100%
		LL	28%	72%	100%
4	MCA	UL	19%	68%	80%
		LL	43%	77%	96%

Table No: 31

Shows the rate of motor recovery (FIM Scores) in the extremities of stroke cases at various cerebral artery territory levels

S. no	Territory of cerebral artery involved	Motor Recovery (FIM Score)		
		On admission	On discharge	On follow up
1	ACA*	-	-	-
2	PCA*	-	-	-
3	Basilar UL artery	71%	100%	100%
	LL	46%	72%	97%
4	MCA UL	43%	76%	86%
	LL	38%	75%	92%

*: No cases at these levels of study.

From the above tables (30 & 31) it is learnt that neurological and functional motor recovery on discharge and follow up are better if the basilar artery territory is involved. Motor recovery is fair in the cases of middle cerebral artery involvement.

Table No: 32

Shows the distribution of Scandinavian stroke scale scores at different stages of stroke

S.no	SSS Score grade	No. of cases on admission	No. of cases on discharge	No. of cases on follow up
1	Mild(45-58)	3	17	17
2	Moderate(30-44)	10	1	3
3	Severe(15-29)	6	2	0
4	Very severe(0-14)	1	0	0

The above distribution table proves the beneficial effects of the "intense sensory motor stimulation" in augmenting the stroke recovery. At the time of admission, only three cases were in the mild grade (45-48) of Scandinavian stroke scale score. But at time of discharge and follow up 17 cases have moved to mild grades of Scandinavian stroke scale. This shows the magnitude of neurological recovery with intense sensory motor stimulation.

Table No: 33

Shows the distribution of Functional independence measure scores at different stages of stroke

S.no	FIM Score grade	No. of cases on admission	No. of cases on discharge	No. of cases on follow up
1	<50	3	0	0
2	50-75	6	1	0
3	75-100	9	3	3
4	>100	2	16	17

From the above distribution table (33) it is learnt that at time of admission only 2 cases have scored more than 100 in functional independence measure. But at time of discharge and follow up, there are 16 and 17 cases respectively who have scored more than 100 in functional independence measure score. This again proves the efficiency of intense sensory motor stimulation in augmenting motor recovery in stroke cases.

CONCLUSION

The following conclusions are drawn from the above study:

- There was a significant level of neurological and functional recovery in the study group during the time of discharge and in the follow up period, when compared to their neurological and functional status at the time of admission. This shows the effectiveness of intensive sensory motor stimulation in augmenting motor recovery in stroke patients.

- The improvement in the neurological and functional status observed at the time of discharge was maintained during the follow up period.

- Stroke cases with severe neurological damage, as evidenced by low Scandinavian stroke scale score on admission required longer duration of treatment.

- The neurological and functional recovery, as evidenced by the high percentage of Scandinavian stroke scale score and Functional independence measure score on discharge and follow up are maximum in the stroke cases which were admitted for treatment with short time lag.

- In the hemorrhagic stroke cases, the neurological recovery at the time of discharge and follow up are less when compared to thrombotic and embolic stroke cases.

- The neurological and functional recovery on discharge and follow up are better if the lesion is at the level of the cortical than at the capsular level.

- Motor recovery is faster in the lower limbs than the upper limbs

The management of stroke, so long a **"Cinderella condition**" is changing rapidly as new developments appear for acute treatment, rehabilitation and secondary prevention. Stroke is no longer an untreatable or unpreventable condition and it is vital that hospitals design appropriate systems to manage patients in an interdisciplinary environment.

Though this study size was relatively small, a treatment effect was demonstrated. In our study, the maximum improvement in the motor recovery was observed at 3 months. These results improved again slightly and remained constant up to the follow up period.

Significant improvement in motor recovery in the stroke patients indicates that "**intense sensory motor stimulation**" is an effective method of stroke rehabilitation to augment motor recovery. Electrical stimulation is a relatively low cost intervention. Concerns that subjects would find the treatment uncomfortable or inconvenient were unfound. All subjects reported that the sensation of the muscle contraction following electrical stimulation and the resultant movement of the limb was encouraging. No stimulation induced complications requiring medical attention such as burns, falls or fractures occurred for ant patient. A few subjects showed minor skin irritation transiently.

Thus '**intense sensory motor stimulation program'** could achieve a better functional outcome in stroke patients and could shorten the hospital stay for rehabilitation.

BIBLIOGRAPHY

1. Dalal P, Bhattacharjee M, Vairale J, Bhat P.UN Millennium development goals; Can we halt the stroke epidemic in India Ann Indian AcadNerol 2007; 10: 130-6.

2. Dalal P.M. (1981) Japanese circulation J., 46: 621-624 (The First Asian-pacific symposium on stroke).

3. Chollet. F, et al: Mechanism of motor recovery after cerebro vascular accident, Neurology 1999; 155(9): 718-24.

4,5. Dalal P, Bhattacharjee M, Vairale J, Bhat P.UN Millennium development goals; Can we halt the stroke epidemic in India Ann Indian AcadNerol 2007; 10: 130-6.

6. Thompson D.W. Clinical epidemiology of stroke Neurol clin 1996; 14(2): 309-15.

7. Whisnant JP, et al: Classification of cerebro vascular disease. III special report from the National Institute of Neurological disorders and Stroke. Stroke 1990; 21: 637-76.

8. Biller J. Medical management of acute ischemia Neurol clin 1992; 10(1): 63-85.

9. Barsan WG, et al: Urgent therapy for acute stroke: effects of a stroke trail on untreated patients. Stroke 1994:25: 2132-7.

10. Caplan L.R. et al: Should thromolytic therapy be the first line of treatment for acute ischemic stroke? N Engi J med 1997:337; 1309-13.

11. Bath Ef Bath PMW. What is the correct management of blood pressure in acute stroke? Cerebro Vascular Dis 1991; 7:205-13.

12. Linderstom E, et al: Reliability of Scandinavian Neurological stroke scale. Cerebro vasc. Dis 1991; 1:103-7.

13. Franchignomi, et al: Length of stay of stroke rehabilitation in patients; Prediction through the functional independence measure. Ann-I st super-Sanita 1998; 11: 181-3.

14. Bohannon Rw, et al: Rehabilitation goals of patients with hemiplagia. Int J Rehabil res 1998; 11: 181-3.

15. W.J.Oczkowski. Neural network modeling accurately predicts the functional outcome of stroke survivors with moderate difficulties. Arch Phys med Rehabli 1997; 78:340-5.

16. Watson L.D Stages of stroke: a model for stroke rehabilitation. British Journal of Nursing 1998 Jan 11-24; 7(11): 631-40.

17. Kotla M, et al; The profile of recovery from stroke and factors influencing outcome. Stroke 1984; 15; 1039-1044.

18. Lehmann JF, et al: Stroke rehabilitation: outcome and prediction. Arch Phys med Rehabli 1975; 56: 383-389.

19. Andrews k, et al: The rate of recovery from stroke- and its measurement. Int. Rehabli med; 3: 155-161.

20. Bach-Y-Rita P: Central nervous system lesions: Sprouting and unmasking in rehabli 1981; 62; 41-47.

21. Bach-Y-Rita P: Brain plasticity as a basis of development of rehabilitation procedures for hemiplegia. Scand J Rehabli med 1981; 13:73-83.

22. Heinenmann A W, et al: Multivariate analysis of improvement and outcome following stroke rehabilitation. Arch Neurol 1987; 44:1167-72.

23. Ottenbacher K.J, et al: Multivariate analysis of improvement and outcome following stroke rehabilitation research. Arch Neurol 1993; 50:37-44.

24. Stroke unit Trailist collaboration. How do stroke units improve patient's outcome? Stroke 1997; 28: 2139-44.

25. Gersten J W. et al: Comparison of home and clinical rehabilitation for chronically ill and physically disabled persons. Arch phys and Rehabli 1968: 49; 615-42.

26. Richards C L, et al: Task- Specific physical therapy for optimization of gait recovery in acute stroke patients. Arch Phys med Rehabli 1993; 73:612-20.

27. Kwakkel G. et al: effect of intensity of rehabilitation after stroke: stroke 1997; 28: 1550-6.

28. Hofkosj J M: Classical massage. In Basmajain JV (ed): Manipulation, traction and massage, 1985, PP 263-269.

29. Frank C, et al: Physiology and therapeutic value of passive joint motion. Clin Orthop 185: 113, 1984.

30. Liberson WT, et al: Functional Electrotherapy, Arch Phys med 1961; 42:105-5.

31. Faghri PD, et al: The effects of functional electrical stimulation on shoulder subluxation, arm function recovery and shoulder pain in hemiplegic stroke patients. Arch Phys med 1994; 75:73-9.

32. Peckham PH Neural Prostheses: Clinical Applications of functional electrical stimulation in spinal cord injury paraplegia, 1992; 30:96-101.

33. Rushton. Functional electrical stimulation physiol-meas. 1997, Nov; 18(4) 241-75.

34. Low and Reed Electrotherapy explained principles and practice: Edision2. Electrical Stimulation of nerve and muscle p.63.

35. Weingarden et al: Upper limb functional electrical stimulation for walker ambulation in hemiplagia. American Journal of physical medicine and rehabilitation 1997, Jan- Feb; 1976(1); 63-67.

36. Chae J: HartR: Comparison of discomfort associated with surface and percutaneous intra muscular electrical stimulation for persons with chronic hemiplegia; American Journal of Physical Medicine and rehabilitation 1998 Nov-Dec77 (6)516-22.

37. Fall M: Advantages and pitfalls of functional electrical stimulation; Acta-Obstet-Gyynecol-scand-Suppl-1998; 168; 16-21.

38. Sonode L Gip C, Fernacus SE et al. Stimulation with low frequency transcutaneous electrical nerve stimulation increases motor function of the post-stroke paretic arm. Scond J rehabli Med 1998; 30; 95-99.

39. Appelros P. For who is training after stroke most beneficial? Selection method exists and should be used. Lakar Tidningen 1999 May 12; 96(19); 2343-7.

40. Rakos M: Electromyogram-controlled functional electrical stimulation for treatment of the paralyzed upper extremity. Aortif- organs. 1999 may: 23(5) 466-9.

41. Baker L.L, Parker K. Neuromuscular electrical stimulation of the muscles surrounding the shoulder. Phys. Ther; 66, 1930-7.

42. Baker L.L et al: Electrical stimulation of wrist and fingers for hemiplegic patients. Phys. Ther, 59, 1495-9.

43. Mourseler N: Evaluation of patterned stimulation for use in surface functional electrical stimulation systems. Med. Eng.Phys.1998 Jul: 20(5) 319-24.

44. Benton LA et al: Functional electrical stimulation, a practical clinical guide 2nd Ed, Downey (CA); Rancho Los Amigos Rehabilitation Engineering center, 1981.

45. Handa et al: Electrically induced hand movements and their application for daily living. In: Proceedings of the 8th international symposium on external control of human extremities; 1984 Oct 23-25; Dubrovnik 1984.p 169-80.

46. Edwards RHT: Human muscle fatigue: Physiological mechanisms. London, Pitman Medical (Ciba Foundation Symposium 82) 1981.

47. Copper RG et al: Human muscle fatigue: Frequency dependence of excitation and force generation J. Physiol 1998, 397: 585-99.

48. Jones DA et al: Excitation frequency and muscle fatigue: Mechanism responses during voluntary and stimulated contractions. Exp. Neurol 1979; 64: 401-413.

STROKE REHABLITATION PREFORMA

PSG HOSPITALS

PSG INSTITUTE OF MEDICAL SCIENCES & RESEARCH

COIMBATORE

1. Name of the patient :
2. Age :
3. Sex :
4. Occupation :
5. Residential Address :
6. Date of onset of stroke :
7. Acute care management :

- Given Yes/No
- Cranial Imaging Study
  - i. Immediate CT Brain Yes/No
    Hemorrhagic stroke Yes/No
  - ii. Delayed CT Brain Yes/No
    Ischemic Stroke Yes/No
    Lacunar Stroke Yes/No

- Treatment :
  - **i) Drug therapy**
    Anticoagulant- Heparin Yes/No
    Calcium Channel blocking agent- Nimodipine Yes/No
    Thrombolytic agent-
    Streptokinase Yes/No
    Anti-platelet drugs-
    Aspirin Yes/No
    Dipyridamole Yes/No
    Ticolopidine Yes/No

    Cerebro active drug-
    Piratam Yes/No
    Dihydro ergotoxine (Hydergine) Yes/No
    Pyritinol (Encephabol) Yes/No

ii) Physiotherapy:

Breathing Exercise	Yes/No
Antispastic positioning	Yes/No
Supine to sitting	Yes/No
Bed mobility	Yes/No

8. Date of admission to the rehabilitation unit:

ON EXAMINATION

9. CARDIO-VASCULAR SYSTEM

Pulse rate	:	
Blood Pressure	:	
Sitting Position	:	
Lying Position	:	
Heart sound	:	
Murmur		
Systolic	:	Yes/No
Diastolic	:	Yes/No

10. **RESPIRATORY SYSTEM**

Respiratory Rate	:	
Respiratory Rhythm	:	
Vesicular breathe sounds	:	Yes/No
Added sounds	:	Yes/No

11**. ABDOMEN**

Soft	:	Yes/No
Organomegaly	:	Yes/No
Free Fluid	:	Yes/No

12. **NEUROLOGICAL EXAMINATION**

A. HIGHER FUNCTIONS:

Consciousness : on admission on discharge

Alert

Responses to verbal stimuli

Responses to pain

Unresponsive

	on admission	on discharge
Appearance Behavior		
Apprehensive		
Apathetic		
Agitated		
Confused		
Emotional Status		
Depressed		
Exited		
Euphoric		
Elated		
Staple		
Handedness		
Right		
Left		
Sleep pattern		
Normal		
Altered		

B.<u>COGNITIVE FUNCTIONS</u>

<u>ORIANTATION:</u>

TIME	Time	Yes/No	Yes/No
	Date	Yes/No	Yes/No
	Day	Yes/No	Yes/No
	Month	Yes/No	Yes/No
	Year	Yes/No	Yes/No
PLACE	Ward	Yes/No	Yes/No
	Hospital	Yes/No	Yes/No
	District	Yes/No	Yes/No
	Town	Yes/No	Yes/No
	Country	Yes/No	Yes/No
<u>ALERT</u>		Yes/No	Yes/No

		on admission	on discharge
ATTENTION			
	Focused	Yes/No	Yes/No
	Sustained	Yes/No	Yes/No
	Divided	Yes/No	Yes/No
REGISTRATION			
Able to name			
	Three objects	Yes/No	Yes/No
	Two Objects	Yes/No	Yes/No
	One object	Yes/No	Yes/No
RECALL			
Able to recall			
	Three objects	Yes/No	Yes/No
	Two Objects	Yes/No	Yes/No
	One object	Yes/No	Yes/No
MEMORY			
	Recent	Yes/No	Yes/No
	Short term	Yes/No	Yes/No
	Long term	Yes/No	Yes/No
INTELLIGENCE			
Abstract thinking		Yes/No	Yes/No
	Reasoning	Yes/No	Yes/No
	Judgment	Yes/No	Yes/No
	Attention	Yes/No	Yes/No
	Calculation	Yes/No	Yes/No
PERCEPTION			
	Delusion	Yes/No	Yes/No
	Hallucination	Yes/No	Yes/No
	Illusion	Yes/No	Yes/No
	Obsession	Yes/No	Yes/No

C.**VISUO SPATIAL FUNCTIONS**

	on admission	on discharge
Visual Neglect	Yes/No	Yes/No
Apraxia		
Ideomotor	Yes/No	Yes/No
Ideational	Yes/No	Yes/No
Construction	Yes/No	Yes/No
Dressing	Yes/No	Yes/No
Limb-Kinetic	Yes/No	Yes/No
Oral- Buccal- Lingual	Yes/No	Yes/No
Agnosia		
Visual	Yes/No	Yes/No
Tactile	Yes/No	Yes/No
Auditory	Yes/No	Yes/No

D.**LANGUGE:**

2 Objects correctly named	Yes/No	Yes/No
Obeying 3 stage commands	Yes/No	Yes/No
Response to written commands	Yes/No	Yes/No
Able to write a sentence	Yes/No	Yes/No
Able to repeat	Yes/No	Yes/No
Able to draw a complex diagram	Yes/No	Yes/No

E. **SPEECH**:

Aphasia	Yes/No	Yes/No
Dysphasia		
Broca's non-fluent	Yes/No	Yes/No
Wernick's fluent	Yes/No	Yes/No
Conduction dysphasia	Yes/No	Yes/No
Echolalia	Yes/No	Yes/No
Pure Word Blindness	Yes/No	Yes/No
Pure Word dumbness	Yes/No	Yes/No
Pure Word deafness	Yes/No	Yes/No

	on admission	on discharge
Dysarthria		
Rigid dysarthria (Extra pyramidal lesion)	Yes/No	Yes/No
Spastic dysarthria (UMN Lesion)	Yes/No	Yes/No
Ataxic Dysarthria (Cerebellar lesion)	Yes/No	Yes/No
Lesion in the muscle Producing speech (LMN Lesion)	Yes/No	Yes/No
Aphonia:		
Central	Yes/No	Yes/No
Disorders of larynx & Vocal cords	Yes/No	Yes/No
Mutism	Yes/No	Yes/No

F.CLINICAL EXAMINATION OF THE CRANIAL NERVES:

a. OLFACTORY NERVE

Normal	Yes/No	Yes/No
Anosmia	Yes/No	Yes/No
Parosmia	Yes/No	Yes/No
Hyperosima	Yes/No	Yes/No
Cacosmia	Yes/No	Yes/No
Olfactory hallucinations	Yes/No	Yes/No

b. OPTIC NERVE

Acuity of vision:		
Near vision	Yes/No	Yes/No
Distant Vision	Yes/No	Yes/No
Field of vision:		
Normal	Yes/No	Yes/No
Hemianopia	Yes/No	Yes/No
Homonymous		

	on admission	on discharge
Hemianopia	Yes/No	Yes/No
Heteronymous Hemianopia		
Binasal	Yes/No	Yes/No
Bitemporal	Yes/No	Yes/No
Colour Vision:		
Red	Yes/No	Yes/No
Green	Yes/No	Yes/No
Blue	Yes/No	Yes/No

c. OCULOMOTOR NERVE

d. TROCHLEAR NERVE

c. ABDUCENT NERVE

Ptosis	Yes/No	Yes/No
Pupil:		
Normal	Yes/No	Yes/No
Miosis	Yes/No	Yes/No
Mydriasis	Yes/No	Yes/No
Pupillary reflex	Yes/No	Yes/No
Light reflex	Yes/No	Yes/No
Accommodation reflex	Yes/No	Yes/No
Strabismus	Yes/No	Yes/No
Nystagmus	Yes/No	Yes/No
Action of Ocular muscles		
Superior rectus	Yes/No	Yes/No
Inferior rectus	Yes/No	Yes/No
Medial rectus	Yes/No	Yes/No
Inferior oblique	Yes/No	Yes/No
Superior oblique	Yes/No	Yes/No
Lateral rectus	Yes/No	Yes/No

F.TRIGEMINAL NERVE

	on admission	on discharge
Motor**:**		
Masseter	Yes/No	Yes/No
Temporalis	Yes/No	Yes/No
Sensory:		
Pain, touch sensations felt on the skin over the face	Yes/No	Yes/No
Reflex		
Corneal reflex	Yes/No	Yes/No
Jaw reflex	Yes/No	Yes/No

g. **FACIAL NERVE**

Motor

Muscles acting:		
Frontails	Yes/No	Yes/No
Corrugator supercilli	Yes/No	Yes/No
Nasalis	Yes/No	Yes/No
Orbicularis oculi	Yes/No	Yes/No
Lavator Labii superiors	Yes/No	Yes/No
Lavator Labii interiors	Yes/No	Yes/No
Zygomaticus	Yes/No	Yes/No
platysma	Yes/No	Yes/No
Buccinator	Yes/No	Yes/No
Risorius	Yes/No	Yes/No
Sensory:		
Taste sensation in the anterior 2/3 of the tongue	Yes/No	Yes/No
Sweet	Yes/No	Yes/No
Salt	Yes/No	Yes/No
Secretory:		
Lacrimation	Yes/No	Yes/No
Noso lacrimal reflex	Yes/No	Yes/No
Salivation	Yes/No	Yes/No

	on admission	on discharge
Reflex :		
Corneal reflex	Yes/No	Yes/No
Bell's Palsy:	Yes/No	Yes/No

h.VESTIBULO COCHLEAR NERVE

Auditory function

Human voice test	Yes/No	Yes/No
Watch test	Yes/No	Yes/No
Tuning fork tests:		
Rinne's test		
AC>BC	Yes/No	Yes/No
BC>AC	Yes/No	Yes/No
Weber's test		
No lateralization	Yes/No	Yes/No
Lateralized to the normal ear	Yes/No	Yes/No
Lateralized to the affected air	Yes/No	Yes/No
Absolute Bone Conduction test		
Equal to the patient and the examiner	Yes/No	Yes/No
Increased to the patient	Yes/No	Yes/No
Decreased to the patient	Yes/No	Yes/No

Vestibular function

Positional vertigo	Yes/No	Yes/No
Oculocephalic reflex	Yes/No	Yes/No

h GLOSSOPHARYNGEL NERVE AND VAGUS NERVE

	on admission	on discharge
Taste sensation in the posterior 1/3 of the tongue	Yes/No	Yes/No
Palatal reflex:		
Elevation of the soft palate both sides	Yes/No	Yes/No
No movement of uvula	Yes/No	Yes/No
Uvula pulled to the healthy side	Yes/No	Yes/No
Gag reflex:		
Reflex contraction of pharynx	Yes/No	Yes/No
Gag reflex absent	Yes/No	Yes/No

j. SPINAL ACCESSORY NERVE:

Sternomastoid:		
Rotation of the head to the opposition side	Yes/No	Yes/No
Trapezius:		
Shrugging of the shoulder	Yes/No	Yes/No

k. HYPOGLOSSAL NERVE:

Strength of the tongue normal	Yes/No	Yes/No
Tongue wasting	Yes/No	Yes/No
Abnormal movement	Yes/No	Yes/No
Deviation of the tongue	Yes/No	Yes/No
Fasciculation	Yes/No	Yes/No
Jaw jerk		
Normal	Yes/No	Yes/No
Exaggerated	Yes/No	Yes/No

G.CLINICAL EXAMINATION OF THE MOTOR SYSTEM:

	Right		Left	
	On Adm	On Dis	On Adm	On Dis
a. Nutrition				
Upper Limp:				
Normal	Yes/No	Yes/No	Yes/No	Yes/No
Wasting	Yes/No	Yes/No	Yes/No	Yes/No
Lower Limb:				
Normal	Yes/No	Yes/No	Yes/No	Yes/No
Wasting	Yes/No	Yes/No	Yes/No	Yes/No

b. Tone: (Modified Ashworth scale of spasticity)

UPPER LIMB:

Shoulder

- Flexor -
- Extensor -
- Abductor -
- Adductor -
- Int. rotator -
- Ext. rotator -

Elbow:

- Flexor –
- Extensor –

Forearm:

- Supinator –
- Pronator –

Fingers:

- Flexor –
- Extensors –

Wrist:

- Dorsiflexors –
- Palmar flexors –

LOWER LIMB:

Tone:

(Modified Ashworth scale of spasticity)

Hip:	Right		Left	
	On Adm	On Dis	On Adm	On Dis
Flexor	-			
Extensor	-			
Abductor	-			
Adductor	-			
Int. rotator	-			
Ext. rotator	-			

Knee:

Flexor	-			
Extensor	-			

Ankle:

Dorsiflexors	-			
Palmar flexors	-			
Invertor	-			
Everter	-			

C.POWER (MRC grading)

UPPER LIMB:

Shoulder

Flexor	-			
Extensor	-			
Abductor	-			
Adductor	-			
Int. rotator	-			
Ext. rotator	-			

Elbow:

Flexor	-			
Extensor	-			

Forearm:

Supinator	-			
Pronator	-			

	Right		Left	
	On Adm	On Dis	On Adm	On Dis
Wrist:				
Dorsiflexors -				
Palmar flexors -				
Fingers:				
Flexors -				
Extensors -				

VOLITIONAL MOVEMENT:
UPPER LIMB:

Shoulder

- Flexion -
- Extension -
- Abduction -
- Adduction -
- Int. rotation -
- Ext. rotation -

Elbow:

- Flexion -
- Extension -

Forearm:

- Supination -
- Pronation -

Wrist:

- Dorsiflexion -
- Palmar flexion -

Fingers:

- Flexion -
- Extension -

LOWER LIMB:
POWER: (MRC grading)

Hip:

- Flexor -
- Extensor -

Abductor -
Adductor -
Int. rotator -
Ext. rotator -

Knee:

Flexor -
Extensor -

Ankle:

Dorsiflexors -
Palmar flexors -
Invertor -
Everter -

VOLITIONAL MOVEMENT:

Hip:

Flexion -
Extension -
Abduction -
Adduction -
Int. rotation -
Ext. rotation -

Knee:

Flexion -
Extension -

Ankle:

Dorsiflexion -
Palmar flexion -
Inversion -
Eversion -

M. CLINICAL EXAMINATION OF THE SENSORY SYSTEM:

a. Extero captive Sensation

	Right		Left	
	On Adm	On Dis	On Adm	On Dis
Pain	Yes/No	Yes/No	Yes/No	Yes/No
Light Touch	Yes/No	Yes/No	Yes/No	Yes/No
Tempurature	Yes/No	Yes/No	Yes/No	Yes/No

b. Proprioceptive Sensation

Position Sense	Yes/No	Yes/No	Yes/No	Yes/No
Vibration Sense	Yes/No	Yes/No	Yes/No	Yes/No

c. Cortical Sensation

Tactile localization	Yes/No	Yes/No	Yes/No	Yes/No
Two points discrimination	Yes/No	Yes/No	Yes/No	Yes/No
Stetrognosis	Yes/No	Yes/No	Yes/No	Yes/No
Graphesthesia	Yes/No	Yes/No	Yes/No	Yes/No

i. **THE REFLEXES (Grading)**

SUPERFICIAL

- Corneal
- Conjunctival
- Palatal
- Pharyngeal
- Upper
- Lower
- Cremasteric
- Plantar

DEEP

- Biceps jerk
- Triceps jerk
- Supinator jerk
- Finger flexion reflex

Hoffman reflex
Wartenberg's sign
Rossolimo's reflex
Knee jerk
Ankle jerk

J. CONDITION OF THE UPPER LIMB:

	On Admission	On Discharge
a. SYNERGY PATTERN		
Flexor synergy	Yes/No	Yes/No
Extensor synergy	Yes/No	Yes/No
b. VOLITIONAL MOVEMENT:		
Shoulder	Yes/No	Yes/No
Elbow	Yes/No	Yes/No
Wrist	Yes/No	Yes/No
Finger	Yes/No	Yes/No
c. HAND FUNCTIONS:		
Non- Prehensile:		
Touch	Yes/No	Yes/No
Tap	Yes/No	Yes/No
Press	Yes/No	Yes/No
Lift	Yes/No	Yes/No
Push	Yes/No	Yes/No
Prehensile activities:		
Precision:		
Three –jaw chuck	Yes/No	Yes/No
Key grip	Yes/No	Yes/No
Power grips:		
Hook grip	Yes/No	Yes/No
Spherical grip	Yes/No	Yes/No

K. CONDITION OF THE LLOWER LIMB:

a. SYNERGY PATTERN:

	On Admission	**On Discharge**
Flexor synergy	Yes/No	Yes/No
Extensor synergy	Yes/No	Yes/No

b.VOLITIONAL MOVEMENT:

Hip	Yes/No	Yes/No
Knee	Yes/No	Yes/No
Ankle	Yes/No	Yes/No

c. GAIT

Normal	Yes/No	Yes/No
Dragging the foot	Yes/No	Yes/No
Circumduction gait	Yes/No	Yes/No
Hip flexion	Yes/No	Yes/No
Knee flexion	Yes/No	Yes/No
Ankle dorsiflexion	Yes/No	Yes/No

13. BLADDER:

Incontinent	Yes/No	Yes/No
Continent	Yes/No	Yes/No

14. BOWEL**:**

Incontinent	Yes/No	Yes/No
Continent	Yes/No	Yes/No

15. SCANDINVIAN STROKE SCALE:

	On Admission	On Discharge	On follow-up
1. Consciousness	-		
2. Orientation	-		
3. Eye movement	-		
4. Facial palsy	-		
5. Speech	-		
6. Arm motor power	-		
7. Hand motor power	-		
8. Leg motor power	-		
9. Gait	-		
	---	---	---
Total –			
	---	---	---

Scandinavian Stroke Scale Score:

Score		Stroke grade
0-14	-	Very Severe
15-29	-	Severe
30-44	-	Moderate
45-58	-	Mild

16**. FUNCTIONAL INDEPENDENCE MEASURE**

	On Admission	On Discharge	On follow-up
a. Self - Care			
Eating	-		
Grooming	-		
Bathing	-		
Dressing upper body	-		
Dressing lower body	-		
Toileting	-		
b. Sphincter Control:			
Bladder management	-		
Bowel management	-		

c. Transfers

Bed, Chair, Wheel Chair -
Toilet -
Bath room -

d. Locomotion

Walk -
Stairs -

e. Communication

Comprehension -
Expression -

f. Social Cognition

Social interaction -
Problem solving -
Memory -

Total FIM Score -

---

17. **PREDICTORS OF FUNCTIONAL OUTCOME AFTERSTROKE**:

1. Age	Less than 65	:	Yes/No
2. Educational level	Educated	:	Yes/No
3. Prior stroke		:	Yes/No
4. Coma on set		:	Yes/No
5. Cognitive function	Poor	:	Yes/No
	Good	:	Yes/No
6. Hemianopia		:	Yes/No
7. Language Function	Poor	:	Yes/No
	Good	:	Yes/No
8. Sensory function	Intact	:	Yes/No
9. Bowel incontinence		:	Yes/No
10. Bladder incontinence		:	Yes/No
11. Stroke onset- Rehabilitation admission			
	Short interval	:	Yes/No
	Prolonged interval	:	Yes/No
12. Congestive cardiac failure		:	Yes/No
13. Medical co- morbidities		:	Yes/No
14. Motivated		:	Yes/No

15. Family involment and support		:	Yes/No
16. Deep tendon reflexes return within 48 hours		:	Yes/No
17. Flaccid period	Short	:	Yes/No
	Prolonged	:	Yes/No
18. Onset of motion following stroke			
	Early	:	Yes/No
	Late	:	Yes/No
19. Proprioception and tactile sense			
	Intact	:	Yes/No
	Lost	:	Yes/No
20. Posture and balance			
	Equilibrium	:	Yes/No
	Disequilibrium	:	Yes/No

18. RISK FACTORS FOR STROKE IDENTIFIED:

a.	Risk factors modifiable by life style changes	Cigarette smoking	:	Yes/No
		Hyperlipidemia	:	Yes/No
		Obesity	:	Yes/No
		Heart disease	:	Yes/No
b.	Risk factors modifiable by medical means			
		Hypertension	:	Yes/No
c.	Non- modifiable risk factors	Age	:	Yes/No
		Sex	:	Yes/No
		Race	:	Yes/No
		Previous Stroke	:	Yes/No

19. INVESTGATIONS DONE:

a. BLOOD**:**

Total count	**:**
Differential Count	**:**
ESR	**:**
Hemoglobin	**:**
Bleeding time	**:**
Clotting time	**:**
Viscosity	**:**
PCV	**:**

Urea :
Sugar :
Fasting :
Postprandial :

b. <u>SERUM:</u>

Creatinine :
Uric acid :
Triglycerides :
Cholesterol :
Lipid profile :
LDL :
VLDL :
HDL :

c. URINE
Routine :

d. ECG :
e. ECHO :
f. CAROTID DOPPLER :
g. CT BRAIN :

20. <u>FINAL DIAGNOSIS</u>

A case of _____ sided hemi paresis / hemiplegia due to the involvement of _____ sided cerebral hemisphere _____ days duration.

_____ Stroke (Pathology)

_____ Territory involvement

Level of Lesion_____

21. MANAGEMENT

(A) DRUG THERAPY:

a. Calcium channel Blocking agent	:	Nimodipine	Yes/No
b. Antiplatelet agent	:	Aspirin	Yes/No
		Ticlopidine	Yes/No
c. Haemrrheological agent	:	Pentoxyphylline	Yes/No
d. Antispastic drugs	:	Carisoprodol	Yes/No
		Diazepam	Yes/No
		Dantrolene sodium	Yes/No
		Tizanidine	Yes/No
e. Antihypertensive drugs	:		
f. Antidiabetic drugs	:		
g. Hypolipidemic drugs	:		

(B) PHYSIOTHERPY:

I.General Physiotherapy Exercises

a. Breathing exercise
b. Roll on the bed
c. Pelvic bridging training
d. Truncal balance training

II. Motor re-education program for the Lower Limb

i) If the paralyzed lower limb is flaccid:

a) Olive oil massage (EFFLEURAGE) to the _____ lower limb.
b) PROM Exercise to the all joints in the _____ lower limb
c) PROM Exercise to the_____ lower limb in the movement pattern.
d) Electrical Stimulation:

Specifications:

Pulse width	:	100-300m.sec
Intensity	:	100V
Frequency	:	30-50 Cycles/Sec (Hz)
Duration	:	10minutes for each muscle

Muscles stimulated

Gluteus maximum
Quadriceps
Tibialis anterior

ii) If the paralyzed lower limb is spastic:

a. Antispastic positioning of the spastic_____ lower limb.
b. Olive oil massage (EFFLEURAGE)
c. Relaxation exercise to the _____ lower limb.
d. Electrical Stimulation (stimulation of antagonists)

Specifications:

Pulse width	:	100-300m.sec
Intensity	:	100v
Frequency	:	30-50 Cycles/Sec (Hz)
Duration	:	10minutes for each muscle

iii) AFTER THE RECOVERY OF VOLOTIONAL MOVEMENTS in the affected limb:

a. to continue Olive oil massage (EFFLEURAGE) to the _____ lower limb.
b. Electrical stimulation, as specified to

Gluteus maximus
Quadriceps
Tibialis Anterior

c. To REEDUCATE the movement pattern in the _____lower limb.

In Supine position, lift the affected leg and taking the knee to the chest with ankle dorsiflexion and vice versa.
In Supine position facilitate ankle

Dorsiflexion
Plantar flexion
Inversion
Eversion

Ankle' Dorsiflexion inhibition' present Yes/No
"Strumpell Phenomenon" facilitated Yes/No

Complex Movement pattern:

Hip abduction with knee extension
Hip abduction with knee flexion
Hip abduction with knee extension
Hip abduction with knee flexion
Internal rotation of the hip
External rotation of the hip.

d. Truncal Balance Training:

Sit up training from side lying posture
Stand up training from sitting posture
To sit with midline orientation
To sit with weight shifted forward
To sit with weight shifted backward
To sit weight shifted sideways

METHODLOGY OF AMBULATION

e. WEIGHT BEARING TRAINING:

Standing from sitting position
Kneel standing
Squatting
Stepping on and off with the feet alternatively
Stepping over objects of small heights

f. Standing training

To stand with wide base
To stand with narrow base
To weight bear on the normal leg
To weight bear on the affected leg

g. Gait training:

-with mobility aids
-without mobility aids
To walk on heels
To walk on toes
Rapid walking
Running
Stair climbing
Walking training with carrying objects

h. STRENGTHENING PROGRAMME to lower limb, once the patients gets volitional control of the lower limb.

III. MOTOR REEDUCATION PROGRAMME FOR THE AFFECTED UPPER LIMB

(The patients is instructed to use the affected limb as much as possible)

a. If the affected upper limb is flaccid:

a) Olive oil massage (Effleurage) to the _____upper limb.
b) Rom Exercise to the all joints in the _____upper limb
c) PROM Exercise to the_____ upper limb in the
d) movement pattern.
e) Sling applied to the flaccid arm to prevent shoulder
f) subluxation.
g) Interlock the fingers and raise the arm overhead
h) Weight bearing in the outstretched forearm and wrist
i) Auto assisted arm movements.
j) Electrical stimulation:

Specifications:

Pulse width	: 100-300m.sec
Intensity	: 100v
Frequency	: 30-50 Cycles/Sec (Hz)
Duration	: 10minutes for each muscle
Muscles stimulated	:

Deltoid
Supraspinatus
Biceps, Triceps
Common flexors of forearm
Common extensors of forearm
Intrinsic muscles of hand

b. If the affected upper is spastic:

a) Anti spastic positioning of the spastic------- upper limb.
b) Olive oil massage (EFFLEURAGE) to the _____ upper limb.
c) Relaxation exercises to the _____ UPPER LIMB.
d) “Finger Spreader” to the abduct and extend the fingers
e) Electrical stimulation (stimulation of antagonists)

Specifications:

Pulse width : 100-300 m.sec
Intensity : 100v
Frquency : 30-50 Cycles/Sec (Hz)
Duration : 10minutes for each muscle

C. AFTER THE RECOVERY OF VOLITIONAL MOVEMENTS IN THE AFFECTED UPPER LIMB:

a) To continue Olive oil massage (EFFLEURAGE) to the ____ upper limb.
b) Electrical stimulation
as specified to
Deltoid
Supraspinatus
Biceps
Triceps
Common flexors of forearm
Common extensors of forearm
Intrinsic muscles of hand
c) Universal extension of the upper limb to facilitate dorsiflexion of the wrist.
d) Passive extension of the thumb to promote wrist extension.
e) To reeducate movement pattern in the upper limb

Arm supported on a high table and teaches
Shoulder flexion
Shoulder abduction
Scapular movements
Scapular protraction in side lying
Scapular protraction in sitting
Supination and pronation of the forearm with elbow supported.

In lying posture, palm of the hand on the forehead, stabilize the elbow while extending it to reach the hand upward and rectum palm to the forehead.

To take the hand to the chin
To take the hand to the opposite knee in sitting posture
To take the hand to the same side shoulder
To take the hand to the opposite side shoulder
To take the hand to the back
To lift the hand to the back

To lift the arm overhead
Wrist flexion and extension
Finger flexion and extension
Finger abduction with wrist extension

Complex Movement pattern taught:

Horizontal abduction of the shoulder and external rotation
Elbow extension and supination

Automatic gesturing encouraged with affected hand

f. Strengthening Program to the upper limb once the patients gets good volitional control of the upper limb.

IV) TO IMPROVE HAND FUNCTIONS:

To practice

Finger tip pinch
Finger side pinch
Spherical grasp
Cylindrical grasp
Hook grip

Holding an object in the affected hand
To pick up, hold and release objects of varying sizes, shapes and weight.

V) ADL TRAINING:

Toileting
Brushing
Bathing
Dressing
Upper part of the body
Lower part of the body
Grooming
Eating