CHAPTER 2

REVIEW LITERATURE

Pat 1 Definition of stroke

The World Health Organization (WHO) has defined stroke as s condition with ‘ rapidly developing clinical signs of focal loss of cerebral function, with symptoms lasting more than 24 hours or leading to death, with no apparent cause other than that of vascular origin.’ (World Health Organization, 1989)

Stroke is an acute onset of neurological dysfunction due to an abnormality in cerebral circulation with resultant signs and symptoms that correspond to involvement of focal areas of the brain (World Health Organization, 1989). The term cerebrovascular accident (CVA) is used interchangeably with stroke to refer to cerebrovascular conditions that accompany either ischemic or hemorrhagic lesions. Clinically, a variety of deficits must persist for at least 24 hours. Motor deficits are characterized by paralysis (hemiplegia) or weakness (hemiparesis), typically on the side of the body opposite the site of the lesion.

Part 2 Epidemiology

Epidemiology is “the study of distribution and determinants of disease frequency” in human population (MacMahon & Pugh, 1970). Stroke is the most common life-threatening neuralgic disease and is the third leading cause of death in the United States, after heart disease and cancer. Although stroke is more often disabling than lethal, 150,300 deaths were attributed to stroke in 1988 (American Heart Association, 1991). In the elderly, the segment of the population where most strokes occurs, it was also a major source of disability leading to institutionalization. Stroke is major health problem in developed countries. It is presently among the top four leading cause of death in ASEAN countries, with the crude death rate varying from 10.9/100,000 (Thailand) to 54.2/100,000 (Singapore). There is little data on

stroke incidence or prevalence: incidence is 161/100,000 in Vietnam; prevalence ranges from 415/100,000 (Vietnam) to 690/100,000 (Thailand) (Venketasubramanian, 1998). In Thailand, Public Health Statistics show that stroke has been on the increase (Granger & Hamilton, 1993). A great majority of stroke patients in rehabilitation improve in function (Viriyavejakul, 1990), but the improvement is quite variable from one patient to the other (Johnston, Kirshblum, Zorawitz, & Walker, 1993). Approximately, 80% of stroke patients survive the acute phase. Although some patients regain their walking ability, 30% to 66% of the survivors are no longer able to use the affected arm (Kwakkel, Kollen, & Wagenaar, 1999). The consequences after stroke are not only persistent neurological impairment, but also lifetime disability that needs rehabilitation to enable optimal function that will overcome the patient's disability.

Part 3 Etiological categories

Atherosclerosis is a major contributory factor in stroke. It is characterized by plaque formation with an accumulation of lipids, fibrin, complex carbohydrates, and calcium deposits on arterial walls that leads to progressive narrowing of blood vessels. Interruption of blood flow by atherosclerotic plaques occurs at certain sites of predilection. These generally include bifurcations, constrictions, dilation, or angulations of arteries. The most common sites for lesions to occur are at the origin of the common carotid artery or at its transition into the middle cerebral artery, at the main bifurcation of the middle cerebral artery, and at the junction of the vertebral arteries with the basilar artery.

Two main mechanisms result in stroke. Strokes can be ischemic, the result of thrombus, embolism, or conditions that produce low systemic perfusion pressures. The resulting lack of cerebral blood flow (CBF) deprives the brain of needed oxygen and glucose, disrupts cellular metabolism, and leads to injury and death of tissues. A thrombus results from platelet adhesion and aggregation on plaques. Cerebral thrombosis refers to the formation or development of a blood clot or thrombus with in the cerebral arteries or their branches. It should be noted that lesions of extra cranial

vessels (carotid or vertebral arteries) could also produce symptoms of stroke. Thrombi lead to ischemia, or occlusion of an artery with resulting infraction or tissue death (atherothrombotic brain infraction (ABI)). Thrombi can also become dislodged and travel to a more distal site in the form of an intraartery embolus. Cerebral embolus (CE) are traveling bits of matter formed elsewhere that are released into the bloodstream and travel to the cerebral arteries where they lodge in a vessel, producing occlusion and infarction. The most common etiology results from cardiovascular disease (valvular disease, myocardial infarction, arrhythmias, congenital heart disease). Occasionally systemic disorders may produce septic, fat, or air emboli that affect the cerebral circulation. Ischemic strokes may also results from low systemic perfusion, the result of cardiac failure or significant blood loss with resulting systemic hypotension. The neurological deficits produced with systemic failure are global in nature with bilateral neurological deficits.

Strokes can also be hemorrhagic, with abnormal bleeding into extravascular areas of the brain secondary to aneurysm or trauma. Hemorrhage results in increased intracranial pressures with injury to brain tissues and restriction of distal blood flow. Intracerebral hemorrhage (IH) is caused by rupture of cerebral vessel with subsequent bleeding into the brain. Primary cerebral hemorrhage (nontraumatic spontaneous hemorrhage) typically occurs in small blood vessels weakened by atherosclerosis producing an aneurysm. Subarachnoid hemorrhage (SH) occurs from bleeding into the subarachnoid space typically from a saccular or berry aneurysm affecting primarily large blood vessels. Developmental defects that produce weakness in the blood vessel wall are major contributing factors to the formation of an aneurysm. Hemorrhage is closely linked to chronic hypertension. Arteriovenous malformation (AVM) is another congenital defect that can result in stroke. AVM is characterized by a tortuous tangle of arteries and veins with agenesis of an interposing capillary system. The abnormal vessels undergo progressive dilatation with age and eventually bleed in about 50 percent of cases. The resulting hemorrhage can be either subarchnoid or intracerebral. Sudden and severe cerebral bleeding can result in death within hours, because intracranial pressures rise rapidly and adjacent cortical tissues are displaced or compressed (Saladin, 1996). Ischemic stroke is the most common

type of stroke, accounting for 61 to 81 percent of all strokes. Hemorrhagic strokes account for 12 to 24 percent of strokes (Post-Stroke Rehabilitation Clinical Practice Guideline, 1996).

Part 4 Risk factors

Cardiovascular diseases affecting the brain and heart share a number of common risk factors important to the development of atherosclerosis. Major risk factors for stroke are hypertension, heart disease, and diabetes. In patients with ABI, 70 percent have hypertension, 30 percent coronary heart disease, 15 percent congestive heart disease, 30 percent peripheral arterial disease, and 15 percent diabetes. This coexistence of vascular problems increases significantly with the age of the patient. The risk for stroke is especially strong in patients with systolic and diastolic blood pressures elevated above 160/95 mmHg. Patients with marked elevations of hematocrits are also at an increased risk of occlusive stroke owing to a generalized reduction of CBF. Cardiac disorders such as rheumatic heart valvular disease, endocarditis, arrhythmias (particularly artrial fibrillation), or cardiac surgery significantly increase the risk of embolic stroke. Transient ischemic attacks (TIAs) are another important risk factor for stroke. Although only 10 percent of strokes are preceded by TIAs, about 36 percent of individuals who experience one or more TIAs will go on to develop a major stroke within 5 years (American Heart Association., 1996; Post-Stroke Rehabilitation Clinical Practice Guideline, 1996).

Part 5 Pathophysiology

Interruption of blood flow for only a few minutes sets in motion a series of pathoneurological events. Complete cerebral circulatory arrest results in irreversible cellular damage with a core area of focal infarction within minutes. The area surrounding the core is termed the ischemic prenumbra and consists of viable but metabolically lethargic cells. The ischemia triggers a number of damaging and potentially reversible events including the release of cascades of chemicals. The release of excess glutamate, an excitatory neurotransmitter, causes changes in calcium

ion distribution with additional calcium influx and overload in intracellular calcium. This in turn results in the sustained activation of destructive calcium sensitive enzymes producing additional cell death, generally within hours. There is an extension of the infarction into the prenumbra area. Research efforts are currently directed toward development of drugs that might reverse the metabolic changes of the ischemic brain following treatment with glutamate receptor antagonists (Guberman, 1994).

Ischemic brain edema, an accumulation of fluids, begins within minutes of the insult and reaches a maximum by 3 to 4 days. It is the result of tissue necrosis and widespread rupture of cell membranes with movement of water from the blood into brain tissues. The swelling then gradually subsides, generally disappearing by 3 weeks. Significant edema can elevate intracranial pressures, leading to secondary brain damage and neurological deterioration from contralateral and caudal shifts of brain structures (brainstem herniation). Clinical signs of elevating intracranial pressure (ICP) include decreasing level of consciousness (stupor and coma), widened pulse pressure, increased heart rate, irregular respirations (Cheyne-Stokes respirations), vomiting, unreacting pupils (cranial nerve III signs), and papilledema. Cerebral edema is the most frequent cause of death in acute stroke and is characteristic of large infarcts involving the middle cerebral artery (Hachinski & Norris, 1985).

Part 6 Anatomical categories

CBF varies with the patency of the vessels. Progressive narrowing secondary to atherosclerosis decreases blood flow. As in coronary heart disease, symptomatic changes generally result from a restriction of flow greater than 80 percent. The symptomatology of stroke id dependent on a number of factors, including

1. The location of the ischemic process
2. The size of the ischemic area
3. The nature and functions of the structures involved

4. The availability of collateral blood flow

Symptomatology may also depend on the rapidity of the occlusion of a blood vessel because slow occlusions may allow collateral vessels to take over, whereas sudden events do not.

6.1 Cerebral blood flow

CBF is controlled by a number of auto regulatory mechanisms (cerebral) that modulate a constant rate of blood flow through the brain. These mechanisms provide homeostatic balance, counteracting fluctuations in systolic blood pressure while maintaining a normal flow of 50 to 60 milliliters per 100 grams of brain tissue per minute. The brain has high-energy requirements and very little metabolic reserves. It requires a continuous, rich perfusion of blood to deliver oxygen and glucose to the tissues. Cerebral flow represents approximately 17 percent of available cardiac output. Chemical regulation of CBF occurs in response to changes in blood concentrations of carbon dioxide or oxygen. Vasodilation and increased CBF are produced in response to an increase in $Paco_2$ or a decrease in Pao_2, while vasoconstriction and decreased CBF are produced by the opposite stimuli. Blood flow is also altered by changes in the blood pH. A fall in pH (increased acidity) produces vasodilation, and a rise in pH (increased alkalinity) produces a decrease in blood flow. Neurogenic regulation alters blood flow by vasodilating vessels in direct proportion to local function of brain tissue. Released metabolites probably act directly on the smooth muscle in local vessel walls. Changes in blood viscosity or intracranial pressures may also influence CBF. Changes in blood pressure produce minor alterations of CBF. As pressure rises, the artery is stretched, resulting in contraction of smooth muscle in the vessel wall. Thus, the patency of the vessel is decreased, with a consequent decrease in CBF. As pressure falls, contraction lessens and CBF increases. Following stroke, auto regulatory mechanisms may be impaired (Curtis & Porth, 1998; Guberman, 1994).

Knowledge of cerebral vascular anatomy is essential to understand the symptomatology, diagnosis, and management of stroke. Extracranial blood supply to the brain is provided by right and left internal carotid arteries and by the right and left vertebral arteries. The internal carotid artery begins at the bifurcation of the common carotid artery and ascends in the deep portions of the neck to the carotid canal. It turns rostromedially and ascends into the cranial cavity. It then pierces the dura mater and gives off the ophthalmic and anterior choroidal arteries before bifurcating into the middle and anterior cerebral arteries. The anterior communicating artery communicates with the anterior cerebral arteries of either side, giving rise to the rostral portion of the circle of willis (Figure 1). The vertebral artery arises as a branch off the subclavian artery. It enters the vertebral foramen of the sixth cervical vertebra and travels through the foramina of the transverse processes of the upper six cervical vertebrae to the foramen magnum and into the brain. There it travels in the posterior cranial fossa ventrally and medially and unites with the vertebral artery from the other side to form the basilar artery at the upper border of the medulla. At the upper border of the pons, the basilar artery bifurcates to form the posterior cerebral arteries and the posterior portion of the circle of Willis. Posterior communicating arteries connect the posterior cerebral arteries with the internal carotid arteries and complete the circle of Willis.

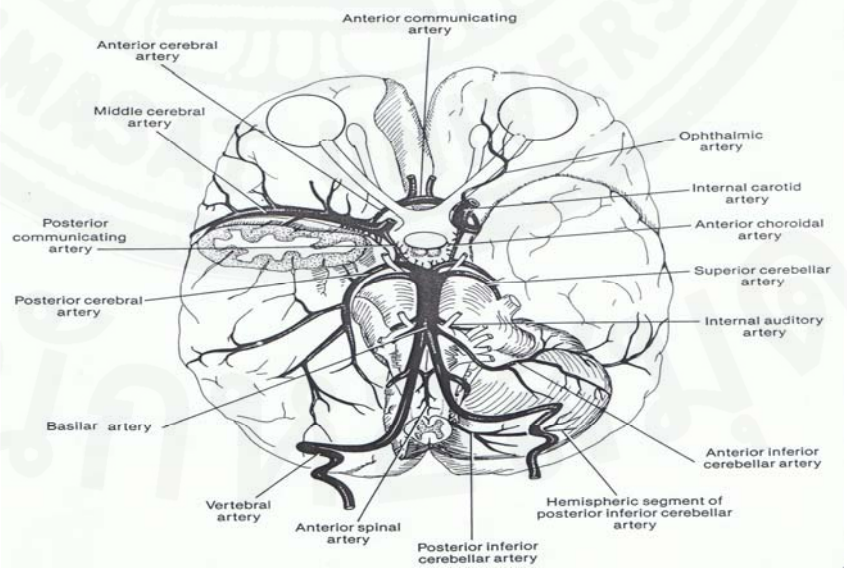

Figure 1 Cerebral circulation: circle of willis.

6.2 Vascular syndromes

6.2.1Anterior cerebral artery syndrome

The anterior cerebral artery (ACA) is the first and smaller of two terminal branches of the internal carotid artery. It supplies the medial aspect of the cerebral hemisphere (frontal and parietal lobes) and subcortical structures, including the basal ganglia (anterior internal capsule, inferior caudate nucleus), anterior fornix, and anterior four fifths of the corpus callosum (Figure 2). Because the anterior communicating artery allows perfusion of the proximal anterior cerebral artery from either side, occlusion proximal to this point results in minimal deficit. More distal lesions produce more significant deficits. The most common characteristic of ACA syndrome is contralateral hemiparesis and sensory loss with greater involvement of the lower extremity because the somatotopic organization of the medial aspect of the cortex includes the functional area for the lower extremity.

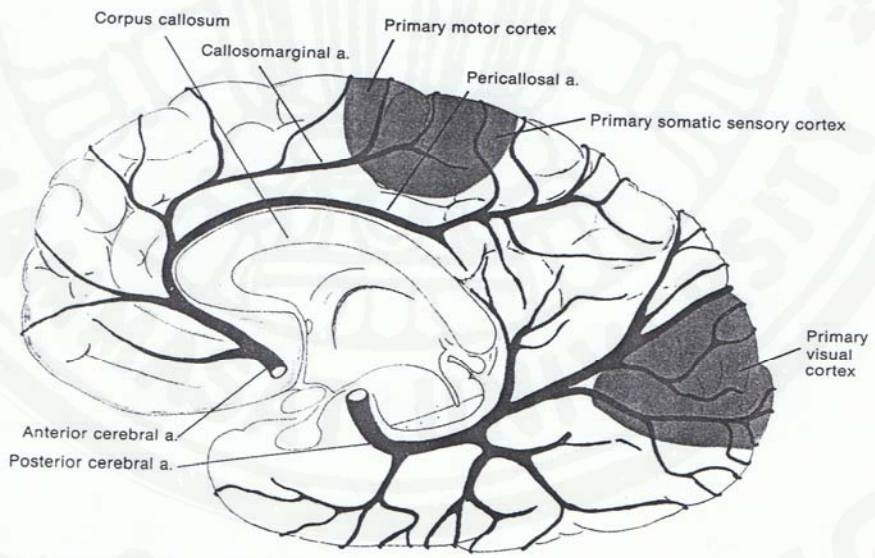

Figure 2 Cerebral circulation: A diagram of a midsagittal view of the brain illustrates the distribution of the anterior and posterior cerebral arteries.

6.2.2 Middle cerebral artery syndrome

The middle cerebral artery (MCA) is the second of two main branches of the internal carotid artery and supplies the entire lateral aspect of the cerebral hemisphere (frontal, temporal, and parietal lobes) and subcortical structures, including the internal capsule (posterior portion), corona radiata, globus pallidus (outer part), most of the caudate nucleus, and the putamen (Figure 3). Occlusion of the proximal MCA produces extensive neurological damage with significant cerebral edema. Increased intracranial pressures typically lead to loss of consciousness, brain herniation, and possibly death. Middle cerebral artery stroke describes the sudden onset of focal neurologic deficit resulting from brain infarction or ischemia in the territory supplied by the middle cerebral artery (MCA).

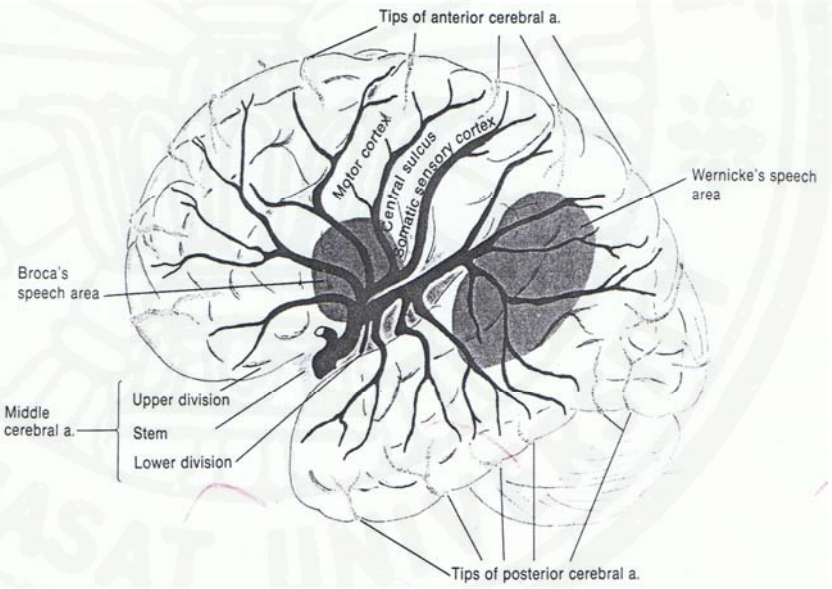

Figure 3 Cerebral circulation: A diagram of a lateral view of the brain illustrates the distribution of the middle cerebral artery.

6.2.3 Internal carotid artery syndrome

Complete occlusion of the internal carotid artery produces massive infarction in both middle cerebral and anterior cerebral arterial territories. Extensive cerebral edema occurs and frequently leads to coma and death. Incomplete occlusions can produce a mixture of middle cerebral and/or anterior artery symptoms.

6.2.4 Posterior cerebral artery syndrome

The two posterior cerebral arteries (PCAs) arise as terminal branches of the basilar artery and each supplies the corresponding occipital lobe and medial and inferior temporal lobe (see figure 3). It also supplies the upper brainstem, midbrain, and posterior diencephalons, including most of the thalamus. Occlusion proximal to the posterior communicating artery typically results in minimal deficits owing to the collateral blood supply from the posterior communicating artery (similar to ACA syndrome). Occlusion of thalamic branches may produce hemianesthesia (contralateral sensory loss) or thalamic sensory syndrome (thalamic pain) (a persistent and unpleasant hemibody sensation). Occipital infarction produces homonymous hemianopsia, visual agnosia, prosopagnosia (inability to recognize faces), or, if bilateral, cortical blindness. Temporal lobe ischemia results in an amnesic syndrome with memory loss. Involvement of subthalamic branches may involve subthalamic nucleus or its pallidal connections, producing a wide variety of deficits. Contralateral hemiplegia occurs with involvement of the cerebral peduncle.

6.2.5 Vertebrobasilar artery syndrome

The vertebral arteries arise from the subclavian arteries and travel into the brain a long the medulla where they merge at the inferior border of the pons to form the basilar artery. The vertebral arteries supply the cerebellum (via the medullary arteries). The basilar artery supplies the pons (via pontine arteries), the internal ear (via labyrinthine arteries), and the cerebellum (via the anterior inferior and superior cerebellar arteries). The basilar artery then terminates at the upper border of the pons giving rise to the two posterior arteries (Figure 2).

Complete occlusion of the basilar artery is a catastrophic event. Patients typically experience occipital headache, diplopia, progressive quadriplegia, bulbar paralysis, coma, and frequently death. Locked-in syndrome (LIS) results from ventral pontines and is defined as quadriplegia and anarthria with preserved consciousness and sensation. Thus the patient cannot move or speak but remains alert and oriented. Only one voluntary movement, vertical gaze remains. Communication can be established via vertical eye movements. Mortality rates are high (59%), and those patients that do survive are usually left with severe impairments associated with brainstem dysfunction (Bauby, 1997).

Occlusions of the vertebrobasilar system can produce a wide variety of symptoms with both ipsilateral and contralateral signs, because some of the tracts in the brainstem will be crossed and others will not. Numerous cerebella and cranial nerve abnormalities also typically occur. Several different specific brainstem syndromes may result, with lateral medullary (Wallenberg's) syndrome being one of the most common.

Extracranial injuries to the vertebral arteries as they travel through the cervical spine can also produce vertebrobasilar signs and symptoms. Forceful neck motions (e.g. whiplash or aggressive neck manipulation) are among the more common types of injuries.

Part 7 Functional disability

Functional mobility skills are typically impaired following stroke and vary considerably from individual to individual. During the acute stroke phase (within the first 3 weeks), 70 to 80 percent of patients demonstrate mobility problems in ambulation while 6 months to 1 year later the figures are reversed, with 70 to 80 percent of patients able to walk independently, with or without assistive devices (Dobkin, 1996). Basic ADL skills such as feeding, bathing, dressing, and toileting are also compromised during acute stroke, with 67 to 88 percent of patients

demonstrating partial or complete dependence. Independence in ADLs also improves with time with about 24 to 53 percent of survivors requiring partial or total assistance 6 months to 1 year later. The ability to perform functional tasks is influenced by a number of factors. Motor and perceptual impairments have the greatest impact on functional performance, but other limiting factors include sensory loss, disorientation, communication disorders, and decreased cardiorespiratory endurance (Mill & DiGenio, 1983).

The MCA is by far the largest cerebral artery and is the vessel most commonly affected by cerebrovascular accident (CVA). The MCA supplies most of the outer convex brain surface, nearly all the basal ganglia, and the posterior and anterior internal capsules. Infarcts that occur within the vast distribution of this vessel lead to diverse neurologic sequelae.

Patients with middle cerebral artery stroke syndrome (MCA stroke syndrome) may have some basic functional disability: motor, cognition and mood findings, as follows (American Heart Association, 1996, Gubermen, 1994, Kwakkel, Kollen, & Wagenaar, 1999, Post-Stroke Rehabilitation Clinical Practice Guideline, 1996, & World Health Organization, 1989):

1. Main trunk occlusion of either side yields contralateral hemiplegia, eye deviation toward the side of the MCA infarct, contralateral hemianopia, and contralateral hemianesthesia. Eye and head deviation toward the side of the lesion is probably due to damage of the lateral gaze center (Brodmann area 8), or it can represent classic neglect, particularly when the right MCA is involved.

2. Trunk occlusion involving the dominant hemisphere causes global aphasia, whereas involvement of the nondominant hemisphere causes impaired perception of deficits (anosognosia) resulting from the stroke and more qualitative deficits of speech, as discussed later in this article.

3. Superior division infarcts lead to contralateral deficits with significant involvement of the upper extremity and face and partial sparing of the contralateral leg and foot.

4. Inferior division infarcts of the dominant hemisphere lead to Wernicke's aphasia. Such infarcts on either side yield a superior quadrantanopsia or homonymous hemianopia, depending on the extent of infarction. Right inferior branch infarcts also may lead to a left visual neglect. Finally, resultant temporal lobe damage can lead to an agitated and confused state.

5. Specific neurologic sequelae

5.1. Loss of consciousness - Initially this is rare after MCA stroke, but it occurs slightly more often than in vertebrobasilar strokes (8.4% versus 5.7%). Loss of consciousness most often is attributable to seizures, but it may result from secondary edema and subsequent brainstem herniation.

5.2. Hemiparesis and hemiplegia

5.2.1. Surprisingly, assigning clear-cut syndromes of weakness to specific territories of MCA infarct has posed a significant challenge. The prognosis of such motor deficit also has not completely been elucidated, with case reports of remarkable recovery from dense limb involvement.

5.2.2. Partial hemiparesis patterns have been mapped more readily to certain MCA territory infarcts. The National Institute of Neurological and Communicative Disorders and Stroke (NINCDS) data bank project gathered pilot data from 488 patients with unilateral hemisphere strokes. The following conclusions arose from the analysis of the project data:

5.2.2.1. Equivalent weakness of the hip, foot, shoulder, and hand was the most common finding among the patients in the NINCDS project, accounting for 71.2% of cases.

5.2.2.2. Hemiparesis with distal predominance describes another 23.5% of cases, with weakness of the lower face, lower legs, toes, fingers, and forearm and sparing of the forehead and proximal muscles of the upper and lower extremities. The resultant deficit is believed to

be due to the large representation of the affected muscles in the homunculus.

5.2.2.3. Faciobrachial paresis describes weakness of the lower face, jaw, tongue, oropharynx, and ipsilateral upper extremity. The weakness of the upper extremity is often more pronounced in the distal musculature of the hand and forearm. These deficits result from ischemic insult of the insula and operculum.

5.2.3. Although uncommon, movement disorders such as athetosis, chorea, and dystonia have been described as sequelae of MCA territory stroke.

5.3. Visual deficits

5.3.1. Hemianopia has long been known to accompany the syndrome following a large MCA infarct; yet, only the superior portion of the optic radiation is supplied by the MCA. The resultant hemianopia is probably due to a massive infarct with subsequent edema affecting adjacent structures.

5.3.2. Quadrantanopsia can be attributed to a parietal infarct affecting the deep fibers of the upper optic radiation; however, this condition is rare.

5.4. Neglect

5.4.1. Neglect in classic form has been attributed to parietal insult, but data from positron emission tomography (PET) scanning reveal that frontal lesions can cause similar but more transient sequelae.

5.4.2. At times, visual neglect is difficult to distinguish from hemianopia. Subtle signs (eg, a patient who responds to a stimulus from the left by turning right and also fails to blink upon threatening stimuli to the affected side) can aid in diagnosing neglect. Patients with visual neglect often have difficulty naming objects presented on the affected side.

5.4.3. Motor neglect with underuse of the side contralateral to the cerebral insult appears much like a hemiparesis. Special efforts must be made by the examiner to encourage the patient to demonstrate strength and dexterity. Typically, the patient has delayed withdrawal to noxious stimuli, fails to place the affected hand in the lap when seated, and falls heavily to the affected side with no apparent effort to minimize impact.

5.5. Autonomic dysfunction

5.5.1. Autonomic disturbance after MCA stroke often can be evidenced by contralateral edema of the hand and foot arising within hours of the infarct and lasting up to 2 weeks. This edema is in contrast to the dependent edema that develops subacutely in the distal aspect of a plegic extremity.

5.5.2. Excessive sweating contralateral to the territory of an MCA stroke can be indicative of a larger lesion, affecting deep and superficial branches.

6. Left-hemisphere (dominant) infarction - The left cerebral hemisphere is dominant for speech and language in more than 95% of right-handed individuals. Defining cerebral dominance for left-handed individuals is more difficult, but most left-handed patients also appear to have a dominant left hemisphere. One study analyzing left-handed patients with aphasia showed that 60% had lesions confined to the left hemisphere. Other studies reveal bilateral speech representation in as many as 15% of left-handed patients.

6.1. Aphasia

6.1.1. Ischemic injury to the sylvian fissure of the dominant hemisphere is the lesion most likely to lead to dysphasia. Describing deficits in speech may be easier if pathologies are categorized as fluent versus nonfluent. In this context, fluent does not describe correct use of language or grammar but simply the ability to produce sounds readily. Nonfluent dysphasia describes a deficit in which a difficulty in producing words or sounds is appreciated.

6.1.2. Surprisingly, studies have revealed patients with only mild speech deficits, despite localized infarcts in cerebral areas thought to be essential for speech and language. Such studies suggest a major role of deeper structures, particularly the thalamus, in this function.

6.1.3. Broca's aphasia, also termed expressive or motor aphasia, describes the ability to comprehend written and spoken language, with nonfluent or impaired expression of either spoken or written language.

6.1.3.1.The infarct responsible for Broca's aphasia encompasses the insula and frontoparietal operculum.

6.1.3.2.Global aphasia can be assumed wrongly in these patients if the examiner does not use comprehension testing with simple questions. Initially, the patient's profound impairment is difficult to differentiate from a global aphasia, and only later does a speech disturbance arise that is isolated to writing (agraphia) and speech production.

6.1.3.3.Dyspraxia describes the impaired cooperation of the oropharyngeal and respiratory elements necessary for speech. Individuals with dyspraxia have a hesitant and somewhat telegraphic verbal response.

6.1.3.4.Agrammatism describes the shortened speech patients use to communicate. These individuals sometimes utter only individual words to communicate an idea.

6.1.4. Wernicke's aphasia, also termed receptive or sensory aphasia, is caused most often by occlusion of the lower division of the MCA bifurcation or one of its branches. Patients with Wernicke's aphasia vocalize smoothly and with expression, but they demonstrate paraphasias or speech with distorted phonetic structure, word substitution, and additional prefixes and suffixes. The speech is fluent but can be without understandable words.

6.1.4.1.The infarct responsible for a classic Wernicke's aphasia includes the dominant posterior temporal, inferior parietal, and lateral temporo-occipital regions.

6.1.4.2.Contrary to the manifestation of motor aphasia, speech is rich in words but is missing key words and ideas and may be perseverative. The patients demonstrate pure-word deafness, with the inability to repeat words, along with alexia, the inability to recognize or comprehend written language.

6.1.5. The classic cause of conductive aphasia is thought to be a disruption of neural pathways or of the arcuate fasciculus connecting the motor and sensory areas concerned with speech. The clinical features of conductive aphasia are not explained completely by this theory. Distinguishing a conductive aphasia is an especially difficult challenge for the clinician.

6.1.5.1. Patients with conductive aphasia have significant difficulty repeating unfamiliar phrases and words and demonstrate much better auditory and written comprehension than do individuals with Wernicke's aphasia; however, patients with conductive aphasia are more likely to recognize the deficit and to make an effort to self-correct.

6.1.5.2. Anatomically, insult to the isolated arcuate fasciculus is believed to be responsible for the symptoms; however, scant case reports actually document such a correlation. In fact, patients with the described syndrome more frequently have more superficial infarcts involving 1 or 2 recently discovered tracts.

6.2. Apraxia

6.2.1. Apraxia refers to the inability to perform a previously learned task despite preserved strength, vision, and coordination. The condition is due to an insult to the dominant hemisphere.

6.2.2. When referring to apraxia, Mohr states, "Motor engrams (programs) that guide skilled acts have either been lost or cannot be accessed."[4,5] Generally, the ability is impaired rather than eliminated; thus, the term dyspraxia is more appropriate.

6.2.3. The most common form of apraxia is ideomotor apraxia, in which a disconnection is thought to exist between the cortex containing plans for movement and the cortex responsible for execution. On verbal command, the patient is uncoordinated in or is unable to perform simple tasks, such as imitating the use of a hammer and nail. Often, the patient performs the actual task with much greater precision. Aphasia and apraxia occur independently, and the cortex responsible for motor planning is thought to be in the superior parietal lobe.

6.2.4. Ideational apraxia describes an impaired ability to complete more complex multistep tasks, such as obtaining a glass of water. Not all experts agree that ideational and ideomotor apraxias are distinct entities.

6.2.5. Callosal apraxia is similar to ideomotor apraxia but only involves the nondominant arm.

6.2.6. Limb-kinetic apraxia refers to an impaired clumsy manipulation of objects in such tasks as combing one's hair. Limb-kinetic apraxia can be accompanied by ataxia, choreoathetosis, spasticity, and weakness. Even after repeated efforts, performance only slightly improves.

6.2.7. Oral-buccal-lingual apraxia describes an impaired ability to perform complex movements of the tongue and face upon command. Often these movements are performed spontaneously. This condition coexists with Broca's aphasia in 90% of patients; however, the 2 disorders often exist independently.

7. Right-hemisphere (nondominant) infarction - Motor deficits following infarction of the nondominant hemisphere parallel those described previously for dominant-hemisphere lesions. Additionally, lesions of the nondominant hemisphere can lead to a variety of behavioral abnormalities. These behavioral deficits correlate much less to location and extent of the infarction than do deficits following infarcts of the dominant hemisphere, and some are predictive of an unfavorable long-term outcome after rehabilitation. Insults of the nondominant hemisphere can affect attention, leading to impersistence and neglect.

7.1. Extinction - This describes inattention to one stimulus when 2 stimuli are presented simultaneously. Generally, the ignored stimulus is on the left side.

7.2. Neglect - According to Schwartz and colleagues, neglect is "a lack of responsively to stimuli on one side of the body, in the absence of any sensory or motor deficit severe enough to account for the imperceptions." Such unilateral neglect occurred in 29% of patients with right-sided brain damage versus 12% of patients with left-sided brain damage among a stroke population studied by Battersby and coauthors. In severe cases, the patient

often ignores tactile, visual, and auditory stimuli on the left side and is turned chronically to the right side. When asked to bisect lines, the patient often does this far to the right of center. Unilateral spatial neglect is a subtler deficit, in which the patient may fail to read words or recognize figures to the left of midline. More sizable infarcts lead to anosognosia or imperceptions of field neglect and imply a much less favorable prognosis.

7.3. Impersistence - This term is used to describe an inability to persist in performing motor tasks; it is often accompanied with visuomotor and visuospatial deficits. This impairment places the patient at risk for an unfavorable rehabilitation outcome.

7.4. Dressing apraxia - This finding is much more common in cases of right-hemisphere infarcts and is attributable to difficulty distinguishing right from left and up from down. The patient is unable to dress without assistance despite having no apparent hemiplegia that would prevent the performance of this function.

7.5. Topographic memory deficit - This term is used when individuals become lost in familiar surroundings. The finding often follows right-hemisphere insults.

7.6. General confusion and delirium - These findings often are more commonly appreciated in patients with damage to the nondominant hemisphere. The central role the right hemisphere plays in attention, vigilance, and distinguishing stimuli is probably responsible for this common presentation.

7.7. Confabulation or unintentional fabrication of information - This is largely due to an inability to recognize errors, disinhibition, and memory deficits. These deficits all are common with damage to the nondominant hemisphere and to the frontal lobe.

7.8. Constructional apraxia - This defines a difficulty in manipulating objects in space. This type of apraxia can be appreciated by having affected patients copy designs or build 3-dimensional models. This tendency is more common with right-sided lesions than with left-sided lesions, as is evident in a population of 67 patients with constructional apraxia studied by Piercy and

colleagues. In this group, 25 had left-sided damage and 42 had damage to the right hemisphere. The apraxia of the patients with a dominant-hemisphere infarct often is described as decreased attention to detail. The apraxia with right-sides damage is consistent with neglect, in which features to the left of midline are ignored.

7.9. Allesthesia - This term describes sensory referral. For example, a patient touched on the left side feels the touch on the right.

7.10. Aprosody, lack of intonation in speech, and affective agnosia - These terms refer to the inability to perceive or comprehend emotional intonation of speech. The 2 deficits often coexist and correlate with lesions in the right temporoparietal region.

Table 1 presents the clinical manifestations of MCA syndrome. The most common characteristics of MCA syndrome are contralateral spastic hemiparesis and sensory loss of the face, upper extremity (UE), and lower extremity (LE), with the face and UE more involved than the LE. Lesions of the parieto-occipital cortex of the dominant hemisphere (usually the left hemisphere) typically produce aphasia. Lesions of the right parietal lobe of the nondominant hemisphere (usually the right hemisphere) typically produce perceptual deficits (e.g., unilateral neglect, anosognosia, apraxia, and spatial disorganization). Homonymous hemianopsia (a visual field defect) is also a common finding. The MCA is the most common site of occlusion in stroke.

Table 1 Clinical manifestations of middle cerebral artery syndrome.

Signs and Symptoms	Structures Involved
Paresis of contralateral face, arm, and leg (leg is least affected)	Primary motor cortex and internal capsule
Sensory impairment over the contralateral face, arm, and leg (pain, temperature, touch, vibration, position, two-point discrimination, stereognosis)	Primary sensory cortex and internal capsule
Motor speech disorder (expressive-aphasia-telegraphic halting speech)	Broca's cortical area in the dominant hemisphere
Wernicke's or receptive aphasia (fluent but often jargon speech, poor comprehension)	Wernicke's cortical area in the dominant hemisphere
Perceptual problems such as unilateral neglect, apraxias, depth perception problems, spatial relation difficulties	Parietal sensory association cortex
Homonymous hemianopia	Optic radiation in internal capsule
Loss of conjugate gaze to the opposite side	Frontal eye fields or their descending tracts
Ataxia of contralateral limb(s) (sensory ataxia)	Parietal lobe

Part 8 Recovery from stroke

Recovery from stroke is generally fastest in the first weeks after onset, with measurable neurological and functional recovery occurring in the first 1 to 3 months after stroke. Patients continue to make functional gains at a reduced rate for up to 6 months or longer after insult. Some patients may demonstrate prolonged recovery with improvements occurring over a period of years, especially in the areas of language and visuospatial function (Panel, 1996). Rates of improvement will vary across management categories: patients suffering minor stroke recover rapidly with few or no residual deficits whereas severely impaired individuals demonstrate more limited recovery. An important finding is that recovery has been demonstrated even in patients with extensive central nervous system (CNS) damage and advanced age (Dombovy & Bach-y-Rita, 1988). Early recovery is generally thought to be the result of resolution of local vascular and metabolic factors. Thus the reduction of edema, absorption of damaged tissue, and improved local circulation allows intact neurons that were previously inhibited to regain function. CNS plasticity is thought to account for continuing recovery. In the presence of cell death, functional reorganization of the CNS (function-induced plasticity) occurs. The stimulation from active rehabilitation and an enriched environment plays an important part in brain repair and recovery (Stein, 1995).

The process of recovery from stroke usually follows a stereotyped series of stages leading to a final stage of recovery that varies with the individual patient.

8.1 Stages of recovery

Several clinical researchers have systematically described the sequence of functional changes a patient undergoes following strokes. A highly detailed report was published by Twitchell (Twitchell, 1951); his sample included 121 patients, all expect three having suffered either thrombosis or embolism of one of the cerebral

vessels. However, his study excluded patients who were in coma for a long period of time, or who exhibited severe aphasia for more than week.

Twitchell (Twitchell, 1951) noted "…a remarkable uniformity in the steps of recovery in different cases." The process of motor recovery followed a general pattern: immediately following the onset of hemiplegia there was a total loss of voluntary movement in the involved extremities and a loss of diminution of the tendon reflexes. While resistance to passive movement was decreased, Twitchell considered that it was seldom so complete as to be called flaccidity. Within 48 hours tendon reflexes became more active on the involved side, and within a short time resistance to passive movements began to increase. Adductors flexors were chiefly involved in the upper extremities and adductors and extensors in the lower. As spasticity increased, clonus appeared (between 1 and 38 days following the onset of hemiplegia).

Six to 38 days after onset of hemiplegia the first willed movements (shoulder and hip flexion) appeared. A flexor synergy developed which, for finger flexion, included flexion of the upper extremity as a whole (shoulder, elbow, wrist, and fingers). Soon afterward, an extensor synergy developed.

As the power of willed flexion of the shoulders and elbows further increased, a stage was reached where spasticity in these muscles abruptly lessened. Spasticity completely disappeared when complete recovery of the power of willed effort occurred.

Voluntary movement in the lower limb also began with flexor and extensor synergies. Twitchell emphasized that although the recovery process could be divided into phases or stages, it was continuous and the gradual appearance of new factors resulted in overlapping at different stages.

When the first movement patterns appeared, a latent period of 2-5 second existed between the times the command to execute a movement was given and the time the actual movement tool place. The movement began with a very slow speed, but it increased toward the end of the limited range. Also, relaxation required 1-3 second. All movements could be performed with greater speed and accuracy if the patient was allowed to watch his hand and limb. If vision was excluded, movements were carried out more slowly and with less dexterity. Ataxia or tremor was increased by excluding vision even in the absence of a defect in sensation.

The process of recovery described above did not occur in all cases. For example, Twitchell noted that 3 patients exhibited a restoration of movement of the arm in an opposite manner: willed movement returned first in the hand while movement in the elbow and shoulder returned later. Also, only mild to moderate spasticity was noted in 13 patients.

Another study of the process of recovery was provided by Brunnstrom (Brunnstrom, 1970). She also noted an almost stereotyped sequence of events that take place during recovery. She had divided the process into a number of stages:

(a) Immediately following the acute episode, flaccidity is present and no movements of the limbs on the affected side can be initiated.

(b) As recovery begins, the basic limb synergies or some of their components may appear as associated reactions, or minimal voluntary movement responses may be present. Spasticity appears at this stage.

(c) The patient gains voluntary control over synergies; spasticity increases.

(d) Some movement patterns out of synergy are mastered, and spasticity begins to decline.

(e) If progress continues, more difficult movement combinations are learned as the basic synergies lose their dominance over motor acts.

(f) With the disappearance of spasticity, individual joint movement become possible and co-ordination approaches normal. Brunnstrom (Brunnstrom, 1970) considers that as a last step normal motor function may be restored.

The neural mechanisms underlying each of the stages of recovery are not thoroughly understood. A portion of the recovery that occurs following a nonfatal stroke is due to the resolution of local factors (e.g., edema and tissue debris), but another part is due to neural mechanisms of recovery. Furthermore, the process of recovery can continue for months or years. Evidence is accumulating that the brain is dynamic and plastic and that the eventual degree of recovery is to a large extent dependent upon functional demand and specific rehabilitation procedures. For example, recovery of a hemiplegia upper extremity may require not only the neural substrate for return of function, but also the functional demand (in animal experiments this has been obtained by forced usage), and a rehabilitation program that is based upon all the known factors in recovery.

8.2 Brain plasticity

Brain plasticity refers to the capacity to modify structural organization and functioning. Plasticity permits enduring functional changes to take place. It is one of two fundamental properties of the nervous system; the other is its excitability, which related to rapid changes that leave no trace in the nervous system (Konorski, 1961).

Bethe (Bethe, 1930) considered plasticity as a general principle of living organisms: the ability to adapt to changes and to meet the dangers of life. It is the capacity of the central nervous system to reorganize following insult and to restore adequate function. The question of CNS plasticity is intimately linked with concepts of cerebral localization, and some functions are more localized than others. For example, Luria (Luria, 1963) considers that the localization of such processes as visual and auditory perception in circumscribed sensory areas is less likely than the localization of the respiratory or patellar reflex. However, he notes that in the cortical representation of the special sense such as vision and audition, the cortical projections are only a small part of the functional system of that part of the brain. The high specificity of the neuronal structures that project a particular receptor system to the cortex underlies the fact that lesions in these areas often lead to irreversible defects, and compensation is possible only within very narrow limits.

Bathe (Bethe, 1930) developed concepts of CNS plasticity that influenced many other workers. One of his demonstrations involved the assessment of functional reorganization following removal of one, two, or three limbs of an amphibian. The animal continued to move about by recoordinating in a new manner. Bethe's work on plasticity led to the conclusion that the high degree of plasticity in man and higher vertebrates is due not to regeneration but to dynamic reorganization and adaptation to new circumstances. Studies that demonstrate the unmasking of previously unrecognized pathways following peripheral nerve or central nervous system lesions support this view.

Since the turn of the century Foerster (cited in Zulch, (Zulch & Olfrid, 1969)) had emphasized the role of physical therapy in effecting recovery from CNS lesions. He further showed that a greater capacity for improvement is produced with deficits either due to lesions in the less highly differentiated parts of the brain, or in which there is a bilateral representation of receptors. One of outstanding clinical exponents of CNS plasticity, Foerster, as well as Bethe (Bethe, 1930) and others, considered that recovery of function takes place through reorganization of the remaining parts of the CNS.

In the second quarter of this century, surgical relocation of muscle demonstrated the CNS capacity to reorganize complex reflexes and motor functions. For example, Weiss and Brown (Weiss & Brown, 1941) transposed the biceps femoris muscle (a flexor) to the extensor side of a knee joint to substitute for the weakened or lost action of a paralyzed quadriceps muscle. Initially, the muscle contracted only in the flexor phase, but " …surprisingly few trials were required to make the transplant suddenly contract in the extensor phase." After further trials, the muscle operated only the extensor phase. Even then, however, temporary relapses into the old flexor association occur repeatedly, even years after the operation. These relapses seem to be favored by fatigue, lack of concentration, automaticity of movement, etc. Weiss and Brown suggested that the adjusted use of the transplant is based not on the substitution of a permanent extensor association for its former flexor association in the elementary motor mechanisms, but rather on the development in higher centers of

a new type of action that can effectively override the innate coordinative association without abolishing them.

For many years studies with stroke patients revealed unexpected and unexplained recovery that, in retrospect, could be interpreted in terms of brain plasticity, but generally were not. The brain was considered to be rigid, unchanging organ. Climente (Clemente, 1976) has pointed out that although everyone admitted that an individual can learn-thus showing functional plasticity-learning was considered an abstract function related to the mind and not the brain. Once the physical neural patterns of the nervous system had developed, i.e., once the fiber connections had become established, alterations in neuronal geometry were not considered possible. Clemente considers that a change in thinking occurred as the gap between the mind had never been considered rigid, only the structure of the brain. It followed naturally that since the brain was the organ of the mind, and that since the brain was composed of cells, that plasticity could only be the result of cellular events in the central nervous system.

One particularly important change in the concept of the brain as a malleable organ resulted from the demonstration that, although the individual brain cells did not regenerate, the cell processes, axons and dendrites were highly responsive to functional demand. Cragg (Cragg, 1968) pointed out that, in the rat visual cortex, cell bodies occupy only 3% of the volume of the cortex. Thus 97% of the cortex is made up primarily by dendrites, axons, and glia. The large difference between the weight of the brain at birth and at maturity is due to the development of these three structures, since the number of cells does not increase. There is evidence that the development of dendrites and dendritic spines, as well as axons and synapses, depends on functional demand (Bech-y-Rita, 1972). Furthermore, Buell and Coleman (Buell & Coleman, 1979) have demonstrated that such growth appears to occur even in 80-yr-old humans.

Early recovery of function following a stroke may be due to the resolution of local factors, such as the reduction of cerebral edema, the absorption of damaged tissue and the improvement of local circulation. However, there is no evidence that these factors play a role in long-term recovery of function: Brodal (Brodal, 1973) considered that they may be important during, at most, the first 2 months, and Wall (Wall, 1980) concluded that, although they may play a crucial role immediately following the event, it is highly unlikely that they are crucial in the days and weeks following the lesion.

Recovery of function can continue for months or years. But how is brain damage repaired and function re-established? In essence, the damage is not repaired, the brain being incapable of regeneration by mitotic duplication, a principal repair mechanism of other body tissues such as the liver. It also appears to be incapable of promoting significant growth of cut axons, such as occurs in peripheral nerves. Therefore, other neural mechanisms must be responsible for the capacity of the brain to recover function.

Interest continues to grow in the role of neuroplasticity in recovery of function following CNS lesions among the mechanisms invoked are:

(a) Regenerative and collateral aprouting. Changes in connections between neurons

(b) Unmasking of pathways. Changes in sensitivity of synaptic transmission have been identified by various terms, such as supersensitivity, alterations of the balance of excitation and inhibition, relatively inefficient synapses, substitution, vicarious function, and unmasking of pre-existing but functionally depressed pathways.

(c) Dissipation of diaschisis. Diaschisis is the depressed function or loss of functional continuity between various centers or neuron tracts.

(d) Regeneration

Diaschisis dissipation mechanisms are not well known and regeneration probably plays a minimal role, if any, in recovery of function (Bach-y-Rita, 1981).

Part 9 Rehabilitation management

The World Health Organization (WHO) as a coordinated process that enhances activity and participation has defined rehabilitation (World Health Organization., 2001). Rehabilitation is a process of educating the disabled person in order to support him/her in coping with family, friends, work and leisure as independently as possible (Barnes, 2003).

Rehabilitation begun early in the acute stage optimizes the patient's potential for functional recovery. Early mobilization prevents or minimizes the harmful effects of deconditioning and the potential for secondary impairments. Functional reorganization is promoted through stimulation and use of the affected side. Learned nonuse of the hemiplegic extremities and maladaptive patterns of movement are prevented. Mental deterioration, depression and apathy can be reduced through the fostering of a positive outlook on the rehabilitation process. Patients need to be presented early on with an organized plan of care that addresses their individual goals and stresses resumption of normal ADLs. It is equally important that patients receive adequate information and know that various forms of support are available, if they need them.

Rehabilitation can begin as soon as the patients are medically stabilized, typically within 72 hours. Patients may be admitted to a specific stroke unit or neurological unit with rehabilitation services. Evidence supports the benefits of such services in significantly improving functional outcomes when compared to patients not receiving those services (Heyes & Corroll, 1986; Langhorne, 1993).

Patients with moderate or severe residual deficits generally require intensive rehabilitation services to assist them in functional recovery. Patients are referred to acute rehabilitation (inpatient hospital rehabilitation) if they can tolerate an intensity of services consisting of two or more rehabilitation disciplines. Less intensive services can be obtained from subacute rehabilitation programs, generally found in facilities or in a subacute hospital unit. Services in subacute units are variable, ranging from 1

hour of services 2 to 3 times per week to daily, shorter duration services. Post acute rehabilitation services can also be delivered at home or in an outpatient facility. Generally the patient who benefits from these services demonstrates less severe involvement and more functional mobility than those requiring hospitalization (Post-Stroke Rehabilitation Clinical Practice Guideline, 1996).

Optimal timing of rehabilitation based on individual patient readiness is an important consideration. A number of factors appear to be related to rehabilitation readiness, including the side of the lesion. There is some evidence to suggest that patients with right hemiplegia may respond more favorably to earlier comprehensive rehabilitation efforts. Patients with left hemiplegia who suffer more cognitive-perceptual deficits and generally have longer rehabilitation stays may benefit from additional preadmission time to allow for cognitive and perceptual-motor reorganization. Equally important factors that influence the timing of rehabilitation efforts include medical stability, motivation, patient endurance, recovery, and ability to learn. In an era of time-limited payment for comprehensive rehabilitation services, selecting the optimal time for rehabilitation training may prevent unnecessary patient failures and improve long-term functional outcome (Johnston & Keister, 1984; Novack, Satterfield, & Connor, 1984).

Goals and outcomes of physical therapy during rehabilitation adapted from the Guide to Physical Therapist Practice (American Physical Therapy Association, 1999) include the following:

1. Changes associated with recovery are monitored.
2. Tolerance to position and activities is increased.
3. Upright (out of bed) and weight-bearing status is improved.
4. Risk of secondary impairments and reoccurrence of condition is reduced
5. Joint integrity and mobility is maintained.
6. Awareness of the hemiplegic side and motor function (motor control and motor learning) are improved.
7. Trunk control, symmetry, and balance are improved.

8. Strength, power, and endurance are increased.
9. Functional independence in ADLs and functional mobility are increased.
10. Aerobic capacity and endurance are increased
11. Patient, family, and caregiver knowledge and awareness of the diagnosis, prognosis, interventions, and goals and outcomes are increased.
12. Care is coordinated with patient, family, and caregivers, and other professionals.
13. Safety of patient, and family, caregivers is improved.
14. Placement needs are determined.
15. Self-management of symptoms is improved.
16. Sense of well-being is improved.
17. Awareness and use of community resources are improved.

Rehabilitation: Interventions

Recovery from stroke and learning is based on the brain's capacity for reorganization and adaptation. An effective rehabilitation plan capitalizes on this potential and encourages functional use of the involved segments. Activities are selected that are meaningful and important to the patient. Optimal motor learning can be ensured through attention to a number of factors, most importantly, strategy development, feedback, and practice.

1.Strategy development

The therapist first assists the patient in learning the desired task (cognitive stage). More specifically, critical task elements and successful outcomes and goals are identified. The desired task is demonstrated at the ideal performance speeds. The patient then begins to practice. If the task has a number of interrelated steps, practice of component parts may precede practice of the whole task. It is important, however, not to delay practice of the integrated task because this may interfere with effective transfer of learning. The therapist should give clear, simple verbal instructions; do not

overload the patient with excessive or wordy commands. Reinforce correct performance and intervene when movement errors become consistent. Manual guidance or VCD can be used to assist the patient through the activity especially for positioning or postural tasks. Active participation of the affected side should be encouraged early. Practicing the movements on the unaffected side first can yield important transfer effects to the affected side. Simultaneous practice of similar movements on both sides (bilateral activities) can also improve learning and promote integration of the two sides of the body. Visualization of the movement components (mental practice) can help some patients in initially organizing the movement.

As initial practice progresses, the patient is asked to assess performance and identify problems, specifically, what difficulties exist, what can be done to correct the difficulties, and what movements can be eliminated or refined. If a complex task is practiced, the patient is asked to identify if the correct components were performed, how the individual components fit together, and if they were appropriately sequenced. If the patient is unable to provide an accurate assessment of problems, the therapist can prompt the patient in decision-making and utilize demonstration to help identify problems. For example, if the patient consistently falls to the right while standing, questions can be directed toward this problem (e.g., “In what direction did u fall?” “What do you need to do to prevent yourself from falling?”). The patient is thus actively involved in developing problem solving skills (self-monitoring and self-correction of movements). These skills are essential in ensuring independence and generalizability of learning to other environments and variations.

2.Feedback

Feedback can be intrinsic (naturally occurring as part of movement response) or extrinsic (provided by the therapist). During early motor learning the therapist will provide extrinsic feedback (e.g., verbal cueing, manual cueing) to shape performance. It is important to monitor performance carefully and provide accurate feedback. The patient’s attention should be directed to naturally occurring intrinsic feedback. During early intervention visual inputs are critical for motor learning. Having the patient watch the movement can facilitate this. If the patient needs glasses, make sure they

are worn during therapy. Use of a mirror can be an effective adjunct for some patients to improve visual feedback, especially during postural and positioning activities. It is, however, contraindicated in patients with marked visuospatial perceptual impairments. During later learning (associative phase), proprioception becomes important for movement refinement. This can be encouraged by early and carefully reinforced weight bearing (approximation) on the affected side during upright activities. Additional proprioceptive inputs (manual contacts, tapping, stretch, tracking resistance, antigravity postures, or vibration) can be used to improve feedback and stimulate learning. The patient should be encouraged to “feel the movement” while learning to distinguish correct movement responses from incorrect ones. Exteroceptive inputs (light rubbing, stroking) may be used to provide additional sources of sensory inputs, particularly where distortions of proprioception exist. As treatment progresses, the emphasis again shifts from extrinsic to intrinsic feedback and to self-monitoring and correcting movement responses. Great care must be taken to avoid sensory bombardment or feedback dependence. This requires careful assessment during each treatment session. Therapists should also limit use of immediate feedback to allow the patient adequate time for introspection. Pain and fatigue (either mental or physical) should also be avoided, because each will result in decreased performance.

3.Practice

The therapist organizes the patient’s schedule to ensure practice sessions are appropriate to foster learning. Repetition of the desired task will improve performance and motivation. This is important to ensure that the patient experience success. Rest periods should be provided. Most hospitalized patients initially require a distributed practice schedule owing to limited endurance. Staff and family efforts should be coordinated to ensure consistency of practice during off-therapy times. The patient should be progressed and challenged with task variation or a new task as soon as the previous one has been mastered or almost mastered. Variable practice (practice if similar or related task) will improve learning, specifically retention and generalizability of skills, and should be instituted as soon as possible. The patient

should be encouraged to self-monitor practice sessions and recognize when fatigue may be setting in and rest is required.

Careful attention to the learning environment will also yield important therapeutic gains. Distractions should be reduced and a consistent and comfortable environment provided in which the patient can exercise. Initially this will be a closed environment with no distractions. Later the environment can be varied, providing an appropriate level of contextual interference. Thus the patient is progressed toward performing the same skill in an open, variable environment. The patient should be assisted in transferring skills to real-life environments.

Treatment sessions should begin and end on a positive note, ensuring the patient has success in treatment and continuing motivation. Family and caregivers should be taught supportive techniques. Finally the therapist should communicate support and encourage the patient; recovery from stroke is an extremely stressful experience and will challenge the abilities of both patient and family.

4.Motor relearning program for stroke

Carr and Shephard (Carr & Shepherd, 1987) have developed a motor relearning program for stroke that incorporates many aspects of motor learning theory and provides practical guidelines for retraining functional skills (e.g., balanced sitting and standing, transfer skills, gait, etc.). Their approach focuses on task-specific learning, and the development of active movement control through effective use of feedback and practice. Facilitation techniques are deemphasized in favor of verbal instruction, demonstration, and manual guidance. The approach is based on four distinct steps:

a. Analysis of task: the therapist carefully observes and analyses the patient’s performance and identifies missing components.
b. Practice of the missing component: The therapist explains the missing components, their relationship to the task, and assists the patient in identification of the goal. Practice of the missing components occurs

with emphasis on careful instruction, demonstration, verbal and visual feedback, and manual guidance to facilitate learning.

c. Practice of task: the task, outcomes, and goals are carefully explained and practiced. There is a continuing emphasis on use of instruction, demonstration, verbal and visual feedback, and manual guidance to facilitate learning of the required task. Flexibility is encouraged.
d. Transference of training: the task is practiced in varying environmental contexts, shifting learning from structured environments to more open real-life environments.

5. Remediation/facilitation approaches

Traditional remediation/facilitation approaches have as their primary focus the use of therapeutic exercises and neuromuscular facilitation techniques to reduce sensorimotor deficits and promote motor recovery and improved function. The affected body segments are targeted to prevent learned nonuse and overcompensation by intact segments. Thus, training using these approaches requires some degree of voluntary movement control. Developmental postures and activities are used to limit the degrees of freedom and focus functional training on specific body segments. The three approaches most commonly used include: neurodevelopmental treatment (NDT), movement therapy in hemiplegia, and proprioceptive neuromuscular facilitation (PNF).

6. Compensatory training (functional) approach

The focus of compensatory training approach is on the early resumption of functional independence using the uninvolved or less involved segments for function. For example, the patient with left hemiplegia is taught to dress using the right upper extremity. Central to this approach is the concept of substitution. The patient is first made aware of movement deficiencies (cognitive awareness id developed). Changes are then made in the patient's overall accomplish the task are suggested, simplified, and adopted. The patient practices and relearns the task using the new pattern. The patient then practices the new pattern in the environment in which the function is

expected to occur. Energy conservation techniques are incorporated to ensure the patient can complete all daily tasks.

The second central tenet of this approach is modification of the environment (adaptation) to facilitate relearning of skills, ease of movement, and optimal performance. For example, the patient with unilateral neglect is assisted in dressing by color-coding of shoes (red tape in the left shoe, yellow tape on the right shoe). The wheelchair brake toggle is extended and color-coded to allow the patient easy identification.

One of the major criticisms of this approach is the focus on uninvolved segments that may suppress recovery and contribute to learned nonuse of impaired segment. For example, the patient with stroke fails to learn to use the involved extremities. Focus on task-specific learning may also lead to the development of splinter skills (skills that cannot be easily generalized to other environments or to variations of the same task). However, a compensatory training approach may be the only realistic approach possible when the patient presents with severe impairments and functional losses.

Part 10 Outcome

1. The Barthel Index (BI) (Sulter, Steen, & Keyser De, 1999; Uyttenboogaart, Stewart, Vroomen, Keyser De, & Luijckx, 2005)

The Barthel Index activities of daily living is a weighted scale of 10 items of basic ADL including feeding, bathing, grooming, dressing, bladder and bowel control, chair/bed transfer, ambulation, and stair climbing. The range possible score of the BI is 0 to 100 and equal or more than 95 is independence care.The main aim is to establish degree of independence from any help, physical or verbal, however minor and for whatever reason. The cut off score for BI with the highest sum of sensitivity and specificity was 95 (sensitivity 85.6%; specificity 91.7%).

2. Modified Rankin Scale (Sulter, et al., 1999; Uyttenboogaart, et al., 2005)

This scale provides an assessment of the degree of disability. Minor strokes are considered Grades 0 to 2, while major strokes are Grades 3 to 5, while fatal is 6.

Grade 0:no symptoms at all

Grade 1: no significant disability despite symptom: able to carry out usual duties

Grade 2: slight disability: unable to carry out all previous activities but able to look after own affairs without assistance

Grade 3: moderate disability: requiring some helps, but able to walk without assistance

Grade 4: moderate/severe disability: unable to walk without assistance, and unable to attend to own bodily needs without assistance

Grade 5: severe disability: bedridden, incontinent, and requiring constant nursing care and attention

Grade 6:fatal

3. The EQ-5D (Brooks, Rabin, & Charro de, 2005)

The role of the EQ-5D is mainly to function as a generic instrument for measurements of health-related quality of life (HRQOL). For economic studies of the cost-utility variety a key feature of the EQ-5D is fully used, which are that peoples' health states can be transformed to single number representing utilities.

EQ-5D descriptive system

On this part of EQ-5D, the respondent is asked to indicate his/her health state by ticking in the box against the most appropriate statement in each dimension. The 5 EQ-5D dimensions comprise 3 levels, (1) no problem (2) some problem (3) severe problem, generating a total of 243 theoretically possible health states.

EQ VAS

The EQ VAS offers a simple method for obtaining and scoring self-rating of current health status. The VAS scale used is a vertical 20-cm. This part can be used in conjunction with the 5-digit classification of the descriptive system to build an

accurate profile of the respondent's health status. The EQ VAS has endpoints of 100 (best imaginable health state) at the top and 0 (worst imaginable health state) at the bottom. The respondent rates his/her current health state on the EQ VAS by drawing a line from the box marked 'your own health state today' to the appropriate point on the EQ VAS.

Part 11 Factors that influence rehabilitation outcome

1. Time-course of recovery

Although most motor and functional recovery occurs in the first 3 months after stroke (Duncan, Goldstein, Matchar, Divine, & Feussner, 1998; Wade & Hewer, 1987). Delay in initiating therapy after stroke appears to reduce eventual functional recovery. For example, Black et al (Black, Markowitz, & Cianci, 1975), in a study recovery after cortical lesions in monkeys, found that when therapy was delayed 4 months, recovery after 6 months of therapy was 67% of preoperative function, versus 82% recovery of the group in which therapy was initiated immediately. However, their study revealed other results that may have clinical relevance, especially when the total amount of therapy that a patient can receive is severely limited for insurance or other economic reasons: when therapy was begun immediately after surgery, recovery of function after 1 week of therapy reached 9% of preoperative function. When therapy was delayed 4 months, recovery after 1 week of therapy reached 50% of preoperative function.

In the study of the effect of rehabilitation on stroke outcome, Lehmann et al (Lehmann, Delatour, Fowler, Warren, & Arnhold, 1975) showed that significant functional gains obtained by rehabilitation are maintained at follow-up. Furthermore, significant gains could be obtained in a sample admitted 3 months and even a year after the onset of stroke. Thus, the timing of the rehabilitation program may be important.

2. Mind-body interactions

The human mind is capable of altering physiological functions. The broad area of behavioral medicine is based on this fact. But, in general, mind-body reactions are not sufficiently appreciated in designing treatment environments or the treatment itself (Bach-y-Rita, 1980).

3. Plasticity in the mature and aged brain

A large number of studies have revealed plasticity in the young brain. However, plasticity is a characteristic of normal brains of all ages. Recent studies suggest that dendritic trees of cortical neurons can grow extensively, even in old age, and it would be expected that growth of synapses would accompany the dendritic tree expansion. In one of these studies, Buell and Coleman (Buell & Coleman, 1979) showed that in layer-II pyramidal neurons in the human parahippocampal gyrus dendritic trees in nondemented aged persons (average age 79.6 years) were more extensive than in the brains of younger adults (average age 51.2 years). Most of the difference results from increase in the number and average length of terminal segments of the dendritic tree. Cells with shrunken dendritic trees were found in all brains but Buell and Coleman (Buell & Coleman, 1979) suggested that in the nondemented aging brain, one population of neurons dies and regresses, while another survives and grows. In another study, Carlen et al (Carlen, 1978) found that computed tomography scans revealed cerebral atrophy in the brain of 8 chronic alcoholics; the 4 who then abstained and showed functional improvement also showed partial recovery may have been due to regrowth of axons and dendrites of neurons that were damaged but not killed by ethanol use. These studies provide morphological evidence for plasticity in the mature and aged brain.

4. Learning theory

At the end of the last century Bryan and Harter (Bryan & Harter, 1899), after analyzing the learning of telegraph operators (sending and receiving), suggested that resumption of progress following a plateau is dependent on the organization of material into larger associated units, either perceptual or motor. Acquisitions of the primarily sensory skill, such as reading or receiving Morse code, and acquisition of a

motor skill such as typing or sending Morse code, are remarkably similar processes. Each is a slow process and has several plateaus or periods of no learning. They considered that resumption of progress following a plateau was dependent on the organization of material into larger units, either perceptual or motor.

Miller (Miller, 1956) pointed out that only about 6 to 8 objects can be held in the immediate memory at one time, and that it is immaterial whether the objects represent many or a few of the organism's receptors, or whether the units or 'chunks' contain a few or many bits of information. Miller calculated the maximum capacity for transmission in several sensory systems and found that their various channel capacities were similar with an average of 2.6 bits; that is, as the number of stimulus categories or the amount of stimulus uncertainly was increased, the amount of information transmitted reached this plateau. This corresponds to about six alternatives as a limiting number of values for the stimulus, beyond which the subject will be confused.

The rather rigid limits imposed by the inaccuracy of our absolute judgments of simple magnitudes can be expanded by the use of both simultaneous and successive discriminations. There are several ways by which the limits of the channel capacity for absolute judgments can be exceeded: for example, we can make relative rather than absolute judgments or we can arrange the task so as to permit one to make a (temporal) sequence of absolute judgments, thereby involving memory (Miller, 1956).

The absolute judgment and the span of memory are quite different kinds of limitations that are imposed on our ability to process information. Absolute judgment is limited by the amount of information (bits), whereas the "items" or "chunks" limit immediate memory. The number of bits of information is constant for absolute judgments, and the number of chunks of information is constant for immediate memory.

In normal learning, more and more of the information extraction processes become automatic and unconscious, and the "chunking" process described by Miller

(Miller, 1956) allows the number of bits per chunk to increase. Is the difficulty that a hemiplegic patient experience in putting together the individual components of gait (which has been relearned in the rehabilitation program) into smooth, coordinated gait related to deficits in “chunking?” Does the stroke patient learn by means of the alternating acquisition-consolidation phases (described since the last century), with each consolidation phase appearing as a plateau, during which he/she is incapable of learning new material? As used presently in the study of the learning process of a stroke patient may lead to therapeutic innovations, such as:

(a) Discharge (to home or to a nursing home) during consolidation (plateau) phases, with return to an active rehabilitation program when enters an acquisition phase
(b) More appropriate use of personnel
(c) Inclusion of therapeutic home program

5. Hierarchical organization and bilaterality of the brain

In a recent comprehensive discussion of neuroanatomy in relation to brain rehabilitation, Moore (Moore, 1980) emphasized the rehabilitation advantages of considering the nervous system from a hierarchical (archi-paleo-neo structures) point of view. Each of these levels has inherent properties that are relevant to rehabilitation. For example, the majorities of the neuronal processes projecting from nuclear centers of the archi systems do so bilaterally, and are highly multisynaptic in character. The pathways from the paleomammalian system tend to decussate and exert their major influences contralaterally, but also usually have minor but important ipsilateral components. They have fewer multisynaptic connections. The neosystems are principally contralateral (except for the neocerebellar systems which are principally ipsilateral). These neosystems have the most direct fiber connections between higher and lower centers. The neosystems are the most vulnerable to lesion, which produce primarily contralateral symptomatology. However, Moore (Moore, 1980) emphasizes the bilaterality of the brain: commissural for the bilateral coordination of the nervous system of the entire body. There is an increase, rather than a decrease, of commissural interneurons as the phylogenetic scale is ascended, in spite of the increasing cephalization of function.

6. Functional rehabilitation program

Functional programs that vary according to the patient's prior interest offer specific advantages. Advocates of home-based stroke rehabilitation suggest several advantage: satisfying patient choice, reducing the risks associated with inpatient care through reductions in length of hospital stay, the home setting being more focused toward rehabilitation outcomes, and saving in direct costs (Lafferty, 1996). In a review of home-based rehabilitation and care, Tamm M. (Tamm, 1999) debates the issue as to whether the home is the best place for rehabilitation. Although plople frequently express the desire to be at home rather than in institutionalized care, the home is a person's personal refuge and their private sanctuary.

The factors responsible for the superiority of organized stroke rehabilitation may include the supports from the family system, the initiation of earlier rehabilitation, home environment of rehabilitation, individually tailored program with audiovisual materials and close follow-up (Anderson, et al., 2000; Fey, De Weerdt, & Selz, 1998; Studenski, Duncan, Perera, Reker, & Lai, 2005).

Part 12 Previous studies

Duncan et al, 1998 (Duncan, Richards, Wallace, Stoker-Yates, & Pohl, 1998) were (1) to develop a home-based balance, strength, and endurance program; (2) to evaluate the ability to recruit and retain stroke subjects; and (3) to assess the effects of the interventions used. Twenty minimally and moderately impaired stroke patients who had completed inpatient rehabilitation and who were 30 to 90 days after stroke onset were randomized to a control group or to an experimental group that received a therapist-supervised, 8-week, 3-times-per-week, home-based exercise program. The control group received usual care as prescribed by the patients' physicians. Baseline and postintervention assessments included the Fugl-Meyer Motor Assessment, the Barthel Index of Activities of Daily Living (ADL), the Lawton Scale of Instrumental ADL, and the medical outcomes study-36 health status measurement. Functional assessments of balance and gait included a 10-meter walk, 6-minute walk, and the Berg Balance Scale. Upper extremity function was evaluated by the Jebsen test of

hand function. Of 22 patients who met study criteria, 20 completed the study and 2 refused to participate. The experimental group tended to improve more than the control group in motor function (Fugl-Meyer Upper Extremity: mean change in score, 8.4 versus 2.2; Fugl-Meyer Lower Extremity: 4.7 versus –0.9; gait velocity: median change, 0.25 versus 0.09 m/s; 6-minute walk: 195 versus 114 ft; Berg Balance Scale: 7.8 versus 5; and medical outcomes study – 36 health status measurement of physical function: 15.5 versus 9). There were no trends in differences in change scores by the Jebsen test of hand function, Barthel Index, and Lawton Instrumental ADL scale. This study demonstrated that a randomized, controlled clinical trial of a poststroke exercise program is feasible. Measures of neurological impairments and lower extremity function showed the most benefit. Effects of the intervention on upper extremity dexterity and functional health status were equivocal. The lasting effects of the intervention were not assessed.

Fey et al, 1998 (Fey et al, 1998) were to investigate the effect of a specific therapeutic intervention on arm function in the acute phase after stroke. In a single-blind, randomized, controlled multicenter trial, 100consecutive patients were allocated to either an experimental group that received an additional treatment of sensorimotor stimulation or to a control group. The intervention was applied for 6 weeks. Patients were evaluated for level of impairment (Brunnström-Fugl-Meyer test) and disability (Action Research Arm test, Barthel Index) before, midway, and after the intervention period and at follow-up 6 and 12 months after stroke. Patients in the experimental group performed better on the Brunnström-Fugl-Meyer test than those in the control group throughout the study period, but differences were significant only at follow-up. Results on the Action Research Arm test and Barthel Index revealed no effect at the level of disability. The effect of the therapy was attributed to the repetitive stimulation of muscle activity. The treatment was most effective in patients with a severe motor deficit and hemianopia or hemi-inattention. No adverse effects due to the intervention were found. Adding a specific intervention during the acute phase after stroke improved motor recovery, which was apparent 1 year later. These results emphasize the potential beneficial effect of therapeutic interventions for the arm.

Holmqvist et al, 1998 (Holmqvist, Koch, Kostualas, Holm, & Widsell, 1998) was to evaluate rehabilitation at home after early supported discharge from the Department of Neurology, Huddinge hospital, for moderately disabled stroke patients in southwest Stockholm. The patients were eligible if they were continent, independent in feeding, had mental function within normal limits, and had impaired motor function and/or aphasia 1 week after stroke. Patients were randomized either to early supported discharge with continuity of rehabilitation at home for 3 to 4 months or to routine rehabilitation service in a hospital, day care, and/or outpatient care. The home rehabilitation team consisted of two physical therapists, two occupational therapists, and one speech therapist; one of the therapists was assigned as case manager for the patient. The rehabilitation program at home emphasized a task- and context-oriented approach. The activities were chosen on the basis of the patient's personal interests. Spouses were offered education and individual counseling. A total of 81 patients were followed up for a minimum of 3 months. Patient outcome was assessed by the Frenchay Social Acitivity Index, Extended Katz Index, Barthel Index, Lindmark Motor Capacity Assessment, Nine-Hole Peg test, walking speed over 10 meters, reported fails, and subjective dysfunction according to the Sickness Impact Profile. Patient use of hospital and home rehabilitation service and patient satisfaction with care were studied. Overall there were no statistical significant differences in outcome. Multivariate logistic regression analysis suggested a systematic positive effect for the home rehabilitation group in social activity, activities of daily living, motor capacity, manual dexterity, and walking. A considerable difference in resource use during such a 3-month period was seen. A 52% reduction in hospitalization was observed: from 29 days in the routine rehabilitation group to 14 days in the home rehabilitation group. Patient satisfaction was in favor of the latter group. Early supported discharge with continuity of home rehabilitation services for the majority of moderately disabled stroke patients during the first 3-month period after acute stroke is not less beneficial than routine rehabilitation.

Studenski et al, 2005 (Studenski, et al., 2005) examined therapeutic exercise effects on functioning and quality of life (QOL) outcomes in subacute stroke survivors. This is a secondary analysis of a single-blind RCT of a 12-week program

versus usual care. Baseline, post-treatment and 6-month post-treatment daily functioning and QOL were assessed by Barthel Index, Functional Independence Measure, instrumental activities of daily living, medical outcome study short-form 36-item questionnaire (SF-36), and Stroke Impact Scale (SIS). Of 100 randomized subjects, 93 completed the postintervention assessment, (mean age 70; 54% male; 81% white; mean Orpington Prognostic Score 3.4), and 80 had 6-month post-treatment assessment. Immediately after intervention, the intervention group improved more than usual care in SF-36 social function (14.0 points; P=0.0051) and in SIS (strength (9.2 points; P=0.0003), emotion (5.6 points; P=0.0240), social participation (6.6 points; P=0.04880, and physical function (5.0 points; P=0.0145). Treatment was marginally more effective on Barthel score (3.3 points; P=0.0510), SF-36 (physical function (6.8 points; P=0.0586), physical role function (14.4 points; P=0.0708), and SIS upper extremity function (7.2 points; P=0.0790). Effects were diluted 6 months after treatment ended. This rehabilitation exercise program led to more rapid improvement in aspects of physical, social, and role function than usual care in persons with subacute stroke. Adherence interventions to promote continued exercise after treatment might be needed to continue benefit.

Several studies have been conducted to examine the rehabilitation in stroke patients but the results have been inconsistent. Previous studies have documented that patients usually have significant residual physical disability, functional impairment and reduced quality of life (Anderson, et al., 2000; Fey, et al., 1998; Studenski, et al., 2005). One study has shown that the motor function gradually returns as the result of only spontaneous recovery. In Duncan et al, 1998 (Duncan, et al., 1998) reported no difference in Barthel Index score. Therefore, the stroke rehabilitation programs may not improve outcomes (Dobkin, 1989). Several authors (Studenski, et al., 2005; Young & Forster, 1993) suggested that home rehabilitation is more effective and cheaper. Rehabilitation begun early in the acute stage optimizes the patient's potential for functional recovery. Early mobilization prevents or minimizes the harmful effects of deconditioning and the potential for secondary impairments. Recovery from stroke and learning is based on the brain's capacity for reorganization and adaptation. An effective rehabilitation plan capitalizes on this potential and encourages functional use

of the involved segments. Activities are selected that are meaningful and important to the patient. Optimal motor learning can be ensured through attention to a number of factors, most importantly, strategy development, feedback, and practice. Rehabilitation can begin as soon as the patients are medically stabilized, typically within 72 hours. Patients may be admitted to a specific stroke unit or neurological unit with rehabilitation services. Evidence supports the benefits of such services in significantly improving functional outcomes when compared to patients not receiving those services (Heyes & Corroll, 1986; Langhorne, 1993; Studenski, et al., 2005; Young & Forster, 1993).

In most developed countries, there is a heavy reliance on hospitals for the acute care, whereas the home rehabilitation of patients with stroke is limited (Anderson, Jamrozik, & Stewart-Wynne, 1994; Young, 1994). Because inpatient rehabilitation in Thailand is not widely available, the demand for home rehabilitation model is increasing. Therefore, the model for effective home rehabilitation for stroke will help improving stroke care and may be applied to other countries. To date, there is no randomized controlled trial study in assessing effectiveness of home rehabilitation program for ischemic stroke.

Therefore, we would like to evaluate the effectiveness of home rehabilitation program for ischemic stroke. We postulated that the program would be able to improve activity daily living (ADL), reduce disability and increase quality of life of stroke patients.